BEFORE THE PUBLIC UTILITIES COMMISSION

OF THE STATE OF SOUTH DAKOTA

IN THE MATTER OF THE APPLICATION BY PREVAILING WIND PARK, LLC FOR A PERMIT OF A WIND ENERGY FACILITY IN BON HOMME COUNTY, CHARLES MIX COUNTY, AND HUTCHINSON COUNTY, SOUTH DAKOTA, FOR THE

PREVAILING WIND PARK PROJECT

EL18-026

PREFILED TESTIMONY OF RICHARD R. JAMES

ON BEHALF OF INTERVENORS

EXHIBIT
Int. I-1

009205

Q: Please state your name, title, affiliation, and address.

A: My name is Richard R. James. I am the Principal Acoustician for E-Coustics Solutions, LLC, in Okemos, Michigan.

Q: What is the purpose of your testimony?

A: I am testifying to the acoustic issues of appropriate thresholds for audible and in-audible wind turbine sound at non-participating properties in the footprint of the proposed Prevailing Wind Park Project (PWPP) and to the computer modeling used by the applicant to assess impact of noise.

Q: What is your educational and professional background?

A: I have a degree in Mechanical Engineering with emphasis on noise control and acoustics. I have attached a set of documents that provide the details of my professional work. (See Exhibit 1.) The first page of that packet summarizes my work with focus on wind turbines since 2006 when I formed my current company, E-Coustic Solutions, LLC, (E-CS). It summarizes my published papers and qualifications to speak to wind turbine noise measurement, modeling and the impact of wind turbine noise on people in various jurisdictions. The next page is an excerpt from a Business Week article on my work with my clients using a computer model I developed with my first company based on the work I did for my undergraduate thesis. This model was accepted in government hearings in 1976. It was capable of modeling both in-facility worker noise and community noise. I was one of the first acousticians to use computer models for new facility design long before there were established national standards for such work. Other parts of the package cover my professional credentials and affiliations, list my publications and list hearings that I have participated in over the past 10 years.

Q: What experiences have you had that qualify you as a health expert in cases involving wind turbine noise?

A: I began looking at wind turbine noise as a special case of noise source shortly after closing my last company in 2006. Several early projects resulted in media exposure and I began to get requests from many places, some international, to advise local agencies or intervenors on proper siting methods. Because of that early work I have been involved in many major lawsuits about wind turbine noises where I have had access to not only my research work but also that of the opposing acousticians through discovery. I was also involved with the early studies that found that modern utility scale wind turbines emitted a pressure pulsation caused by the blade when it passes in front of the tower back in 2009.

This experience led to my work for the intervenors in the Wisconsin Brown County Shirley Wind case which Mr. Hessler referred to in his written testimony submitted in prior proceedings before the PUC. Subsequent to that I have been associated with other acousticians around the world, such as Steven Cooper of Australia's Acoustics Group who have reproduced my work and expanded upon it.

This experience gives me a unique set of experiences that I have used to advise my clients for projects currently under development or for lawsuits related to existing projects.

Q: What materials have you reviewed in this matter?

A: I have reviewed:

1. The sound study conducted by Burns & McDonnell Engineering Company, dated May 18, 2018;
2. The contour maps of the Project depicting the 45 dBA Leq boundaries from the sound study model;
3. The pre-filed testimony of Chris Howell, summarizing his and the Burns and McDonnell Engineering report assessing noise from the Prevailing Wind Park Project (PWPP);
4. The pre-filed testimony of Dr. Mark Roberts regarding Prevailing Wind Park;
5. The testimony of David M. Hessler, dated May 4, 2018, regarding his review of the Dakota Range Wind Project and recommendations for noise thresholds;
6. The testimony of David M. Hessler, dated March 28, 2018, regarding the Crocker Wind Farm; and
7. Bon Homme County's Article 17, regulation of wind energy systems (WES).

Q: After reviewing those materials, what is your overall impression regarding any potential health risks posed by the proposed Project?

A: The project, as proposed, has a significant potential to cause adverse health effects related to sleep disturbance and annoyance to audible sounds from the wind turbines, especially at night. The recommended thresholds by Howell and Hessler of 45 dBA Leq, are not appropriate for rural communities. This is especially true for communities that have no prior experience with utility scale noise sources operating 24/7/365 that produce fluctuating, pulsatile, tonal infra and low frequency sound. Wind turbine noise emissions have specific characteristics that make them more likely to cause these adverse effects than other common rural noise sources. Thus, criteria intended for urban/suburban communities where traffic noise is the typical nighttime noise source (urban hum) are not

suitable for communities were people have an expectation of quiet. People in rural communities have lifestyles that are based on the quiet nature of most rural communities at night. This is reflected in ANSI-ASA S12.9 Part 4 "Noise Assessment and Prediction of Long-term Community Response" Appendix F, which cautions:

> **"F.3.4.1** In newly created situations, especially when the community is not familiar with the sound source in question, higher community annoyance can be expected. This difference may be equivalent to up to 5 dB.
>
> **"F.3.4.2** Research has shown that there is a greater expectation for and value placed on "peace and quiet" in quiet rural settings. In quiet rural areas, this greater expectation for "peace and quiet" may be equivalent to up to 10 dB.
>
> **"F.3.4.3** The above two factors are additive. A new, unfamiliar sound source sited in a quiet rural area can engender much greater annoyance levels than are normally estimated by relations like equation F.1. **This increase in annoyance may be equivalent to adding up to 15 dB to the measured or predicted levels." (Emphasis added)**

The community's response to the wind turbine noise will be as if the wind turbines were 15 dB louder than what is being predicted. This caution was in the EPA's 1974 Levels Document and also is present in current ISO standards followed in the EU and other countries. It is accepted acoustical practice that is overlooked by wind energy developers and their consultants.

Q: Are there sound level limits that you find more appropriate for rural communities?

A: In 2008 I worked with George Kamperman, one of the senior acousticians who led in the development of community noise limits for urban and suburban communities in the 1960s and 70s, to determine what the proper sound limits should be for wind turbines in quiet rural communities. Wind turbines were never considered when the community noise limits were set and especially it was not anticipated that they would be located in quiet rural areas near homes. So we decided to apply the same type of analysis to wind turbine noise as had been done for other common community noise sources in the past. We looked at when the turbines would operate, what the nighttime background sound levels would be in the receptor's location, and how much sound they emit in each frequency band. Then applying methods for calculating sound propagation that reflect how low frequency sound differs from higher frequency sound, we estimated the distances needed to prevent the noise of ten (10) wind turbines of the 1.5 MW class common in the late 2000s from causing nighttime annoyance inside a home with windows open.

We determined that the maximum sound level for audible sounds should be 35 dBA

(Leq) and 50 dBC, especially for nighttime wind turbine noise. We also limited the new noise source to be no more than 5 dBA louder than the pre-operational background sound level at night. Typical nighttime background sound levels are under 30 dBA (L90) in these communities so the 35 dBA acts as an upper limit.

The Kamperman/James document was subsequently reviewed in a paper titled: "Noise: Wind Farms," by three experts (Shepherd (Psychoacoustics), Hanning (Sleep Medicine Specialist) and Thorne (low frequency acoustician)) and published in the 2012 edition of the Encyclopedia of Environmental Management. They review the special character of wind turbine noise and in the Appendix update the criteria that Mr. Kamperman and I prepared in 2008 to address the fluctuating character of wind turbine noise. I have attached a copy as Exhibit 2 of their paper for the details behind these criteria.

Q: Are there other acousticians who have made similar recommendations for noise thresholds in rural communities?

A: Yes, there are many who have made similar recommendations. In 2017, Dr. Paul Schomer, the Emeritus Director of the Acoustical Society of America's Standards Committee published a paper titled: "A possible criterion for wind farms" at the 173rd meeting of the Acoustical Society of America. (*See* Exhibit 3.) Dr. Schomer, in his capacity as Director of the ASA Standards Committee has directed the work of the American National Standards Institute (ANSI) groups that produce the S12 consensus standards on how to measure noise and how noise affects people for over 30 years.

In his 2017 paper, he reviews how proper application of the ANSI standards for assessing the impact of a new noise source on a community to avoid adverse impacts results in a criterion of 36 to 38 dBA Leq. Dr. Schomer explains how the character of wind turbine noise requires lower limits than other common community noise sources.

He also bases his recommendation on the findings of a major study conducted by Health Canada (the Canadian equivalent to the US Centers for Disease Control (CDC). That study looked at a sample of just under 2000 people living within 3-5 km of six wind projects in Ontario. It found that the percent of people who report they are highly annoyed by wind turbine noise jumps dramatically from less than 2% when the modeled sound levels are 35 dBA Leq or less to over 10% for levels between 35 and 40 Leq. Health Canada defines High Annoyance to noise as an adverse health effect in accordance with the World Health Organization (WHO) and other bodies. The limits for new wind projects in Canada are set at 40 dBA Leq (worst case one hour). Thus, if PWPP is permitted to produce higher sound levels, it should be expected that annoyance will also be higher for those closest to the turbines.

Other countries, such as the U.K., Australia, and New Zealand, also use 40 dBA Leq

as the upper limit for wind turbine projects. Some, like Germany and other European countries have limits of 35 dBA Leq for rural communities. Limits like these have not prevented wind energy development in those countries. The developers have to select locations where there is sufficient distance to prevent noise from exceeding the limits or work out private easement contracts with neighbors.

Q: Has the use of a limit of 40 dBA Leq been found adequate to prevent adverse effects?

A: No. This might be anticipated from the Health Canada finding that 10% of people find sound levels in the range of 35 to 40 dBA Leq are highly annoyed, increasing to about 14% for higher sound levels. Jurisdictions that set the threshold at 40 dBA Leq must deal with ongoing complaints, threats of legal action and other indicators that 40 dBA Leq is not sufficiently protective. Proper siting criteria can prevent this.

Q: How can, what appears to be a small change in sound level from 40 Leq to my 35 dBA Leq or Dr. Schomer's 36-38 dBA Leq, make such a difference in acceptability?

A: While it may appear that the difference is only a few decibels, it is important to remember that a 3 dB change in sound levels represents a doubling or halving of the acoustic energy. Thus, a change from 40 dBA to 37 dBA Leq is equivalent to turning off half of the wind turbines in a project designed to meet the 40 dBA Leq limit. This implies that the 3 dB change increases the setback distances by a substantial amount.

Based on my experience reviewing Ontario projects designed for 40 dBA Leq the closest homes to wind turbines have setbacks of about 1800 feet. To meet a 37 dBA Leq limit these setbacks would be increased to about 2500 feet. To meet the 35 dBA Leq limit the setback distance would be on the order of 3600 feet. To prevent annoyance during nighttime periods from multi-turbine projects Mr. Kamperman and I calculated the setback would need to be 1.25 miles (2km).

This is primarily because the rural areas are so quiet at night that even at these distances wind turbines can be audible inside homes where people are sleeping, especially those that sleep with windows open. To avoid this disturbance, the people would need to change their behavior to how suburban people cope with noise by having windows closed much of the time and using air conditioning for summer cooling.

In parts of Germany and Poland noise limits have been replaced with arbitrary setback distances based on the diameter of the wind turbine's rotors. The setbacks are equivalent to ten (10) times the rotor diameter. Thus, for a wind turbine with a 110 meter diameter blade the setback would be about 3600 feet. This is equivalent to the setbacks

derived for 35 dBA Leq limits discussed above but avoids the complexity of sound modeling.

Q: Should these limits be applied to the property lines or to the homes?

A: I am a strong supporter of property rights and believe that noise that exceeds known safe levels should not be imposed on people just because they live near a neighbor who wishes to host wind turbines. This position influences my response to this question.

If a person owns property that is primarily agricultural with a residential home, they should still have the entire property protected to prevent future restriction on how the land can be used. For example, in the future they decide to subdivide their property for residential purposes. If the limit was set to the home, it is possible that the future development would be in a location where the noise is excessive for residential land use. If the limits are set for the homes, not the property lines, then wind project's noise emissions physically trespass on the neighbor's property without any compensation for the non-participating neighbor. The phrase "Noise Trespass" has been used in states like Michigan and Ohio were the debate over setting limits for the property line vs home are debated.

The question may be easier to answer if we look at other forms of pollution than noise. Take water pollution for example. If a farmer raises livestock and that livestock causes pollution of a stream passing through the property, the adjacent property owner is deprived from using the stream for normal purposes. In most states that I am aware of, the pollution is controlled at the emitter's property line. The same should be true for noise pollution. The landowner hosting the wind turbine may have a right to have a wind turbine on his/her property but does not have any rights to allow that sound energy to trespass onto the properties of neighbors. The obligation to prevent that trespass is on the property owner hosting the wind turbine(s) and the utility operator.

There is nothing that prevents the utility developer from working out an agreement with non-participating property owners to compensate them for allowing higher sound levels on parts of their property that are between the home and property line that they know will not be used for residential developments. Thus, the property line should be the default for protecting neighbors. If the utility developer/operator is willing to provide compensation for the "Noise Trespass" they can work out arrangements to protect that part of the property that is residential or may become residential in the future.

Q: What other characteristics of wind turbine sound emission affect adjacent properties?

A: The limits using dBA criteria are focused on sound that is in the speech frequency range. Sounds that are heard. The A-weighting process de-emphasizes low frequency

sounds from 500 Hz and below. That includes sound that is felt. Like the bass beat from a neighbor's home when they play the stereo loud. Modern utility scale wind turbines like those proposed for PWPP have most of their acoustic energy in the range from under 1 Hz to 500 Hz that is ignored by the dBA calculations. This sound is called infrasound (0-20Hz) and low frequency sound (20-250Hz). Low frequency sounds, including infrasound, are problematic because they propagate much further than higher frequency sound with little loss of energy. That results in people hearing a rumble (very low frequency noise) or roar (low frequency sound above 100Hz) that penetrates their homes, especially at night when the house is quiet. Infra and low frequency sounds are not blocked by normal home construction methods for walls, roofs and windows.

Infra sound is a special case of low frequency sound where the energy has to be very high for the sound to be audible, but some people can "feel" the sound as body vibrations, pressure changes, migraines, tinnitus, dizziness, and other non-auditory effects. This is not limited to wind turbines. It also is a characteristic of helicopter sound emissions or large fans in high rise office buildings when they need maintenance. (In that last case the term is Noise induced Sick Building Syndrome.)

Dr. Schomer's 2015 paper titled: "A theory to explain some physiological effects of the infrasonic emissions at some wind farm sites" (attached as Exhibit 4) explains how these inaudible levels of wind turbine sound, which are presented as pressure pulsations inside of homes, can trigger these non-auditory sensations and symptoms. The phrase "Wind Turbine Syndrome" was coined by Dr. Nina Pierpont, MD. to describe them. These symptoms cannot be explained as occurring due to audible sound levels in the speech frequency range. See the attached Exhibit 5, which is a one-page summary of wind turbine blade pass frequency and effects, for an explanation of how these pulsations are produced.

Mr. Hessler refers to a study in his written testimony that he participated in for the Wisconsin Public Service Commission for the Shirley Wind Project in Brown County Wisconsin. That study was conducted in the homes of my clients who had filed complaints with the WI PSC during a hearing on a second wind project in another part of the state. The study that Mr. Hessler points to was designed by me for my clients and accepted by the PSC. I developed the test protocol, selected the homes to be tested, and picked the acousticians who would conduct it. Because the complainants were my clients, I did not participate, but was given full access to the data and did an independent analysis for the PSC which confirmed the presence of pulsating infrasound.

This study confirmed that inside the homes, wind turbine pulsations created by the loss of lift on the blades as the blade passes into the wind deficit region in front of the tower

was present at levels almost the same as outside the homes. I have attached as Exhibit 6 a set of graphs showing the infrasound that I prepared for the Brown County Health Department showing the infrasound using two types of instrumentation. The graph on the first page shows the spectrograms from multi-hour micro barometer tests in the home with the highest infra sound during the test Mr. Hessler describes. (This was R1 of the study at 3600 feet from the nearest wind turbine). The infrasound pulsations are seen as horizontal bands of energy and are explained in the notes. The last page shows a simultaneous test at R1 and another home located about four (4) miles away where the occupants experience pressure related headaches when the turbines are operating even though none of the wind turbines are visible. The infrasound traces are still present at this distance although somewhat attenuated. It is this ability to propagate long distances that makes the infra sound component of wind turbine noise so problematic.

Brown County's Health Department declared the entire region within 2.5 miles of the Shirley Wind project to be a "Human Health Hazard" zone. This is an official classification under Wisconsin law.

The owners of two of the homes (R1 at 3600 feet and R3 at one mile) abandoned their homes shortly after the project started to operate due to symptoms that included nausea and dizziness. Those homes are still vacant. R2 was abandoned to the mortgage company who resold it to a different family.

Q: Has this study been duplicated?

A: Yes, several times by myself and other acousticians, most notably Steven Cooper of Australia's Acoustics Group. Cooper's Cape Bridgewater study is very detailed and lengthy but can be obtained at http://www.pacifichydro.com.au/english/our-communities/communities/cape-bridgewater-acoustic-study-report/.

He finds that the test subjects in his three test homes were able to reliably sense the starting and stopping of the wind turbines without visual cues. One test subject was functionally deaf due to childhood illness damaging the auditory nerves. This test subject was able to sense the operation of distant wind turbines without any auditory or visual cues. Mr. Hessler refers to this study as one that resulted in him rethinking his position on inaudible infrasound as a source of adverse health effects.

Dr. Schomer references this study in his paper (referenced earlier) and also conducted a peer review of it. His peer review concludes:

> "The results are that there is a cause and effect relationship between turbine power output and subject response, and, at the same time there is no correlation between subject

response and either sound level or vibration level. These results show that there is a non-visual, non-audible pathway by which wind turbine emissions can cause some specific effects in some people. These results say nothing about the nature of these effects. Nothing internal to the body is discussed. We again reiterate to government and to wind farm operators, if you don't believe the results, replicate the study using clearly independent consultants.

"Some may ask, this is only 6 people, why is it so important? The answer is that up until now windfarm operators have said there are no known cause and effect relations between windfarm emissions and the response of people living in the vicinity of the windfarm other than those related to visual and/or audible stimuli, and these lead to some flicker which is treated, and "some annoyance with noise." This study proves that there are other pathways that affect some people, at least 6. The windfarm operator simply cannot say there are no known effects and no known people affected. One person affected is a lot more than none; the existence of just one cause-and-effect pathway is a lot more than none. It only takes one example to prove that a broad assertion is not true, and that is the case here. Windfarms will be in the position where they must say: "We may affect some people." And regulators charged with protecting the health and welfare of the citizenry will not be able to say they know of no adverse effects. Rather, if they choose to support the windfarm, they will do so knowing that they may not be protecting the health and welfare of all the citizenry."

Q: Has this been duplicated in a controlled laboratory test?

A: Yes. Mr. Hessler references such a study in his testimony. This was reported in a paper presented by Steve Cooper at the Acoustical Society of America's December 2017 conference and published in the Proceedings of Meetings on Acoustics (POMA) in a paper titled: "Subjective perception of wind turbine noise - The stereo approach."

Steve Cooper designed a laboratory where he could accurately reproduce the sounds he measured in the Cape Bridgewater homes in both frequency and time domain, down to 3 Hz. He created an audio sample from one of his Cape Bridgewater measurements that reproduced the pulsations at the infrasonic rate of the blade pass frequency. He did blind testing of people who included some who live in wind projects and by others who did not think they were sensitive to such sounds.

Cooper's controlled experiments reproduced the acoustical characteristics found inside homes where sensitive people have filed complaints of sensations and other non-auditory complaints. Inaudible sound pulsations occurring at infrasonic rates emitted

by wind turbines were shown to cause perceptible sensations in test subjects who self-identified as being sensitive to wind turbine infra sound. Those who self-identified as being sensitive to wind turbine infra sound were able to reliably detect when the sample was played or not and could also detect the direction from which the sound came (blindfolded and sitting in a swivel chair). Some of the test subjects who did not identify as "sensitive" were also able to detect the presence of the infra sound pulsations.

Mr. Cooper's study shows that:

1. It is possible to reproduce in a controlled laboratory experiment the acoustic characteristics of wind turbine sound pressure pulsations occurring at infrasonic rates found in homes of people living near utility scale wind turbines who have filed complaints of adverse sensations and health effects.

2. These inaudible acoustic conditions reliably trigger in self-identified "sensitive people" sensations and adverse effects associated with the complaints by people who live in or near the footprint of utility scale wind turbines.

Wind turbine sound emissions consisting of dynamically modulated pressure pulsations at infrasonic rates synchronized to the blade pass frequency were shown to cause sensations and other adverse effects under controlled laboratory conditions.

There are other studies of this type being conducted but they do not use a real audio sample from a home where people have reported the sensations. Those studies rely on what is being called a "surrogate sample" that does not include the dynamically modulated pressure pulsations, they only reproduce the frequency and sound pressure levels measured in the homes. Thus, they do not include the most important characteristic of pulsating noise. These studies report that the test subjects do not respond to the sound. This is a strong piece of evidence that it is the pulsations and not the infra and low frequency sound levels that are important in producing sensations. It also explains why people do not report these sensations when exposed to steady infra sound from the natural environment.

Q: Do you have any comments on the Burns-McDonnell Sound Study for the Prevailing Wind Park Project?

A: Yes. First as indicated by my testimony above I disagree with the idea that a threshold of 45 dBA Leq is protective for people living near the wind project. Second, I reviewed the information on the computer model prepared for the report. I find the model is deficient in many ways. One significant way is that it fails to include two important sets of tolerances. The sound power data used as input to the model is derived using a method that has about a ± 2 dB tolerance for measurement repeatability. This tolerance should have been added to the sound power levels used as input to the model to account for known

variability in measurement data. Also, the model uses the formulas and protocols from ISO 9613-2 which states it is not applicable for noise sources that are more than 30 meters above the ground or receiver elevation. Even if the model was appropriate for wind turbine noise the model has known tolerances of ± 3 dBA. This should have also been applied as an adjustment to the Burns-McDonnell sound model. Given these two tolerances the predicted sound levels are as much as 5 dBA low.

Further, the values used for ground attenuation are not disclosed. The proper value for ground attenuation is "0" to turn off any calculations of ground effect. This is because the height of the wind turbines means that the sound emitted by them radiates directly from the blades to the homes without interaction with the ground. The ISO ground attenuation calculations are intended for ground-based noise sources where the sound radiates along a line from source to receiver just above the ground.

Dr. Schomer has in the past, identified additional problems with wind turbine noise prediction using the ISO model methods. He was a member of the committee that developed the ISO 9613-2 standard and its ANSI equivalent (ANSI/ASA S12.62). He has repeatedly stated in hearings and conferences that the model does not properly predict the propagation of low frequency noise. The ISO model range for accuracy is focused on sound in the frequencies that are most important for other types of ground-based community noise sources. In testimony he gave for the White Pines project in Ontario he stated that the model is likely to underestimate the sound propagation from wind turbines by as much as 11 dBA. This is in addition to the issue of tolerances for the calculations. As I have stated above I have also measured wind turbines operating at levels 10 dBA Leq or more above the predicted sound levels.

Q: What does this mean for the Prevailing Wind Park project?

A: It means that the predicted sound levels at receptors in and near the PWPP are <u>at least 5 dBA less</u> than what should be expected if the project was operating and the sounds measured and compared to the model's predictions. I have conducted such studies and routinely find that the wind turbines exceed the modeled sound levels by 5 dBA and in some cases, especially when the operating mode includes high blade angles or wind turbulence, the model under predicts by 10 or more dBA.

The flaws in the model make it likely that if the project is approved as designed there will be many complaints of annoyance and some of adverse health effects along the lines of what occurred at Shirley Wind and Cape Bridgewater.

Before any decisions are made on permitting this project the applicant should be required to submit a new model that applies the known tolerances to the input data. It should also show the contour lines for 30, 35, and 40 dBA. These new sound levels should

then be viewed as indicators of what the community will experience on a day when the wind turbines are operating under optimum conditions for the lowest noise emissions. They are not precision predictions. Review of the model should be done keeping in mind that the operating values can be as much as 10 dB higher than what is predicted, under operating conditions that would be considered normal.

The likely complaint times will be at night when winds at the blades are strong with high wind shears at the hub elevation, but calm or no winds at the ground (called a stable nighttime atmosphere). Studies have shown that these weather conditions occur as frequently as 2 out of 3 nights during warm seasons. Since the ground level winds are calm there is no wind induced noise or leaf rustle to mask the wind turbine noise. This condition is recognized in many jurisdictions (e.g. Ontario) as the "worst-case" condition for complaints.

Q: Do you have any comments on Dr. Roberts' testimony.

A: Yes, however I understand the Dr. Punch will be addressing that testimony in more detail. What I would add is that, in my opinion as an acoustician, Dr. Roberts is not qualified to speak to the issue of acoustics or human response to wind turbine noise. Acoustical engineers are trained in how to measure sound and relate those measurements to human and community response. I saw nothing in his background that qualifies him to speak to these issues.

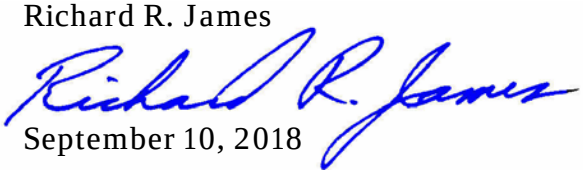
Dr. Roberts' testimony is not reliable when read by an experienced acoustician who understands the particular character of wind turbine noise that leads to it being highly annoying at sound levels well below other common community noise sources.

Q: Do you have anything further to add at this time?

A: The foregoing written testimony is to be presented to the South Dakota Public Utilities Commission for SD PUC Docket EL 18-026.

I reserve the right to revise and expand upon these written comments during the hearing.

Richard R. James



September 10, 2018

Bio Materials for: Richard R. James
Ver: Nov. 8, 2017

Mr. James is the Owner and Principal Consultant for E-Coustic Solutions, LLC, of Okemos, Michigan. He has been a practicing acoustical engineer for over 40 years. He started his career as an acoustical engineer working for the Chevrolet Division of General Motors Corporation in the early 1970s. His clients include many large manufacturing firms, such as, General Motors, Ford, Goodyear Tire & Rubber, and others who have manufacturing facilities where community noise and worker noise exposure occur. In addition, he has worked for many small companies and private individuals. He was actively involved with the Institute of Noise Control Engineers (INCE) since its formation in the early 1970's. He was a full Member from early in the 1990's through 2017.

His academic credentials include a degree in Mechanical Engineering (BME) from General Motors Institute, Flint Michigan (now Kettering Institute). He has been an adjunct Instructor to the Speech and Communication Science Department at Michigan State University from 1985 to 2013 and an adjunct Professor for the Department of Communication Disorders at Central Michigan University from 2012 through 2017. In addition, Mr. James served on the Applied Physics Advisory Board of Kettering Institute from 1997 to 2007.

Specific to wind turbine noise, he has worked for clients in over 60 different communities.

He has provided written and oral testimony in approximately 30 of those cases. He has also authored or co-authored four papers covering wind turbine noise topics including:

- Criteria for wind turbine projects necessary to protect public health (2008),
- Demonstrating that wind turbine sound immissions are predominantly comprised of infra and low frequency sound (2011), and
- A peer reviewed historical review of other types of low frequency noise sources with similar sound emission characteristics, such as large HVAC systems (fans) which caused noise induced Sick Building Syndrome and other noise sources that have known adverse health effects on people exposed to their sound. (2012).
- A peer reviewed literature review of research spanning 40 years showing wind turbines cause risks of adverse health effects from both audible and inaudible sound emissions (2016).

He has been qualified as an expert in acoustics for hearings and court proceedings in several countries. Examples of recent qualifications are:

Jurisdiction	Before	Qualified as:
Ontario, CA (January 2014)	Ministry of Environment (MOE) and Environmental Review Tribunal (ERT)	Qualified to provide evidence on matters related to acoustics and noise control engineering and wind turbines
Alberta, CA (Dec. 2013)	Alberta Utilities Commission (AUC)	An acoustical engineer and acoustician with expertise in the field of sound including noise, low frequency noise, sounds emitted from industrial wind turbines and human response to noise.
Michigan, US	Michigan Circuit Court	1. acoustician with expertise in measurement of wind turbine noise and its effects on people. (Dec. 2013) 2. acoustician qualified to opine that the plaintiff's symptoms were caused by the defendant's wind turbines. After special Daubert Hearing (Dec. 2013)

EXHIBIT Int. I-1(a)

Revised: Nov. 8, 2017

Computers to quiet the factory

Ever since the Occupational Safety & Health Administration (OSHA) adopted industrial noise standards in 1971, plant engineers have been struggling to reduce the ear-piercing din in factories. But it is a tough job. The hundreds of machines inside a factory produce different sounds, each of which interacts differently with nearby equipment and partitions. Even skilled acoustical engineers often misjudge the effort needed to get down to the noise level the government permits. And because noise control is often expensive, mistakes can be costly.

Now, however, many corporations are turning to computer models to make sure their noise-control efforts will be cost-effective. Spurred by the falling cost of computer time and the high price of noise control, companies are using models to ensure that newly built plants will comply with OSHA noise rules. Managers are also using computers to test whether modifications of an existing plant will actually reduce noise. And executives are finding that models enable them to contest ineffective noise-control measures proposed by the government.

The computer's advantage. A noise model is based on equations that state in mathematical form the same laws of physics that consultants have traditionally used to forecast sound levels. These equations predict, for example, the effects of bouncing sound off a wall or absorbing it in acoustical tile. To apply a model to a particular plant, a consultant first measures the several different noises emitted by each machine, then records the size, nature, and placement of noise barriers such as walls and ceilings. When these data are fed into the noise model, engineers can get information about the noise level anywhere in the plant.

Without the computer, a noise consultant must calculate intuitively. The advantage of having a model is in being able to track interactions among a larger number of variables to predict the noise level at each station. A model developed by Total Environmental Systems Inc. in East Lansing, Mich., can cope with 3,500 noise sources and 250 partitions. "There are no more than 10 people in the country who can intuitively evaluate 100 variables," says Richard R. James, TES vice-president.

Many company officials are enthusiastic about the success of these models. Using a computer model developed by TES, General Motors Corp. found that it could slash by 25% its expected use of noise-reducing material in the body-fabricating area of its new Oklahoma City assembly plant. Tests made after the plant was built showed that the model had predicted the actual noise level in the plant to within 2 decibels, a high degree of accuracy. "We can't afford trial and error," notes Woodford L. Van Tifflin, the engineer who oversees GM's noise control system.



Peter Yates

Engineers James and Van Tifflin: Using models to plan noise control.

Costs. The average machine shop could not afford the $50,000 it cost GM to have TES model a 750,000-sq.-ft. portion of its Oklahoma City plant. The TES prices for less complex jobs start at $18,000. But even clients paying the highest fees say that the savings from modeling more than cover the costs. "Modeling prices are not out of line," argues Robert F. Birdsall, a Ford Motor Co. environmental engineer. Ford recently completed noise modeling for its new Batavia (Ohio) transaxle plant, slated to be in production by the 1981 model year.

Most of the modeling of existing plants is aimed at preventing OSHA citations for excessive noise. But modeling also helps a company fight alleged violations of noise standards. Stanadyne Inc. in Windsor, Conn., recently used a model to show that the government overstated—by a factor of 20—the effectiveness and thus the feasibility of noise control measures that it claimed Stanadyne should have used at its Bellwood (Ill.) plant to keep workers from being exposed to more than 90 decibels. The model's results played a key role in a judge's Dec. 28 decision in favor of the company, claims Stanadyne's attorney, Columbus R. Gangemi Jr. Testimony based on a model "is easier for the court to understand and easier to defend" than traditional expert testimony based on engineering analysis alone, he says.

Saving time. In addition to eliminating the cost of unnecessary or ineffective noise-control measures, modeling husbands executive time. The model can generate a noise map of a new plant using colors and contour lines to indicate the sound level at each worker station. Additional maps then can display the impact of various noise-reduction strategies. So, rather than having to wade through statistical tables or try to follow complex oral explanations, managers can see at a glance what areas in the plant have noise problems and the effect of potential solutions. "It puts complex information into a meaningful summary," says Ford's Birdsall.

Although users of noise modeling are enthusiastic, there is still some skepticism in the acoustical consulting community. These doubts persist despite the widespread use of modeling to cope with other forms of industrial pollution (BW—Oct. 29). "It could be a gimmick," says Paul Jensen, manager of the industrial noise division at Bolt Beranek & Newman, an acoustical consulting firm in Cambridge, Mass. Jensen contends that it is more important to consider the worker. "The problem with the model is that it doesn't say a darn thing about the worker—where he is, how he moves in and out of noisy areas."

Many other consultants, though, contend that Jensen overstates the case against modeling. "We use it successfully for companies having 5 to 1,000 employees," counters Thomas D. Miller, vice-president of Donley, Miller & Nowikas Inc. in East Hanover, N. J. But he cautions that modeling, like any mathematical simulation, is only as valid as the data base and operating assumptions on which it is built. ■

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BIOGRAPHICAL SKETCH

NAME	POSITION TITLE	BIRTHDATE
Richard R. James	Principal Consultant, E-Coustic Solutions, LLC (2006-)	3/3/48

ACADEMIC CREDENTIALS

INSTITUTION	DEGREE/POSITION	YEAR	FIELD
General Motors Institute, Flint, MI	B. Mech. Eng.	1966-1971	Noise Control Engineering
Michigan State University, East Lansing, MI	Adjunct Instructor	1985-2013	Acoustics and Effects of Noise on People
Central Michigan University, Mount Pleasant, MI	Adjunct Professor	2012-2017	Wind Turbine Noise and its Impact on People

RESEARCH AND PROFESSIONAL EXPERIENCE:

Richard R. James has been actively involved in the field of noise control since 1969, participating in and supervising research and engineering projects related to control of occupational and community noise in industry. In addition to his technical responsibilities as principal consultant, he has developed noise control engineering and management programs for the automotive, tire manufacturing, and appliance industries. Has performed extensive acoustical testing and development work in a variety of complex environmental noise problems utilizing both classical and computer simulation techniques. In 1975 he co-directed (with Robert R. Anderson) the development of SOUND™, an interactive acoustical modeling computer software package based on the methods that would be later codified in ISO 9613-2 for pre and post-build noise control design and engineering studies of in-plant and community noise. The software was used on projects with General Motors, Ford Motor Company, The Goodyear Tire & Rubber Co., and a number of other companies for noise control engineering decision making during pre-build design of new facilities and complaint resolution at existing facilities. The SOUND™ computer model was used by Mr. James in numerous community noise projects involving new and existing manufacturing facilities to address questions of land-use compatibility and the effect of noise controls on industrial facility noise emissions. He is also the developer of ONE*dB(tm) software. He was also a co-developer (along with James H. Pyne, Staff Engineer GM AES) of the Organization Structured Sampling method and the Job Function Sound Exposure Profiling Procedure which in combination form the basis for a comprehensive employee risk assessment and sound exposure monitoring process suitable for use by employers regulated by OSHA and other governmental standards for occupational sound exposure. Principal in charge of JAA's partnership with UAW, NIOSH, Ford, and Hawkwa on the HearSaf 2000tm software development CRADA partnership for world-class hearing loss prevention tools.

1966-1970	Co-operative student: General Motors Institute and Chevrolet Flint Metal Fabricating Plant.
1970-1971	GMI thesis titled: "Sound Power Level Analysis, Procedure and Applications". This thesis presented a method for modeling the effects of noise controls in a stamping plant. This method was the basis for SOUNDTM.
1970-1972	Noise Control Engineer-Chevrolet Flint Metal Fabricating Plant. Responsible for developing and implementing a Noise Control and Hearing Conservation Program for the Flint Metal Fabricating Plant. Member of the GM Flint Noise Control Committee which drafted the first standards for community noise, GM's Uniform Sound Survey Procedure, "Buy Quiet" purchasing specification, and guidelines for implementing a Hearing Conservation Program.
1972-1983	Principal Consultant, Total Environmental Systems, Inc.; Lansing, MI. Together with Robert R. Anderson formed a consulting firm specializing in community and industrial noise control.
1973-1974	Consultant to the American Metal Stamping Association and member firms for in-plant and community noise.
1973	Published: "Computer Analysis and Graphic Display of Sound Pressure Level Data For Large Scale Industrial Noise Studies", Proceedings of Noise-Con '73, Washington, D.C.. This was the first paper on use of sound level contour 'maps' to represent sound levels from computer predictions and noise studies.

Nov. 1973	Published: "Isograms Show Sound Level Distribution in Industrial Noise Studies", Sound & Vibration Magazine
1975	Published: "Computer Assisted Acoustical Engineering Techniques", Noise-Expo 1975, Atlanta, GA which advanced the use of computer models and other computer-based tools for acoustical engineers.
1976	Expert Witness for GMC at OSHA Hearings in Washington D.C. regarding changes to the "feasible control" and cost-benefit elements of the OSHA Noise Standard. Feasibility of controls and cost-benefit were studied for the GMC, Fisher Body Stamping Plant, Kalamazoo MI.
1977-1980	Principal Consultant to GMC for the use of SOUND(tm) computer simulation techniques for analysis of design,layout, and acoustical treatment options for interior and exterior noise from a new generation of assembly plants. This study started with the GMAD Oklahoma City Assembly Plant. Results of the study were used to refine noise control design options for the Shreveport, Lake Orion, Bowling Green plants and many others.
1979-1983	Conducted an audit and follow-up for all Goodyear Tire & Rubber Company's European and U.K. facilities for community and in-plant noise.
1981-1985	Section Coordinator/Speaker, Michigan Department Of Public Health, "Health in the Work Place" Conference.
1981	Published: "A Practical Method for Cost-Benefit Analysis of Power Press Noise Control Options", Noise-Expo 1981, Chicago, Illinois
1981	Principal Investigator: Phase III of Organization Resources Counselors (ORC), Washington D.C., Power Press Task Force Study of Mechanical Press Working Operations. Resulted in publishing: "User's Guide for Noise Emission Event Analysis and Control", August 1981
1981-1991	Consultant to General Motors Corporation and Central Foundry Division, Danville Illinois in community noise citation initiated by Illinois EPA for cupola noise emissions. Resulted in a petition to the IEPA to change state-wide community noise standards to account for community response to noise by determining compliance using a one hour L_{eq} instead of a single not-to-exceed limit.
1983	Published: "Noise Emission Event Analysis-An Overview", Noise-Con 1983, Cambridge, MA
1983-2006	Principal Consultant, James, Anderson & Associates, Inc.; Lansing, MI. (JAA), Together with Robert R. Anderson formed a consulting firm specializing in Hearing Conservation, Noise Control Engineering, and Program Management.
1983-2006	Retained by GM Advanced Engineering Staff to assist in the design and management of GM's on-going community noise and in-plant noise programs.
1984-1985	Co-developed the 1985 GM Uniform Plant Sound Survey Procedure and Guidelines with James H. Pyne, Staff Engineer, GM AES.
1985-2013	Adjunct instructor in Michigan State University's Department of Communicative Sciences and Disorders from 1985-2013
1986-1987	Principal Consultant to Chrysler Motors Corporation, Plant Engineering and Environmental Planning Staff. Conducted Noise Control Engineering Audits of all manufacturing and research facilities to identify feasible engineering controls and development of a formal Noise Control Program.
1988-2006	Co-Instructor, General Motors Corporation Sound Survey Procedure (Course 0369)
1990	Developed One*dB(tm), JAA's Occupational Noise Exposure Database manager to support Organizational structured sampling strategy and Job Function Profile (work-task) approach for sound exposure assessment.
1990-1991	Co-developed the 1991 GM Uniform Plant Sound Survey Procedure and Guidelines with James H. Pyne, Staff Engineer, GM AES. Customized One*dB(tm) software to support GM's program.
1990-2006	Principal Consultant to Ford Motor Company to investigate and design documentation and computer data management systems for Hearing Conservation and Noise Control Engineering Programs. This included bi-annual audits of all facilities.
1993-2006	GM and Ford retain James and JAA as First-Tier Partners for all non-product related noise control services.
1993	Invited paper: "An Organization Structured Sound Exposure Risk Assessment Sampling Strategy" at the 1993 AIHCE

1993	Invited paper: “An Organization Structured Sound Exposure Risk Assessment Database” at the Conference on Occupational Exposure Databases, McLean, VA sponsored by ACGIH
1994-2001	Instructor for AIHA Professional Development Course, “Occupational Noise Exposure Assessment”
1996	Task Based Survey Procedure (used in One*dB(tm)) codified as part of ANSI S12.19 Occ. Noise Measurement
1995-2001	Coordinate JAA’s role in HearSaf 2000tm CRADA with NIOSH, UAW, Ford, and HAWKWA
1997-2007	Board Member, Applied Physics Advisory Board, Kettering Institute, Flint, Michigan
2000	Use of structured, interactive interviews in retrospective noise exposure assessment in an occupational epidemiologic study, Prince, Waters, Anderson, and James, JASA,, April 2000
2002-2006	Member American National Standards Institute (ANSI) Accredited Standards Committee S12, Noise
2006	Closed James, Anderson and Associates, Inc. (JAA) and founded E-Coustic Solutions (E-CS)
2006-Present	Consultant to local communities and citizen’s groups on proper siting of Industrial Wind Turbines. This includes presentations to local governmental bodies, assistance in writing noise standards, and formal testimony at zoning board hearings and litigation.
2008	Paper on “Simple guidelines for siting wind turbines to prevent health risks” for INCE Noise-Con 2008, co-authored with George Kamperman, INCE Bd. Cert. Emeritus, Kamperman Associates.
2008	Expanded manuscript supporting Noise-Con 2008 paper titled: “The “How To” Guide To Siting Wind Turbines To Prevent Health Risks From Sound”
2009	"Guidelines for Selecting Wind Turbine Sites," Kamperman and James, Published in the September 2009 issue of Sound and Vibration.
2010	Punch, J., James, R., Pabst, D., "Wind Turbine Noise, What Audiologists should know," Audiology Today, July-August 2010
2011	Jerry L. Punch, Jill L. Elfenbein, and Richard R. James, "Targeting Hearing Health Messages for Users of Personal Listening Devices," Am J Audiol 0: 1059-0889_2011_10-0039v1
2011	Bray, W., HEAD Acoustics, James, R., "Dynamic measurements of wind turbine acoustic signals, employing sound quality engineering methods considering the time and frequency sensitivities of human perception," invited paper for Noise-Con 2011, Portland OR
2012	James, R., "Wind Turbine Infra and Low Frequency Sound: Warning Signs that were not Heard," April 2012, Bulletin of Science, Technology and Society
2012	Appointed to position as Adjunct Professor in the Department of Communication Disorders at Central Michigan University.
2014	Negative Health Effects of Noise from Industrial Wind Turbines-Parts 1-3, Punch, J, James, R., Hearing Health Technology Matters, http://hearinghealthmatters.org/hearingviews/2014/wind-turbine-noise-evidence-health-problems/
2016	Punch, J. L., James, R.R., “Wind turbine noise and human health: a four-decade history of evidence that wind turbines pose risks,” Journal of Hearing Health and Technology Matters, October 4, 2016, http://hearinghealthmatters.org/journalresearchposters/files/2016/09/Final-Final-16-09-30-Wind-Turbine-Noise-Final-Manuscript-HHTM-Punch-James.pdf.

Professional Affiliations/Memberships/Appointments

Research Fellow - Metrosonics, Inc.	American Industrial Hygiene Association (through 2006)
National Hearing Conservation Association (through 2006)	Institute of Noise Control Engineers (Member through 2017)
American National Standards Institute (ANSI) S12 Working Group (through 2006)	Founder and Board Member of the Society for Wind Vigilance, Inc.
Adjunct Professor, CMU 2012-2017	Adjunct Instructor, MSU 1985-2013

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List of Recent Publications

Sept. 5, 2017

2000 JASA, April 2000, Prince, Waters, Anderson, and James, Use of structured, interactive interviews in retrospective noise exposure assessment in an occupational epidemiologic study

2008 Paper on guidelines for siting wind turbines to prevent health risks for INCE Noise-Con 2008, co-authored with George Kamperman, Kamperman Associates.

2008 Expanded manuscript supporting Noise-Con 2008 paper titled: "The 'How To' Guide To Siting Wind Turbines To Prevent Health Risks From Sound"

2009 "Guidelines for Selecting Wind Turbine Sites," Kamperman and James, Published in the September 2009 issue of Sound and Vibration.

2010 Punch, J., James, R., Pabst, D., "Wind Turbine Noise, What Audiologists should know," Audiology Today, July-August 2010

2011 Jerry L. Punch, Jill L. Elfenbein, and Richard R. James , "Targeting Hearing Health Messages for Users of Personal Listening Devices," Am J Audiol 0: 1059-0889_2011_10-0039v1

2011 Bray, W., HEAD Acoustics, James, R., "Dynamic measurements of wind turbine acoustic signals, employing sound quality engineering methods considering the time and frequency sensitivities of human perception," invited paper for Noise-Con 2011, Portland OR

2012 James, R., "Wind Turbine Infra and Low Frequency Sound: Warning Signs that were not Heard," April 2012, Bulletin of Science, Technology and Society, http://bsts.sagepub.com, DOI:10.1177/0270467611421845

2014 Negative Health Effects of Noise from Industrial Wind Turbines-Parta 1-3, Punch, J, James, R., Hearing Health Technology Matters, http://hearinghealthmatters.org/hearingviews/2014/wind-turbine-noise-evidence-health-problems/

2016 Punch, J. L., James, R.R., "Wind turbine noise and human health: a four-decade history of evidence that wind turbines pose risks," Journal of Hearing Health and Technology Matters, October 4, 2016, http://hearinghealthmatters.org/journalresearchposters/files/2016/09/16-10-21-Wind-Turbine-Noise-Post-Publication-Manuscript-HHTM-Punch-James.pdf.

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See Appendix A for proposed criteria

Noise: Windfarms

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Bob Thorne
Massey University, New Zealand

Abstract
Windfarms consist of clusters of wind turbines, which, when placed in populated areas, are associated with intrusive and unwanted sound. A relatively new noise source; wind turbine noise has characteristics sufficiently different from other, more extensively studied, noise sources to suggest that preexisting noise standards are not appropriate. Though research into the human impacts of wind turbine noise has appeared only in the last decade and in small quantity, the data suggest that, for equivalent exposures, wind turbine noise is more annoying than road or aviation noise. Furthermore, the particular characteristics of wind turbine noise may be likely to cause sleep disruption. As with other impulsive noise sources, time-aggregated noise metrics have limited utility in protecting public health, and a cluster of metrics should be used in order to estimate potential threat. At this time, however, the quantity and quality of research are insufficient to effectively describe the relationship between wind turbine noise and health, and so legislation should apply the precautionary principle or conservative criteria when assessing proposed windfarm developments.

INTRODUCTION

Planning authorities, environmental agencies, and policy makers in many parts of the world are seeking information on possible links between wind turbine noise and health in order to legislate permissible noise levels or setback distances. Concurrently, larger and noisier wind turbines are emerging, and consent is being sought for progressively larger windfarms to be placed even closer to human habitats. While noise standards can effectively and fairly facilitate decision-making processes if developed properly, the current standards on offer suffer severe conceptual difficulties. Specifically, noise metrics considered by many in the industry as best practice may in fact relate little to health outcome variables such as annoyance or sleep disruption. In this entry, we describe the physical characteristics of wind turbine noise, review the impact of such noise on humans, and critique current approaches to mitigation.

INDUSTRIAL WIND TURBINES

Industrial wind turbines transform kinetic energy from the wind into electricity, a practice dating back over 100 years. Structurally, wind turbines can be decomposed into three key components (Fig. 1). First, wind turbines possess a rotor, consisting of one or more blades designed to rotate when exposed to wind. The rotor can be thought of as a type of sail, catching wind in order to induce movement. Depending on the axis of blade rotation, wind turbines can be categorized as either horizontal-axis (the most common) or vertical-axis turbines. The second major component is the generator or "dynamo." The generator component includes a gearbox to regulate the speed of the dynamo and components to change blade pitch and plane of rotation with respect to wind direction. The dynamo can be used as a motor to maintain rotation at very low wind speeds. Third, there is a tower supporting the rotor and, typically, the generator. The size of a wind turbine can be specified either as a dimension (e.g., tower height measured from the ground to the top of a blade at its highest point) or as an electrical output (e.g., watts). Currently, turbines range from approximately 2 to 200 m high and from approximately 50 W to 6 MW.

Wind turbines can be erected in isolation or in sets and be located either onshore (i.e., terrestrial) or offshore (i.e., marine), though the latter is associated with higher construction costs. Industrial-scale wind energy generation, involving the saturation of an optimum number of wind turbines in a fixed area of land, gives rise to the concept of the "windfarm" or "wind park." Wind energy developers seek areas that have good consistent wind flow and close access to energy grids. The proliferation in the number of windfarms established globally in the past decade has been largely driven by environmental concerns such as climate change, renewableness and sustainability, and strategic energy considerations relating to the depletion of fossil fuels.[1] However, in the absence of large-scale electricity

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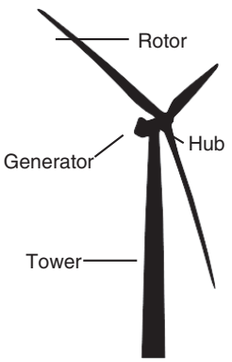


Fig. 1 Components of a typical horizontal-axis wind turbine.

storage devices (i.e., batteries), the contribution of wind energy to a nation's electricity needs is likely to be peripheral. Another barrier is social acceptance, with reviewed social surveys indicating citizens supporting renewable energy in principle but opposed to having windfarms in their immediate vicinity due to visual impacts on the landscape, shadowflicker from the blades, and fears of noise-induced annoyance and sleep disruption.

ACOUSTIC PROFILE OF WIND TURBINE NOISE

The sound generated from a windfarm is qualitatively different from any sound source commonly met in the environment, can rapidly switch from being stationary to nonstationary, and can vary by as much as 20 dB within a single minute. When it interferes with human activities, wind turbine sound becomes a type of noise. Analysis of windfarm noise poses distinct challenges, including the identification of acoustic energy that can be directly attributed to the turbines and the detection of special audible characteristics, including distinct tonal complexes and modulation effects. Windfarm noise is often a broadband low-amplitude noise constantly shifting in character ("waves on beach," "rumble-thump," "plane never landing." etc.). In this respect, windfarm noise is not like, for example, traffic noise or the continuous hum from plant and machinery. When assessed, wind turbine noise is often related to either wind speed (m/s) or electrical output (watts) and typically increases with both.

When the wind reaches a blade, it flows both over and under the blade. The part of the airflow with momentum great enough to break away forms trailing vortexes and turbulence behind the blade, producing a set of sound sources. The power of each sound source depends on the strength of the turbulence, which in turn depends on the speed of airflow; the compressibility and viscosity of the air; the design and surface texture (roughness) of the blade; the wind speed; and the velocity of the blade at that point. The faster the blade rotates, the earlier the breakup in the boundary vortexes and the greater the interaction between the vortexes emanating by adjacent wind turbines. An amplification of potential noise occurs when two or more turbines are, or nearly are, synchronous, such that the blade passing pulses coincide and then go out of phase again.[2] With exact synchronicity, there is a fixed interference pattern; with near synchronicity, concurrent arrival of pulses will change over time and place.

Noise emissions from modern wind turbines are primarily due to turbulent flow and trailing edge sound, blade characteristics, blade/tower interaction, and to a lesser degree, mechanical processes. The most commonly used description of wind turbine noise is the A-weighted

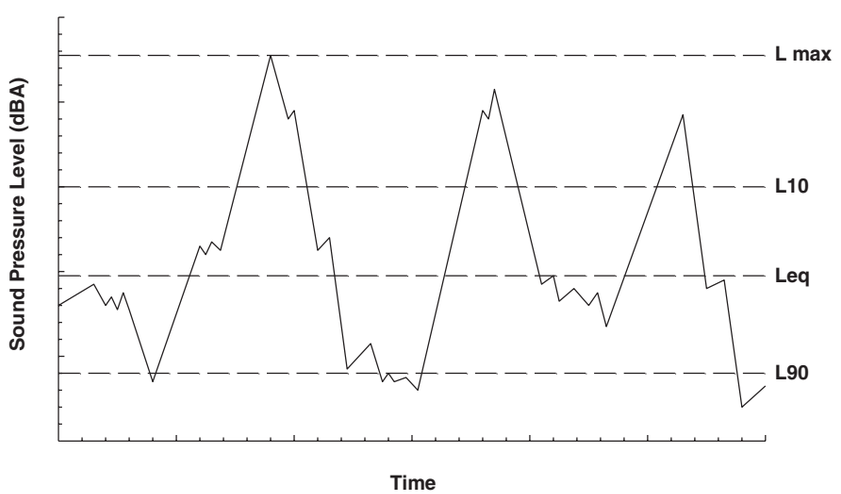


Fig. 2 Chart illustrating different noise descriptors. L10 is the level exceeded 10% of the time, while L90 is the level exceeded 90% of the time. The time-average (equivalent continuous) sound pressure level, Leq, represents the average acoustic energy across a defined measurement epoch.

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sound pressure level, which is expressed in decibels (notated dBA). The most commonly used noise compliance assessment methods for windfarms involve the "time-average" sound level L_{Aeq} or the background sound level L_{A90}. These levels are quite different as the time-averaged ambient sound level includes all noises from near and far. The difference between these levels, and other common levels, is illustrated in Fig. 2. The chart shows that sound levels change over time and that any derived sound level index is a summary of fluctuating levels in that time period. In a relatively short time period, such as 10 minutes, the unique noise events such as bangs or thuds from turbines shifting in the wind may be captured. If the time period is relatively long, for example, an hour, then evidence of unique short-term noise events is reduced because the sound energy is "averaged" over the whole hour, and the single-value A-weighted level will not represent short-term variations in sound character. If extraneous noise (e.g., insect noise) is included in the wind turbine measurement, its contribution to the overall level must be determined, though how this is undertaken remains a challenge.[3]

The A-frequency–weighted sound pressure level or "sound level" is the most common sound descriptor and is reputedly analogous to our hearing at medium sound levels. This is not strictly true, and the A-weighting has a significant restriction in that it does not permit measurement or assessment of low-frequency sound (i.e., 20 to 250 Hz). For more complex situations where dominant tonal components are significant (i.e., special audible characteristics), a procedure for determining tonal adjustment requiring one-third octave band frequency or narrow-band analysis is needed. These assessment procedures require the "C" weighting for low frequency or the unweighted (also known as "Z") response to measure both low-frequency and infrasonic sound. Whereas the dBC metric is able to include low-frequency sounds such as the audible rumble and thump from wind turbines, the dBZ response is more suitable for infrasound measurements (i.e., typically inaudible energy below 20 Hz). Fig. 3 presents a third octave band analysis of outdoor wind turbine noise recorded over a 6-hr period. Other measures include assessments for tonality or low-frequency sound referenced to third octave bands and the "G" weighting for infrasound. Aside from physical measures of amplitude (e.g., dBA), wind turbine noise can be quantified with a variety of other acoustical and objective psychoacoustic measures, including amplitude modulation (for example, 100 msec samples of peak, time-average, or fast response), sound quality (including audibility, dissonance, roughness, fluctuation strength, sharpness, tonality), loudness (for steady, time-varying, and impulsive sounds), and unbiased annoyance.[4]

Certification of wind turbine noise is undertaken in accordance with the International Standard IEC 61400-11:2002.[5] Emission levels are to be reported as A-weighted time-averaged (L_{Aeq}) sound levels in one-third octave bands. Audibility is calculated by reference to tones. An informative chapter in IEC 61400-11 states the following: "In addition to those characteristics of wind turbine noise described in the main text, this emission may also possess some, or all of the following: infrasound; lowfrequency noise; impulsivity; low-frequency modulation of broad band or tonal noise; other, such as a whine, hiss, screech, or hum, etc., distinct pulses in the noise, such as bangs, clatters, clicks or thumps, etc." Unfortunately, many of these parameters are not reported by the turbine manufacturer and cannot be predicted with the simple

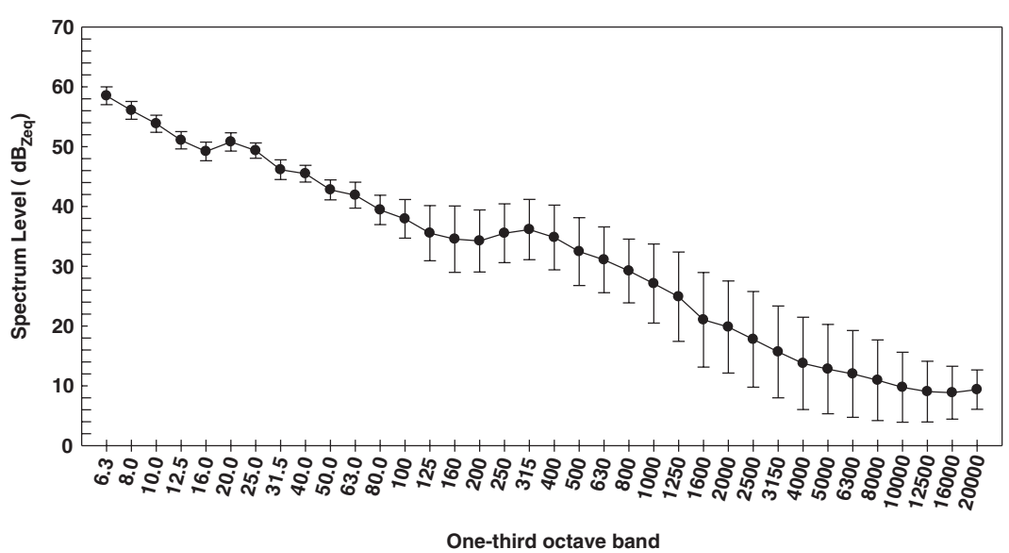


Fig. 3 One-third octave band analysis of time-average unweighted sound pressurelevel (dB_{Zeq}) for wind turbine sound measured from 7:00 PM to 1:00 AM outside of a residence.

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Table 1 Factors affecting the prediction of wind farm noise levels at a receiver.[a]

- The true sound power level of the turbine(s) at the specified wind speed
- The reduction in sound level due to ground effects
- The increase or reduction in sound level due to atmospheric (meteorological) variations and wind direction
- The variation due to modulation effects from wind velocity gradient
- Increase and reduction in sound levels due to wake and turbulence modulation effects due to turbine placement and wind direction
- Increased sound levels due to synchronicity effects of turbines in phase due to turbine placement and wind direction
- Building resonance effects for residents inside a dwelling

[a]A conservative set of noise predictions should take all factors into account.

calculation methods currently available. The prediction of windfarm sound levels is most often referenced to national or international standards that have been based on ISO 9613-2.[6] The propagation method is calculated with the receivers being downwind from the noise source(s). All prediction models have uncertainty to their accuracy of prediction. Table 5 of the ISO 9613-2 standard gives an estimated accuracy for broadband noise of ±3 dB at between 100 and 1000 m. This is due to the inherent nature of the calculation algorithms that go into the design of the model, the assumptions made in the implementation of the model, and the availability of good source sound power data. The ISO 9613-2 method holds for wind speeds of between approximately 1 and 5 m/s, measured at a height of 3 to 11 m above the ground. However, wind turbines are sound sources that operate at higher wind speeds than allowed for under the standard, and an accuracy of ±7 dB can be expected.[3] Ultimately, the received noise levels at residences will vary subject to varying meteorological conditions in the locality (e.g., wind speed and direction, wind shear, temperature, humidity, inversions), among other factors (see Table 1), all of which must be accounted for when measuring or modeling wind turbine noise levels.

THE HUMAN IMPACTS OF WIND TURBINE NOISE

A Psychological Description of Wind Turbine Noise

At the psychological level of description, wind turbine noise is most frequently characterized as a swishing or lashing sound or less commonly as thump/throb, low-frequency rumble, or a rustling sound.[7,8] Wind turbines produce noise with an impulsive character[9] and while the actual cause of the swishing or thumping has not yet been fully elucidated, it has been demonstrated that the swishing or thumping pattern is common with larger turbines[10] and may result from a fluctuating angle of attack between the trailing edge of the rotor blade and wind, or wind speed inequalities across the area being swept by the rotor blades.[11] It is thought that the swishing sound may be linked to activity in the 2000 to 4000 Hz band, with the pace of the rotor blades determining the degree of amplitude modulation.[12] Unfortunately, such amplitude-modulated sounds are generally attenuated poorly by background noise, especially so in rural areas.[13] Further, because human sensory systems behave as contrast analyzers, fluctuations in the incoming stimulus field tend to direct attention and so are more easily detected. Thus, amplitude-modulated sounds such as wind turbine noise are readily perceived and difficult to filter out, making them especially intrusive.[14] The loudness of a wind turbine depends on a number of factors, including wind speed, sound-attenuating materials between the turbines and the receiver, other masking sounds, the season, and time of day. The loudness of a modern 2 to 3 MW wind turbine can be compared to a car on a motorway, autobahn, or freeway,[15] with a sound power level of 94 to 104 dBA at a windspeed of 8 m/s.[16] Wind turbine noise is perceived louder at night and during the summer months and when the wind is blowing from the direction of the turbines toward the receiver.[7,8]

Quantifying the Health Impacts of Wind Turbine Noise

Elucidating a causal mechanism between an environmental event and health is a complicated undertaking, and noise effects are commonly "indirect" as opposed to "direct." According to the biomedical model of health (Fig. 4a), a direct health effect implies a direct pathological relationship between an environmental parameter (e.g., noise level) and a target organ. An alternative approach (Fig. 4b) distinguishes between direct health effects and psychosomatic illness, the latter indicting that any physiological illness coinciding with the onset of wind turbine noise is caused by a negative psychological response to the noise and not the noise *per se*. Thus, anxiety or anger in the presence of wind turbine noise induces stress and strain that, if maintained, can eventually lead to adverse health effects. A counter argument to this approach is that some individuals are simply more susceptible to noise than other individuals, which fits with the general concept of biological and physical variation. In the field of epidemiology, the differential susceptibilities of individuals are known as risk factors or vulnerabilities, with noise sensitivity being one risk factor related to negative responses to intrusive noise. A second challenge to the psychosomatic approach comes from documented instances of individuals who initially welcomed wind turbines into the community but who later campaigned to have them removed due to undesirable noise exposure.[17] Lastly, the veracity of psy-

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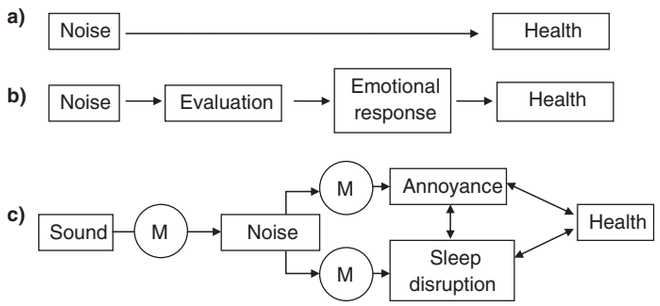


Fig. 4 Three models representing the relationship between noise and health: the biomedical model (a) stipulating a direct causal relationship and indirect models (b and c) containing moderators and mediators.

chosomatic arguments lessens in the face of feasible biological mechanisms describing the relationship between health and noise.[18]

An alternative and more accepted approach would be to adopt the World Health Organization's (WHO's) definition of health:[19] "A state of complete physical, mental and social well-being and not merely the absence of disease or infirmity." The forerunner of the biopsychosocial model, the WHO's definition states that optimal human functioning is determined by the interplay of biological, environmental, psychological, and social factors. Fig. 4c displays a model consistent with the WHO's approach, in which the impact of noise is moderated by environmental, psychological, and social factors. A context-relevant model proposed by van den Berg and colleagues,[8] based on previous wind turbine literature, takes a similar shape to that presented in Fig. 4c. They dichotomize moderators (denoted "M" in Fig. 4c) into environmental moderators (e.g., degree of urbanization, house type, and ambient sound level) or psychological and demographic moderators (e.g., age, gender, education, employment status, attitudes to wind energy, noise sensitivity, and whether the individual receives a monetary return from the turbines). Other models linking wind turbine sound and health have been proposed[20] but can be considered extensions of that presented in Fig. 4c.

As a new source of noise, the impact of wind turbine noise is understandably understudied relative to aviation and road traffic noise. Consequently, little data exist with which to assess the impacts of wind turbine noise on health, a state of affairs compounded by rapid development of wind turbine technology, in which data collected for smaller and less powerful turbines are not generalizable to larger, more modern turbines.[9,21] To date, there have been two approaches to collecting wind turbine noise impact data, either epidemiological studies relying on masked surveys or direct clinical case studies.[22] Both approaches typically focus on the emotional impacts of noise (i.e., annoyance), upon sleep disruption, and/or the degradation of well-being and increases in stress that arise from sleep disturbance and annoyance. Irrespective of approach, however, case studies,[23–25] and epidemiological studies[7,8,20] have provided evidence that, like road traffic and aviation noise, wind turbine noise can be associated with negative health outcomes.

Wind Turbine Noise and Annoyance

People generally respond more negatively to man-made noise than to natural sounds,[26] and this generalization holds true for wind turbine noise.[16] From a psychological perspective, chronic exposure to community noise can impact health through information overload, overarousal, loss of coping strategies, loss of privacy, and loss of perceived control. These mechanisms give rise to a number of subjective responses to noise, of which the most common is annoyance. As a psychological stressor,[27] noise annoyance can express itself through malaise, fear, threat, uncertainty, restricted liberty, excitability, or defenselessness.[28] Furthermore, annoyance may be accompanied by intense anger, especially if one believes that they are being harmed unnecessarily. Thus, the term "annoyance" is often misinterpreted by the layperson as a feeling brought about by the presence of a minor irritant. The medical usage, in contrast, exists as a precise technical term and defines annoyance as a mental state capable of degrading health and well-being,[29,30] and it is classified as an adverse health effect by the WHO.[31]

There have been few studies estimating the health impacts of windfarms, with a series of studies undertaken in Scandinavia contributing the most to current knowledge. A seminal Swedish study undertaken by Pedersen and Persson Waye[7] sought to document the prevalence of wind turbine–induced annoyance and, further, to generate dose–response relationships between the two. Respondents were located between 150 and 1200 metres from the nearest wind turbine and were classified into noise exposure categories (see Fig. 5). A significant relationship between dose (dBA) and annoyance was reported, but the variability in annoyance scores explained by noise level was small (adjusted $R^2 = 0.13$). Those reporting annoyance indicated a daily or nearly-everyday intrusion of windfarm noise. Those describing the noise as "swishing" were more

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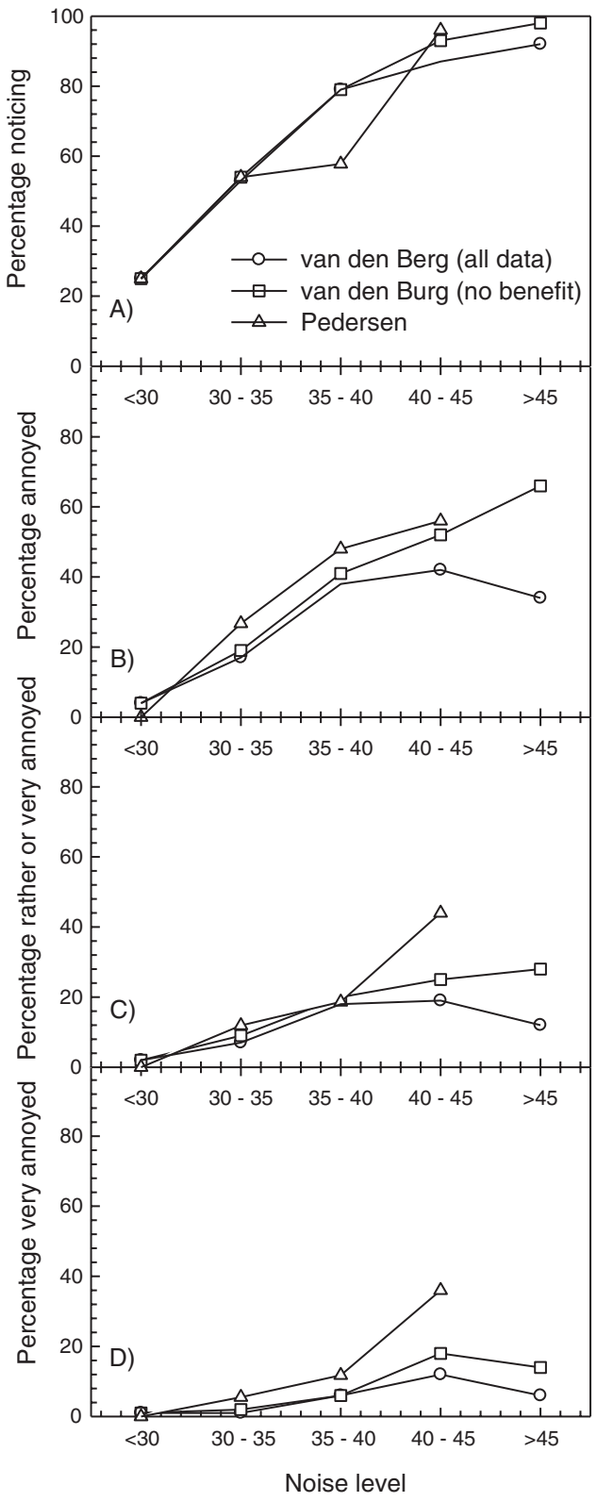


Fig. 5 Perception of wind turbine noise as a function of noise level for three sets of data:Tables 7.25 (complete data set) and 7.26 (no economic benefit of turbines) from van den Berg et al.[8] and Pedersen and Persson Waye's[7] Table V. Plot A is percentage noticing the noise, while plots B to D are for annoyance. Plot B includes data from plots C and D, and plot C includes data from plot D.

likely to report noise annoyance, a finding replicated in a subsequent study reporting a high correlation ($r = 0.664$) between the swishing sound and annoyance.[14] Among those who noticed the noise, 11.2% reported being annoyed when indoors. A small but significant correlation was found between noise annoyance and noise sensitivity, with approximately 50% of the rural-dwelling respondents describing themselves as noise sensitive. Those making negative appraisals of the wind turbines, for example, as visually incongruent with the landscape, were at higher risk of an annoyance response. On the basis of their data, the authors undertook follow-up studies[14–16,22] supporting their conclusion that wind turbine noise maybe more potent than other categories of environmental noise (e.g., road or aviation) and appealed for further studies to determine why this might be. In a later report, Pedersen[22] suggests that coping strategy may moderate the relationship between wind turbine noise and stress.

Van den Berg et al.[8]analyzed data from 725 Dutch nationals residing within 2.1 km of a wind turbine and who were exposed to calculated outdoor noise levels between 24 and 54 dB(A). Approximately 60% of the sample could hear the turbines outdoors, while 33% reported that they could hear the wind turbines indoors. Of the 45% ($n = 231$) who noticed the sound of the rotor blades, 24.7% were not annoyed, 25.8% were slightly annoyed, 19.5% were rather annoyed, and 29.9% were very annoyed. The sound level explained approximately 25% of the variability in annoyance scores, and those who compared the noise to an amplitude modulation (i.e., swishing or lashing) were more likely to be annoyed, though this is not a novel finding.[14,32,33] Fig. 5 plots the data from van den Berg et al., presenting proportions of detection and elicited annoyance as a function of noise level, for their entire dataset (Fig. 5, circles) and for those receiving no economic benefit (Fig. 5, squares). Note that, for those receiving no economic benefit, a monotonic relationship is evident, while a non-monotonic function occurs when individuals benefiting financially from the turbines are included. Van den Berg[8] reports that this depreciation in annoyance of those benefiting economically can be explained by the control they have over the wind turbines, such that they can impede their operation if noise levels increase. Finally, it was reported that annoyance was positively correlated with stress scores, though a causal relationship could not be inferred.

It is accepted that both the physical parameters of the noise and the psychological characteristics of the listener combine to produce noise annoyance.[34] On the physical side, the relatively high annoyance levels elicited by wind turbine noises (e.g., swishing or thumping) may be explained by the increased fluctuation of the sound, up to 4 to 6 dB for a single turbine operating in a stable atmosphere.[11] Individuals are also highly sensitive to changes in frequency modulation variations of approximately 4 Hz or greater.[4] Noting that amplitude-modulated sound is known to be more annoying than unmodulated sound, Lee et al.,[34] in a laboratory setting, demonstrated that

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amplitude-modulated wind turbine noise was consistently judged to be more annoying than its unmodulated counterpart. Thus, the dominant acoustic driver of annoyance is likely to be noise dynamics rather than noise level. Other physical parameters linked to annoyance include terrain complexity, with rural terrain associated with greater annoyance than urban areas, possibility due to more complicated terrain exhibiting various focusing or defocusing effects and greater ground reflection.

While there is a strong correlation between the sound pressure level (i.e., amplitude) of a sound wave and the perceived loudness of a sound, there is no one-to-one mapping between sound pressure level and the psychological responses that individuals have to a sound.[35] Many non-acoustical factors determine how annoyed one will become toward a source of noise.[36–38] Thus, the response of the individual to the sound is just as important as the parameters of the acoustic wave, and the "people" side of noise should not be omitted from acoustical reports. Table 2 lists, in no particular order, non-acoustical factors found to influence levels of noise annoyance.[39] In relation to windfarms, the personal factors listed in Table 2 have been found to strongly influence how exposed individuals perceive the noise.[16] In addition, perceptions of amenity, individuals seeking refuge from urban noise, or the lower ambient sound levels typical of the rural environment may explain why annoyance responses are higher in rural as opposed to urban settings.[13,16]

When considering wind turbine noise and annoyance data emerging from the literature, a number of risk factors are evident, including an effect of age and educational status but not gender.[8] Employment status was also linked to wind turbine noise–induced annoyance in one study, possibly due to impeded restoration,[16] but to date, there are no data meaningfully comparing ethnicity or national groups (but see Pedersen et al.[40]). The general public view wind turbines as necessary but ugly,[14] and it is possible that the visual impact of a windfarm can interact with noise level to cause moderate annoyance. This amplification of annoyance is possibly due to a violation of the landscape–soundscape continuum constructed by those who choose to live in areas that later contain windfarms,[41] or alternatively, multisensory engagement may enhance detection and identification of wind turbine noise.[42] The degree of influence of the visual aspects of windfarms has yet to be determined, with laboratory studies suggesting that it is wind turbine noise and not the visual impact that underlies the annoyance response,[41] while epidemiological studies suggest that the visual effects are nontrivial. [40]

Table 2 Non-acoustical factors influencing the degree of annoyance to noise.

- Perceived predictability of the noise level changing
- Perceived control, either by the individual or others
- Trust and recognition of those managing the noise source
- Voice, the extent to which concerns are listened to
- General attitudes, fear of accidents, and awareness of benefits
- Personal benefits, how one benefits from the noise source
- Compensation, how one is compensated due to noise exposure
- Noise sensitivity
- Home ownership, concern about plummeting house values
- Accessibility to information relating to the noise source

Source: From Flin dell and Stallen.[39]

Wind Turbine Noise and Sleep

The deleterious effects of noise on sleep and the consequences of sleep loss are well documented and are a major concern for governments.[43] In comparison with road, rail, and aircraft noise, there is little research on the effects of wind turbine noise on sleep. However, there is no doubt that wind turbine noise can and does disturb the sleep of those living nearby. Sleep disruption is the predominant symptom in the thousands of anecdotal cases reported in the press and on the Internet and is confirmed by more structured surveys.[25] The quantity, consistency, and ubiquity of complaints has been taken as *prima facie* epidemiological evidence of a causal link between wind turbine noise, sleep disruption, and ill health.[44]

Early investigations into wind turbine noise and sleep are difficult to interpret as researchers used imprecise outcome measures, generally relying on recalled sleep disturbances such as difficulty in initiating or returning to sleep, which tends to underestimate the magnitude of the noise impact and its consequences.[45] One of the earliest studies (n = 128) reported that approximately 16% of respondents living at calculated outdoor turbine noise exposures exceeding 35 dB L_{Aeq} stated that wind turbine noise disturbed their sleep.[7] A New Zealand study of 604 households within 3.5 km of a windfarm found that 42 reported occasional and 26 frequent sleep disturbance.[46] The largest wind turbine noise study to date, "Project WINDFARMperception,"[8] concluded that turbine noise was more of an annoyance at night and that interrupted sleep and difficulty in returning to sleep increased with both indoor and outdoor calculated noise levels. Even at the lowest noise levels, 20% of 725 respondents reported disturbed sleep at least one night per month. In a meta-analysis[40,47] of three European datasets (n = 1764),[7,8,16] there was a clear increase in levels of sleep disturbance with dB L_{Aeq} in two of the three studies. In one study, an increment in self-report sleep disturbance occurred between 35 and 40 dBA, while in the other, it occurred between 40 and 45 dBA.

More recent research into wind turbine noise and sleep includes two studies reported by Nissenbaum, Aramini, and Hanning.[48] In the first, a pilot study, a structured questionnaire was administered to 22 subjects living 370 to 1100 m from twenty-eight 1.5mW turbines and a control group (n = 28) living at least 4.5 km from the nearest turbine. The study group had clinically and statistically

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worse sleep disturbance, headache, vestibular symptoms, and psychiatric symptomatology. The second study, using validated questionnaires, administered the Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Score (ESS), and Short-form health survey (SF36) to 79 subjects living between 375 and 6600 m from two windfarms. Those living within 375–1400 m reported worse sleep, were sleepier, and had worse SF36 mental summary scores than those between 3 and 6.6 km from a turbine. Psychiatric symptom scores (irritability, stress, anger, hopelessness, and anxiety) were significantly greater, as was a composite mental health score. They were also more likely to report headaches, nausea (31.6% vs. 12.2%), and a willingness to move away. Modeled dose–response curves of both sleep and health scores against distance from nearest turbine (Fig. 6a–c) were significantly related after controlling for gender, age, and household clustering. There was a sharp increase in effects between 1 and 2 km. This study is the first to use appropriate sleep outcome measures[45] and to use a control group. While the sample size is modest ($n = 78$), it is convincing evidence that wind turbine noise adversely affects sleep and health for those living within 1.5 km of turbines.

Mechanisms explaining the effects of wind turbine noise on sleep have been considered, but would benefit from further empirical support.[45] Noise of any description can interfere with sleep by preventing the onset of sleep either at sleep initiation or at the return to sleep after a spontaneous or induced awakening. The amplitude, character, and associations of the noise are all important as is the noise sensitivity of the individual and the psychological response to the noise. In this respect, wind turbine noise seems to be particularly annoying, possessing an impulsive nature with short bursts of low-frequency sound, making it audible 10–15 dBA below background level.[38,49] Nocturnal atmospheric stability ensures that wind turbine noise is maintained while ground level ambient noise diminishes. Indoor noise levels for most noise sources can be reduced by closing windows; however, the low-frequency content of wind turbine noise means that it may be more audible indoors than outdoors. Additionally, during warmer months, windows are more likely to stay open to control thermal parameters, whence the inability to control or modify wind turbine noise will contribute to the annoyance and, presumably, the effect on sleep onset.[16]

Noise may also cause awakenings and arousals. Arousal is a brief lightening of sleep that is not recalled. Sleep becomes fragmented and, if enough arousals occur, induces the same consequences as reduction of total sleep time. Awakenings are arousals of sufficient degree for wakefulness to be reached and long enough (greater than 10 sec) to be recalled. Arousals are more likely than awakenings,

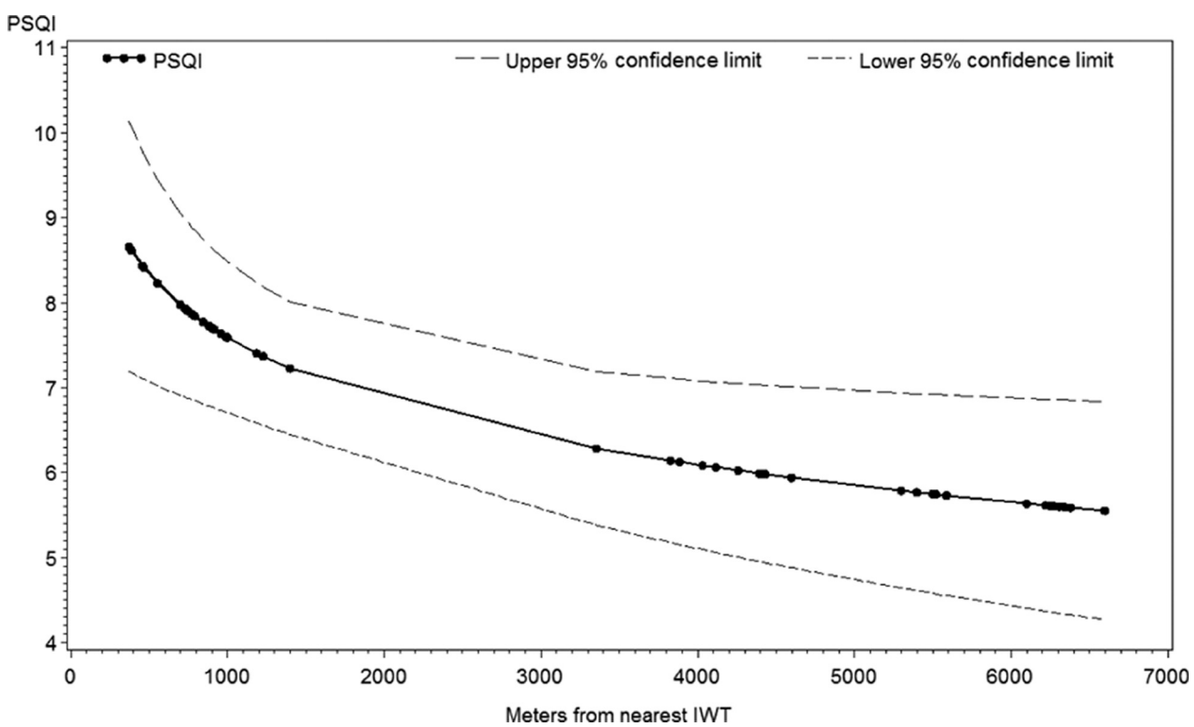


Fig. 6a Mean Pittsburgh Sleep Quality Index (PSQI) scores as a function of setback distance. The dashed lines are 95% confidence intervals.
Source: From Nissenbaum, Aramini, and Hanning.[48]

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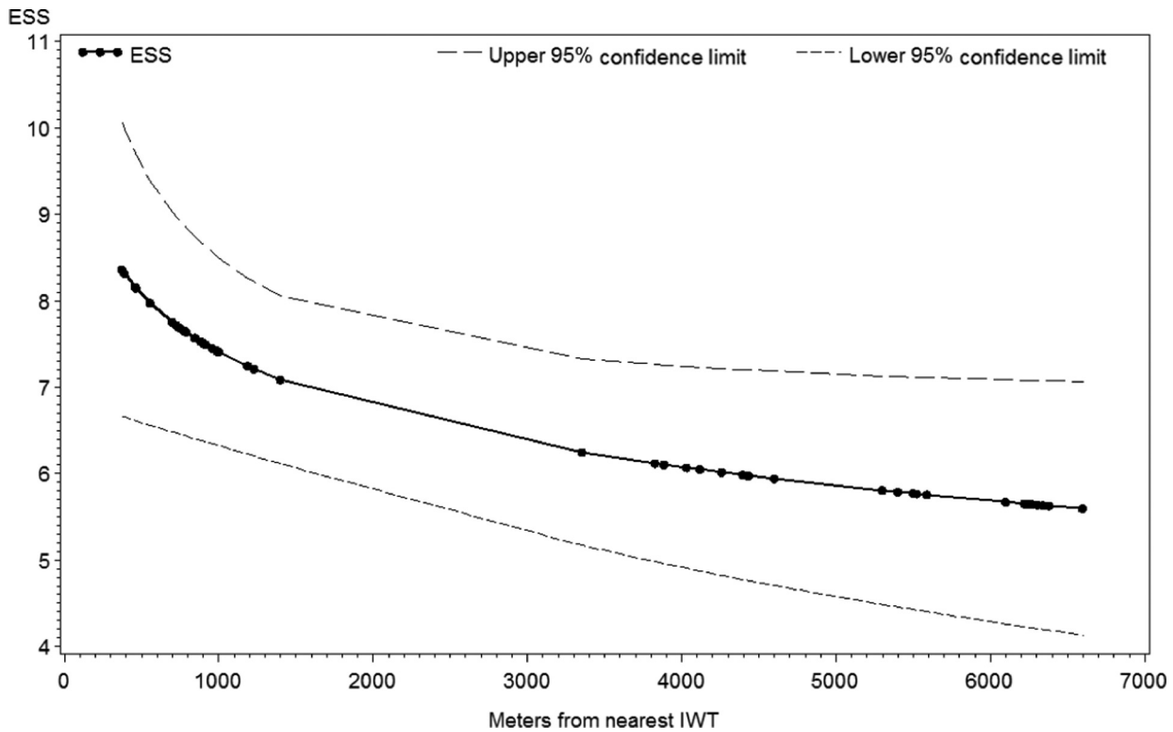


Fig. 6b Mean Epworth Sleepiness Scale (ESS) scores as a function of setback distance. The dashed lines are 95% confidence intervals.
Source: From Nissenbaum, Aramini, and Hanning.[48]

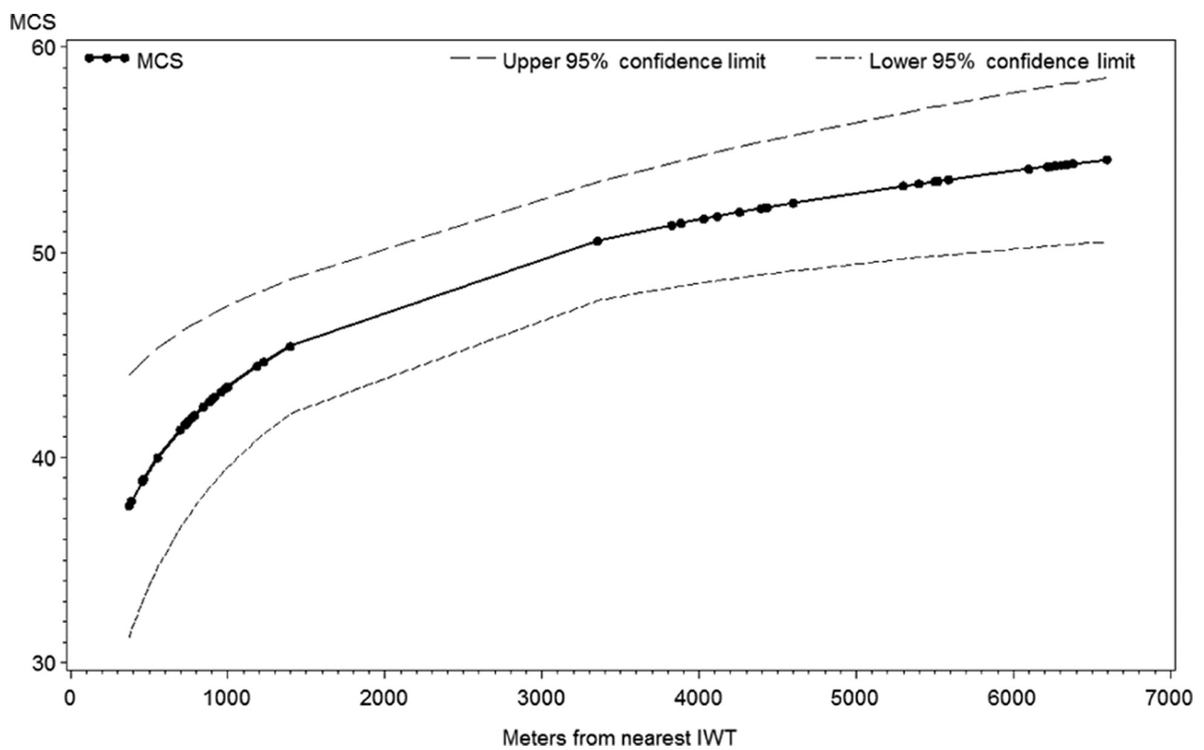


Fig. 6c Mean SF36 mental component score (MCS) as a function of setback distance. The dashed lines are 95% confidence intervals.
Source: From Nissenbaum, Aramini, and Hanning.[48]

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and thus, relying on reported awakenings underestimates the magnitude of the noise effects. The likelihood of an arousal depends upon the volume, character, and duration of the noise as well as the sleep stage and individual propensity (i.e., noise sensitivity). In an investigation into hospital noise, dose–response curves were created for different noises in different sleep stages.[50] Noises with characteristics designed to alert (e.g., telephone, alarms) were more likely to arouse. These noises tend to be impulsive in character, as does wind turbine noise. Noises that were classified as continuous broadband noises (e.g., traffic noise) were less likely to arouse. Another study[51] has shown that subjects with fewer sleep spindles (electrophysiological markers characteristic of stage II sleep) are more easily aroused by noise (Fig. 7). Sleep spindles are taken as a marker of sleep stability and may provide a physiological marker of sleep quality.

To date, there are no electrophysiological studies of wind turbine noise on sleep. However, it is reasonable to expect that, in common with road, rail, and aircraft noise, it will induce arousals, fragmenting sleep, as well as preventing the onset of and return to sleep. The sleep measures used in the study by Nissenbaum, Aramini, and Hanning[48] (i.e., ESS and PSQI) are average scores, determining sleepiness and sleep quality, respectively, over a period of weeks. Thus, occasional sleep disturbance would not alter scores as the sleep loss would have been compensated quickly over one or two nights. The study results imply strongly that sleep was being disturbed to some degree on sufficient nights to prevent compensation occurring, thus leading to persistent daytime symptoms.

Wind Turbine Syndrome

Wind turbine syndrome refers to a cluster of symptoms, which Pierpont,[24] who coined the phrase, claims are associated with exposure to wind turbine noise. Using direct clinical case studies, Pierpont describes the following symptoms to be characteristic of many individuals residing in close vicinity of wind turbines: insomnia, headaches, dizziness, unsteadiness, nausea, exhaustion, anxiety, anger, irritability, depression, memory loss, eye problems, problems with concentration and learning, and tinnitus. Pierpont hypothesizes that wind turbines may affect the vestibular system, that part of the inner ear that plays an important role in the maintenance of balance and stable visual perception. Wind turbines may compromise this system in two ways: first, by the visual disturbance of the moving blades and shadows (i.e., the flicker), and second, by direct vibration of the vestibular system. Such a model would explain why some residents in the close proximity of wind turbines (i.e., less than a kilometer) complain of vertigo, dizziness with nausea, and migraines. Wind turbine syndrome awaits further validation from the medical and scientific establishments, specifically the confirmation of a cause-and-effect relationship between wind turbine noise and vestibular function.

Wind Turbine Noise and Low-Frequency/ Infrasound Components

Recent enquiry has focused on the impacts of low-frequency (20–200 Hz) and infrasonic frequencies (typically taken as

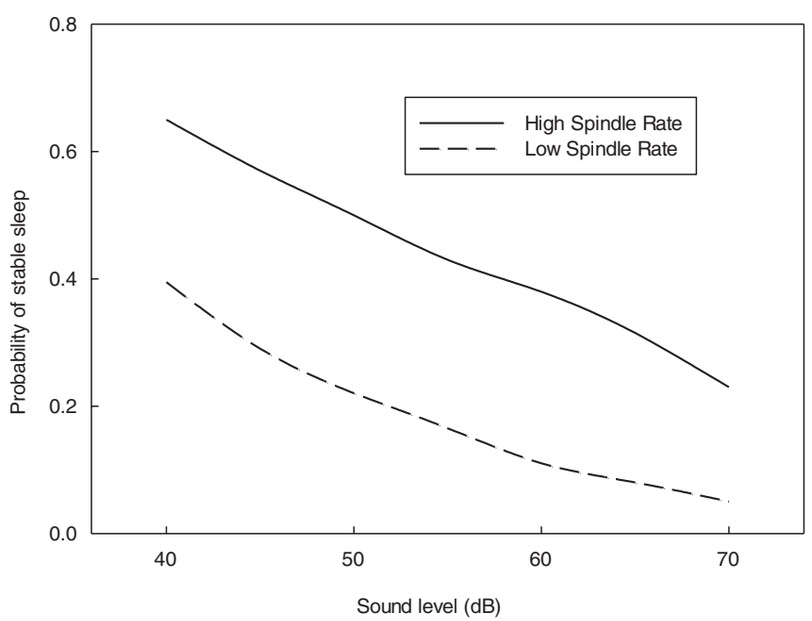


Fig. 7 Sleep stability as a function of sound level for noise-resistant (high-spindle) and noise-sensitive (low-spindle) groupings.
Source: Estimated from Dang-Vu et al.[51]

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below 20 Hz) being emitted by wind turbines. Infrasound is characterized by fluctuating pressure sensations at the eardrum, is atonal and countable, and is of a level proportional to wind speed.[21] Low-frequency acoustic waves emitted by wind turbines may be amplified by ground reflection and originate from varying lift forces as the rotors travel through spaces differing in wind speed and density.[21] Compared with medium (i.e., 250 to 4000 Hz) and high frequencies (above 4000 Hz), low-frequency energy decays slowly with distance, is less attenuated by conventionally designed structures, causes certain building materials to vibrate, and can sometimes resonate within rooms and undergo amplification. The effect of air absorption must also be taken into account, in which higher frequencies are attenuated at a greater rate as a function of distance, resulting in a shifting of the spectrum toward lower frequencies. The relationship between low-frequency wind turbine noise and building type creates an interesting proposition in which the low-frequency sound may be louder inside a dwelling than out,[21,52] and the assumption that walls and windows attenuate sound by 15 dB may not be applicable to frequencies below 200 Hz.

Research has shown that low-frequency noise increases cortisol levels in those who are sensitive to noise[12] and disturbs rest and sleep at levels below noise otherwise free from lower-frequency components.[31] Low-frequency noise and infrasound are known disturbers of sleep; however, the contribution, if any, of the low-frequency noise emissions of wind turbines to the sleep disturbances they induce remains to be scientifically determined. Beyond infrasound, the phenomenon of vibroacoustic disease is worthy of note. Humans chronically exposed to infrasound may exhibit elevated cortisol levels and generalized cell damage: a condition known as vibroacoustic disease.[53] A number of human and animal models explaining how infrasound can lead to cardiovascular and respiratory disease have been proposed[54] and applied to wind turbine noise.[55] The phenomenon of vibroacoustic disease is supported by correlational evidence coupled with a thoroughly detailed mechanism. However, further research is required to establish the veracity of this approach to human health within and beyond the wind turbine context.

MITIGATION

There are multiple ways in which to reduce the impacts of audible and inaudible wind turbine noise. The first, and often the most effective, method is to control audible noise at the sound source. Thus, mechanical solutions invite technologies designed to attenuate wind turbine noise or to shift its spectral character in order to eliminate salient tonal characteristics. To safeguard health is more difficult, however, because wind turbine noise is largely aerodynamic in origin,[7] and it is not possible to obtain solutions that completely attenuate the noise at its source. Having minimized the noise through the implementation of technology, other approaches are often required, normally involving the application of noise standards to limit exposure levels or the determination of "safe" setback distances to mitigate noise impact. Still other approaches involve the positioning of wind turbines around preexisting noise generators,[15] in remote areas away from human habitations, or using social processes to determine wind turbine location.[27,56]

Regulating Permissible Noise Level

Permissible or safe exposure levels are often set in national noise standards, which may or may not be specific to wind turbine noise. These standards may serve one of two purposes, or sometimes both, with noise compliance guidelines naturally emerging from the two. The first purpose relates to methodologies for the physical quantification of the noise. This may involve standardized procedures for measuring noise from preexisting windfarms or detailing accepted mathematical models affording noise predictions of a planned windfarm. The second purpose is to determine what exposure levels can be considered safe and to clearly state criteria to this effect. However, there are a number of flaws inherent in wind turbine noise standards, including the metrics used to represent the noise, oversimplified modeling approaches that yield unrealistically low predictions of noise levels representing "best case" conditions,[5] or stimulus-oriented approaches that fail to account for human factors.[3,57]

There exists, in respect to levels-based noise standards, disagreement as to the relevance of physical measures such as dBA to human response,[58] not only for windfarm noise (Pedersen, 2008b) but also for traffic and aviation noise. Of the few parametric studies that have been published,[7,8] only marginal dose–response relationships between wind turbine noise intensity and health measures have emerged. For example, Pedersen[22] noted that stress was not related to wind turbine noise level but rather noise annoyance. Persson Waye and Öhtrsöm[12] reported that annoyance ratings varied for five distinct recordings of wind turbine noise, even though all five had equivalent noise levels. Others note that both laboratory and field studies have consistently found that the equivalent dBA measure fails to account for the relationship between wind turbine noise and annoyance.[14]

To some degree, then, it must be accepted that there is an uncoupling between wind turbine noise level and human response. A hitherto rarely measured characteristic of wind turbine noise is amplitude modulation, whereby noise levels fluctuate periodically as a function of blade passing frequency. Lee et al.[34] recommend that standardized metrics based on the modulation depth spectrum be developed and used in conjunction with sound levels. Other approaches to measuring amplitude modulation have existed for some time[4,59] but have yet to be seriously applied to the wind turbine noise context. However, the inability to

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account for amplitude modulation arises primarily due to the time-averaged dBA levels applied by noise standards, and arguably, smaller sampling epochs of around 100 msec should be adopted as best practice in order to record the amplitude modulation inherent in turbine noise.[60,61] The New Zealand Standard[62] applies a penalty for amplitude modulation, but does not describe an objective assessment. Furthermore, using aggregated metrics that average noise level over long periods underestimates the effect of peak levels and crest factors, important when considering sleep disturbance.

For the most part, the acceptable noise limits recommended by noise standards are derived from WHO guidelines.[31,63,64] However, as Fig. 8 demonstrates, using recommended noise levels from guidelines based on transport data risks exposing the population to unacceptable levels of noise. It follows that the Ldn (the "day–night" level in the United States) or Lden (the "day–evening–night" level in Europe) measures, derived from the measured L_{Aeq} sound level can be used in a wind farm context, but with caution.[65] Inspection of Fig. 8 suggests that, relative to transport guidelines, at least a 10 dBA penalty should be placed on wind turbine noise. The differences in annoyance ratings between wind turbine noise and transport noise maybe accounted for by amplitude modulation, the typical location of windfarms (e.g., rural areas), or the over-representation of noise-sensitive individuals. A recent meta-analysis of three epidemiological studies revealed a consistent trend in wind turbine noise exposure and both annoyance and sleep disruption.[22] On the basis of her analysis, Pederson recommends that outdoor levels should not exceed 40 dBA, though this level could be more-or-less depending on situational factors, that is, ambient noise levels or the building's construction materials. When noise is continuous, the WHO[31] stipulates an indoor limit of 30 dBA, though for noises containing lower frequencies (e.g., wind turbine noise), a lower limit still is recommended. Thus, careful examination of the lower end of the frequency spectrum is important when judging appropriate exposure to wind turbine noise, and the use of dBC or spectral analysis in one-third octave bands or narrow bands is necessary.

In the comparison of global wind turbine noise level standards, there exist two chief methodologies, namely, sound levels not to be exceeded (usually in dBA) or a not-to-be-exceeded limit derived from the sum of the preconstruction ambient limit and a constant (e.g., L_{A90}+10 dBA). Critique of both these approaches can be found in Thorne.[3] The fact that noise limits differ between, and even within, a country is testament to the impoverished research database guiding their development or the political sensitivities around wind turbine placement. Examples of noise limits are presented in Table 3, and the variability in guidelines is evident. Based on the authors' collective experience, an interim guideline, providing a conservative noise limit capable of protecting the health of the public and suscep-

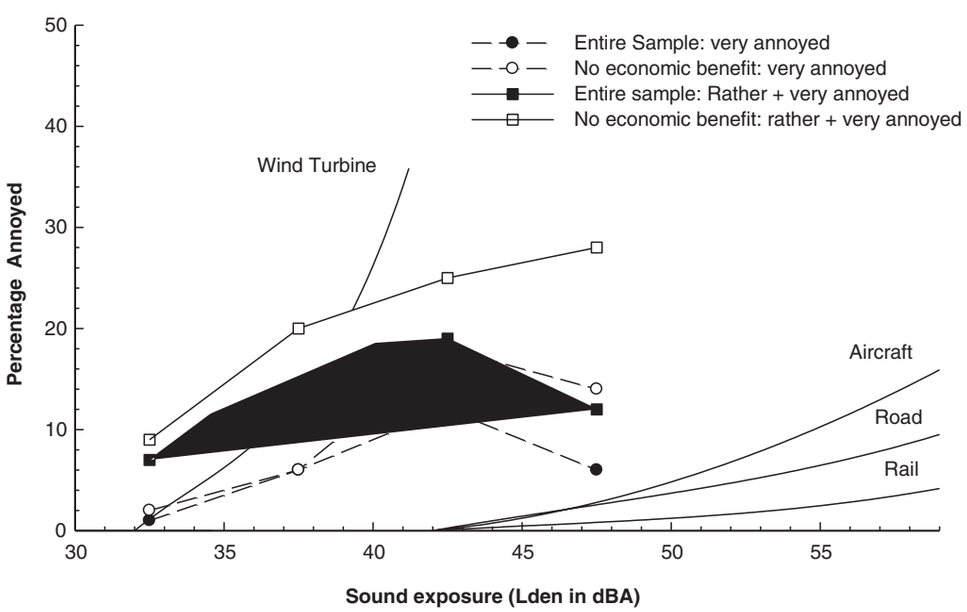


Fig. 8 Annoyance plotted as a function of noise level for four theoretical models (rail, road, and air parameters: Miedema and Oudshoorm;[66] wind turbine parameters: Pedersen et al.;[7] and four sets of data obtained from Tables 7.24–7.26 of van den Berg et al.[8]). For the data, closed symbols are for the entire sample, while open symbols are for those who identified that they had no economic interest. Circles represent the percentage of "very annoyed" responses, while squares represent the sum of "very annoyed" and "rather annoyed" responses.

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Table 3 A comparison of wind turbine noise guidelines taken from nine countries.

Country	State	Limit (dBA)	Background plus constant
Australia	Victoria	L_{A90} 35 or 40	L_{A90} + 5 dBA
	South Australia	L_{Aeq} 35 or 40	L_{A90} + 5 dBA
Australia	Queensland	L_{Aeq} 30 indoors	Health and well-being criteria
Canada	Ontario	L_{Aeq} 40 to 51	
Denmark		40	
France			Day: L_{A90} + 5 dBA Night: L_{A90} + 3 dBA
Netherlands		40	
New Zealand		L_{A90} 35, 40	L_{A90} + 5 dBA
United Kingdom		Day: 40 Night: 43	L_{A90} + 5 dBA
United States	Illinois	Day: 50 Night: 46	
	Michigan	55	
	Oregon	35	

tible individuals, would be a sound level of L_{Aeq} 35 dBA outside the residence and below the individual's threshold of hearing inside a residence. More specific guidelines are presented in Appendix A of this document.

Regulating Setback Distances

A setback distance is defined as the minimum distance between a dwelling and the closest wind turbine required to protect the health of the inhabitants. One difficulty is whether such setback distances can be standardized, as they will differ depending on a number of factors, including turbine type, terrain, and climate. Lee et al.[34] report that the perception of amplitude-modulated noise decreases with distances beyond a kilometer, though others claim that amplitude-modulated turbine noise can be heard up to 4 km away from the source.[67] Setback distances maybe based on noise level, which, as discussed in the preceding section, maybe an invalid approach. Instead, a better approach may be to link setbacks to turbine type. Møller and Pedersen,[21] investigating the detection and annoyance of lower-frequency sound emitted from wind turbines, suggest that, for flat terrain, the minimum setback distance for modern turbines (2 to 3.6 MW) should be between 600 and 1200 metres. Other approaches rely on the establishment of dose–response curves relating a health outcome variable (e.g., annoyance or disturbed sleep) and distance (e.g., Fig. 6). Medical professionals have proposed setback distances of 2.4 km[23,24] or 1.5 km.[45] Other research recommends a minimum of 2 km if wind turbines are sited in rough terrain.[3,20]

CONCLUSION

Windfarms have significant potential for sleep disruption and annoyance due to the intermittent nature and amplitude modulation of their sound emissions, even though exposure may be of low amplitude. The interactions between ambient levels, amplitude modulation, and the tonal character of windfarm noise overlaid within a soundscape are complex and difficult to measure and assess in terms of health and individual amenity. Additionally, currently employed sound level measurement and prediction approaches for complex noise sources of this nature are only partially relevant to environmental risk assessment. Aside from acoustic parameters, other factors such as noise sensitivity or amenity expectations may also predict the human response to wind turbine noise. Unfortunately then, for policymakers, there appears to be no proportional relationship between wind turbine noise levels and health, as these outcome factors will be influenced by characteristics associated with both the noise and the listener.[39]

As a relatively new source of intrusive noise, there is little research to draw upon when judging if a proposed windfarm constitutes a health threat to the exposed public. A liberal approach to assessing health impact will involve the application of previous knowledge obtained from other noise sources (e.g., road, aviation). A conservative approach, consistent with the precautionary principle, will consider wind turbine noise more potent than these other harmful noise sources. Thus, at this time, a constellation of acoustic and social metrics should be taken at preexisting wind farms in order to assess potential threat. Peak and crest noise levels, level metrics assessing low-frequency contributions (e.g., dBC), and amplitude modulation indices constitute the acoustic measures of importance. It should also be remembered that predicted levels derived from computer models represent estimates and not precise values, are constrained by numerous assumptions, contain substantial uncertainty, and as such should not constitute the sole criteria for wind turbine positioning. What form the social measures will take is yet to be elucidated, but research suggests that noise sensitivity[67] and procedural fairness[27] are the best approaches to minimize the health impacts and facilitate social acceptance of windfarms.

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APPENDIX A

'Proposed Wind Turbine Siting Sound Limits', a revision by Thorne, R, of the Kamperman James criteria (2008) to include updates to ISO 1996-2 and UK Court of Appeal (Hulme re: Den Brook).

1. Audible Sound Limit
   - **a.** No wind turbine or group of turbines shall be located so as to cause an exceedance of the pre-construction/operation background sound levels by more than 5 dBA. The background sound levels shall be the L_{A90} sound descriptor measured during a pre-construction noise study during the quietest time of evening or night. All data recording shall be a series of contiguous ten (10) minute measurements. L_{A90} results are valid when L_{A10} results are no more than 15 dBA above L_{A90} for the same time period. Noise sensitive sites are to be selected based on wind development's predicted worst-case sound emissions in L_{Aeq} and L_{Ceq} which are to be provided by the developer.
   - **b.** Test sites are to be located along the property line(s) of the receiving non-participating property(s).
   - **c.** A 5 dB penalty is applied for tones as defined in IEC 61400-11 at the turbine and ISO1996-2 at any affected residence.
   - **d.** A 5 dB penalty is applied for amplitude modulation as defined following. When noise from the wind farm has perceptible or audible characteristics that are perceived by the complainant as being cause for complaint, or greater than expected, the measured sound level of the source shall have a 5 dB penalty added. Audible characteristics include tonal character measured as amplitude or frequency modulation (or both); and tonality (where the tonal character/tonality of noise is described as noise with perceptible and definite pitch or tone). Amplitude modulation is the modulation of the level of broadband noise emitted by a turbine at blade passing frequency. Amplitude modulation will be deemed greater than expected if the following characteristics apply:
     - **i)** A change in the measured L_{Aeq}, 125 ms turbine noise level of more than 3 dB (represented as a rise and fall in sound energy levels each of more than 3 dB) occurring within a 2 second period.
     - **ii)** The change identified in (i) above shall not occur less than 5 times in any one minute period provided the L_{Aeq}, 1 minute turbine sound energy for that minute is not below 28 dB.
     - **iii)** The changes identified in (i) and (ii) above shall not occur for fewer than 6 minutes in any hour.

Noise emissions are measured outside a complainant's dwelling and shall be measured not further than 35 metres from the relevant building, and not closer than within 3.5 metres of any reflective building or surface, or within 1.2 metres of the ground.

2. Low Frequency Sound Limit
   - **a.** The L_{Ceq} and L_{C90} sound levels from the wind turbine at the receiving property shall not exceed the lower of either:
     - **i)** L_{Ceq} -L_{A90} greater than 20 dB outside any occupied structure, or
     - **ii)** A maximum not-to-exceed sound level of 50 dBC measured as the background sound level (L_{C90}) from the wind turbines without other ambient sounds for properties located at one mile or more from State Highways or other major roads or measured as the background sound level (L_{C90}) for properties closer than one mile.
     - **iii)** These limits shall be assessed using the same night-time and wind/weather conditions required in 1(a). Turbine operating sound emissions (L_{Aeq} and L_{Ceq}) shall represent worst case sound emissions for stable night-time conditions with low winds at ground level and winds sufficient for full operating capacity at the hub.

3. General Clause
   - **a.** Sound levels from the activity of any wind turbine or combination of turbines shall not exceed L_{Aeq} 35 dB within 100 feet of any noise sensitive premises.
   - **b.** The monitoring shall include all the sound levels as required by these noise conditions and shall include monitoring for the characteristics described in Annex A of IEC 61400-11including infrasound, low-frequency noise, impulsivity, low-frequency modulation of broad-band or tonal noise, and other audible characteristics. Wind speed and wind direction shall be measured at the same location as the noise monitoring location.

4. Requirements
   - **a.** All instruments must meet ANSI or IEC Class 1 integrating sound level meter performance specifications.
   - **b.** Procedures must meet ANSI S12.9, IEC61400-11 and ISO1996-2
   - **c.** Procedures should meet ANSI, IEC and ISO standards applicable to the measurement of sound or its characteristics.
   - **d.** Measurements must be made when ground level winds are 2m/s (4.5 mph) or less. Wind shear in the evening and night often results in low ground level wind speed and nominal operating wind speeds at wind turbine hub heights.

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e. IEC 61400-11 procedures are not suitable for enforcement of these requirements except for the presence of tones near the turbine.

5. Definitions
   ANSI S12.9 Quantities and Procedures for Description and Measurement of Environmental Sound, Parts 1 to 6.
   IEC 61400-11 *Wind turbine generator systems—Part 11: Acoustic noise measurement techniques.*
   ISO 1996-2 *Acoustics—Description, measurement and assessment of environmental noise—Part 2: Determination of environmental noise levels*.
   L_{A90}, L_{A10} Statistical measures calculated under ANSI S12.9.
   L_{Aeq}, L_{Ceq} Time average levels calculated under ANSI S12.9 or ISO 1996-2.
   Noise sensitive premises includes a residence, hotel, hostel or residential accommodation premises of any type.

6. References
   ANSI S12.9-2008, Quantities and Procedures for Description and Measurement of Environmental Sound—Part 6: Methods for Estimation of Awakenings Associated with Outdoor Noise Events Heard in Homes, 2008.
   Hulme V Secretary of State (2011). Hulme v Secretary of State for Communities and Local Government and Anor. Approved Judgment in the England and Wales Court of Appeal (Civil Division) on Appeal from the High Court of Justice, Case No: C1/2010/2166/QBACF. Neutral Citation Number [2011] EWCA Civ 638; Available from: www.bailii.org/ew/cases/EWCA/Civ/2011/636.html.
   IEC 61400-11Wind Turbine Generator Systems—Part 11: Acoustic Noise Measurement Techniques, 2nd ed. (International Technical Commission, Geneva, 2002 plus Amendment 1 2006).
   International Standards Organization, (2007). ISO 1996-2 second edition, Acoustics—description, assessment and measurement of environmental noise-part 2: Determination of environmental noise levels. International Standards Organization.
   Kamperman, G. W.; James, R. R. In: Proceedings of the 30th Annual Conference of ASME's Noise Control and Acoustics Division, 28–31 July 2008, Dearborn, Michigan, USA.

REFERENCES

1. Warren, C.R.; Lumsden, C.; O'Dowd, S.; Birnie, R.V. 'Green on green': Public perceptions of wind power in Scotland and Ireland. J. Environ. Plann. Manage. **2010**, *48* (6), 853–875.
2. Van Den Berg, G.P. Wind-induced noise in a screened microphone. J. Acoust. Soc. Am. **2006**, *119* (2), 824–833.
3. Thorne, R. The problems with "noise numbers" for wind farm noise assessment. Bull. Sci., Technol. Soc. **2011**, *31*, 262–290.
4. Zwicker, E.; Fastl, H. *Psychoacoustics: Facts and Models*; 2nd Edition, Springer-Verlag, Berlin Heidelberg, New York, 1999.
5. International Electrotechnical Commission. *IEC 61400-11 Wind Turbine Generator Systems—Part 11: Acoustic Noise Measurement Techniques, 2nd Ed.*; International Technical Commission: Geneva, 2002 plus Amendment 1, 2006.
6. International Standards Organization. *ISO 9613-2 Acoustics—Attenuation of Sound during Propagation Outdoors—Part 2: General Method of Calculation*; International Organization for Standardization: Geneva, 1996.
7. Pedersen, E.; Persson Waye, K. Perception and annoyance due to wind turbine noise—A dose–response relationship. J. Acoust. Soc. Am. **2004**, *116*, 3460–3470.
8. Van den Berg, G.P.; Pedersen, E.; Bouma, J.; Bakker, R. *Project WINDFARM perception. Visual and Acoustic Impact of Wind Turbine Farms on Residents*. FP6-2005-Science-and-Society-20. Specific Support Action Project no. 044628. Final report; University of Groningen: Holland, 2008.
9. Van Den Berg, G.P. Effects of the wind profile at night on wind turbine sound. J. Sound Vib. **2004**, *277* (4–5), 955–970.
10. Stigwood, M. Large wind turbines—are they too big for ETSU-R-97? *Wind Turbine Noise*; Institute of Acoustics: Bristol, 2009.
11. Van Den Berg, G.P. The beat is getting stronger: The effect of atmospheric stability on low frequency modulated sound of wind turbines. J. Low Freq. Noise Vib. Active Control **2005**, *24* (1), 1–24.
12. Persson Waye, K.; Öhtrsöm, E. Psycho-acoustic characters of relevance for annoyance of wind turbine noise. J. Sound Vib. **2002**, *250* (1), 65–73.
13. Arlinger, S.; Gustafsson, H.A. *How a Broadband Noise with Constant Sound Pressure Level Masks a Broadband with Periodically Varying Sound Pressure Levels*; Department of Technical Audiology, Linkoping University: Sweden, 1988.
14. Pedersen, E.; Waye, K.P. Wind turbines—Low level noise sources interfering with restoration? Environ. Res. Lett. **2008**, *3* (1), 1–5.
15. Pedersen, E.; van den Berg, F.; Bakker, R.; Bouma, J. Can road traffic mask sound from wind turbines? Response to wind turbine sound at different levels of road traffic sound. Energy Policy **2010**, *38* (5), 2520–2527.
16. Pedersen, E.; Persson Waye, K. Wind turbine noise, annoyance and self-reported health and well-being in different living environments. Occup. Environ. Med. **2007**, *64* (7), 480–486.
17. Martin, M. (2008). *Statement of Evidence in Chief of Mr Murray Martin In the Matter of an Appeal Under Sections 120 and 121 of that Act Between Motorimu Wind Farm Ltd (Appellant) and Palmerston North City Council, Environment Court*, ENV-2007-WLG-000098.
18. Lercher, P. Environmental noise and health: An integrated research perspective. Environ. Int. **1996**, *22*, 117–129.

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19. World Health Organization. *Preamble to the Constitution of the World Health Organization as Adopted by the International Health Conference*; WHO: New York, 1948.
20. Shepherd, D.; McBride, D.; Welch, D.; Dirks, K. N.; Hill, E. M. Evaluating the impact of wind turbine noise on health-related quality of life. Noise and Health, **2011**, *13* (54), 333–339.
21. Møller, H.; Pedersen, C.S. Low-frequency noise from large wind turbines. J. Acoust. Soc. Am. **2011**, *129* (6), 3727–3744.
22. Pedersen, E. Health aspects associated with wind turbine noise—Results from three field studies. Noise Control Eng. J. **2011**, *59* (1), 47–53.
23. Harry, A. *Wind Turbines, Noise and Health*; 2007, http://www.flat-group.co.uk/pdf/wtnoise_health_2007_a_barry.pdf (accessed July 2011).
24. Pierpont N. *Wind Turbine Syndrome: A Report on a Natural Experiment*; K Selected Publications: Santa Fe, New Mexico, 2009.
25. Krogh, C.M.E.; Lorrie Gillis, L.; Kouwen, N.; Aramini, J. WindVOiCe, a self-reporting survey: Adverse health effects, industrial wind turbines, and the need for vigilance. Bull. Sci., Technol. Soc. **2011**, *31*, 334–345.
26. Alvarsson, J.J.; Wiens, S.; Nilsson, M.E. Stress recovery during exposure to nature sound and environmental noise. Int. J. Environ. Res. Public Health **2010**, *7* (3), 1036–1046.
27. Maris, E.; Stallen, P.J.; Vermunt, R.; Steensma, H. Noise within the social context: Annoyance reduction through fair procedures. J. Acoust. Soc. Am. **2007**, *121* (4), 2000–2010.
28. Niemann, H.; Maschke, C. *WHO LARES: Report on Noise Effects and Morbidity*; World Health Organization: Geneva, 2004.
29. European Union. *Noise Policy of the European Union—Year 2*; Luxembourg, 2000, www.ec.europa.eu/environment/noise/pdf/noise_expert_network.pdf (accessed August 2011).
30. European Commission. *Relating to the Assessment and Management of Environmental Noise*, European Parliament and Council Directive 2002/49/EC; 2002, http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2002:189:0012:0025:EN:PDF (accessed August 2011).
31. Berglund, B.; Lindvall, T.; Schwela, D.H. *Guidelines for Community Noise*; The World Health Organization: Geneva, 1999.
32. Hayes, M.; McKenzie, A. *The measurement of Low Frequency Noise at Three UK Windfarms*; Hayes Mckenzie Partnership Ltd, report to the Department of Trade and Industry: London, England, 2006.
33. Berglund, B.; Nilsson, M. Intrusiveness and dominant source identification for combined community noises. In *Fechner Day 1997*, Proceedings of the 13th Annual Meeting of the International Society for Psychophysics, Poznan, Poland, Aug 16–19, 1997; Preis, A., Hornowski, T., Eds.; The International Society for Psychophysics, 1997.
34. Lee, S.; Kim, K.; Choi, W.; Lee, S. Annoyance caused by amplitude modulation of wind turbine noise. Noise Control Eng. J. **2011**, *59* (1), 38–46.
35. Shepherd, D. Wind turbine noise and health in the New Zealand context. In: *Sound, Noise, Flicker and the Human Perception of Wind Farm Activity*; Rapley, B., Bakker, H., Eds.; Atkinson and Rapley: Palmerston North, 2010; ISBN 978-0-473-16558-1.
36. Kuwano, S.; Seiichiro, N. Continuous judgment of loudness and annoyance. In *Fechner Day 90*, Proceedings of the Sixth Annual Meeting of the International Society of Psychophysics, Wurzburg, Germany, Aug 18–22, 1990; Muller, F., Ed.; International Society of Psychophysics.
37. Johansson, M.; Laike, T. Intention to respond to local wind turbines: The role of attitudes and visual perception. Wind Energy **2007**, *10*, 435–45.
38. Nelson, D. 2007. Perceived loudness of wind turbine noise in the presence of ambient sound. In Proceedings of the Fourth International Meeting on Wind Turbine Noise, Rome, Italy, Apr12–14, 2011; INCE Europe; ISBN: 978-88-88942-33-9.
39. Flindell, I.; Stallen, P.J. Non-acoustical factors in environmental noise. Noise Health **1999**, *3*, 11–16.
40. Pedersen, E.; van den Berg, F.; Bakker, R.; Bouma, J. Response to noise from modern wind farms in The Netherlands. J. Acoust. Soc. America **2009**, *126*, 634–643.
41. Pheasant, R.J.; Fisher, M.N.; Watts, G.R.; Whitaker, D.J.; Horoshenkov, K.V. The importance of auditory–visual interaction in the construction of 'tranquil space.' J. Environ. Psychol. **2010**, *30*, 501–509.
42. Calvert, G.A. Crossmodal processing in the human brain: Insights from functional neuroimaging studies. Cereb. Cortex **2001**, *11* (12), 1110–1123.
43. World Health Organization. *WHO Technical Meeting on Sleep and Health*; World Health Organization, Regional Office for Europe: Bonn, 2004.
44. Phillips, C.V. Properly interpreting the epidemiologic evidence about the health effects of industrial wind turbines on nearby residents. Bull. Sci., Technol. Soc. **2011**, *31*, 303–315.
45. Hanning, C.D.; Nissenbaum, M. Selection of outcome measures in assessing sleep disturbance from wind turbine noise. In Proceedings of the Fourth International Meeting on Wind Turbine Noise, Rome, Italy, Apr 12–4, 2011; INCE Europe; ISBN: 978-88-88942-33-9.
46. Phipps, R. A.; Amati, M.; McCoard, S.; Fisher R.M. (2007). Visual and Noise Effects Reported by Residents Living Close to Manawatu Wind Farms: Preliminary Survey Results. New Zealand Planners Institute Conference, Palmerston North, 27–30 March 2007
47. Pedersen, E. Effects of wind turbine noise on humans. In Proceedings of the Third International Meeting on Wind Turbine Noise, Aalborg, Denmark, June 17–19, 2009; INCE Europe.
48. Nissenbaum, M.; Aramini, J.; Hanning, C. Adverse health effects of industrial wind turbines: A preliminary report. In Proceedings of 10th International Congress on Noise as a Public Health Problem (ICBEN), London, UK, July 24–28, 2011; Foxwoods, C.T., Ed; International Commission on Biological Effects of Noise.
49. Bolin, K. *Wind Turbine Noise and Natural Sounds-Masking, Propagation and Modelling*; Doctoral Thesis; Royal Institute of Technology: Stockholm, 2009.
50. Solet, J.M.; Buxton, O.M.; Ellenbogen, J.M.; Wang, W.; Carballiera, A. *Evidence-Based Design Meets Evidence-Based Medicine: The Sound Sleep Study*; the Center for Health Design: Concord, California, 2010.
51. Dang-Vu, T.T.; McKinney, S.M.; Buxton, O.M.; Solet, J.M.; Ellenbogen, J.M. Spontaneous brain rhythms predict

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sleep stability in the face of noise. Curr. Biol. **2010**, *20*, R626–7.
52. Casella, S. *Low Frequency Noise Update*; DEFRA Noise Programme, Department of the Environment, Northern Ireland Scottish National Assembly for Wales, 2001; 1–11.
53. Castelo-Branco, N.A.A.; Alves-Pereira, M. The clinical stages of vibroacoustic disease. Aviat., Space Environ. Med. **1999**, *70* (3), A32–9.
54. Castelo-Branco, N.A.A.; Alves-Pereira, M. Vibroacoustic disease. Noise Health **2004**, *6* (23), 3–20.
55. Alves-Pereira, M.; Castelo-Branco, N.A.A. In-home wind turbine noise is conducive to vibroacoustic disease. In Proceedings of the Second International Meeting on Wind Turbine noise, Lyon, France, Sept 20–21, 2007.
56. Gross, C. Community perspectives of wind energy in Australia: The application of a justice and community fairness framework to increase social acceptance. Energy Policy **2007**, *35*, 2727–2736.
57. Shepherd, D.; Billington, R. Mitigating the acoustic impacts of modern technologies: Acoustic, health and psychosocial factors informing wind farm placement. Bull. Sci., Technol. Soc. **2011**, doi:10.1177/0270467611417841.
58. Fidell, S. The Schultz curve 25 years later: A research perspective. J. Acoust. Soc. of Am. **2003**, *114* (6), 3007–3015.
59. Ando, Y.; Pompoli, R. Factors to be measured of environmental noise and its subjective responses based on the model of auditory-brain system. J. Temporal Des. Archit. Environ. **2002**, *2* (1), 2–12.
60. Lundmark, G. Measurement and analysis of swish noise: A new method. In Proceedings of the Fourth International Meeting on Wind Turbine Noise, Rome, Italy, Apr 12–4, 2011; INCE Europe; ISBN: 978-88-88942-33-9.
61. Hulme. *Approved Judgment in the Court of Appeal (Civil Division) on Appeal from the High Court of Justice, Case No: C1/2010/2166/QBACF*, Neutral Citation Number [2011] EWCA Civ 638, www.bailii.org/ew/cases/EWCA/Civ/2011/636.html (accessed August 2011).
62. New Zealand Standards. *NZS6808:2010 Acoustics—Wind farm noise*; Standards New Zealand: Wellington, 2010.
63. World Health Organization, Regional Office for Europe. *Night Noise Guidelines for Europe*; World Health Organization: Geneva, Switzerland, 2009, http://www.euro.who.int/document/e92845.pdf (accessed August 2011).
64. World Health Organization. *Burden of Disease from Environmental Noise*; Bonn: World Health Organization: Geneva, 2011, http://www.euro.who.int/__data/assets/pdf_file/0008/136466/e94888.pdf (accessed August 2011).
65. Pedersen T.H, 2007, The "Genlyd" Noise Annoyance Model Dose-Response Relationships Modelled by Logistic Functions. Delta Acoustics & Electronics on behalf of the Ministry of Science, Technology and Innovation, Denmark.
66. Di Napoli, C. Long distance amplitude modulation of wind turbine noise. In Proceedings of the Fourth International Meeting on Wind Turbine Noise, Rome, Italy, Apr 1–14, 2011; INCE Europe; ISBN: 978-88-88942-33-9.
67. Miedema, H.M.E.; Oudshoorm, C.G.M. Annoyance from transportation noise: Relationship with exposure metrics DNL and DENL. Environ. Health Perspect. **2001**, *109* (4), 409–416.

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Noise: Paper 4aNSb3

A possible criterion for wind farms

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Opposition to wind farm noise is not abating and shows no sign of doing so in the future. In a January 2017 paper in Sound and Vibration, Hessler, Leventhal, Walker and Schomer come together to report that independently they have come to about the same conclusion for a proper threshold of wind turbine noise. The same A-Weighted criterion has shown to come up in a variety of independent ways. This paper is not for pie in the sky desires for no sound. Rather, it attempts to recommend a criterion to use for determining the limits of wind turbine noise. This criterion is based off of the data of four independent sources: (1) CTL, (2) ANSI S12.9 Part 4, (3) Michaud et al. (2016), and (4) a State of Minnesota Department of Commerce survey of criteria set in various foreign countries and provinces. This paper recommends the use of A-weighting and a 24-hour Leq as the metric. 36-38 dB is recommended for the criterion.

EXHIBIT
Int. I-1(c)

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1. INTRODUCTION

A. BACKGROUND

Like most other industries or sources of transportation, noise and noise criteria are a matter of consternation to all sides involved in the siting and development of wind farms. Industry wants the permitted acoustical levels as high as possible, the community wants them as low as possible, and the municipality or county wants to maximize the dollars in their budget. For the past 10 or 15 years there has been an evolution towards developing a metric and criterion for wind turbine noise. Many turbines were built with what turns out to be rather high levels. They were designed with the community level being set at 50 or even 55 dB (A). Gradually, these levels have decreased, but friction between the community groups, the developers of the wind farms, and local government continues to this day.

B. PURPOSE

The purpose of this paper is to explain and evaluate the metric by which the community response to wind turbine noise is gauged and the limits appropriate to that response function. Chapter II deals with selection of the metric, and Chapter III presents the data and methods used to establish criteria and a criterion, based on the metric selected.

C. APPROACH

The approach to the selection of a metric is pragmatic. When looking at the present situation, A-weighting is the only appropriate metric for most noise sources. Based on all that we know, it could well be that C-weighting is preferred, or even Z-weighting or lower would be an improvement. But pragmatically, what is in use today and has corresponding response functions is A-weighting. These issues are dealt with in Chapter II.

In the second and more major part of the paper, various *independent* references and their procedures are used to find data on which to base the selection of a recommended criterion. These data come from four very independent sources. The use of four totally independent sources of data, independent from each other and independent from the issues at hand cannot be stressed enough. For example, the community tolerance level (CTL) was developed based on road traffic and airport noise, totally independent of wind turbine noise (WTN), totally independent of American National Standards Institute (ANSI) S12.9 Part 4, totally independent of the Health Canada study, and totally independent of the Minnesota Department of Commerce study. Similar statements can be made of each of the four sources, and these four sources are equally independent from the parties concerned (industry, community, and local government). They are totally independent of the results from the ANSI S12.9 Part 4 calculation, because these results were developed without having wind turbines mentioned or included in any way, as this was just a general procedure for environmental noise. Any assessment here is certainly independent from the Minnesota Department of Commerce existing criteria levels. The average and extremes of those data are what they are; nothing we do here can influence that. CTL is derived for other sources and other places, and not constructed for WTN, so its application is totally independent from wind turbine noise sources. The Health Canada data are not totally independent of the issues at hand, but the authors argue that the Health Canada data are equally independent for all three parties. In the same test with the same subjects, the Health Canada study finds that there are no health effects that can be found at the resolution that one gets with about 1200 subjects, but that there are substantial annoyance effects with these same subjects in the same study. One finding for industry, one finding for the community. That is, with the same sampling, the same noise measurements, the same noise predictions, the same surveyors, the same survey instrument, the same subjects, one gets half of the results that in some sense support industry, and half of the results that in some sense support the community. At least to this authors' mind, Health

Canada represents an independent government entity not aligned with any of the three parties. The four sources are as follows:

1.data inherent to community tolerance level (CTL);

2.ANSI S12.9 Part 4

3.data from Health Canada, used to establish the equivalency between wind turbine noise and other noise;

4.the Minnesota Department of Commerce

Note: None of the data was developed by these authors and each of the sources is independent from any of the three primary groups involved: community, developer/operator, and local government. Thus, our approach is to present and explain these sets of data or procedures, and to show how they relate to the general method and the criterion that is ultimately selected.

1. CTL provides a one-number assessment of a set of cluster data from an attitudinal survey. Depending on what is held constant, almost any situation can be compared in decibel units of day-night level (DNL). Keeping with current practice, road traffic noise is used as the baseline. The difference in CTL between a data set under study and road traffic noise is the decibel difference between the two CTL values, respectively.
2. ANSI S12.9 Part 4 is directly used to form a small range of levels for potential development of a criterion.
3. Direct use of the Michaud *et al.* data and other similar international data to set a criterion.
4. Data from a State of Minnesota Department of Commerce survey of criteria set in various foreign countries and provinces.

2. SELECTION OF A METRIC

A. DISCUSSION OF WEIGHTING

As is well known, most sources are assessed using A-weighting with perhaps an adjustment for sound character (e.g. tonal or impulsive). A basic version of this assessment metric has been used since at least 1971 when the first version of ISO 1996 (International Organization for Standardization) was approved. The only source for which A-weighting is not used is high-energy impulsive noise, e.g. sound from demolition, open pit mining and quarrying, sonic booms, and noise from military training. For these sources, C-weighted data are collected, and these data are transformed to equivalent A-weighted levels in terms of equal annoyance (ANSI S12-9, ISO 1996-1).

There is no function that relates C-weighted wind turbine noise to an equivalent A-weighted level, nor is there a function that relates Z-weighting to an equivalent A-weighted level. The C-weighting procedure for high-energy impulsive noise took about 25 years to validate and get into use. Correlation between A-weighting and C-weighting in response to turbine noise has been shown, but this does not show that either of the weightings is correct. There is no conversion tool upon which to develop equivalent A-weighted levels. A response function is required. But it can be observed that a high degree of correlation between A- and C-weighting exists; so high that there is virtually no difference between using C-weighting or A-weighting. When one has a class of sources that all have the same spectrum, then the difference between different linear filters that all measure at least some part of the sound will all be highly correlated with one another. The difference between A-weighting and other weightings is that response functions have been created and scrutinized for A-weighting.

A constant, 24-hour A-weighted equivalent level (Leq) computed over the day and night periods, is the recommended metric, and in nearly all cases, the metric of interest is the nighttime Leq resulting from wind farm operations. So, as with aircraft and other noise categories that are dominated by one kind of source, comparisons can be made from one situation to another because the spectral content has not changed from one situation to another. For example, if one is measuring traffic noise, then the Leq for the hour beginning at 1500 measured on Tuesday should be similar to the hour measured at 1500 on Wednesday.

If the appropriate computational procedures are chosen, then one can install a barrier, have a reasonable chance at predicting a reduction, and subsequently produce a meaningful reduction for the community. That is not the situation with wind farm noise. It has been shown that the correlation from one type of wind turbine to another, and from one size to another, results in a set of numbers that properly order different situations because there is no change to the spectrum from one wind turbine to another. But this is not the case if one performs mitigation and predicts the benefit based on A-weighting. A barrier can be built alongside a highway and the reduction can be predicted. The corresponding decrease in community annoyance can also be predicted, at least to a reasonable degree. We cannot make the same statement about wind farm noise.

The reader should be cautioned not to believe that A-weighting is the correct weighting function for wind farm noise assessment. This simply has not been shown. Currently, however, the A-weighted levels assigned to different community responses seem to fit current wind farms in terms of response and level, at least in terms of annoyance based on attitudinal survey data. A-weighting is not chosen because it has been shown scientifically to be better than other metrics. Rather, it is chosen because at the current state of development, to date, no one has shown any metric to be superior. Even if it were available today, it would still take quite a while to gain acceptance for such a metric.

B. METRIC

The choice of a metric is limited. In principle, all of the readily available noise metrics are those built into sound level meters and other similar devices. The non-time integrating metrics are very limited in the data provided. Lmax and Lmin are two non-integrated choices, but it is clear that Lmax may be something that occurs for a short time every once in a while (e.g., once an hour or once a day). In the class of time-integrated metrics, there are three prominent choices: Leq, Ldn, and Lden. These three are not significantly independent; rather, there are very clear and consistent differences among them. Leq 24-hour is predicated on the assumption that wind farm noise emissions from a given turbine throughout the 24-hour day are more or less constant (read ±1 dB). The question is: how far above Leq must the DNL be such that the calculation of Leq during daytime added to (Leq+10) dB at night equals to DNL? The difference between the numerical value for Leq and DNL when the Leq is held constant is about 6-7 dB. A similar number exists for DENL. DNL or DENL provide no additional information as compared to the simpler, constant 24-hour Leq. Were Leq not a constant, and Ld and Ln are not constant, then a more complicated difference between DNL and 24-hour Leq would be required.

3. METHODS AND PROCEDURES BY WHICH A CRITERION FOR WIND TURBINE NOISE CAN BE SELECTED

A. DIFFERENCES IN COMMUNITY TOLERANCE LEVEL (CTL) BETWEEN ROAD TRAFFIC AND WIND TURBINE NOISE

At this point, it is proposed that a relationship between percent highly annoyed and various nighttime Leq levels be established. However, the recent papers by Fidell et al. and Schomer et al. relate percent highly annoyed to DNL. These two papers also introduce the concept of community tolerance level (CTL). This paper will establish the relationship between nighttime Leq, CTL, and DNL for wind turbine noise. Once that is done, we will compare various DNL and CTL levels with wind farm levels. As a part of this comparison, we will include the transformation of CTL or DNL data to nighttime Leq in order to have valid comparisons. First, DNL will be discussed, followed by CTL.

Up until the introduction of CTL, all community attitudinal survey data were analyzed by using linear regression analysis. There was no underlying functional relation. With CTL, it is hypothesized that the community response to environmental noise is similar to the basic human loudness function where loudness is proportional to the independent variable raised to the 0.3 power. Secondly, it is hypothesized

that the functional form of a relationship is a transition function, and for the sake of simplicity, the simplest form of a transition function is used: e^{-v}. It becomes:

$$\%HA = 100 * e^{-1/(10^{\left(\frac{Ldn-Lct+5.306}{10}\right)^{0.3}})} \quad (1)$$

where 5.306 is an arbitrary constant K. The property of K is such that when Ldn=Lct, then Lct corresponds to the 50th percentile for %HA. That is, for purposes of convenience, the value of CTL for a given community is standardized at the midpoint of the exponential function. A CTL value thus corresponds to the DNL value at which half of the people in a community describe themselves as highly annoyed by transportation noise exposure. As Fidell *et al*. (2011) show, the constant 5.306 follows from the definition of CTL as the midpoint of the exponential function. That is, when DNL = CTL, the %HA = 50%. (Definition of CTL at a point other than 50% on the exponential function would merely result in a change to the constant 5.306, with no loss of generality.)

Fidell *et al.* (2011) gives the percent highly annoyed as a function of DNL for all noise caused by airport operations. Schomer *et al.* (2012) does the same for highway and railroad noise. The convention is that all noises are compared to road traffic noise. The difference in the value of K between any source and road traffic yields the numerical difference in dB between the two situations. For example, the CTL for all road traffic is 78 dB and the CTL for all aircraft is 73 dB. So, aircraft is 5 dB less tolerable than road traffic noise. CTL can quantify the difference between any two situations one wants to consider. For example, one could look at the difference between nighttime and daytime, the difference between hilly country and flat country, the difference between urban, suburban, and rural, or the difference between communities on the ocean and those landlocked.

Michaud *et al.* (2016) calculates the CTL for wind turbine noise to be 62 DNL. That is, 16 dB must be added to the DNL of road traffic noise to make it equivalent to that of wind turbine noise. Michaud et al. also calculate the CTL for each of his two study areas, Prince Edward Island and Ontario, independently. In addition, they calculate the CTL for other surveys that provide the necessary data to calculate the CTL (Pedersen et al. 2004, 2007, 2009; Yano et al. 2013). Michaud shows that the CTL for Ontario is very similar to the CTL for Pederson et al., 2004 and Yano et al. 2013. The CTL for PEI is shown to be very similar to the CTL for Pederson et al 2007 and 2009. The CTL for Ontario is about 7.5 dB lower than the CTL for PEI. They also compute the average CTL for windfarms and that is what is used herein.

B.USE THE DIRECT HEALTH CANADA AND THER COMPARABLE INTERNATIONAL SURVEY DATA OF %HA AT VARIOUS TURBINE NOISE LEVELS

This method is the simplest, it says that the %HA at a certain dB(A) is exactly what is measured. There are three data points provided by the Health Canada analysis: the ranges are from [30-35) dB, [35-40) dB, and [40-46) dB. The corresponding %HA are 1%, 10%, and 14%.

In this paper, several primary sources of data are used to develop the functional relationship and select the criteria. Once a DNL is chosen as the metric, the second step is to establish percent highly-annoyed as a function of DNL. This %HA can then be compared to the results from Michaud *et al*. to form a criterion.

C. USE THE S12.9 TO DIRECTLY DEVELOP A CRITERION

ANSI S12.9 Part 4 uses DNL as its primary metric. ANSI S12.9 Part 6 establishes 55 DNL as the criterion for start of impact from noise. Part 4 also establishes the adjustment of 10 dB for quiet rural areas, i.e. the criterion drops to 45 DNL. In terms of a 24-hour A-Leq, this criterion drops to 39 dB. So,

we find 39 dB to be a criterion, independent of the noise source. This derivation never mentions wind turbine noise.

D. USE THE MINNESOTA DEPARTMENT OF COMMERCE FINDINGS

Minnesota, like 29 other states (reference 2 from Haugen 2011), has a state renewable energy objective that calls for "25% of the state's electrical energy to come from renewable sources including wind energy by 2025 (reference 3 from Haugen 2011)." "While many people support wind energy, some have become concerned about possible impacts to their quality of life due to wind turbines, including noise, shadow flicker, and visual impacts…" Because of these concerns surrounding wind power, the state set out to survey a variety of players in the wind energy industry, from many foreign regions and countries. "For this report, a variety of professionals working on renewable energy issues within national and regional governments, wind energy associations, wind energy development companies, and other areas were contacted by email."

The Minnesota findings are shown in Figure 1. This figure shows national and regional wind farm limits in two different kinds of areas: (1) residential and other noise sensitive areas, and (2) all other areas. These are represented in the figure as a solid blue bar for the sensitive areas, and a solid green bar going above the blue for the other areas. Only 3 of the 19 jurisdictions are above 40 dB: Spain, Portugal, and the Netherlands, and the average is 36 dB.

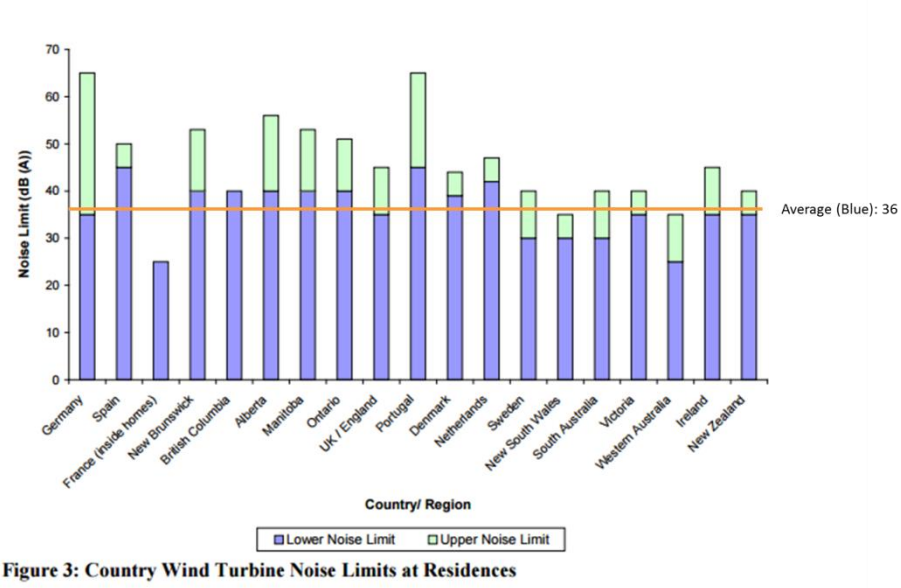


Figure 1: International wind turbine noise limits obtained by the Minnesota Department of Commerce

4. EVALUATION OF CURVES EQUATING DNL TO %HA

In this report, data from six different sources are examined in an attempt to develop a %HA criterion for wind turbine noise (and most other noises): Schultz, the Committee on Hearing, Bioacoustics, and Biomechanics (CHABA), the Federal Interagency Committee on Noise (FICON), CTL (Fidell *et. al.*,

Schomer *et. al.*), Miedema and Oudshoorn (2003), and Miedema and Vos (1997). Schultz, CHABA, and FICON are all based on the Schultz's 1978 synthesis of social surveys on noise annoyance, with the CHABA curve being virtually identical, and FICON being mysteriously low in the relevant DNL interval (60-75 DNL). Miedema and Oudshoorn is an improved version of Miedema and Vos, and along with CTL is used in the current version of ISO 1996-1. Schultz, CHABA, and FICON use data from a combination of aircraft and road traffic noise sources to arrive at their %HA values, whereas CTL, Miedema and Vos, and Miedema and Oudshoorn all make a distinction between aircraft and road traffic. The curve given by Miedema and Vos is shown in the figure for reference as a dashed blue line, but is not included in the analysis that follows because they are two variant data fits to the same data base by the same organization, and using both of them could bias the calculations that follow.

These five sources and their %HA from 50 to 70 DNL in 5 dB increments are shown in Table 1. In this table, Miedema and Oudshoorn and CTL both have separate equations for road traffic and air traffic. CHABA and FICON each use their own single equation for all modes of transportation; planes, trains, and automobiles. Research has conclusively shown that aircraft sound is more annoying than other sound for the same numerical value, which implies that the DNL values Schultz, CHABA, and FICON attribute to a corresponding percentage of high annoyance must be biased high for use with road traffic. And conversely, the %HA for aircraft noise must be biased low. Part A of Figure 2 shows the five functions described for road traffic noise, and Part B shows the five functions described for aircraft noise. From the figures, it would seem that the biased low is a much stronger factor than the biased high. In fact, from the data, one would be tempted to say there is no bias high, but from the logic, this seems to be impossible. As shown in Figure 2A, the Schultz, CHABA, and FICON curves fit somewhat closely to the road traffic curves, but understates the %HA value. For aircraft noise (Figure 2B), %HA values are understated by a very large amount, nominally 15%.

ROAD:					
Group	**M&O**	**CTL**	**CHABA**	**FICON**	**SCHULTZ**
50	3.8	0.7	2.3	1.7	1.3
55	6.6	3.1	4.6	3.3	3.9
60	10.6	8.6	8.7	6.5	8.5
65	16.5	17.6	15.2	12.3	15.2
70	25.1	29.2	24.5	22.1	24.6
AIR:					
Group	**M&O**	**CTL**	**CHABA**	**FICON**	**SCHULTZ**
50	5.3	3.1	2.3	1.7	1.3
55	11	8.6	4.6	3.3	3.9
60	18.6	17.6	8.7	6.5	8.5
65	27.8	29.2	15.2	12.3	15.2
70	38.5	41.9	24.5	22.1	24.6

Table 1: %HA values at different DNL levels for 5 sources

There is no doubt that both Schultz and CHABA represent excellent researchers and excellent organizations. Their results differ from more recent results by Miedema and Oudshoorn, Fidell, and

Schomer. The only conclusion one could come to is that the two databases being analyzed are not the same, and that is known to be the case. The database used by Schultz contained 11 clustering surveys, of which six were aircraft, four were road traffic, and one was railroad. In contrast, the three more recent curves are based on a much larger database. Fidell used 43 aircraft surveys for his work, and Schomer used 39 road traffic surveys and 11 railroad surveys, totaling 93 surveys used to create the CTL method. Miedema and Oudshoorn is based upon a similar quantity of data. A large quantity of the data is used both for CTL and Miedema and Oudshoorn. For a variety of reasons, the authors of this paper will use the methods based on the larger database, Miedema and Oudshoorn, CTL, and CHABA.

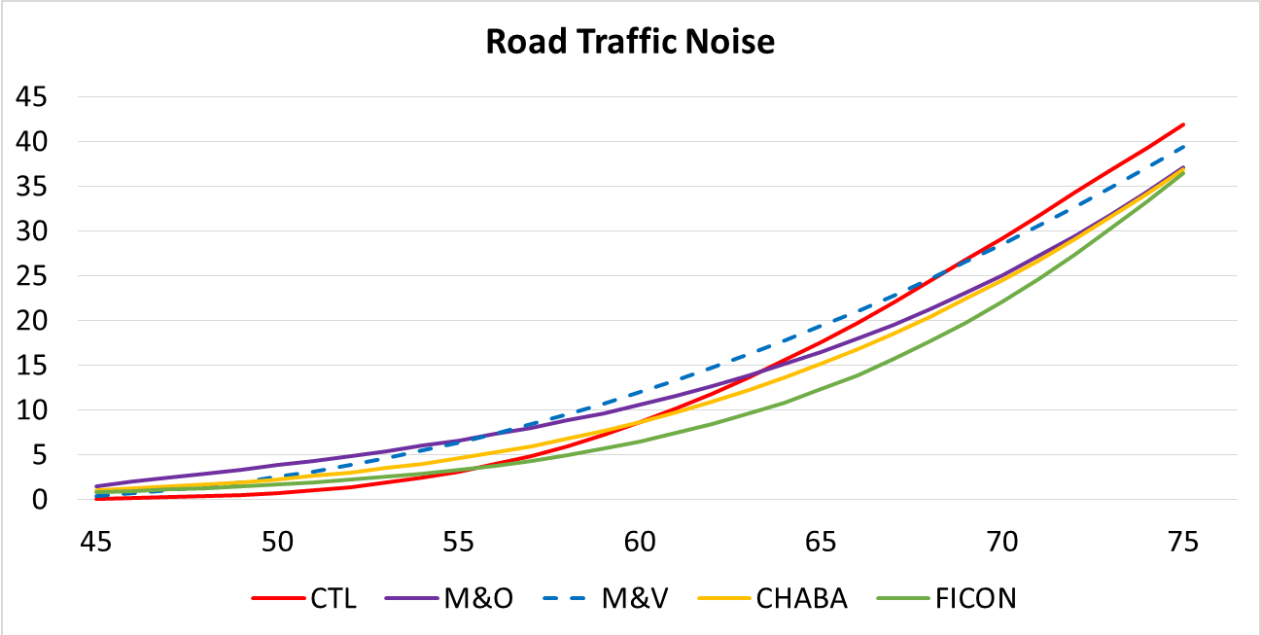


Figure 2A: 5 curves for determining %HA for road traffic noise

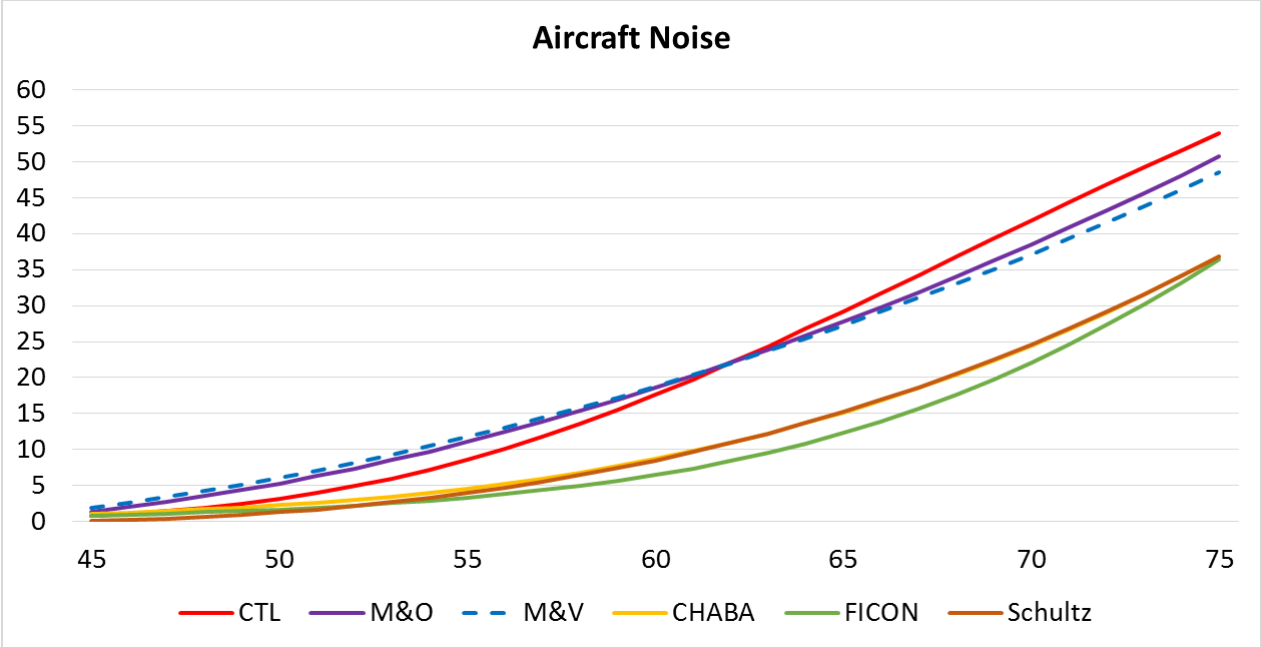


Figure 2B: 6 curves for determining %HA for aircraft noise

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5.WHAT IS THE ACCEPTABLE LIMIT FOR PERCENT HIGHLY ANNOYED (%HA)?

A. ESTABLISHING A FUNCTION FOR %HA vs DNL

Since the purpose of this report is to establish data and relations for the selection of a wind turbine noise criterion. In this section, four independent methods are given with which to establish a relation by which to judge wind turbine noise annoyance. During at least the last several years, it has been common to use road traffic noise as the "yardstick" by which other noises are measured. Miedema and Vos (1997), Miedema and Oudshoorn (2003), Fidell *et al.* and Schomer *et al.*, as well as ISO 1996-1 all use road traffic noise for this purpose.

In 2005, Schomer examined the metrics and criterions used by nearly every federal agency and board, by recommendations in national standards, and by international recommendations such as those made by the World Health Organization. These, and multiple other sources agree to 55 DNL as an acceptable criterion for road traffic noise. Therefore, we will use 55 DNL as our intermediate criterion. The term "intermediate" is used because the real issue is annoyance and not decibels. It is very common to relate %HA to decibels, but it is almost always decibels that are measured and not annoyance. For a DNL of 55 dB, 4 different estimates of %HA were found in the literature. CTL equates 55 DNL with about 3% HA, Miedema and Oudshoorn equates 55 DNL with about 7% HA, for road traffic and aircraft noise separately, and CHABA predicts about 5% for a DNL of 55, for both air and road traffic combined. Herein, we will be using the average of these four estimates, which is 5%.

B. CHOOSING A CRITERIA

1. The first method, the method that is dependent on %HA, relates the data from Health Canada to the 5% value established above. Michaud *et al.* (2013) writes that "Consistent with Pedersen et al. (2009), the increase in wind turbine annoyance was clearly evident when moving from [30–35) dB to [35–40) dB, where the prevalence of wind turbine annoyance increased from 1% to 10%. This continued to increase to 13.7% for areas where WTN levels were [40–46] dB." Michaud relates 3 different values for %HA values with 3 corresponding decibel levels: 1%HA is related to 32.5 dB(A), and 10%HA is related to 37.5 dB(A). Therefore, 5%HA would be related to a value between 32.5 and 37.5 dB(A), most likely around 35 dB(A). With this method, a 5%HA criterion is related to 35 dB(A). A more conservative criterion is given by the doubling of the %HA from 5 to 10%. For this second %HA limit, the corresponding dB(A) level is 37.5 dB(A).

2. The second method compares CTL for road traffic noise to CTL for wind turbine noise. The average CTL for road traffic noise (Schomer *et al.* 2012) is 78.3 dB. In comparison, the average CTL for wind turbine noise is 62 dB. So, a 16 dB difference is found between wind turbine noise and the traffic noise "yardstick." To complete this comparison, one must have a value for an acceptable DNL for road traffic noise. Here, a range of DNL is considered: 55-60 dB. Subtracting 16 yields a range of 39-44 dB for wind turbine noise. As per section II-B above, 6-7 dB is subtracted from DNL in order to calculate Leq. This subtraction yields a range of 32-38 dB as a limit for wind turbine noise.

3. A third method to develop a criterion is to directly apply ANSI S12.9 Parts 4 and 5. Part 5 recommends a DNL of 55 dB for residential areas as a limit based on the start of impact. Part 4 recommends a 10 dB

penalty on the limits for quiet rural areas. Most wind farms are built in quiet rural areas, so this penalty is applicable in this case. In a quiet rural area, the DNL limit becomes 45 dB. But this is DNL, to get to Leq we must subtract 6-7 dB, so that the recommendation becomes an Leq of 38-39 dB.

4. Data published by the Minnesota Department of Commerce, shown in Figure 1, give noise limits for sensitive rural areas and non-sensitive areas. As an example of land use designations, wind turbine noise limits in South Australia are based on the highest level applicable between: rural areas at 35 dB(A), non-rural areas at 40 dB(A), or 5 dB(A) above background measured as L90. The average value of the noise limits for sensitive areas given by the Minnesota report is about 36 dB(A).

6. ANALYSIS AND CONCLUSIONS

Four independent data sources are used to create four estimates of an acceptable 24-hour A-weighted Leq criterion for wind turbine noise. Two methods use 5% highly annoyed as the estimated start of impact for a receiving person. The remaining methods examine both adjustments to a recommended DNL indicating start of impact, and an analysis of existing wind turbine noise limits. The four estimates of a criterion are listed below:

1. 5% HA is shown to be a very approximate average to a criterion for %HA. In order to be conservative, the range from 5 to 10% is considered herein. Applying a 5% HA value to the Health Canada data gives a limit between 32.5 dB and 37.5 dB, or about 35 dB(A). Applying a 10% HA value to the Health Canada data gives a limit of 37.5 dB(A) (Michaud *et al.* 2016b).

2. A 16 dB difference is found between the CTL for road traffic noise and WTN, and if the metric is Leq, then the difference between WTN and Leq is another 6-7 dB, for a total of 22-23 dB difference. Comparing the CTL for wind turbine noise to the CTL for road traffic at the lower limit of 55 DNL for road traffic suggests a limit of 32-33 dB(A). Comparing the CTL for wind turbine noise to the CTL for road traffic at the upper limit of 60 DNL for road traffic suggests a limit of 37-38 dB(A).

3. Applying ANSI S12.9 Parts 4 and 6 to determine the level at which impact will start in a quiet, rural area gives a limit of 38-39 dB(A).

4. The average of existing worldwide limits found in the Minnesota Department of Commerce report for sensitive areas is about 36 dB.

As applicable, Table 2 lists the minimum, average, and maximum Leq criteria for wind turbine noise for each of the four methods above:

	Minimum (dB)	Average (dB)	Maximum (dB)
1-%HA		35	37.5
2-CTL	32		38
3-ANSI		38	39
4-MN DoC		36	
AVERAGE	**32**	**36.3**	**38.2**

Table 2: Minimum, average, and maximum Leq criteria

The average of the top-end values is about 38 dB(A) and the average of the middle values is about 36 dB(A). The minimum level, 32 dB, is not emphasized. These four sets of independent data result in criteria recommendations that are remarkably close to one another, lending support to a 24-hour A-weighted Leq wind turbine noise criterion in or around the range of 36-38 dB(A).

References

CHABA (1977). "Guidelines for Preparing Environmental Impact Statements on Noise." Committee on Hearing, Bioacoustics, and Biomechanics, Assembly of Behavioral and Social Sciences, National Research Council, National Academy of Sciences, Washington D.C.

Fidell, S., Mestre, V., Schomer, P., Berry, B., Gjestland, T., Vallet, M., and Reid, T. (2011). A first-principles model for estimating the prevalence of annoyance with aircraft noise exposure. J. Acoust. Soc. Am. **130**, 791.

FICON (1992). "Federal Agency Review of Selected Airport Noise Analysis Issues," Federal Interagency Committee on Noise, Washington, D.C.

Haugen, K. M. B. (2011). International Review of Policies and Recommendations for Wind Turbine Setbacks from Residences: Setbacks, Noise, Shadow Flicker, and Other Concerns. Minnesota Department of Commerce: Energy Facility Permitting, St. Paul, MN.

ISO 1996-1 (2003). "Acoustics—Description, measurement and assessment of environmental noise—Part 1: Basic quantities and assessment procedures," International Organization for Standardization, Geneva, Switzerland.

Michaud, D. S., Feder, K., Keith, S. E., Voicescu, S. A., Marro, L., Than, J., Guay, M., Denning, A., McGuire, D., Bower, T., Lavigne, E., Murray, Brian J., Weiss, Shelly K., van den Berg, F,. (2016a). "Exposure to wind turbine noise: Perceptual responses and reported health effects." J. Acoust. Soc. Am. 139 (3), 1443-1454.

Michaud, D. S., Feder, K., Keith, S. E., Voicescu, S. A., Marro, L., Than, J., Guay, M., Denning, A., Bower, T., Lavigne, E., Whelan, C., Janssen, S. A., Leroux, T., van den Berg, F,. (2016b). "Personal and situational variables associated with wind turbine noise annoyance" J. Acoust. Soc. Am. 139 (3), 1455-1466.

Miedema, H. M., & Oudshoorn, C. G. (2001). Annoyance from transportation noise: relationships with exposure metrics DNL and DENL and their confidence intervals. *Environmental Health Perspectives*, *109*(4), 409–416.

Miedema, H., and Vos, H. (1998). "Exposure-response relationships for transportation noise," J. Acoust. Soc. Am. 104, 3432–3445.

Schomer, P. D. (2005) Criteria for assessment of noise annoyance. Noise Control Eng. J. **53** (4).

Schomer, P., Mestre, V., Fidell, S., Berry, B., Gjestland, T., Vallet, M., and Reid, T. (2012). Role of community tolerance level (CTL) in predicting the prevalence of the annoyance of road and rail noise. J. Acoust. Soc. Am. 131, 2772.

Schultz, T. J. (1978). "Synthesis of social surveys on noise annoyance," J. Acoust. Soc. Am. 64(2), 377–405.

A theory to explain some physiological effects of the infrasonic emissions at some wind farm sites

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For at least four decades, there have been reports in scientific literature of people experiencing motion sickness-like symptoms attributed to low-frequency sound and infrasound. In the last several years, there have been an increasing number of such reports with respect to wind turbines; this corresponds to wind turbines becoming more prevalent. A study in Shirley, WI, has led to interesting findings that include: (1) To induce major effects, it appears that the source must be at a very low frequency, about 0.8 Hz and below with maximum effects at about 0.2 Hz; (2) the largest, newest wind turbines are moving down in frequency into this range; (3) the symptoms of motion sickness and wind turbine acoustic emissions "sickness" are very similar; (4) and it appears that the same organs in the inner ear, the otoliths may be central to both conditions. Given that the same organs may produce the same symptoms, one explanation is that the wind turbine acoustic emissions may, in fact, induce motion sickness in those prone to this affliction.
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[BSF] Pages: 1356–1365

I. INTRODUCTION

For at least four decades there have been reports in the scientific literature of people experiencing motion sickness-like symptoms attributed to low-frequency sound and infrasound. For example, Dawson (1982) makes the following points:

"Apart from the matter of acoustic fatigue in buildings and other structures, the main problem arising from excessive low frequency noise concerns people who can be disturbed, annoyed, made wretched or ill by acoustic insult to a degree which can be disruptive on a local scale and which nationally produces significant economic and social penalties."

He adds that: "[With] low frequency noise some people can be distressed to an extreme degree while others remain quite unaffected."

"Once a person has displayed some sensitivity to low frequency noise, further exposure lowers the sensitivity threshold."

"Any sensitivity is exacerbated by the presence of other stresses. The low frequency sensitivity syndrome includes: Feelings of irritation, unease, stress, undue fatigue, headache, nausea, vomiting, heart palpitations, disorientation, swooning, prostration."

Fifteen years later, Tesarz *et al.* (1997) reports much the same scenario: "In case studies of persons sensitive to low frequency noise, symptoms such as pressure on the eardrum or a pulsating feeling on the eardrum have been the most consistent result. Other symptoms that have been reported in both field and experimental studies are tiredness, irritation and uneasiness, difficulties to concentrate, headache, nausea and dizziness…."

Adopting the conclusions of Tesarz, Annex C, Clause C.1 of ISO 1996-1 (2003) states "…that the perception and the effects of sounds differ considerably at low frequencies as compared to mid or high frequencies." The text goes on to list six reasons for these differences. Two of these reasons are: (1) "perception of sounds as pulsations and fluctuations," and (2) "complaints about feelings of ear pressure." These are the same two effects as those listed in the preceding text by Tesarz as "most consistent."

Now these same problems are appearing in the vicinity of wind farms, and as in 1982 and earlier, nobody understands how these problems arise; nor is it understood why only a fraction of the population is affected.

The purpose of this paper is to provide a foundation upon which the reported effects of infrasound from wind turbines may be investigated. This paper presents a theory upon which needed investigations can go forward. The Appendix outlines some elements of a research statement.

II. DATA FROM A PROBLEM SITE

A. Observations from people affected by the installation of wind turbines

One wind farm that is experiencing these problems is in Shirley, WI. Here three families have abandoned their homes

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because family members who became ill after installation of the turbines could not acclimate to the situation.[1] Because of these conditions in Shirley, a study was conducted with the proposed test plan calling for the wind farm owner to cooperate fully in supplying operational data and by turning off the units for short intervals so the true ON/OFF impact of turbine emissions could be documented. The owner declined this request citing the cost burden of lost generation from the eight turbines at the Shirley site.

Four acoustical consulting firms cooperated to jointly conduct this study: (1) Channel Islands Acoustics (ChIA), (2) Hessler Associates, Inc., (3) Rand Acoustics, and (4) Schomer and Associates, Inc.

This study was conducted during a 3-day period in December, 2012. The first task accomplished was to meet with residents having problems with the wind turbine acoustic emissions including members of the three families who had abandoned their homes. These discussions with the residents yielded the following observations:

(1) At most locations where these various symptoms occurred, the wind turbines were generally not audible. That is, these problematic symptoms are devoid of noise problems and concomitant noise annoyance issues. The wind turbines could only be heard distinctly at one of the three residences examined, and they could not even be heard indoors at this one residence during high wind conditions.
(2) Some residents reported that they could sense when the turbines turned on and off; this was independent of hearing or seeing the turbines. This assertion by the residents is readily testable, and a plan to test this assertion is briefly summarized in the Appendix.
(3) The residents reported "bad spots" in their homes but pointed out that these locations were as likely to be "bad" because of the time they spent at those locations as because of the "acoustic" (inaudible) environment. The residents did not report large changes from one part of their residences to another.
(4) The residents reported little or no change to the effects based on any directional factors. Effects were unchanged by the orientation of the rotor with respect to the house; the house could be upwind, downwind, or crosswind of the source.
(5) Many of the residents reported motion sickness like symptoms as adverse effects associated with the wind turbines.

Two of the major implications of these five findings are: (1) Because these residents largely report wind turbines as inaudible, it seems that suggestions some have made that these conditions are being caused by extreme annoyance can be ruled out and (2) the lack of change with orientation of the turbine with respect to the house and the lack of change with position in the house suggest that we are dealing with very low frequencies; frequencies such that the wavelength is a large fraction of the wind-turbine diameter (i.e., about 3 Hz or lower).

It should be mentioned that there are about 120 residences within about 5000 ft of the closest turbine; this suggests that there are about 275 residents. Of these 275 residents, 50 have described adverse effects that they have experienced after the introduction of the wind turbines. It is not known how many of the 120 residences are "participating," but most agreements for participating residences include some form of confidentiality and non-complaint clauses.[2]

The most common complaints are feelings of pressure and pulsations in the ears. And this is very much in accordance with ISO 1996-1 (2003) where, as discussed in the preceding text, these two factors are listed as the most common effects of low-frequency noise. However, in this paper, we are concentrating on sea-sickness like symptoms.

B. Physical measurements

Figure 1 is an aerial photo of the Shirley wind farm. This figure shows the positions of five of the eight wind turbines that make up this site, Nordex N-100 s, and the position of the three abandoned residences. Primary measurements were made at residences 1–3 on consecutive days.

Bruce Walker of Channel Island Acoustics employed a custom designed multi-channel data acquisition system to measure sound pressure in the time domain at a sampling rate of 4000/s where all signals are collected under the same clock. The system is calibrated to be accurate from 0.1 Hz thru 10 000 Hz. Measurements were made both inside and outside the house to gather sufficient data for applying advanced signal processing techniques.

George and David Hessler of Hessler Associates, Inc., employed four off-the-shelf type 1 precision sound level meter/frequency analyzers with a rated accuracy of ± 1 dB from 5 to 10 000 Hz. Two of the meters were used as continuous monitors to record statistical metrics for every 10-min interval over the 3-day period.

Robert Rand of Rand Acoustics observed measurements and documented neighbor reports and physiological effects including nausea, dizziness, and headache. He used a highly accurate microbarograph to detect infrasonic pressure modulations from wind turbine to residences.

Paul Schomer of Schomer and Associates, Inc., observed all measurements. Among other things the following observations are made based on the results of the physical measurements. In particular, these observations are based upon the coherence calculations by Bruce Walker. Figure 2 shows the coherence between the outdoor ground plane microphone and four indoor spaces at residence 2: The living room, the master bedroom, behind the kitchen, and in the basement. The data collected at residence 2 were measured with only 58% of turbine power, although the wind conditions were optimal for turbine operation, and the power was much less than 58% during the measurement periods at R1 and R3.

It is inferred from the residents' observations that the important effects result from very low frequency infrasound of about 3 Hz or lower. We can test this assertion with the data collected at the three residences at Shirley. Only residence 2 was tested during a time when significant power was being generated, so it is the only source of data used herein. Figure 2 shows the coherence between the outdoor ground plane microphone and the four indoor spaces listed above

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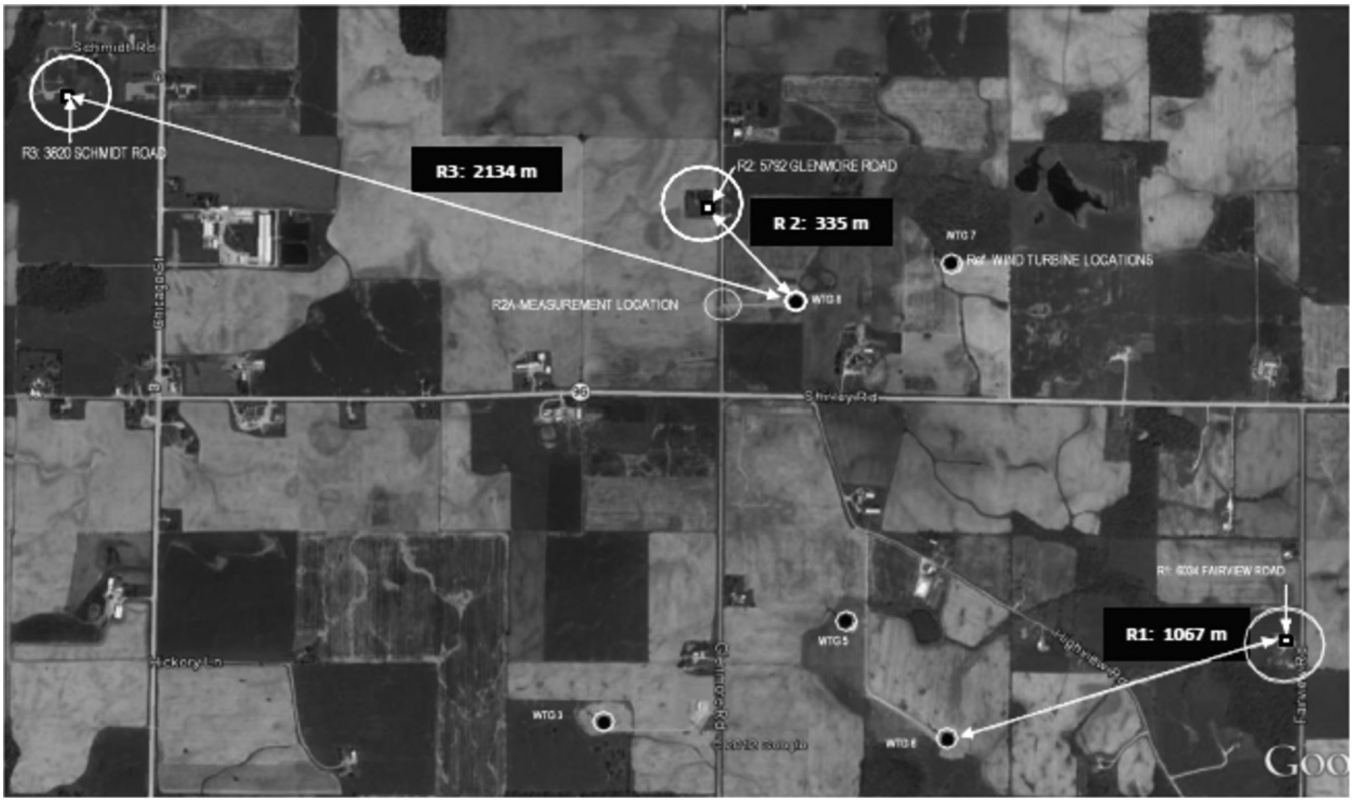


FIG. 1. Aerial photograph of the site showing the three residences and the five closest wind turbines.

for the frequency range from 0.5 to 7 Hz. All of the four spaces exhibit coherence at 0.7, 1.4, 2.1, 2.8, and 3.5 Hz, and in this range, there is no coherence indicated except for these five frequencies. The basement continues, with coherence exhibited at these higher harmonically related frequencies of 4.2, 4.9, 5.6, 6.3, and 7 Hz. The three indoor microphones situated on the first floor exhibit only random zones of high and low coherence as a function of frequency but not so as to correspond to other microphones in the house. That is, above 5 Hz the three indoor microphones exhibit only random periods of coherence, and above 7 Hz the basement microphone exhibits only random periods of coherence. But all four microphones are lock step together in their coherence with the outdoor microphone below about 4 Hz.

As an analysis that is complementary to the coherence plots of Fig. 2, Fig. 3 shows spectral plots of data collected at residence 2. As in the coherence plots, one can see the first several harmonics of the wind-turbine blade-passage frequency, 0.7 Hz, and nothing notable above about 7 Hz. Two channels of measurement are shown on Fig. 3, the outside, ground plane microphone (upper curve), and the indoor microphone in the living room (lower curve). Note that the pressures that result from the acoustic emissions of the wind turbines, when measured indoors, keep growing as the frequency goes lower because the entire house is behaving like a closed cavity.

Based on this analysis of the spectral and coherence data, we conclude that the only wind turbine-related data

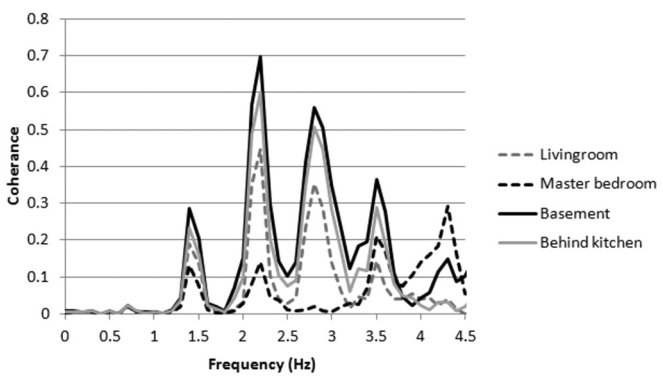


FIG. 2. Coherence between the each of the four indicated rooms with the outdoor-ground plane microphone.

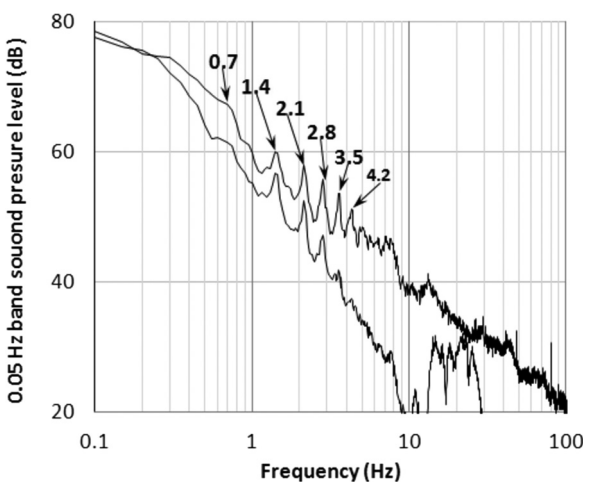


FIG. 3. Spectral plot of the ground-plain outdoor microphone data (upper trace) and indoor data measured in the living room of Residence 2 (lower trace).

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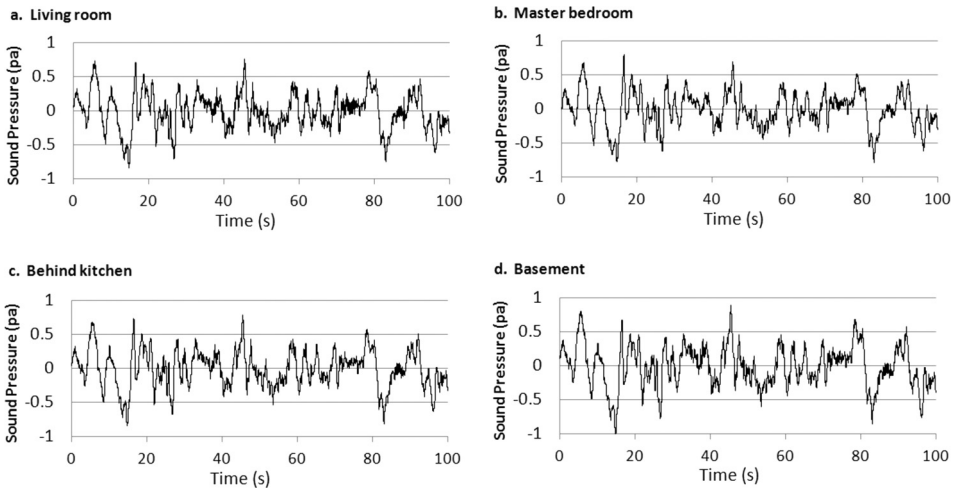


FIG. 4. Sound pressure versus time for of the data collected at the four indoor measurement locations indicated in Fig. 2 and for the first minute of data from the data set used for Fig. 2. Note that the sound pressure versus time is very similar for all indoor locations.

evident in the measurements at residence 2 are the very low frequencies ranging from the blade passage frequency of 0.7 Hz to up to about 7 Hz. This conclusion is consistent with the residents' reports that the effects were similar from one space to another but a little to somewhat improved in the basement, the effects were independent of the direction of the rotor and generally not related to audible sound.

Figure 4 shows the sound pressure level for the first minute of the 10 min represented on Fig. 2, above. This figure, which is sensitive to the lowest frequencies, shows that at these very low frequencies, the sound pressure levels in all four spaces are quite similar. The small changes from different positions in the house also suggests that the house is small compared to the wavelength so that the insides of the house are acting like a closed cavity with uniform pressure throughout being driven by very low-frequency infrasound.

The measurements support the hypothesis developed in the preceding text that the primary frequencies are very low, in the range of several tenths of a hertz up to several hertz. The coherence analysis shows that only the very low frequencies appear throughout the house and are clearly related to the blade passage frequency of the turbine. As Fig. 4 shows, the house is acting like a cavity and indeed at 5 Hz and below, where the wavelength is 60 m or greater, the house is small compared to the wavelength.

While we would have liked to have been able to draw conclusions on measurements at all three sites, that was not possible because the energy company was not generating much power during the measurements of R1 and R3, and even just over 50% during the measurements at R2.[3]

III. THE MOTION SICKNESS HYPOTHESIS

A. The Navy's nauseogenic region

As a starting point we consider a paper by Kennedy *et al.* (1987) entitled: "Motion sickness symptoms and postural changes following flights in motion-based flight trainers." This paper was motivated by Navy pilots becoming ill from using flight simulators. The problems encountered by the Navy pilots appear to be similar to those reported by about five of the Shirley residents. This 1987 paper focused on whether the accelerations in a simulator might cause symptoms similar to those caused by motion sickness or seasickness. Figure 5 (Fig. 1 from the reference) shows the advent of motion sickness in relation to frequency, acceleration level and duration of exposure. To develop these data, subjects were exposed to various frequencies, acceleration levels, and exposure durations, and the Motion Sickness Incidence (MSI) was developed as the percentage of subjects who vomited. Figure 5 shows two delineated regions. The lower region is for an MSI of 10%. The top end of this region is for an exposure duration of 30 min and the bottom end is for 8 hr of exposure. The upper delineated region has the same duration limits but is for an MSI of 50%.

What is important here is the range encompassed by the delineated regions of Fig. 5. Essentially, this nauseogenic condition appears to occur primarily below 1 Hz. Note that the Navy criteria are for acceleration, while in Shirley we are dealing with pressures in a closed cavity, the house. The similarity between force on the vestibular components of the inner ear from acceleration and pressure on these from being in a closed cavity suggests that the mechanisms and frequencies governing the nauseogenic region might be similar for both pressure and acceleration, and much of this paper is concerned with showing the plausibility of the ear responding in like fashion to accelerations of a moving vehicle and acoustic pressures at these same infrasonic frequencies (e.g., 0.7 Hz).

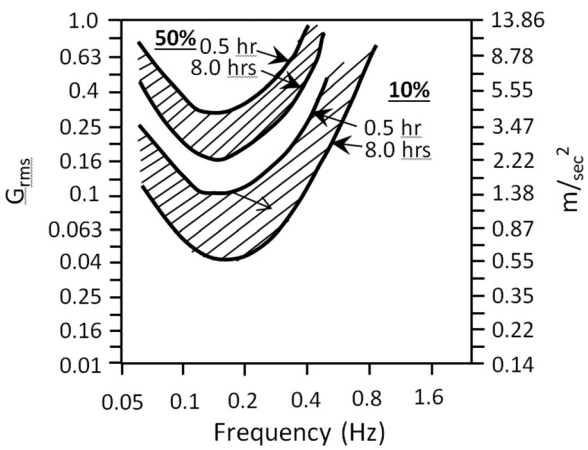


FIG. 5. The nauseogenic region as developed by the U.S. Navy (after Kennedy *et al.*, 1987).

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As the generated electric power of a wind turbine doubles, the sound power doubles and the blade passage frequency decreases by about 1/3 of an octave (Møller and Pedersen, 2011).[4] The wind turbines at Shirley have a blade passage frequency of about 0.7 Hz. This suggests that a wind turbine producing 1 MW would have a blade passage frequency of about 0.9 Hz, and on Fig. 5, a change from 0.7 to 0.9 Hz requires a doubling of the acceleration for the same level of response. Thus it is very possible that this nauseogenic condition has not appeared frequently heretofore because older wind farms were built with smaller wind turbines. However, the 2.5 MW, 0.7 Hz wind turbines clearly have moved well into the nauseogenic frequency range.

B. Motion sickness like symptoms and their implications

We systematically listed the symptoms of low frequency noise, as given by the two papers cited in the preceding text (Dawson, 1982; Tesarz *et al.*, 1997), and on the same basis, we listed the symptoms of sea-sickness, using two journal papers (Stevens and Parsons, 2002; Bittner and Guignard, 1988) and the symptoms listed by the National Health Service (2014) and C-Health (2013). Table I compares the various frequencies of the indicated symptoms of seasickness and low-frequency infrasound sickness from this published literature. The two sets of symptoms are strikingly similar.

Motion sickness, or kinetosis, is generally related to the vestibular, visual, and somatosensory systems (cf. Griffin, 1990). A common theory of the cause of kinetosis is that of sensory conflict: The information received from two or more sensory systems conflict (e.g., visual inputs in a closed room and vestibular inputs from a rolling boat) producing symptoms similar to that of ingesting a poisonous substance. The result is an evolutionary protective response to rid the body of a harmful foreign substance. Thus motion sickness is not really a sickness but rather is a natural reaction to unusual input information.

At the start of this analysis, the working hypothesis was that wind turbine noise somehow, because of the nauseogenic regions similarity, created symptoms that were similar to those of motion sickness. We now have a much simpler hypothesis—just as some people experience motion sickness when watching movies and videos, wind-turbine acoustic emissions trigger motion sickness in those who are susceptible; it is another form of *pseudo-kinetosis*.

TABLE I. Percent of references citing symptom indicated.[5]

	Composite of four sea sickness studies or information papers	Composite of two low frequency "sound" sickness studies
Not feeling well	100	100
Dizziness	100	100
Headache	100	100
Nausea and vomiting	100	100
Sleepiness, drowsiness, and sleep disturbance	75	100
Fatigue and tiredness	75	100
Difficulty thinking	25	50
Irritation	25	100
Sweating	100	0
Pale	75	0

At Shirley, of the 50 people who reported symptoms after the introduction of wind turbines to the area, 5 of those 50 people reported symptoms similar to motion sickness. We simply have no information on other area residents, except for these 50, and do not know how many of the other residents are participating.[3] Based on the sample of 5 of 50, we can say that the incidence of motion sickness symptoms at Shirley is 10% or less, a figure that is clearly in line with the expected percentage of those in the general population affected by motion sickness.[6] In fact, Montavit (2014) indicates that "about 5% to 10% of the population is extremely sensitive to motion sickness; 5% to 15% are relatively insensitive; and about 75% are only subject to it to a 'normal,' i.e., limited degree."

In our meeting with affected residents discussed in the preceding text, it was stated that each person affected by the wind farm noise in the form of motion sickness symptoms was also motion sickness sensitive. The same is true for Rob Rand and Steve Ambrose, who are two acoustical researchers who have themselves reported suffering strong symptoms from low frequency wind-turbine emissions.

As noted in the preceding text, inconsistent proprioception, accelerations, and visual cues may not be resolved and cause a defensive emetic response. For example, during a car trip, nerves and muscle receptors do not register any movement because the body itself is sitting still. The eyes, on the other hand, send the brain a message of fast motion. The equilibrium organ in the inner ear delivers information of curves, acceleration, and/or ascents that contradict the messages from the other two sources. This contradictory flood of impulses and information overburdens a healthy sense of equilibrium that the brain, in turn, interprets as a danger situation. It then releases stress hormones, which in turn create symptoms of dizziness and nausea.

So to induce a sense of motion where none exists and thereby create the sensory conflict that is requisite to induce motion sickness requires that the acoustic signal cause the vestibular system to "tell the brain" it is accelerating when the ocular system is telling the brain there is no motion.

IV. EXCITATION OF THE OTOLITH

A. The middle ear and inner ear

As shown on Fig. 5, the Navy criteria for the likelihood of sea sickness are functions of three factors: (1) Duration of exposure to the motion, (2), amplitude of the acceleration, and (3) frequency of the acceleration. Moreover, because the blade passage frequency has been decreasing and the acoustic power has been increasing as the turbines get larger, one can imagine a future with greater, more frequent problems like those in Shirley (Møller and Pedersen, 2011) (footnote 4). There is one main question that greatly affects the likelihood of this eventuality. This main question relates to the fact that the Navy criteria are based on acceleration, while the wind-turbine acoustic emissions are very low frequency acoustic pressure waves.

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In the following, we show only that it appears that an acoustic wave at 0.5–0.7 Hz can generate a similar response as the signal generated by acceleration at 0.5–0.7 Hz. This discussion analyzes the linear motion sensing function of the ear and explains how the ear could respond to wind turbine emissions. We are concerned primarily with the inner ear.

Figure 6 shows just the inner ear, which contains the cochlea, the organ that transforms the sound wave into locally acting vibration at frequencies ranging from about 10 Hz to about 20 kHz (Obrist, 2011). The inner ear also contains the vestibular system, which controls and facilitates balance and motion. The system of semicircular canals has evolved to be able to sense rotational movements of the head while remaining rather insensitive to forces arising either from translational acceleration of the body or gravity: The cupulae normally have a similar specific gravity to that of the endolymph. The vestibular perception of translational forces originates normally from sensory systems (maculae) located within the utricle and saccule.

As shown in Fig. 7, the classical description for the maculae are flat gelatinous masses (otollithic membrane) covered with minute crystals (otoconia) connected to an area of the utricle and saccule by cells, including hair cells. A suitably oriented translational force will cause the mass to exert a shear force, resulting in a variation in the firing rate of the hair cells. The maculae cover an area of a few square millimeters. They are located on the floor and lateral wall of the utricle and, in an orthogonal plane, on the anterior wall of the saccule (Griffin, 1990).

These six inner ear organs, the cochlea, the three SCCs, the saccule, and the utricle, open into the inner space, the vestibule. The inner ear is divided into distinct fluid-filled chambers containing perilymph and endolymph. A hard bone and fluid (perilymph) surrounds the scala media, which are filled with endolymph, and the only openings to the "outside" are two windows, the round window, which separates the air-filled middle ear from the fluid-filled inner ear by a thin membrane, and the oval window, which connects to the stapes, and also separates the inner ear from the middle ear by means of a thin (round window) membrane (Obrist, 2011).

As the acoustic pressure impinges on the tympanic membrane, it travels through the middle ear and into and through the inner ear from the oval window to the round window. Like a transformer in an electric circuit, the middle ear increases the pressure by 29 dB with a corresponding decrease in velocity. This transformer matches the impedance of air to the impedance of the inner ear fluids. At high frequencies, the tympanic membrane develops modes that affect the transmission of sound across the middle ear. Low frequencies do not create these vibration modes and the membrane vibrates as a "plate." The round window is compliant and responds to the pressure wave that travels up the scala vestibuli and down the scala tympani to create shear forces in the cochlea. These two "tunnels" surround the basilar membrane. Additionally, there is a communication between the scala vestibuli and the vestibular system by means of which acoustic pressure might be transmitted to the otoliths.

B. Classical model of the otolith

We have shown there is a plausible path for the infrasound pressures to reach the inner ear and in particular the otoliths. The classical model of the otolith is shown pictorially in Fig. 7. The otoconial layer is a rather dense, firmer layer of the otolith. It thickens at the surface. The otoconial layer gets its density from embedded calcium carbonate crystals (otoconia). The otoconial layer creates an inertial force when accelerated owing to its mass. This force is transferred to the gel layer (cupula), which then bends the hair cells causing them to transmit signals to the brain. Figure 7 shows in a simple way how the mass in the otoconial layer creates an inertial force that results in shear forces in the cupula and bending of the hair cells coupled into the cupula. So the fundamental measurement by the otolith is the inertial force of the otoconial layer (Grant and Best, 1986); the otolith is measuring force.

C. Calculations of forces acting on the otolith

In this section, we approximate and compare two potential forces acting on the otoliths: (1) Inertial force to accelerations and (2) forces due to the instantaneous pressure in an acoustic wave.

Although the more complete solution for modeling the motion of the otolith is given by a parabolic partial differential equation (Grant and Best, 1986), the frequency response of the otoliths is flat from DC to about 10 Hz (McGrath, 2003), the

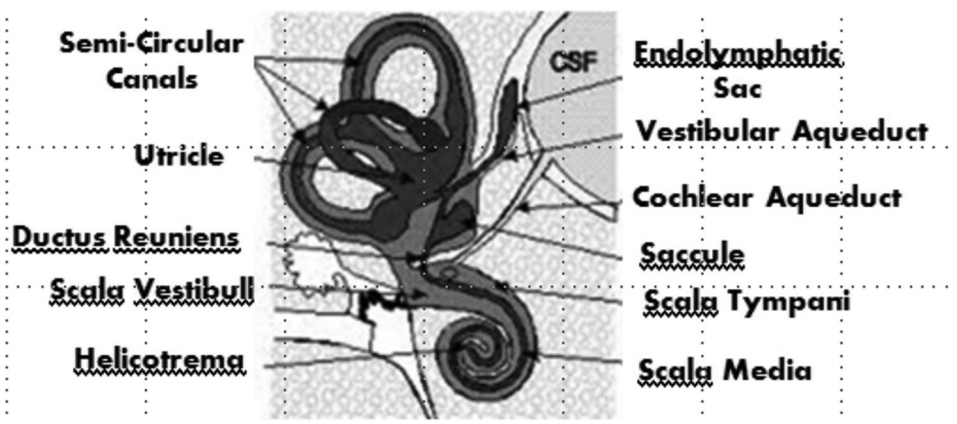


FIG. 6. The inner ear (after Salt, unpublished data).

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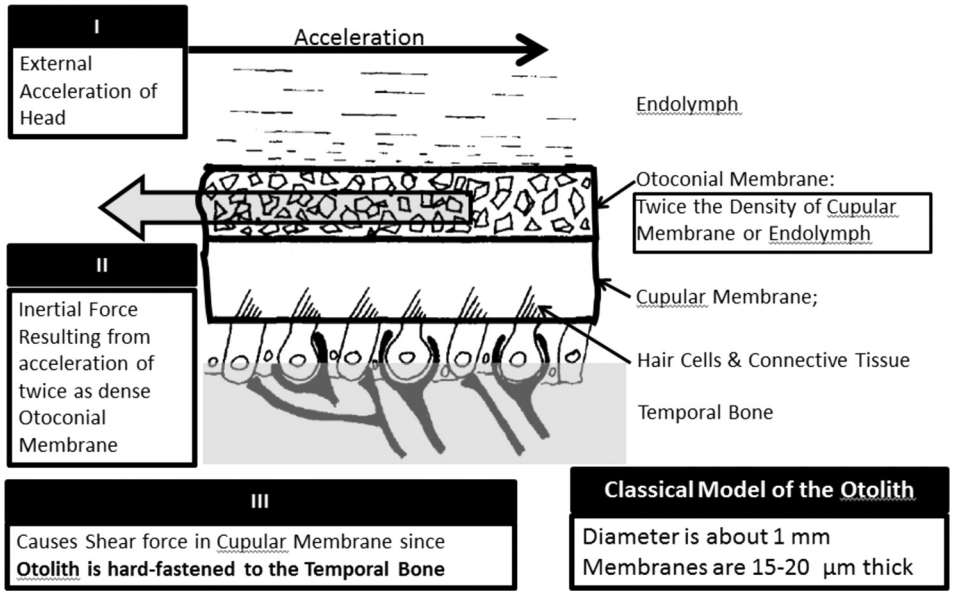


FIG. 7. Schematic sectional drawing of the classical model for the otolith.

position of the poles in the response being functions of assumptions for values of certain parameters describing physical attributes of the layers and their constituents. For an order of magnitude calculation, we simply consider $F = \text{ma}$, where the acceleration is precisely the acceleration of the head, and the mass is the differential density of the otoconial layer minus the density of the surrounding fluid and the cupular membrane times the volume of the otoconial layer. Although calcium carbonate has a density of 2.7 g/cm^3, the density of the otoconial layer is taken to be 2 g/cm^3 because it is a combination of the dense calcium carbonate and the less dense gel material. The density of the cupular membrane and of the endolymph, which has properties given as being similar to water, is taken as 1 g/cm^3, so the differential density is 1 g/cm^3 or 1000 kg/m^3. As can be seen in the classical model of the otoliths (Fig. 7), they are approximated as round and their diameter is about 1 mm. The thickness of the otoconial layer has been given as 15–20 μm (Grant and Best, 1986). Therefore we calculate: the mass = density $*$ height $*$ top surface area or, mass(kg) $= 10^3$ (kg/m^3) $* 18 * 10^{-6}$ m $* \pi * 0.5 * 10^{-3} * \text{m} * 0.5 * 10^{-3} * \text{m} = 18 * \pi/4 * 10^{-9} \approx 1.4 * 10^{-8}$ kg, where density $= 10^3$ (kg/m^3), height $= 18 * 10^{-6}$ m, and top surface area $= \pi * 0.5 * 10^{-3} * \text{m} * 0.5 * 10^{-3} * \text{m}$. With reference to Fig. 7, we take the acceleration to be 5 m/s^2, so the acceleration force,

$$F_{\text{accel}} = 7 * 10^{-8}\text{N}.$$

In terms of the pressure of an acoustic wave, we take the sound pressure level (SPL) to be 54 dB, which corresponds to 0.01 Pa, and because of the "transformer" function of the middle ear, we assume a 29 dB gain in pressure. Therefore the acoustic force, $F_{\text{acous}} = 28 * 0.01 * \pi/4 * 10^{-6}$ N $\approx 22 * 10^{-8}$ N.

D. Excitation of the otoliths

More recent research tends to confirm the model presented in the preceding text for the excitation of the saccule. It is shaped something like an elongated hemi-sphere with the base of the hemi-sphere rigidly attached to the temporal bone and the otoconial layer on the top where under the force of acceleration shear forces can be set up in the cupula. However, there is radically new information about the utricle. Uzun-Coruhlu *et al.* (2007) have used x-ray microtomography and a method of contrast enhancement to produce data revealing "that the saccular maculae are closely attached to the curved bony surface of the temporal bone as traditionally believed, but the utricular macula is attached to the temporal bone only at the anterior region of the macula" (see Fig. 8). This changes the model for excitation of the utricular macula. According to Uzun-Coruhlu *et al.* in the classical view of the utricular macula

"…the sub-surface of utricular macula is implied (if not actually stated) to be rigid; these models do not accommodate the "floating" utricular macula which we have shown and which is consistent with other anatomical evidence (e.g. Schuknecht, 1974). Since the hair cell receptors on the utricular macula are stimulated by forces there would be a major difference in modeling the sensory transduction of the macula to such forces if the forces acted on a tenuously supported flexible membrane or acted on a membrane which is rigidly attached to bone. As an example, modeling the magnitude of utricular hair cell displacement to an increased dorso-ventral g-load during centrifugation will be quite different if the whole membrane is deflected by the g-load or if it remains fixed in place. The latter rigid attachment has been explicitly or tacitly assumed, whereas our results show the macula is not rigidly attached to bone."

"The key information which is now required for realistic modeling of utricular transduction is information about the flexibility of the utricular membrane to determine the extent to which it would be deflected by such forces."

Essentially, Uzun-Coruhlu *et al.* are saying that the excitation of the otolith in the utricle depends on the flexibility of the utricular macula. Because the macula is not rigidly attached to the temporal bone, the classical model (Fig. 7) for excitation of the otolith by acceleration does not work. One way for inertial forces on the otolith to create bending forces is if the stiffness of the utricular membrane varies with position. Then inertial forces on the otolith will make

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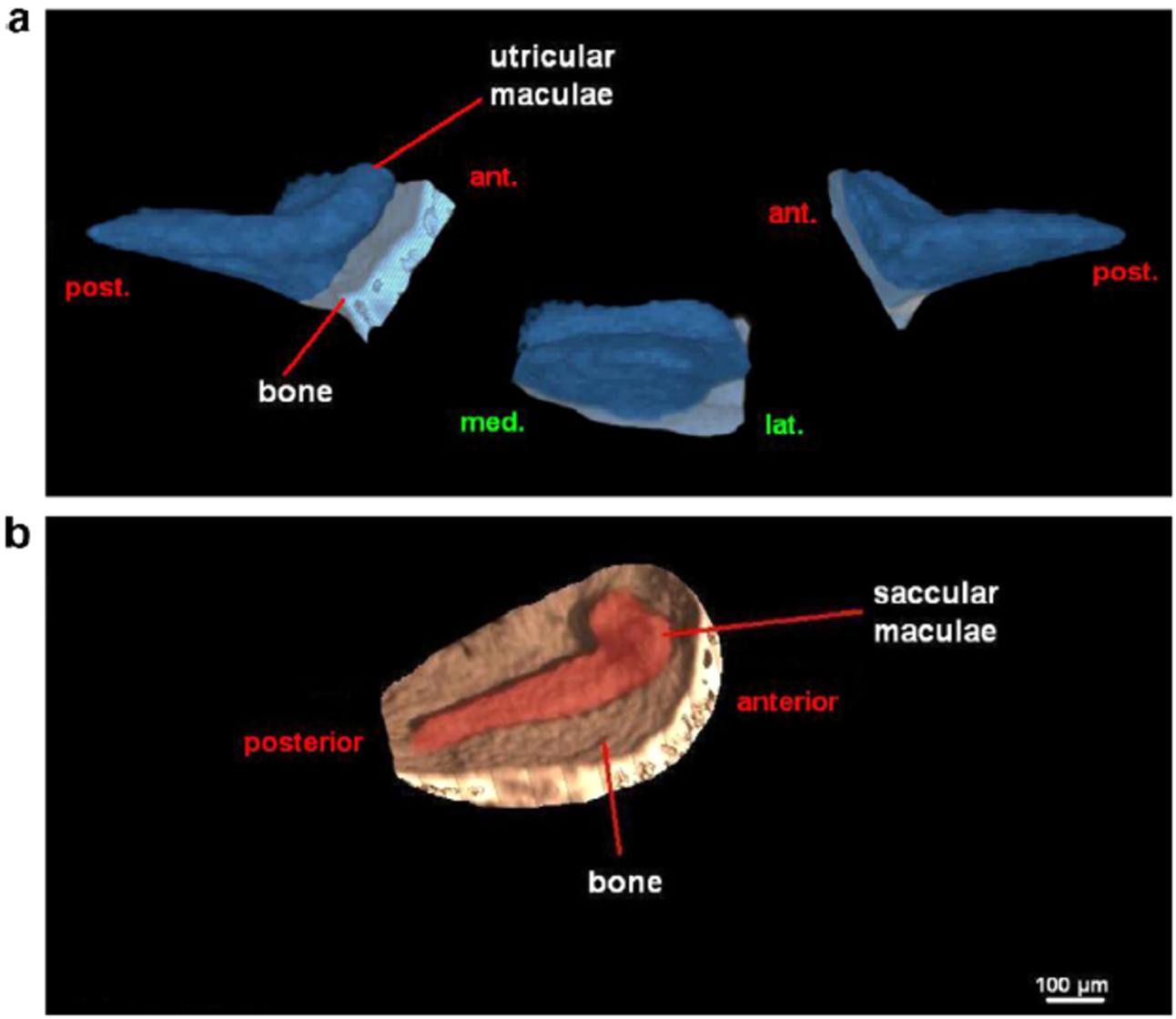


FIG. 8. (Color online) Artist rendered three-dimensional images of the utricular and the saccular maculae of a guinea pig (from Uzun-Coruhlu *et al.*, 2007).

the otolith "bulge" where it is less stiff and contract where it is stiffer, producing bending forces that will trigger the hair cells. Precisely the same thing will happen if the force is externally applied through the endolymph as when the force is internally applied through the otoconial layer. In this model, if there is external force on the utricle, it will expand where it is less stiff and contract where it is stiffer. In particular, the acoustic pressure that reaches the otolith through the eardrum and middle ear pathway described earlier should cause the utricular macula to signal the brain in virtually identical fashion to signals generated by inertial forces, i.e., forces generated by acceleration of the head. That is, the utricular macula should respond in like fashion to acoustic pressure fluctuations and direct acceleration of the head at the same frequency.

E. An example that indicates these theories may be correct

The pressure in the endolymph is a scalar; its "direction" is everywhere normal to the surface. Therefore in contrast to true inertial forces that are vectors, the acoustic pressure will always excite the same hair cells independent of the orientation of the head. So one who experiences this effect should always feel the same motions. And this is exactly what both Steve Ambrose and Rob Rand, who are both acousticians, each experienced. Rob Rand, one of the acoustical researchers on this project, the one who is sensitive to wind turbine acoustic emissions, said of his work in Falmouth, MA in April 2011: "I went outside hoping to feel better. I looked straight at a tree with my eyes, and my brain said the tree was about 20 to 30 degrees elevated and about 20 to 30 degrees to the right. Then I tried to focus on a bush looking straight at it, and again my brain said the bush was off to the right and elevated at about the same angle as before; and the same for the house. For everything I looked at, immediately my brain would say it was elevated and off to the right." Steve Ambrose had exactly the same experience, only not the same angles.

V. CONCLUSIONS

The wind turbine clearly emits acoustic energy at the blade passage frequency, which for the Nordex N100 is

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0.7 Hz and about the first six harmonics of 0.7 Hz. This very low infrasound was only found at R2, but that was the only day in which significant power was being generated (about 58%).

Most residents do not hear the wind-turbine sound; noise annoyance is not an issue. The issue is physiological responses that result from the very low frequency infrasound and that appears to trigger motion sickness mainly in some of those who are susceptible to it. These results suggest a relation between wind turbines and motion sickness symptoms in what appears to be a small fraction of those exposed. This finding does not prove our hypothesis that the otoliths are responding to the wind turbine infrasonic emissions. Rather, we can say that the pathway for inducing this condition appears to be the same as airborne transmission through the middle ear and thence to the vestibular sensory cells, but confirmatory research of the pathway is recommended.

Finally, it is shown that the force generated on the otoliths by the pressure from the infrasonic emissions of the wind turbines is perhaps three times larger than the force that would be generated by an acceleration that was in accordance with the U.S. Navy's nauseogenic criteria (Fig. 5 herein). That is, a 0.7 Hz "tone" at 54 dB produces about the same to three times the force as does a 5 m/s^2 acceleration.

VI. ADDITIONAL RESEARCH AND DATA COLLECTION RECOMMENDATIONS

Research to date has not tended to study the effects on humans reported anecdotally in what is probably a minority of wind farms even though these reports are exactly what is to be expected in accordance with ISO 1996-1 (2003). This paper provides part of the foundation upon which such research could be accomplished. Some of the necessary research is listed below. The first item in the list, perform sensing, is discussed in more detail in the Appendix.

(a) Perform the "sensing" tests outlined in the Appendix of this paper.
(b) Demonstrate electric signals going to the brain that emanate from the otoliths; signals that are in sync with the wind turbine emissions, where depending on method this testing would be done with surrogate species.
(c) Develop an understanding of why this phenomenon seems to affect residents near only a small minority of wind farms.
(d) Establish who is and who is not affected by wind turbine infrasonic emissions in various ways.
(e) Establish why this all occurs.

Currently the wind turbine industry presents only A-weighted octave-band[7] data down to 31 Hz, or, frequently 63 Hz, as a minimum. They have stated that the wind turbines do not produce low frequency sound energies. The measurements at Shirley have shown that low frequency infrasound is clearly present and relevant. As indicated by ISO 1996-1 (2003), A-weighting is inadequate and inappropriate for description of infrasound.

ACKNOWLEDGMENTS

The authors wish to acknowledge the extraordinary effort and trust that went into making the testing at the Shirley wind farm possible. First, there is the extraordinary effort of David and George Hessler and their client, Clean Wisconsin, who made these tests happen at the Shirley wind farm. Coupled with this effort were the extraordinary efforts by Glen Reynolds and Forest Voice who also made this test happen. Additionally, our acknowledgment goes to George Hessler for repeated reviews of the paper with helpful inputs and questions, and much credit is due to Bruce Walker for his development of a custom multi-channel time-domain very low frequency, 0.1 Hz, measurement system necessary for advanced signal processing and analysis between and among channels and his custom reprinting of the coherence and spectral plots herein from Shirley. Additionally, credit goes to Robert Rand for repeatedly being a firsthand source of knowledge about the effects of wind turbine emissions and for general thoughts and ideas. Our acknowledgment goes to Dr. Sarah Laurie (Southern Australia), Dr. Robert McMurtry (Ontario, Canada), and Dr. Jay Tibbitts (central Wisconsin); three physicians from around the world who searched their records to provide information on symptoms and histories. And finally, acknowledgments, Alec Salt for providing key references about the otoliths that led us in the right direction; Sumuk Sundarum, MD Ph.D. internal medicine, for review of an early draft; Stephen Chadwick, MD otolaryngology, for initial ideas and review of an early draft; Paul Schomer's good friend Michael Rosnick, MD family medicine, for correcting a misconception about the Eustachian Tube; and to Paul Schomer's daughter Beth Miller, Ph.D., for initial lessons and information on the anatomy and physiology of the ear.

APPENDIX: A TEST FOR PERCEPTION OF THE ACOUSTIC EMISSIONS FROM WIND TURBINES

In Shirley, residents stated that some of them could sense the turning on and off of the wind turbines without any visual or audible clue. This assertion is readily tested; however, it requires the cooperation of the energy company.

Consider the two houses at Shirley where there is no audible sound; the R-1 house and the R-3 house. The residents of the houses, and others who would be subjects, would arrive at the house with the wind turbines off. The test itself would take something like 2 h to perform. Sometime during the first hour, the wind turbines(s) that had been designated by the residents as the turbines they could sense, might or might not be turned on. It would be the residents' task to sense this "turn on" within some reasonable time designated by the residents–say 10 or 30 min. Correct responses (hits) would be sensing a "turn on" when the turbines were turned on, or sensing no change if they were not turned on. Incorrect responses (misses) would be failure to sense a turn on when the turbines were turned on, or (false alarms) would be "sensing" a turn on when the turbines were not turned on.

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Similar tests could not necessarily be done starting with the turbines initially on because the subjects, when sensitized find it more difficult to sense a turn off.

[1]The family in the closest dwelling, R-2, reported that the wife and their then 2-yr-old son had the problems; the husband did not have problems. This totally stopped upon their leaving the vicinity of the wind turbines.

[2]Traditionally, participating households are those that receive a share of the proceeds in exchange for having wind turbines or ancillary facilities or equipment on their property. As a part of these agreements, these households are required to agree to not complain about the wind turbines. At Shirley, the energy company also had their "good" neighbor policy wherein all residents who were not eligible to be participating were offered payments for agreeing not to make complaints or take any legal action.

[3]A report, including conclusions and recommendation, was written and signed by these five Shirley technical participants. One of the many interested parties and /or legal entities did not like the conclusions and expunged these from the report without obtaining the approval of the authors while retaining the signature block as it was. Both versions were eventually placed in the record and the complete version as written and signed can be found at the following link: http://psc.wi.gov/apps40/dockets/conten/detail.aspx?dockt_id=2535-CE-100c, go to *"Documents"*; then to "January 2, 2013, 8:40 A.M." (Ex. -Forest Voice-Rand2) (Last viewed 9/29/2014).

[4]Møller and Pedersen present data from 41 wind turbines. In Fig. 1, they plot the turbine sound versus power. These 41 data points form two clumps based on power; one at about 700 kW and the second at about 2 MW. Regression lines fit to two measures of the power both show that the sound level is increasing at a rate of about 12 dB for a tenfold increase in power or about 3.6 dB per decade. Normalized spectra for these same two groups exhibit about a one-third of an octave decrease in the spectrum for the higher power relative to the lower power (Sec. D, Fig. 16). There is also a third much smaller clump of 4 turbines with power ratings of about 100 kW that are not used for much in the paper.

[5]A major effort was made to logically group the "symptoms" in Table I. It is possible that this grouping should have gone further and grouped "sleepiness, drowsiness, and sleep disturbance" with "fatigue and tiredness." That combined "symptom" would have resulted in 100% for the two categories that make up the table.

[6]Montavit (2014) states that 5%–10% of the population are "extremely sensitive" and that 5%–15% are "relatively insensitive." So 5%–10% of the population is probably closer to the percentage that we should be using rather than 15%.

[7]One of the reviewers questioned the use of A-weighted octave band levels. The authors also question this, but the IEC standard requires that the data be reported this way and the wind farm industry concurs.

Bittner, A. C., and Guignard, J. C. (**1988**). "Shipboard evaluation of motion sickness incidence and human problem," J. Low Freq. Sound Vib. **7**(2), 50–54.

C-Health (**2013**). "Motion sickness," http://chealth.canoe.ca/channel_condition_info_details.asp?disease_id=183&channel_id=40&relation_id=55627 (Last viewed 12/11/2013).

Dawson, H. (**1982**). "Practical aspects of the low frequency noise problem," J. Low-Freq. Sound Vib. **6**(4), 28–44.

Grant, J. W., and Best, A. W. (**1986**). "Mechanics of the otolith organ-dynamic response," Ann. Biomed. Eng. **14**, 241–256.

Griffin, M. J. (**1990**). "Motion sickness," in *Handbook of Human Vibration* (Elsevier Academic, San Diego, CA), Chap. 7, pp. 271–330.

ISO 1996-1 (**2003**). *Acoustics—Description, measurement, and assessment of environmental noise—Part 1: Basic quantities and assessment procedures* (International Organization for Standardization, Geneva, Switzerland).

Kennedy, R. S., Allgood, G. O., Van Hoy, B. W., and Lilienthal, M. G. (**1987**). "Motion sickness symptoms and postural changes following flights in motion-based flight trainers," J. Low Freq. Noise Vib. **6**(4), 147–154.

McGrath, E. F. (**2003**). "Modeling and monitoring of otolith organ performance in U.S. Navy operating environments. Chapter 2: A simplified mathematical model to predict otolith membrane displacement," Ph.D. thesis, Virginia Polytechnic Institute and State University, Blacksburg, VA.

Møller, H., and Pedersen, C. S. (**2011**). "Low-frequency noise from large wind turbines," J. Acoust. Soc. Am. **129**(6), 3727–3744.

Montavit (**2014**). "Motion sickness," http://www.montavit.com/en/areas-therapy/motion-sickness (Last viewed 9/29/2014).

National Health Service (**2014**). "Motion sickness—NHS choices," http://www.nhs.uk/conditions/Motion-sickness/Pages/Introduction.aspx (Last viewed 9/29/2014).

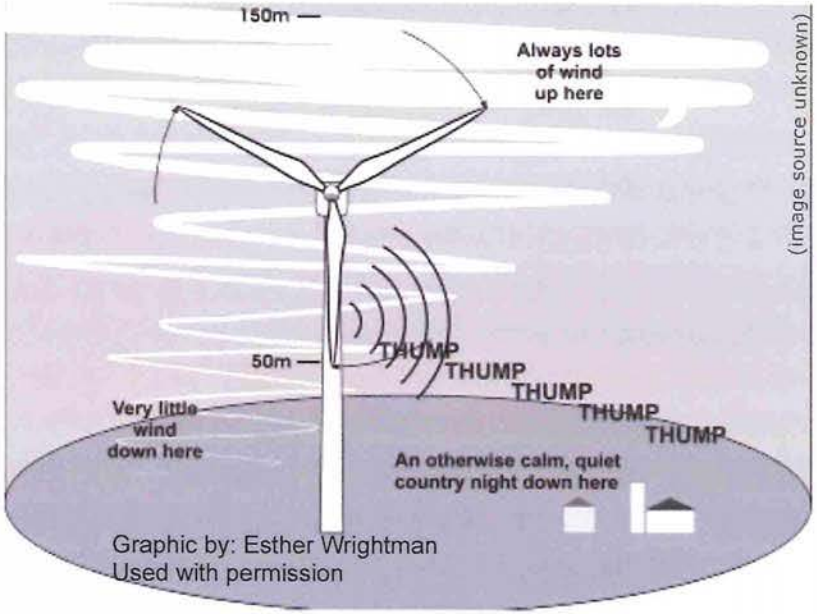
Obrist, D. (**2011**). Fluid Mechanics of the Inner Ear, Habilitation Treatise in Fluid Mechanics at the Department of Mechanical and Process Engineering of ETH Zurich, Zurich, Chaps. 1, 20–22, available at http://e-collection.library.ethz.ch/eserv/eth:5720/eth-5720-01.pdf (Last viewed 9/29/2014).

Stevens, S. C., and Parsons, M. G. (**2002**). "Effects of motion at sea on crew performance: A survey," Mar. Technol. **39**(1), 20–47, available at http://www.nps.navy.mil/orfacpag/resumePages/projects/fatigue/HSISymposium/cdr_pdfs/indexed/1a_3.pdf

Tesarz, M., Kjellberg, A., Landström, U., and Holmberg, K. (**1997**). "Subjective response patterns related to low-frequency noise," J. Low-Freq. Sound Vib. **6**(2), 145–149.

Uzun-Coruhlu, H., Curthoys, I. S., and Jones, A. S. (**2007**). "Attachment of the utricular and saccular maculae to the temporal bone," Hear. Res. **233**, 77–85.

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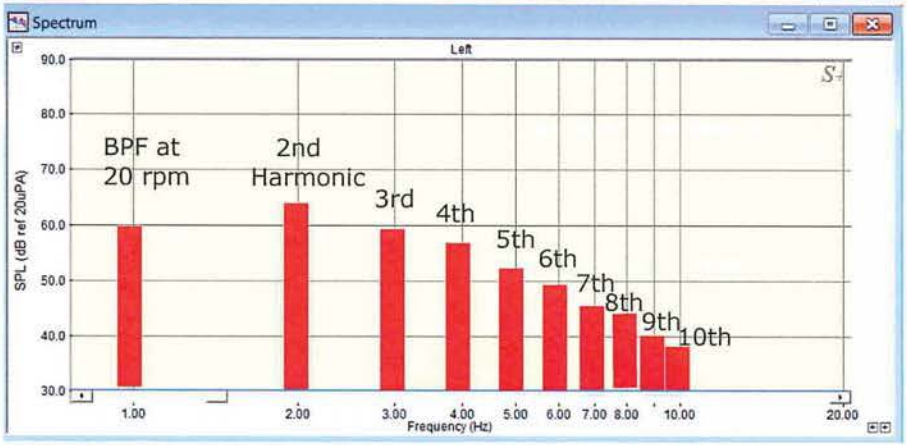


As the blade passes the tower, the low frequency noise and infrasound is generated at a frequency related to the hub's rotation and number of blades. These pressure pulsations appear as tones during analysis but are not heard as tones by most people. Instead they may feel the pressure changes as pulsations, internal organ vibrations, or as a pain (like ear aches or migraines).

This frequency is called the Blade Pass Frequency often abbreviated as BPF.

For modern utility scale wind turbines this frequency is at 1 Hz or lower. A three bladed wind turbine with a hub rotation of 20 revolutions per minute (rpm) has a BPF of 1Hz. This means there is a pressure pulsation emitted into the community once every second. At 15 rpm the BPF is 0.75 Hz and at 10 rpm, 0.5 Hz.





When wind turbine blades rotate past the tower a a short pressure pulse (top graphic) occurs producing a burst of infrasound.

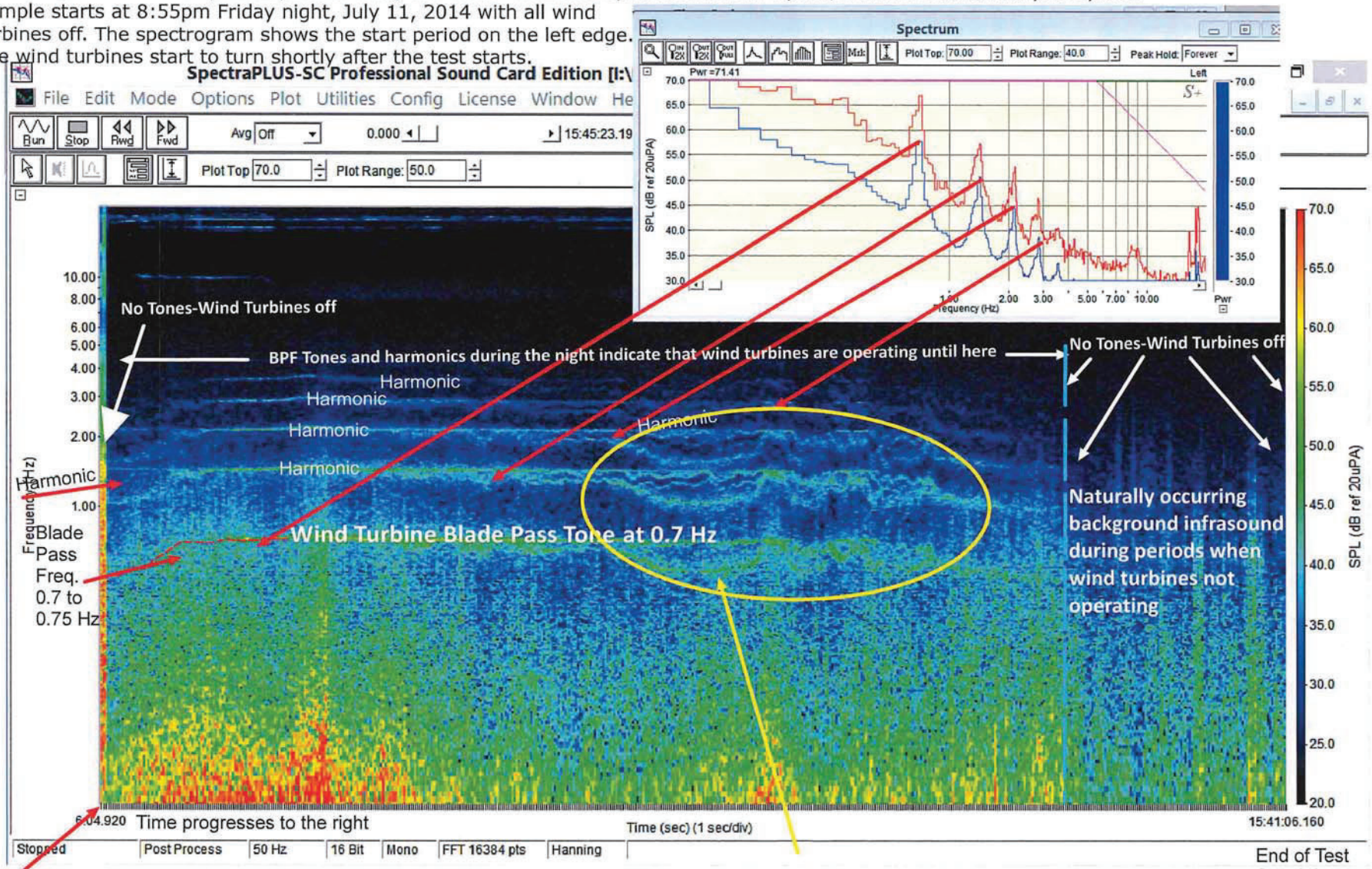
When analyzed the result is a well defined array of tonal harmonics below 10 Hz.
(red bars in figure above)

For impulsive sound of this type the harmonics are all "phase-correlated." This means the peaks of each occur at the same time. Thus, the peaks add together in a linear fashion with their individual maximum sound pressures all coinciding.

Thus, for an impulse having 4 equal amplitude harmonics (BPF, 2nd, 3rd and 4th) each of the same amplitude, the peak level is +12 dB.

10 equal harmonics would produce a peak level of +20 dB.

Baseline micro-barometer test results for side-by-side validation testing overnight in the dining/kitchen area of Home 3 (R1 from Shirley Wind Study)
This test period was sampled in parallel using the GRAS 40AZ microphones with the Apollo/Samurai Sound Analysis System
Sample starts at 8:55pm Friday night, July 11, 2014 with all wind turbines off. The spectrogram shows the start period on the left edge. The wind turbines start to turn shortly after the test starts.



Start of test in R2 Main Floor dining area
Fri. July 11, 2014 at 8:55 pm
Wind turbines were off, but turned on shortly after test started.

The tones from some of the wind turbines deviate from the 0.7 Hz Blade pass frequency indicating that winds or operational changes have altered the rotation speed of some turbines.

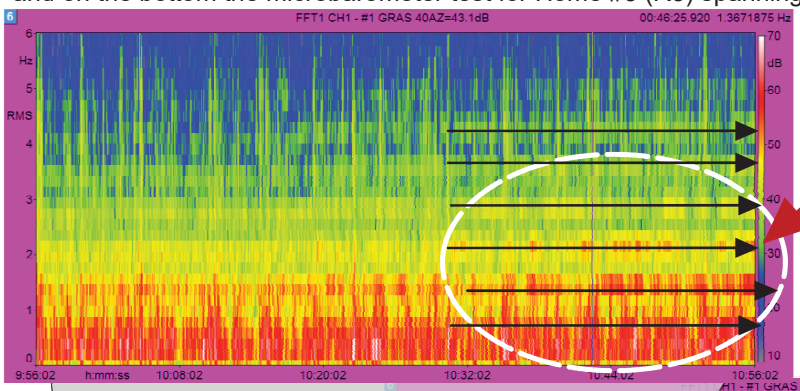
End of Test
Sat. July 12, 2014
at 12:25pm CDT
Turbines were off

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Next two pages present results of side-by-side tests conducted to validate micro barometer reading with acoustic microphone measurements using GRAS 40AZ microphone and Apollo/Samurai hardware/software.

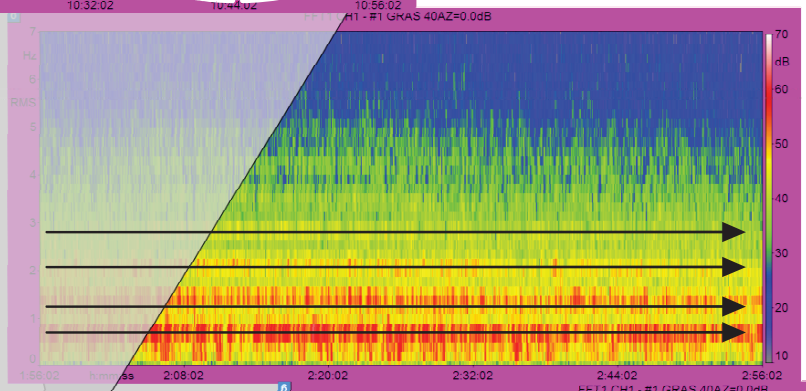
EXHIBIT
Int. I-1(f)

Results of Side-By-Side Validation Tests showing (on top) one (1) hour spectrograms from GRAS/Apollo/Samurai microphone tests and on the bottom the microbarometer test for Home #3 (R3) spanning the entire test period.



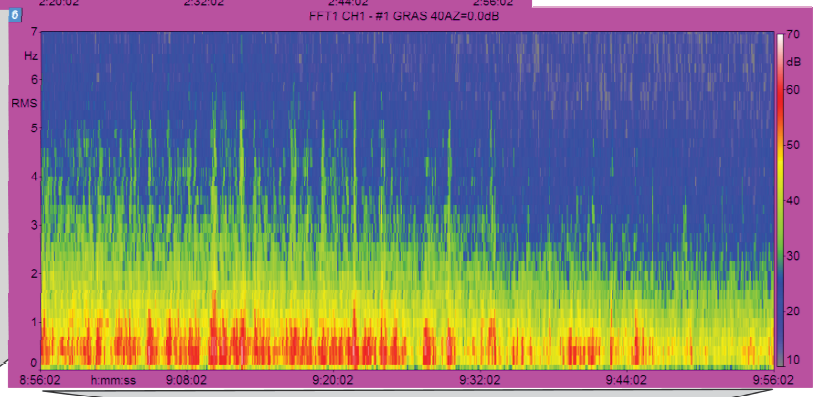
Spectrogram of period during ramp up of wind turbine blade rpm. Tonal characteristics become clearer toward the last third of the test as the microbarometer test shows the wind turbines reaching uniform operating speed.

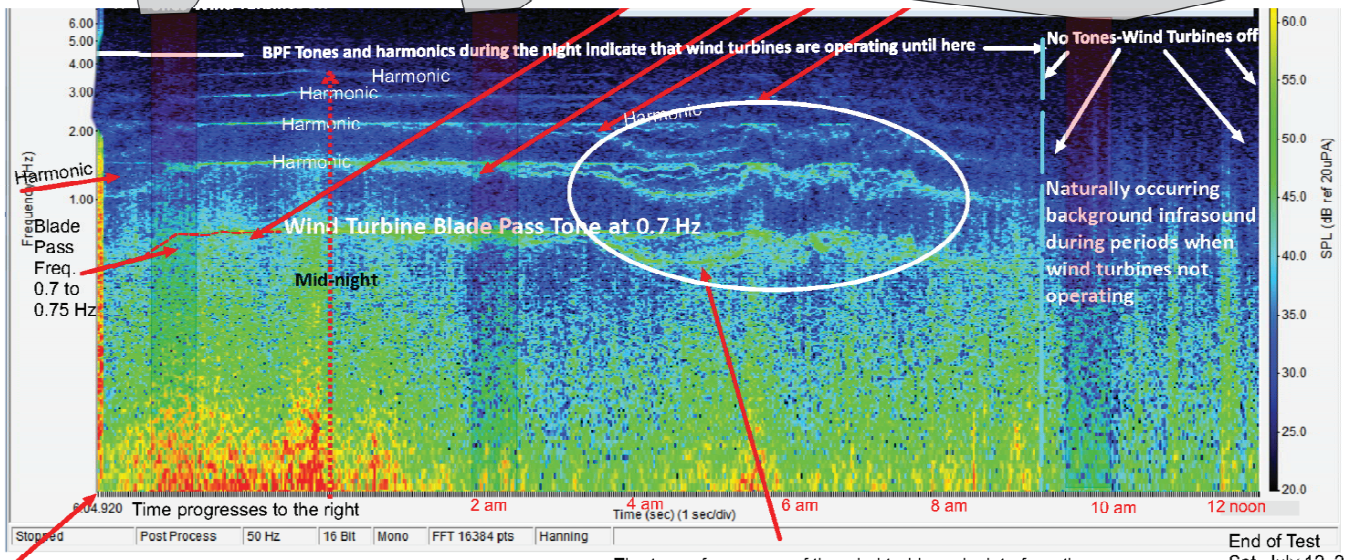
Note: Red on these spectrograms represents sound pressure levels from 50 to 65 dB SPL. This is equivalent to the green through yellow colors for the same range of SPL on the micro barometer spectrogram.



Spectrogram of period during stable operation at 2am of wind turbines shows tones at BPF and at harmonic frequencies corresponding to the SPL of the tones seen in the micro barometer test below.

Spectrogram of period when wind turbines are not in operation (here we see Sat. morning at 9-10am) shows no tones at the BPF or harmonic frequencies.





Start of test in R2 Main Floor dining area
Fri. July 11, 2014 at 8:55 pm
Wind turbines were off, but turned on shortly after test started.

The tones from some of the wind turbines deviate from the 0.7 Hz Blade pass frequency indicating that winds or operational changes have altered the rotation speed of some turbines.

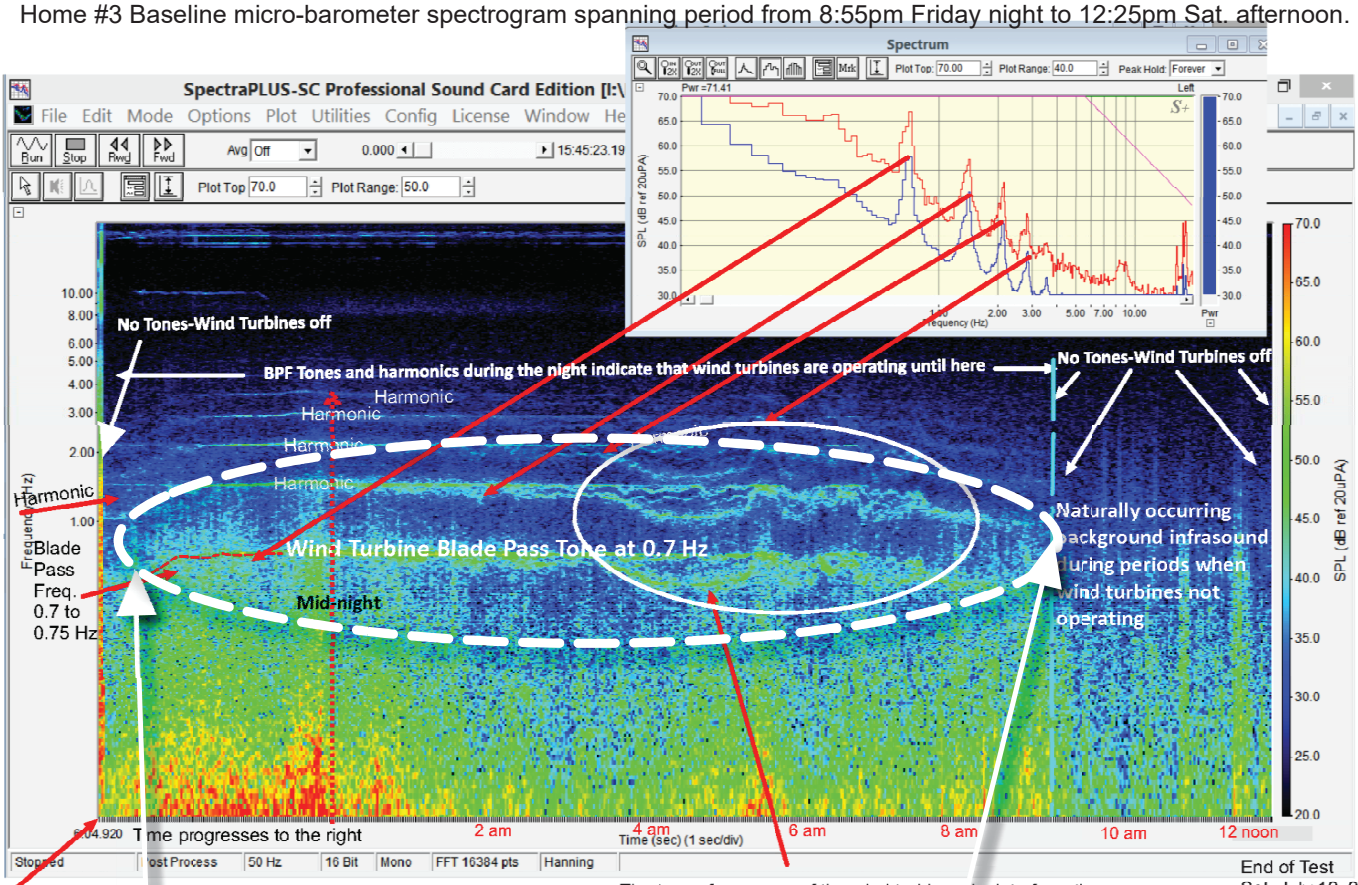
End of Test
Sat. July 12, 2014
at 12:25pm CDT
Turbines were off

Comparison of micro-barometer (bottom) tests overnight inside Home 3 (R1) to microphone spectrograms (3 at top) also inside Home #3 (R1)

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Home #3 Baseline micro-barometer spectrogram spanning period from 8:55pm Friday night to 12:25pm Sat. afternoon.

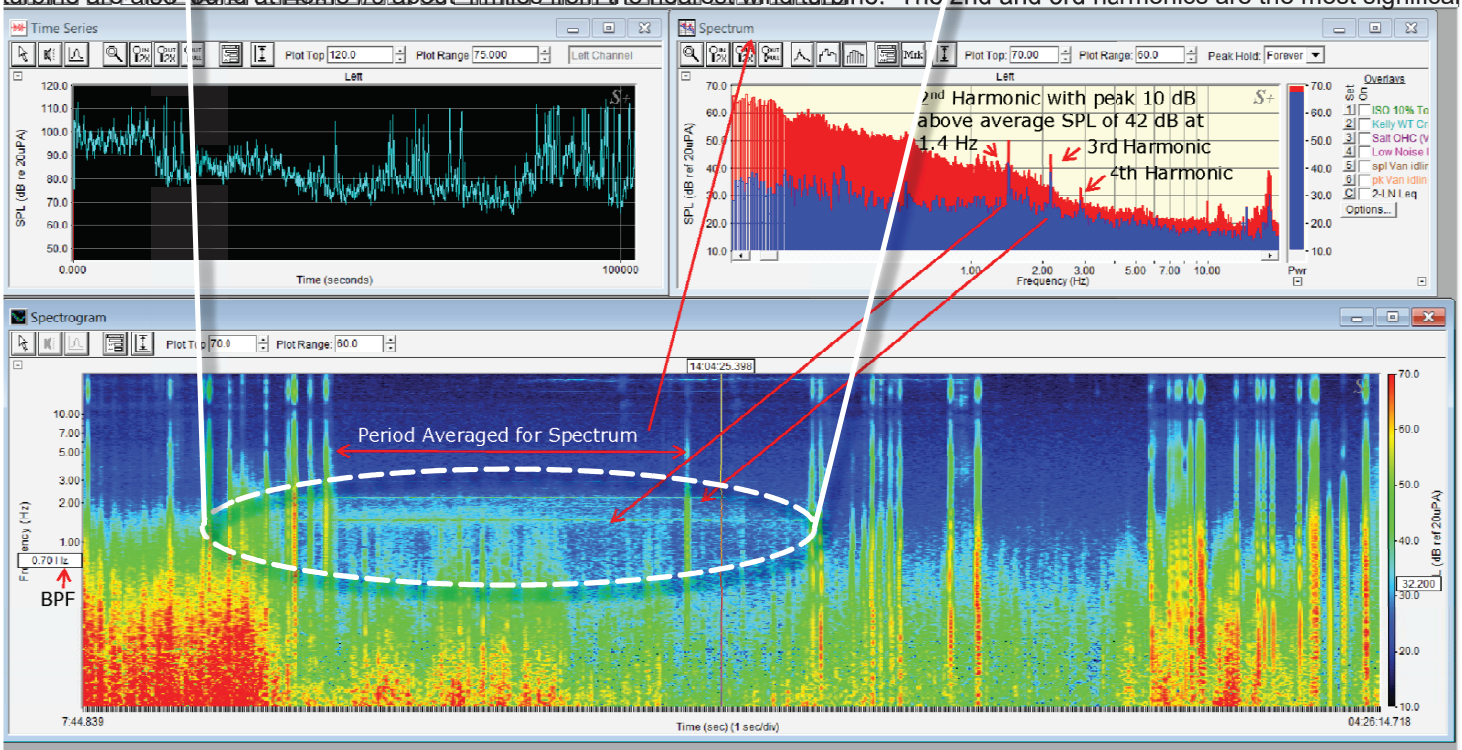


Start of test in R2 Main Floor dining area
Fri. July 11, 2014 at 8:55 pm
Wind turbines were off, but turned on shortly after test started.

The tones from some of the wind turbines deviate from the 0.7 Hz Blade pass frequency indicating that winds or operational changes have altered the rotation speed of some turbines.

End of Test
Sat. July 12, 2014
at 12:25pm CDT
Turbines were off

Home #9 (22,000 ft. from nearest wind turbine) Baseline micro-barometer spectrogram. Test starts at 3:22pm on Friday, July 11, 2014 and ends 33 hours later spanning about double the time as the test at Home #3. Thus, scale is different from the one at Home #3 above. When adjusted for time scale the tones found at Home #3, 3300 feet from the nearest wind turbine are also found at Home #9 about 4 miles from the nearest wind turbine. The 2nd and 3rd harmonics are the most significant.



Comparison of micro barometer test at Home 9, 4+ miles from wind turbines.
Taken concurrent with side-by-side tests inside Home #3 above..

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BEFORE THE PUBLIC UTILITIES COMMISSION

OF THE STATE OF SOUTH DAKOTA

IN THE MATTER OF THE APPLICATION BY PREVAILING WIND PARK, LLC FOR A PERMIT OF A WIND ENERGY FACILITY IN BON HOMME COUNTY, CHARLES MIX COUNTY AND HUTCHINSON COUNTY, SOUTH DAKOTA, FOR THE PREVAILING WIND PARK PROJECT

EL18-026

PREFILED TESTIMONY OF JERRY L. PUNCH

ON BEHALF OF INTERVENORS

EXHIBIT
Int. I-2

Q: Please state your name, title, affiliation, and address.

A: My name is Jerry L. Punch, and I am a Professor Emeritus in the Department of Communicative Sciences and Disorders (CSD) at Michigan State University (MSU) in East Lansing, Michigan. As a retired faculty member, I maintain an office in the Department, which is located in the Oyer Speech and Hearing Building, 126 Red Cedar Road, East Lansing, MI 48824. My home address is 4469 Satinwood Drive, Okemos, MI 48864.

Q: What is the purpose of your testimony?

A: I have been asked to provide testimony as an audiologist on behalf of Intervenors in the matter of the Prevailing Wind Park wind project ("Project"). My testimony as an expert witness will address the potential health risks posed by noise from the Project, if approved according to the application and regulations described in Article 17 of Bon Homme County zoning ordinances and the affidavit of Peter Pawlowski, dated August 9, 2018.

Q: What is audiology?

A: Audiology is the study of hearing and hearing disorders. It is a health-related discipline that focuses on sound, the anatomy and physiology of the ear, hearing disorders, and the clinical aspects involved in diagnosing and treating hearing disorders. As an audiologist, I am knowledgeable of the anatomy and physiology of the ear; sound generation, propagation, and perception; and the ear and how it processes sound. I also have knowledge of research design and interpretation of research findings, and I have had a long-standing interest in community noise issues. This background has led me to understand the relationships between noise and the impacts it can have on human health.

Q: What is your educational and professional background?

A: My full CV is appended as Exhibit 1. I hold a PhD degree in Audiology from Northwestern University and have held a number of professional positions in audiology over the past 50 years. I have had an extensive and eclectic career as a clinical audiologist; clinical supervisor; researcher; teacher; and administrator in academic, professional association, hospital, and industrial settings. My academic coursework included the study of the biological sciences through enrollment in MA and PhD-level courses in anatomy and physiology of hearing and

enrollment in a PhD-level course in physiological psychology. My work experiences include internships and paid employment as an audiologist in multiple otolaryngology clinics as a graduate student; instruction of ENT residents at Indiana University School of Medicine on the clinical aspects of audiology; and instruction of undergraduate-level courses in the anatomy and physiology of hearing. Over the years, I have taught a large variety of undergraduate- and graduate-level courses in clinical audiology. Those courses include a graduate-level course on Research Methods, which I taught at MSU for approximately five years prior to my retirement in 2011. I have also taught a graduate-level seminar on ethics in research and clinical practice. For seven years in the recent past, I served as a representative of the five departments of the College of Communication Arts and Sciences on MSU's Institutional Review Board (IRB). The IRB is charged with reviewing and approving research applications of MSU researchers, with the aim of protecting human subjects who participate in research studies conducted in various disciplines.

Q: What are your current professional credentials and affiliations?

A: I am a member of the American Speech-Language-Hearing Association (ASHA), the American Academy of Audiology, the American Auditory Society, and the Acoustical Society of America (ASA). I hold the Certificate of Clinical Competence in Audiology from ASHA, which I have maintained since 1968 through various formal programs of continuing education. I am also an ASHA Fellow. Fellowship is one of the highest honors the Association bestows. To be awarded Fellowship, nominees must have made outstanding contributions to the discipline of communication sciences and disorders. ASHA Fellows make up less than one percent of the membership of that national organization. Although I am officially retired from MSU, I maintain an office in my academic department and continue to conduct audiological research and to consult on wind turbine projects as a health expert.

Q: What experiences have you had that qualify you as a health expert in cases involving wind turbine noise?

A: I have had a considerable number of such experiences. Since about 2009, I have coauthored a review article on wind turbine noise in *Audiology Today*, served as Chairperson of the Wind and Health Technical Work Group, at the invitation of the Michigan Department of Energy, and

presented invited comments in public hearings and hearings of zoning boards and commissions in several states, including Michigan, Illinois, Indiana, and New York. I coauthored a three-part, invited blog on the *HearingHealthMatters.org* website (Punch & James, 2014). I have been qualified as a health expert in MI by meeting the legal challenge in a Daubert hearing, and served as a health-expert witness in legal cases at local, state, and federal levels in Ohio, Wisconsin, Michigan, Iowa, Illinois, Oregon, Indiana, and New York. This information is detailed in the Forensic Activities section of my CV. I have interviewed multiple individuals and families who have reported adverse health effects, including some who have abandoned homes or are considering abandonment because of health complaints due to wind turbine noise. I have conducted ongoing reviews of the scientific literature on the health effects of wind turbine noise, and in 2016 I coauthored an extensive peer-reviewed article on the HearingHealthMatters.org website with Richard James. The title of that article is *Wind turbine noise and human health: a four-decade history of evidence that wind turbines pose risks*, which I append as Exhibit 2. That paper contains all of the literature references in my testimony. The purpose of the 2016 article was to review the scientific literature that disputes 12 positions commonly taken by the wind industry. Among those positions are statements suggesting that acoustic energy below audible threshold cannot harm people ("What you can't hear can't hurt you"), the complaints are based on psychological expectations, and that there is not sufficient scientific evidence to establish a cause-effect relationship between wind turbine noise and adverse health effects.

Q: What materials have you reviewed in this matter?

A: I have reviewed Bon Homme County's Article 17, drafted on July 27, 2015 to regulate wind energy systems (WES); the sound study conducted by Burns & McDonnell Engineering Company, dated May 18, 2018; the 45-dBA Contour maps of the Project; the direct testimony of Chris Howell, summarizing his noise assessment in the matter of Prevailing Wind Park; the direct testimony of David M. Hessler, dated May 4, 2018, regarding the Dakota Range Wind Project; the pre-filed supplemental testimony of Dr. Mark Roberts regarding Prevailing Wind Park; the direct testimony of David M. Hessler, dated March 28, 2018, regarding the Crocker Wind Farm; and the affidavit of Peter Pawlowski, signed August 9, 2018.

Q: After reviewing those materials, what is your overall impression regarding any potential health risks posed by the proposed Project?

A: In my opinion, those materials paint an overly optimistic picture by indicating or suggesting that limiting wind turbine noise to an average level of 45 dBA will avoid significant adverse health impacts and significant community annoyance. Based on my professional background and experience with people living near existing wind projects, numerous anecdotal reports, the scientific literature, papers presented at scientific and professional meetings, and governmental and agency reports, I believe that a substantial proportion of people living in the vicinity of the proposed Project can be expected to experience not only annoyance, but also a variety of adverse health effects. Those effects, which vary widely among affected individuals, are commonly observed worldwide. They include sleep disturbance, annoyance, headaches, dizziness, vertigo, nausea, motion sickness, ear and bodily sensations, fatigue, stress, depression, memory deficits, inability to concentrate, and reduced quality of life. In a given individual, these effects can occur alone or in combination with other effects. In short, a design goal of a 45 dB average level will not adequately protect the health of residents who live in the boundaries of the proposed Project.

Q: You seem to imply that not all residents will be affected adversely. In what percentage of residents would you expect these adverse reactions to occur?

A: Certainly, not everyone will experience or report negative consequences. Landowners who lease their farmland to host wind turbines ("participants") are less likely than others to complain, partially because they earn an income from their leasing agreements with the wind company, but also because they are often constrained by lease agreements that restrict them from complaining or speaking negatively about their experiences. Likewise, not all non-participants will experience negative impacts, or they may not overtly complain if they do. Some of these individuals have signed waiver agreements with the wind company, occasionally accompanied by a financial payment, which virtually ensures that they will be less likely to complain. One factor that makes the noise tolerable for many people is that the noise is intermittent because the wind is often not sufficiently strong to run the turbines. For almost all exposed residents, though, the turbines inevitably generate relatively a loud thumping, or whooshing, noise, and some residents experience ill effects from the low-

frequency noise and infrasound. The result, for what I would estimate at being around 15%-25% of exposed residents, is extreme annoyance and sleep disturbance. In the longer term, some of the other symptoms I've mentioned begin to emerge. In some cases, a few residents may suffer serious cardiovascular problems such as high blood pressure.

Q: Some of the symptoms you describe seem naturally to occur with aging. How can wind turbine noise be distinguished from aging and pre-existing conditions as the cause of such complaints?

A: One line of evidence comes from the World Health Organization (WHO, 2009), which focuses primarily on low-frequency community noise. That organization states that, based on multiple research studies, such noises can lead to stress, and subsequently to health problems. The pathways from noise to adverse health effects may be direct or indirect. It indicates that several studies have established a closer relationship between subjective responses to community noise and cardiovascular outcomes when the annoyance is sleep-related than when it is non-sleep-related (p. 78). In addition, there are many anecdotal and scientific reports of residents who have experienced sleep disturbance, as well as headaches, dizziness, ear pain or pressure, and inability to concentrate, when near the turbines. When they leave the project area temporarily or for a few days or more, their symptoms subside, and when they return, those symptoms, including sleep disturbance, reappear. Similar observations can be made regarding pre-existing conditions, which are sometimes reported to worsen after turbines become operational. If it can be determined that the additional stresses experienced when near the turbines can be relieved by leaving the area, and that they reoccur when the individual returns to the area, that is a good indication that the turbines are responsible for their deteriorating state of health. The scenario in which symptoms subside and recur with changes in location with respect to the turbines, which many have experienced repeatedly, is similar to the research design known the case-crossover design. Case-crossover studies are described in the 2016 Punch and James paper (Exhibit 2). The types of evidence I've described indicate that there is a strong association between exposure to wind turbines and the health complaints, and they strongly suggest that the link is causative. The main point is that all possible precautionary steps need to be taken to ensure the Project will not substantially impair the health of those living in and around the Project.

Q: How do you view your role in this matter, as it relates to an ability to establish a causative link between wind turbine noise and adverse health impacts?

A: I distinguish between general causation and specific causation, as they differ based on the targets of interest: the general population versus targeted individuals, respectively. Physicians, including those with epidemiological backgrounds, have the medical expertise to diagnose and treat the health symptoms of their individual patients who have been exposed to wind turbine noise. The chief recommendation of physicians who have become involved with patients who suffer adverse health effects from wind turbine noise is to move away from the source of the problem. On the other hand, acousticians, audiologists, occupational health and safety experts, and environmental experts have the expertise to analyze the available research and other evidence needed to conclude that wind turbine noise causes adverse health impacts in the general population. These individuals are often called upon as experts in legal proceedings such as this one. That is the role in which I see myself in this matter.

Q: Dr. Mark Roberts, in his supplemental direct testimony, has testified on the role of epidemiological research in establishing a causative link between wind turbine noise and AHEs. What is your reaction to that testimony?

A: My reaction is essentially the same as that already described in Exhibit 2. Dr. Roberts' testimony rests primarily on his credentials in epidemiology and apparently not on his first-hand experience with people who have been exposed to wind turbine noise over long periods of time. Also, he appears to be acquainted with only that body of literature on the subject that is favorable to the wind industry, and to his testimony in its behalf. He points to peer-reviewed epidemiological research as the only basis for proof of cause-effect relationships. Although he espouses the Bradford Hill criteria as relevant, he essentially dismisses most of the nine criteria by naming them, without discussing their implications. Those criteria, with descriptions from Punch & James, 2016, were: (1) strength (strength of observed relationships), (2) consistency (consistency, or repeatability, of relationships, based on observations by different persons, in different places, under different circumstances, and at different times), (3) specificity (causation is indicated if the association is limited to specific individuals and to particular sites

and types of disease and there are no associations with other factors), (4) temporality (there is a clear temporal relationship between outcomes and periods of exposure and non-exposure), (5) biological gradient (a dose-response relationship exists), (6) plausibility (causation is more likely when certain outcomes are biologically plausible, or possible, a caveat being that plausibility depends on the biologic knowledge of the day; this element is best expressed in the statement: "When you have eliminated the impossible, whatever remains, however improbable, must be the truth" (p. 10), (7) coherence (the cause-and-effect interpretation of data should not seriously conflict with generally known facts of the natural history and biology of the disease), (8) experiment (experimentation or semi-experimental evidence, even if only occasional, can reveal the strongest kind of evidence for causation), and (9) analogy (the recognition that similar cause-effect relationships have occurred under similar conditions). Hill states:

> What I do not believe (is) ...that we can usefully lay down some hard-and-fast rules of evidence that must be obeyed before we can accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question – is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?... No formal tests of significance can answer those questions. Such tests can, and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the 'proof' of our hypothesis (p. 299).

Hill makes this final observation in his essay:

> All scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time (p. 300).

In summary, my reaction to that portion of Dr. Roberts' testimony is that, like many of his epidemiological colleagues who testify on behalf of wind energy projects, he chooses to disregard Hill's intent to emphasize that experimentation (Hill's eighth of nine criteria) is only one of many criteria that are useful is establishing causation between external agents and disease processes.

Q: Can you give specific examples of how the Bradford Hill criteria apply to wind turbine noise and adverse effects on health?

A: Yes, I believe that the available evidence, which includes both research and common-sense observations, meets all nine of the Bradford Hill criteria, and that, in their totality, that evidence supports a causative relationship between wind turbine noise and adverse health effects. This evidence includes, respectively: (1) widespread reports of complaints, (2) consistency of reported symptoms, (3) and (4) concurrence of symptoms with wind turbine operation, (5) an observable dose-response relationship between exposure levels (or distance) and symptoms, (6) the role of disturbances of the hearing and balance mechanisms of the inner ear in causing identified symptoms, (7) coherence with WHO (2009) and other relevant guidelines, (8) in addition to cross-sectional studies, experimentation is established by the fact that symptoms decline or disappear when receptors leave the area and recur when they return to the area, and (9) Sick Building Syndrome as the analogy. Based on these observations, Dr. Roberts' efforts to raise epidemiology as the only cause-and-effect threshold sets the standard so high that we may never expect to reach resolution on this and many similar matters. Dr. Carl Phillips, also an epidemiologist, states in a paper prepared for the Wisconsin Public Service Commission (dated July 3, 2010):

> Some recent commentators (Colby et al. 2009; Roberts and Roberts 2009) have attempted to dismiss this evidence because none of it is based on the epidemiologic study types that they understand. It is true that other study designs would have told us more, and still could. But dismissing the evidence we have makes little sense given that a huge portion of all knowledge, including formal scientific inference, is based on data that is not from studies designed according to certain preferred approaches. It should be obvious that "does not tell us everything we want to know" does not mean "has no information content". Those making this argument either do not understand scientific inference or are pretending they do not. Claiming that there is no evidence even though there are reports of individuals suffering is akin to claiming that there is no evidence that people get injured as a result of text-messaging while engaged in other activities because, even though the pathway is obvious and there are numerous accidents occurring from some activities, there is often not a "real study" that allows us to make various quantitative estimates. (p. 7).

Q: Do you have additional reactions to Dr. Roberts's supplemental direct testimony in this case?

A: Yes, I would like to make one other point. Dr. Roberts raises the nocebo argument. He is arguing that the complaints people make regarding adverse effects of wind turbine noise are psychologically motivated by expectations resulting from negative messages surrounding

turbines. That argument continues to persist as one of the wind industry's primary explanations for adverse health impacts. In our 2016 paper, James and I, after evaluating these claims, concluded that none of these explanations is as plausible as the notion that a variety of adverse reactions are *physiological* effects caused directly or indirectly from exposure to low-frequency noise and infrasound from wind turbines. While psychological expectations and the power of suggestion can influence perceptions of the effects of wind turbine noise on health status, no scientifically valid studies have yet convincingly shown that psychological forces are the major driver of such perceptions. We describe in some detail in our article the scientific shortcomings of the several studies that have been done, all of which conclude that the nocebo effect is the culprit. I encourage interested individuals to read those details.

Q: How does your background qualify you to testify on the general causal mechanism that explains these adverse health effects?

A: First, I would note that two of the seven panelists commissioned by the American Wind Energy Association to conduct the 2009 review of literature by Colby and colleagues on the noise and health effects of wind turbines were audiologists. Audiologists have the educational background to understand the functioning of the inner ear, and it is that knowledge that led me to become interested, over the last decade, in the relationship between ear physiology and the health impacts of infrasound and low-frequency noise from wind turbines on people. Like many others who have studied this relationship, I believe that most of these adverse reactions are mediated by disturbances of the hearing and balance mechanisms of the inner ear resulting from the low-frequency noise emitted by industrial wind turbines. The inner-ear components affected include the cochlea, which is the organ of hearing, and the vestibular system, which includes the semicircular canals, utricle, and saccule. These organs are responsible for balance, or equilibrium. While the cochlea is responsible for the perception of audible sounds, the vestibular system is sensitive to movement and changes in head position, and can be stimulated by infrasound to induce perceptions of unsteadiness, dizziness, vertigo, and motion sickness in some people.

Q: Earlier, you emphasized sleep as being critical to health. How does wind turbine noise lead to sleep disturbance, in your opinion?

A: Wind turbine noise is a significant disruptor of sleep because our ears, unlike our eyes, are always open, especially to unusual or novel stimuli, including "bumps in the night" that might threaten our safety. During operation, the turbines produce audible noise, mostly in the infrasonic and low-to-mid-frequency range. That audible noise results in the perception of both a relatively constant whirling sound and a periodic whooshing sound, caused by a combination of the blade movement against the air and the blades passing in front of the tower. When the three blades are rotating at a typical 20 revolutions per minute, that sound occurs once per second. Those audible sounds can annoy people and disrupt their sleep patterns. The turbines also generate a pulsating sound at infrasonic rates that are based on blade rotational speed, meaning that the sound spikes, or peaks intermittently. These noises, and the unpredictability of the prevailing winds, are responsible for sleep disturbance in a substantial number of people. The peakiness of the noise is especially annoying and disturbing, and is the reason sleep disruption is not adequately predicted from, or correlated with, long-term average decibel levels, designated as LAeq.

Q: If dB LAeq is not used to quantify noise levels of wind turbines, what metric might better predict sleep disturbance?

A: LAmax, or the maximum noise level produced during a given nighttime period, appears to be the optimal measurement metric to protect sleep. The WHO (2009) Night Guidelines suggest that a 40 dB LAmax level should be the maximum allowable level during nighttime hours. That document uses the term "LAmax" a total of 93 times, which is an indication that the WHO considers the concept highly important as a metric for quantifying nighttime noise. If used, any compliance-monitoring procedures should allow some degree of repetition to occur, and to eliminate other noise sources as the origin of the emissions, before noncompliance is declared. Because there are sufficient audible differences among wind turbine noise and other sources of noise—including traffic noise, thunder, wind, and wildlife—the various sources are easily distinguishable.

Q: Are there other noise measurement metrics that could effectively protect sleep?

A: Yes, possibly. Dr. Paul Schomer currently recommends that wind turbine noise should be limited to an average level of 36-38 dBA, based on a 24-hour measurement period. Although he

offers that recommendation for the purpose of avoiding substantial annoyance at all hours of the day and night, it is a potential alternative to 40 dB LAmax in an effort to minimize or avoid sleep disturbance. Dr. Schomer's credentials as the former Director of the Standards Division of the Acoustical Society of America, and his use of four independent sources in deriving his recommendation, give considerable weight to his recommendation. The major concern I have with that approach is that verification is required to show that a 24-hour metric can sufficiently protect sleep during nighttime hours. Wind companies typically prefer to use the Leq metric because it is more easily compared to available data, and generally resist accepting levels lower than 45 or 40 dBA as a design goal for its wind projects.

Q: The Charles Mix County zoning commission seemingly has joined Bon Homme County in establishing minimum setback distances. Rather than establishing the highest permissible noise level to protect the health of residents, would it not be simpler to establish the minimum permissible distance?

A: Undoubtedly, distance is the most effective means of avoiding negative health impacts from wind turbine noise. The short distances from the property line, such as the 500 feet or 1.1 times the system height, whichever is greater, and from residences, such as the 2,000 feet or 3.5 times the system height, whichever is greater, that have been agreed to in this Project are entirely inadequate. Such short distances are intended to reduce risks from physical failures such as blade throw, ice throw, or falling towers. They do almost nothing to protect residents from exposure to low-frequency noise and infrasound. Researchers who have offered distance as an index to obviate health effects have typically recommended 2 kilometers, or 1.25 miles, as a minimally safe distance from the nearest turbine. Although that distance will not prevent annoyance and health effects for everyone, I think it is a reasonable compromise aimed at protecting health and well-being. We have to recognize, though, that studies have shown that some residents within several miles of an industrial wind project complain that the noise is disturbing, presumably because infrasound travels great distances and is not easily attenuated. The problem with distance as a predictor is that different residences at the same distance from the turbines will experience different noise emissions, depending on the turbine array, topography, variable wind speeds, and other factors. In the end, the actual level of noise

emissions is the critical variable that needs to be controlled, as distance in itself cannot assure that the noise will not be invasive for residents in the footprint of the wind project.

Q: In your opinion, is there any important information omitted from, neglected, or erroneously stated in the documents you reviewed for the Prevailing Wind Park project?

A: Yes. Similar to Mr. Hessler's observation in his Dakota Range report, I noticed that an important component missing from the Burns & McDonnell Engineering Company's sound study for this Project is a discussion of the annoyance and adverse health impacts of the Project. Like almost all reports commissioned by wind companies, it does not discuss the fact that annoyance can lead to adverse health effects, as established by Berglund et al. (1999); the WHO (2009); Shepherd, Hanning, and Thorne (2012); and Fast et al. (2016). The WHO (2009) has described annoyance as a critical health effect, in that in some people it is associated with stress, sleep disturbance, and interference with daily living. In fact, the Burns & McDonnell report ignores much of the information in the WHO 2009 guidelines, which were revised downward from the 1999 guidelines as a result of new medical research into adverse health symptoms due to noise. Burns & McDonnell describe wind noise as a masker that can "drown out" the sounds created by the turbines. Although this may be true in rare cases, it is typically not true at night when wind speeds are high at the turbine heights and low at ground level. Also, the design goal of 45 dBA (Bon Homme County ordinance), or 43 dBA (Charles Mix County— Pawlowski affavidit) is higher than what most independent researchers consider protective of health.

Q: Did you find any shortcomings in Mr. Howell's study of background sounds?

A: Yes, in several respects. To me, the most surprising point Mr. Howell made is that he reports measured L90 background sound levels as high as 45 dBA, which is unusually high for a rural area. A table showing all measured levels would have revealed the frequency of such occurrences. Instead, he reports only a range of 21.5-45 dBA. He also understates the sound impact of wind turbine noise by comparing it to levels of normal conversational speech. Comparing the noise from wind turbines to speech using an A-weighted scale is misleading because the levels of low-frequency noise and infrasound from turbines is substantially greater than for speech, as speech energy begins to drop off precipitously at about 150 Hz and below, and the levels of turbine noise continue to rise below that frequency. Using A-weighting

attenuates low frequencies below 1000 Hz, and effectively filters out infrasound, leading to a gross underestimate of infrasonic energy. Also, related to the fact that Bon Homme County does not specify how sound measurements should be performed, Mr. Howell does not indicate whether the design goal is met by measurements over a specified time period. They could be taken over hours, minutes, or days, and could cover the daytime hours, nighttime hours, or a full 24-hour day. Again, it is essential to limit sound levels to those that fully protect residents' sleep, as sleep is a major determinant of good health.

Q: Based on your professional experience and expertise, what restrictions should be placed on the Project to ensure that it will not substantially impair the health of those living around it?

A: As a general rule, no wind turbine should be located closer than 1.25 miles from the property line of any residence. This distance should preferably be applied to all residences, both participating and non-participating. If placed closer to participating residences than 1.25 miles, those residents should be adequately informed, in writing, of the potential for high annoyance and health risks. With regard to permissible noise levels, the WHO recommendation of 40 dBA Leq(night,outside) should not be exceeded at any residence, particularly at non-participating households. To provide adequate protection from sleep disturbance, nighttime noise levels should be limited to 40 dB LAmax. A metric of dB LA10(night, outside), the noise level exceeded 10% during nighttime hours and measured at the façade of the residence, may be a reasonable substitute for LAmax if considered by acoustical experts to be easier to apply for the purpose of compliance.

Q: Does this conclude your testimony?

A: Yes.

The foregoing written testimony is to be presented to the South Dakota Public Utilities Commission for SD PUC Docket EL 18-026.

Dated this 6th day of September 2018.



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Curriculum Vitae
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EDUCATIONAL BACKGROUND

A.A. – 1963 Gardner-Webb College (now Gardner-Webb University)
Boiling Springs, NC USA

B.A. – 1965 Wake Forest College (now Wake Forest University)
Winston-Salem, NC USA
Major: Psychology

M.S. – 1967 Vanderbilt University
Nashville, TN USA
Major: Audiology and Speech Pathology
Master's Thesis: An investigation of the speech discrimination ability of elderly adults
Thesis Advisor: Freeman McConnell, Ph.D.

Ph.D. – 1972 Northwestern University
Evanston, IL USA
Major: Audiology
Doctoral Dissertation: Forward masking under homophasic, antiphasic and other listening conditions
Advisor: Raymond Carhart, Ph.D.

PROFESSIONAL BACKGROUND

TRAINEE AND STUDENT POSITIONS

Audiology Trainee, Veterans Administration, Winston-Salem, NC, June-August 1965
Clinical Audiologist and Supervisor, Bill Wilkerson Hearing and Speech Center, Nashville, TN, February 1967-June 1968

Experience in salaried, part-time positions as ASHA-certified audiologist while a doctoral student/candidate; work settings included Chicago Hearing Society, Veterans

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Int. I-2(a)

Administration Research Hospital, Schwab Rehabilitation Hospital, and ENT practice of Dr. George Sisson, Chicago, IL, Fall 1968-Summer 1971

PROFESSIONAL POSITIONS

Assistant Professor of Audiology, University of Mississippi, Oxford, MS, September 1971-July 1973
Assistant Professor, Memphis State University (now University of Memphis), Memphis, TN, August 1973-April 1975
Research Associate, Biocommunications Laboratory, Department of Hearing and Speech Sciences, University of Maryland, College Park, MD, May 1975-June 1980
Project Director, American Speech-Language-Hearing Association, Rockville, MD, July 1980-June 1981
Director, Research Division, American Speech-Language-Hearing Association, Rockville, MD, July 1980-July 1984
Associate Professor and Chief, Audiology Section, Department of Otolaryngology-Head and Neck Surgery, Indiana University School of Medicine, Indianapolis, IN, July 1984-October 1987
Senior Research Audiologist, Audio-Diagnostics Division, Nicolet Instrument Corporation, Madison, WI, October 1987-December 1989
Associate Professor (tenured), Department of Audiology and Speech Sciences, Michigan State University, East Lansing, MI, January 1990-August 1994
Professor, Chair, and Clinic Director, Department of Audiology and Speech Sciences, Michigan State University, East Lansing, MI, August 1994-May 2000
Professor, Department of Audiology and Speech Sciences (renamed Department of Communicative Sciences and Disorders), Michigan State University, East Lansing, MI, June 2000-May 2011
Professor Emeritus, Department of Communicative Sciences and Disorders), Michigan State University, East Lansing, MI, May 2011-present

ACADEMIC COURSES TAUGHT (in alphabetical order)

Anatomy and Physiology of Hearing
Anatomy and Physiology of Speech and Hearing Mechanisms
Audiological Assessment and Intervention/Rehabilitation
Aural Rehabilitation
Clinical Audiometry/Hearing Assessment/Evaluation Procedures in Audiology (course and lab instruction)
Differential Diagnostic Audiology
Evaluation Procedures in Audiology (also with associated lab)
Hearing Aids/Hearing Amplification and Rehabilitation/Hearing Amplification I & II
Hearing Disorders
Industrial Audiology/Hearing Conservation
Introduction to Audiology
Introduction to Speech and Hearing

Medical Aspects of Audiology
Microcomputer Applications in Audiology and Speech Sciences
Pediatric Audiology/Special Populations in Audiology
Professional Ethics in Communicative Sciences and Disorders
Psychoacoustics
Research Design and Analysis/Research Methods/Research Methods in Communicative Sciences and Disorders
Seminar for Honors Undergraduates: Investigating Hearing Health Risks in the MP3 Generation

PUBLICATIONS

REFEREED ARTICLES

Allen, J. et al. (Vanderbilt Hereditary Deafness Study Group) (1968). Dominantly inherited low-frequency hearing loss. *Archives of Otolaryngology*, *88*, 242-250.
Punch, J.L., & McConnell, F. (1969). The speech discrimination function of elderly adults. *Journal of Auditory Research*, *9*, 159-166.
Wilson, R.H., & Punch, J.L. (1971). Masked spondee thresholds: Variable duty cycle and mask intensity. *Journal of Auditory Research*, *11*, 270-275.
Punch, J., & Carhart, R. (1973). Influence of interaural phase on forward masking. *Journal of the Acoustical Society of America*, *54*, 897-904.
Punch, J.L., & Lawrence, W.F. (1977). Decibel notation with correlated and uncorrelated signals. *Journal of the American Audiology Society*, *3*, 71-79.
Punch, J.L. (1978). Quality judgments of hearing aid-processed speech and music by normal and otopathologic listeners. *Journal of the American Audiology Society*, *3*, 179-188.
Punch, J.L., Lawrence, W.F., & Causey, G.D. (1978). Measurement of attack-release times in compression hearing aids. *Journal of Speech and Hearing Research*, *21*, 338-349.
Punch, J.L. (1978). Masking of spondees by interrupted noise in hearing-impaired listeners. *Journal of the American Audiology Society*, *3*, 245-252.
Punch, J.L., & Howard, M.T. (1978). Listener-assessed intelligibility of hearing aid-processed speech. *Journal of the American Auditory Society*, *4*, 69-76.
Punch, J.L., & Beck, E.L. (1980). Low-frequency response of hearing aids and judgments of aided speech quality. *Journal of Speech and Hearing Disorders*, *45*, 325-335.
Punch, J.L., Montgomery, A.A., Schwartz, D.M., Walden, B.E., Prosek, R.A., & Howard, M.T. (1980). Multidimensional scaling of quality judgments of speech signals processed by hearing aids. *Journal of the Acoustical Society of America*, *68*, 458-466.
Punch, J.L., & Parker, C.A. (1981). Pairwise listener preferences in hearing aid evaluation. *Journal of Speech and Hearing Research*, *24*, 366-374.
Montgomery, A.A., Schwartz, D.M., & Punch, J.L. (1982). Tournament strategies in hearing aid selection, *Journal of Speech and Hearing Disorders*, *47*, 363-372.
Punch, J.L., & Howard, M.T. (1985). Spondee recognition threshold as a function of set size. *Journal of Speech and Hearing Disorders*, *50*, 120-125.
Punch, J.L., & Beck, L.B. (1986). Relative effects of low-frequency amplification on syllable recognition and speech quality. *Ear and Hearing*, *7*, 57-62.

Miyamoto, R.T., McConkey, A.J., Myres, W., Pope, M., & Punch, J.L. (1986). Long-term intracochlear implantation in man. *Otolaryngology-Head and Neck Surgery, 95,* 63-70.

Pope, M.L., Miyamoto, R.T., Myres, W.A., McConkey, A.J., & Punch, J.L. (1986). Cochlear implant candidate selection. *Ear and Hearing, 7,* 71-73.

Punch, J.L., Robbins, A.M., Myres, W., Pope, M.L., & Miyamoto, R.T. (1987). Relationships among selected measures of single-channel cochlear implant performance. *Ear and Hearing, 8,* 37-43.

Punch, J.L. (1987). Matching commercial hearing aids to prescriptive gain and maximum output requirements. *Journal of Speech and Hearing Disorders, 52,* 76-83.

Miyamoto, R.T., Myres, W.A., Wagner, M.L., & Punch, J.L. (1987). Vibrotactile devices as sensory aids for the deaf. *Otolaryngology-Head and Neck Surgery, 97,* 57-63.

Hecox, K.E., & Punch, J.L. (1988). The impact of digital technology on the selection and fitting of hearing aids. *American Journal of Otology* (Supplement), *9*. 77-85.

Punch, J.L. (1988). CROS revisited. *Asha*, 35-37.

Punch, J., Chi, C., & Allan, J. (1990). Signal averaging in real ear probe tube measurements. *Ear and Hearing, 11,* 327-331.

Punch, J.L., Jenison, R.L., Allan, J., & Durrant, J.D. (1991). Evaluation of three strategies for fitting hearing aids binaurally. *Ear and Hearing, 12,* 205-215.

Punch, J.L., & Robb, R. (1992). Prescriptive hearing aid fitting by parameter adjustment and selection. *Journal of the American Academy of Audiology, 3,* 94-100.

Punch, J., & Rakerd, B. (1993). Loudness matching of signals spectrally shaped by a simulated hearing aid. *Journal of Speech and Hearing Research, 36,* 357-364.

Punch, J.L., & Jarrett, A.M. (1994). Hearing aid licensing statutes and the audiologist. *American Journal of Audiology, 3,* 43-54.

Punch, J.L., Robb, R., & Shovels, A.H. (1994). Aided listener preferences in laboratory versus real-world environments. *Ear and Hearing, 15,* 50-61.

Punch, J.L, Shovels, A.H., Dickinson, W.W., Butcher, J., & Snead, C. (1995). Target-matched insertion gain derived from three different hearing aid selection procedures. *Journal of the American Academy of Audiology, 6,* 425-432.

Punch, J.L., Robinson, D.O., & Katt, D.F. (1996). Development of a hearing performance standard for law enforcement officers. *Journal of the American Academy of Audiology, 7,* 113-119.

Arsenault, M.D., & Punch, J.L. (1999). Nonsense-syllable recognition in noise using monaural and binaural listening strategies. *Journal of the Acoustical Society of America, 105,* 1821-1830.

Rakerd, B., Punch, J., Hooks, W., Amlani, A., & VandeVelde, T.J. (1999). Loudness discrimination of speech signals spectrally shaped by a simulated hearing aid. *Journal of Speech, Language, and Hearing Research, 42,* 1285-1294.

Punch, J.L., Rakerd, B., & Amlani, A. (2001). Paired-comparison hearing aid preferences: Evaluation of an unforced-choice paradigm. *Journal of the American Academy of Audiology, 12,* 190-201.

Punch, J.L. (2001). Contemporary issues in hearing aid selection and fitting. *Speech and Hearing Review, 2,* 21-50 (Chinese version); 166-191 (English version) (by invitation).

Punch, J. (2003). A universal hearing aid: Recommended technologic and functional features. *Iranian Audiology, 2(1),* 24-31. (Invited paper).

Punch, J., Joseph, A., & Rakerd, B. (2004). Most comfortable and uncomfortable loudness levels: Six decades of research. *American Journal of Audiology*, *13*, 144-157.
Punch, J., Rakerd, B., & Joseph, A. (2004). Effects of test order on most comfortable and uncomfortable loudness levels for speech. *American Journal of Audiology*, *13*, 158-163.
Holcomb, S.S., & Punch, J. L. (2006). Multimedia Hearing Handicap Inventory: Test-retest reliability and clinical utility. *American Journal of Audiology*, *15*, 3-13.
Amlani, A., Rakerd, B., & Punch, J. (2006). Speech-clarity judgments of hearing-aid-processed speech in noise: Differing polar patterns and acoustic environments, *International Journal of Audiology*, 45, 319-330.
Joseph, A., Punch, J., Stephenson, M., Paneth, N., Wolfe, E., & Murphy, W. (2007). The effects of training format on earplug performance. *International Journal of Audiology*, 46, 609-618.
Callaway, S.L., & Punch, J.L. (2008). An electroacoustic analysis of over-the-counter hearing aids, *American Journal of Audiology*, *17*, 14-24.
Punch, J., Elfenbein, J., & James, R. (2011). Targeting hearing health messages for users of personal listening devices. *American Journal of Audiology*, *20*, 69-82.
Punch, J. & James, R. R. (2016). Wind turbine noise and human health: a four-decade history of evidence that wind turbines pose risks. *Hearing Health & Technology Matters*, http://hearinghealthmatters.org/journalresearchposters/files/2016/09/16-10-21-Wind-Turbine-Noise-Post-Publication-Manuscript-HHTM-Punch-James.pdf.

NON-REFEREED ARTICLES

Causey, G.D., Punch, J.L., Schweitzer, H.C., & Beck, L.B. (Spring 1978). The clinical and acoustic parameters of hearing aid effectiveness. *Bulletin of Prosthetics Research*.
Causey, G.D., Punch, J.L., Schweitzer, H.C., & Beck, L.B. (Spring 1979). The clinical and acoustic parameters of hearing aid effectiveness. *Bulletin of Prosthetics Research*.
Causey, G.D., Punch, J.L., & Schweitzer, H.C. (Fall 1979). The development of improved techniques for the analysis of hearing aid performance. *Bulletin of Prosthetics Research*.
Causey, G.D., & Punch, J.L. (Spring 1980). The clinical and acoustic parameters of hearing aid effectiveness. *Bulletin of Prosthetics Research*.
Punch, J.L. (1980). Self-study of profession's service and training needs in the 1980's. *Asha*, 22, 849-850.
Punch, J.L. (1981). Subjective approaches to hearing aid evaluation. *Hearing Instruments, 32*, 12-14, 65 (Invited paper).
Rees, N.S., Punch, J.L., & Snope, T.L. (1981). Report of Advisory Committee for the self-study project. *Asha*, *23*, 899-902.
Punch, J.L., & Gelatt, J.P. (1982). Federal funding agencies and speech-language-hearing. *Asha*, *24*, 325-331.
Punch, J. (1983). Characteristics of ASHA members. *Asha*, *25*, 31.
Punch, J. (1983). The prevalence of hearing impairment. *Asha*, *25*, 27.
Punch, J. (1983). The geographic distribution of speech-language-hearing personnel. *Asha, 25*, 31.
Punch, J. (1983). Sociodemographic and health characteristics of the hearing-impaired population. *Asha*, *25*, 15.

Punch, J. (1983). Occupational mobility of speech-language pathologists and audiologists: Part I. *Asha*, *25*, 31.
Punch, J.L., & Fein, D.J. (1984). Profile of educational programs in speech-language pathology and audiology. *Asha*, *26*, 43-48.
Karr, S., & Punch, J. (1984). PL 94-142 state child counts. *Asha*, *26*, 33.
Punch, J. (1984). Occupational mobility of speech-language pathologists and audiologists: Part II. *Asha*, *26*, 29.
Punch, J.L. (1984). Salaries in the speech-language pathology and audiology profession. *Asha*, *26*, 41-46.
Mansour, S., & Punch, J. (1984). Research activity among ASHA members. *Asha*, *26*, 41.
Miyamoto, R.T., Myres, W.A., & Punch, J.L. (1987). Tactile aids in the evaluation procedure for cochlear implant candidacy. *Hearing Instruments*, *38*, 33, 36-37.
Miyamoto, R.T., Myres, W.A., Carotta, C.C., Robbins, A.M., Pope, M.L., Punch, J.L., & Steck, J. (November 1987). Cochlear implants in children. *Insights in Otolaryngology*, 2.
Williamson, M., & Punch, J. (1990). Speech enhancement in digital hearing aids. *Seminars in Hearing*, *11*, 68-78.
Punch, J., Chi, C., & Patterson, J. (1990). A recommended protocol for prescriptive use of target gain rules. *Hearing Instruments*, *41*, 12, 14, 16, 18-19.
Punch, J., Birman, M., & Balmer, W. (1990). Comparative evaluation of substitution and equivalent substitution methods of hearing aid analysis. *The Hearing Journal*, *43*, 28-29, 32-33.
Punch, J.L., & Weinstein, B.E. (1996). The Hearing Handicap Inventory: Introducing a multimedia version. *The Hearing Journal*, *49(10)*, 35-36, 38-40, 44-45.
Punch, J.L. (2001). Technologic and functional features of hearing aids: What are their relative costs? *The Hearing Journal*, *54(*6*)*, 32, 34, 36-38, 42, 44.
Punch, J. (2001). Committee Report: A low-cost hearing aid for developing countries. *Hearing International*, *10(3)*, 4-5.
Punch, J. (2002). The differences between analog and digital hearing aids. *Hearing International*, *10(4)*, 4-5 (Part I); *Hearing International*, 11(1), 8 (Part II).
Punch, J. (2002). Technology Note: Linear vs nonlinear hearing aids. *Hearing International*, *11(2)*, 8.
Punch, J. (2002). Technology Note: Output-limiting vs wide dynamic range compression. *Hearing International*, *11(3)*, 8.
Punch, J. (2002). Technology Note: Acoustic modifications in hearing aids. *Hearing International*, *11(4)*, 7.
Punch, J. (2002). Viewpoint: The virtues of virtual learning. *Audiology Today*, *14(5)*, 18.
Punch, J. (2003). Survey of hearing aid manufacturers. *Hearing International*, *12(1)*, 7-8.
Punch, J. (2003). Technology Note: Hearing aids and the audibility index. *Hearing International*, *12(2)*, 8-9.
Punch, J. (2003). Technology Note: Assistive listening devices. *Hearing International*, *12(3)*, 7-8.
Punch, J. (2004). Technology Note: Understanding hearing aid standards organizations. *Audiology Today*, *16(4)*, 15.
Punch, J. (2005). Providing high-quality, low-cost hearing aids for developing countries. *Hearing International*, *14(3)*, 5.

Punch, J. (2006). Providing high-quality, low-cost hearing aids for developing countries: A status report 2. *Hearing International, 14(4)*, 4-5.

Punch, J., James, R., & Pabst, D. (2010). Wind-turbine noise: What audiologists should know. *Audiology Today, 22(4)*, 20-31.

Punch, J, & James, R. (2014). Adverse health effects of noise from industrial wind turbines. HearingHealthMatters.org, Three-part series in Hearing Health & Technology Matters. Part 1: http://hearinghealthmatters.org/hearingviews/2014/wind-turbine-health-problems-noise/, Part 2: http://hearinghealthmatters.org/hearingviews/2014/wind-turbine-noise-evidence-health-problems/, Part 3: http://hearinghealthmatters.org/hearingviews/2014/infrasound-wind-turbine-hearing-health-effects/.

MONGRAPHS

Amlani, A.M., Punch, J.L., & Ching, Y.C. (2002). Methods and applications of the audibility index in hearing aid selection and fitting. *Trends in Amplification, 6*, 81-129.

BOOK CHAPTERS

Punch, J. (2004). High-quality, low-cost hearing aids. In *Hearing Impairment: An Invisible Disability*. Suzuki, J, Kobayashi, T, & Koga, K. (Eds). New York: Springer-Verlag.

Punch, J. (2004). Hearing International project on high-quality, low-cost hearing aids. In *Hearing Impairment: An Invisible Disability*. Suzuki, J, Kobayashi, T, & Koga, K. (Eds). New York: Springer-Verlag.

PRESENTATIONS

INVITED PRESENTATIONS

INTERNATIONAL LEVEL

Punch, J.L. (March 31, 2001). An analysis of hearing aid costs. Invited presentation at conference of Committee on Management and Rehabilitation of Hearing Loss, Hearing International, Singapore.

Punch, J (January 31, 2002). Challenges of developing and distributing a low-cost hearing aid. Invited paper, written by J. Punch and presented by Jun-Ichi Suzuki (J.I. Suzuki & J. Punch: View from the HI Rehabilitation Committee), Hearing International, Pattaya, Thailand.

NATIONAL LEVEL

Punch, J. (December 1-2, 1976). Elements of hearing aid performance and aural rehabilitation. Invited workshop participant, Little Rock, AR.

Punch, J. (March 17-19, 1983). Hearing aid selection: Listener judgments of speech quality and intelligibility. Invited participant at Three Rivers Conference on Communicative Disorders, Pittsburgh, PA.

Punch, J. (April 15-16, 1988). Introduction to digital signal processing concepts and terminology, and Summary: Project Phoenix approach in developing and evaluating the performance of a digital hearing aid. Invited speaker at meeting of Iowa Hearing Aid Society, Des Moines, IA.

Punch, J. (October 1, 1988). Recent advances in digital hearing aids. Invited speaker at program sponsored by George Washington University Medical Center and George Washington University, Washington, DC.

Punch, J. (May 20, 1989). Comparing and contrasting digital hearing aid systems. Invited participant in panel discussion at meeting of Academy of Dispensing Audiologists, Phoenix, AZ.

Punch, J. (June 16, 1989). Digital hearing aids. Invited speaker at Amplification '89 Conference, Central Michigan University, Mt. Pleasant, MI.

Punch, J. (October 26-27, 1990). How can digital hearing aids benefit the hearing impaired? Invited presentation, Iowa Hearing Aid Society, Des Moines, IA.

Punch, J. (March 12-13, 1993). DSP-based hearing aid fitting procedures. Invited presentation at Technology Summit: Sound Advice on Amplification, Starkey Laboratories, Brookland Park, MN.

Bentler, R.A., Fortune, T., Humes, L.E., McCarthy, P, Punch, J., & Van Vliet, D. (November 1994). Hearing aid fittings: A time for change. Invited miniseminar presentation to meeting of American Speech-Language-Hearing Association, New Orleans, LA.

Punch, J. (March 22-25, 1995). A hearing performance standard for police officers. Invited panel presentation at Hearing Conservation Conference III/XX, as part of Forum “The Americans with Disabilities Act: Recruiting, Retaining, and Protecting Workers with Hearing Impairment,” Cincinnati, OH.

Punch, J. (December 7, 2002). Contemporary issues in hearing aid fittings. Invited presention to Palm Springs Hearing Seminars, Palm Springs, CA.

REGIONAL/STATE/LOCAL LEVEL

Punch, J. (April 26, 1980). Hearing aid evaluation: Development of alternative strategies. Invited participant at meeting of Maryland Speech-Language-Hearing Association, Annapolis, MD.

Punch, J. (January 22, 1987). Cochlear implants: Case studies. Invited speaker at Indiana University Faculty Colloquium, Bloomington, IN.

Punch, J. (April 14, 1988). Digital hearing aids. Invited speaker at meeting of Indiana Speech and Hearing Association, Ft. Wayne, IN.

Punch, J. (April 29, 1988). Development and evaluation of a digital hearing aid. Invited speaker at meeting of Speech and Hearing Association of Alabama, Orange Beach.

Punch, J. (July 21, 1988). Signal processing and digital programmable hearing aids. Invited speaker at meeting of Texas Speech and Hearing Association, Vail, CO.

Punch, J. (September 7, 1988). Digital hearing aids. Invited speaker at meeting of audiology staff of Jewish Hospital and Washington University School of Medicine, St. Louis, MO.

Punch, J. (September 23, 1988). Digital signal processing. Invited speaker at Cleveland Hearing and Speech Center, Cleveland, Ohio.

Punch, J. (November 14, 1988). Use of real ear data in hearing aid fitting. Invited speaker at Real Ear Workshop for Aurora Users, Madison, WI.

Punch, J. (March 10, 1989). Integrating digital hearing aids into clinical practice. Invited speaker at meeting of Kentucky Speech and Hearing Association, Lexington, KY.

Punch, J. (March 31, 1989). Digital hearing aids. Invited speaker at meeting of Texas Speech and Hearing Association, El Paso, TX.

Punch, J. (October 26-27, 1990). Software approaches to prescriptive hearing aid fitting. Invited presentation, Iowa Hearing Aid Society, Des Moines, IA.

Punch, J.L. (June 10-12, 1991). Sound, psychophysics, and audition, and federal and state regulations. Two CAOHC training presentations, Buick-Oldsmobile-Cadillac Plant, Lansing, MI.

Punch, J. (September 1991). CCC examination review lectures (2), Sponsored by Michigan Speech-Language Hearing Association, Michigan State University.

Punch, J. (March 12, 1992). Characteristics of well-fitted hearing aids, recent developments in hearing aids, and hearing aid troubleshooting. Invited presentation to Jackson, MI, chapter of Self-Help for the Hard-of-Hearing (SHHH).

Punch, J.L. (May 2, 1993). Hearing loss and hearing aids. Invited presentation to Michigan Department of Social Services, Lansing, MI.

Punch, J.L. (July 24, 1993). Roundtable facilitator at Committee on Institutional Cooperation (CIC) Summer Research Opportunities Program (SROP) Conference, Michigan State University.

Punch, J. (September 18, 1993). CCC examination review lecture, Sponsored by Michigan Speech-Language-Hearing Association, Michigan State University.

Punch, J. (August 8, 1994). Current audiological research in the U.S. Invited presentation to international visitors (London) to Michigan State University.

Punch, J. (September 16, 1995). CCC examination review lecture, Sponsored by Michigan Speech-Language-Hearing Association, Michigan State University.

Punch, J. (March 19, 1999). Student forum. Presented at meeting of Michigan Speech-Language-Hearing Association, Troy, MI.

Punch, J. (June 13, 2000). What about my hearing? Invited presentation at meeting of University Club members, Lunch and Learn Series, Michigan State University, East Lansing, MI.

Punch, J. (October 23-24, 2000). Current practices in hearing aid selection and fitting, Invited presentation at meeting of Hearing International, East Lansing, MI.

Punch, J. (October 24, 2001). Hearing loss and hearing aids. Invited presentation at Valley Court Community Center, East Lansing, MI.

Punch, J. (September 17, 2002). Making the most of your hearing aids. Invited presentation at Foster Community Center. Sponsored by Self Help for the Hard of Hearing, Lansing, MI.

Punch, J. (September 9, 2003). What did you say? Invited presentation at meeting of University Club members, Lunch and Learn Series, Michigan State University, East Lansing, MI.

Punch, J. (March 13, 2004). Advances in hearing aids. Invited presentation at meeting of Ohio Speech-Language-Hearing Association, Columbus, OH.

Punch, J. (July 1, 2004). Hearing loss and hearing aids. Invited presentation at Hanna Community Center, East Lansing, MI.

Punch, J. (October 10, 2006). Hearing conservation. Invited presentation at Department of Music, Michigan State University, East Lansing, MI.

Punch, J. (June 8, 2007). Technological features of modern hearing aids. Invited presentation at 2007 Golden Grads Alumni Breakfast, Michigan State University, East Lansing, MI.

Punch, J. (August 22, 2008). Hearing, hearing loss, and the audiogram. Invited presentation at Michigan Association of Disability Examiners, Lansing Community College West Campus, Lansing, MI.

Punch, J. (October 21, 2008). Hearing Loss and Over-the-Counter Hearing Aids. Invited presentation at Michigan State University Colloquy, University Club, East Lansing, MI.

Punch, J. (November 10, 2008). Hearing Loss and Hearing Aids. Invited presentation at Foster Community Center, Sponsored by Lansing-Area Chapter of Hearing Loss Association of America (HLAA), Lansing, MI.

Punch, J. (March 15, 2010). Invited presentation on variety of personal and professional topics, Sponsored by American Sign Language (ASL) class, Lansing Community College, Arts and Sciences Building, Lansing, MI.

Punch, J. (October 21, 2010). Hearing protection issues. Invited presentation at meeting of Michigan Agricultural Aviation Association, Lansing, MI.

Punch, J. (March 10, 2011). Wind Turbine Information and Issues Forum. Invited presentation at West Shore Community College, Ludington, MI (Mason County), hosted by Great Lakes Renewable Energy Association (GLREA), A Few Friends for the Environment of the World (AFFEW), and West Shore Community College.

Punch, J. L. (June 14, 2011). Does wind turbine noise cause adverse health effects? Invited presentation to Zoning Commission, Riga Township, Palmyra, MI (Lenawee County).

Punch, J. L. (April 25, 2017). Wind Turbines: What's the Noise All About? Invited lecture to Michigan State University class, ECE 491: Special Topics.

SUBMITTED PRESENTATIONS

INTERNATIONAL LEVEL

Punch, J.L. (November 1982). Computer-based information services for ASHA members. The computer and speech and hearing services: Administrative applications. Miniseminar presented at meeting of American Speech-Language-Hearing Association, Toronto, Canada.

Punch, J.L. (June 24-27, 1993). Aided listener preferences in laboratory vs. real-world environments. Paper presented at International Hearing Aid Conference II: Signal Processing, Fitting, and Efficacy, University of Iowa, IA.

NATIONAL LEVEL

Stream, R.W., et al. (1968). Hereditary low frequency hearing loss. Paper presented at meeting of American Speech and Hearing Association, Denver, CO.

Punch, J.L., & D'Agostino, A. (1972). Influence of word number on spondee threshold in normal listeners. Paper presented at meeting of American Speech and Hearing Association, San Francisco, CA.

Punch, J.L., & Rushing, C. (November 1974). Effect of poor-ear occlusion on sound field speech discrimination scores. Paper presented at meeting of American Speech and Hearing Association, Las Vegas, NV.

Punch, J.L., & Page, J. (November 1974). Evaluation of the efficiency of three hearing screening measures. Paper presented at meeting of American Speech and Hearing Association, Las Vegas, NV.

Punch, J.L., & Studebaker, G.A. (November 1975). Comparison of hearing aid and hearing aid-processed frequency response in ear canals. Paper presented at meeting of American Speech and Hearing Association, Washington, DC.

Punch, J.L. (1976). Evaluation of the Zwislocki coupler and KEMAR for use in hearing aid processing. Paper presented at 91st meeting of Acoustical Society of America, Washington, DC.

Punch, J.L., & Lawrence, W.F. (November 1976). Procedural considerations in the measurement of attack-release times. Paper presented at meeting of American Speech and Hearing Association, Houston, TX.

Punch, J.L., & Ciechanowski, J.M. (November 1977). Reliability of paired-comparison quality judgments in hearing aid evaluation. Paper presented at meeting of American Speech and Hearing Association, Chicago, IL.

Punch, J.L., & Howard, M.T. (1977). Listener-assessed intelligibility of hearing aid-processed speech. Paper presented at 94th meeting of Acoustical Society of America, Miami Beach, FL.

Punch, J.L., Montgomery, A.A., & Howard, M.T. (November 1978). Relationships between hearing aid electroacoustic characteristics and speech quality judgments. Paper presented at meeting of American Speech and Hearing Association, San Francisco, CA.

Punch, J.L., Talkin, D.T., & Lawrence, W.F. (November 1978). Measurement and analysis of hearing aid impulse response. Paper presented at meeting of American Speech and Hearing Association, San Francisco, CA.

Punch, J.L., & Parker, C.A. (November 1979). Validity of paired-comparison listener preferences in hearing aid evaluation. Paper presented at meeting of American Speech-Language-Hearing Association, Atlanta, GA.

Punch, J.L., & Beck, E.L. (November 1979). Aided speech quality judgments: Effect of varying low-cutoff frequency. Paper presented at meeting of American Speech-Language-Hearing Association, Atlanta, GA.

Howard, M.T., & Punch, J.L. (November 1980). Spondee threshold as a function of word number. Poster presented at meeting of American Speech-Language-Hearing Association, Detroit, MI.

Beck, L.B., Leatherwood, R.W., & Punch, J.L. (November 1980). Aided low-frequency response: Speech quality and speech intelligibility. Paper presented at meeting of American Speech-Language-Hearing Association, Detroit, MI.

Punch, J.L., Levitt, H., Mahaffey, R.B., & Wilson, M.S. (November 1983). Computer technology: The revolution has started without us. Miniseminar presented at meeting of American Speech-Language-Hearing Association, Cincinnati, OH.

Miyamoto, R.T., McConkey, A.J., Myres, W., Pope, M., & Punch, J.L. (May 1985). Long-term intracochlear implantation in man. Paper presented at meeting of American Neurotologic Society, Miami, FL.

Punch, J.L., & Miyamoto, R.T. (November 1985). Supplementary use of hearing aids by cochlear implantees. Poster presented at meeting of American Speech-Language-Hearing Association, Washington, DC.

Punch, J.L., McConkey, A., Myres, W., Pope, M.A., & Miyamoto, R.T. (November 1985). Relationships among selected pre- and post-implant measures. Paper presented at meeting of American Speech-Language-Hearing Association, Washington, DC.

Punch, J.L. (November 1985). Matching commercial hearing aids to prescriptive gain and SSPL requirements. Paper presented at meeting of American Speech-Language-Hearing Association, Washington, DC.

Miyamoto, R.T., & Punch, J. (October 1986). Current issues in cochlear implantation of adults and children. Miniseminar presented at Crossroads Conference on Communicative Disorders. National Student Speech-Language-Hearing Association. Purdue University, West Lafayette, IN.

Punch, J.L., Miyamoto, R.T., & Myres, W.A. (November 1986). Lipreading ability vs. auditory advantage with cochlear implants. Paper presented at meeting of American Speech-Language-Hearing Association, Detroit, MI.

Brown, D.B., Miyamoto, R.T., & Punch, J.L. (November 1986). Intraoperative ABR monitoring during vestibular nerve sections. Paper presented at meeting of American Speech-Language-Hearing Association, Detroit, MI.

Punch, J.L., Stone, R.E., Jr., Horii, Y., & Miyamoto, R.T. (November 1987). Oral-nasal coupling in the speech of cochlear implantees. Poster presented at meeting of American Speech-Language-Hearing Association, New Orleans, LA.

Myres, W.A., Miyamoto, R.T., & Punch, J.L. (November 1987). Auditory performance of a binaural cochlear implant recipient. Paper presented at meeting of American Speech-Language-Hearing Association, New Orleans, LA.

Punch, J., Chi, C., & Patterson, J. (November 1988). Factors affecting individualized target-gain based prescription of hearing aids. Miniseminar presented at meeting of American Speech-Language-Hearing Association, Boston, MA.

Punch, J., Allan, J., Sammeth, C., Palmer, R., Williamson, M., & Hecox, K. (November 1988). High-frequency limiting: Effects on laboratory and real-world performance. Paper presented at meeting of American Speech-Language-Hearing Association, Boston, MA.

Birman, M., Punch, J., & Balmer, W. (November 1988). Equivalent substitution method vs. a commonly used electroacoustic method. Paper presented at meeting of American Speech-Language-Hearing Association, Boston, MA.

Allan, J., Punch, J., & Chi, C. (November 1988). Signal averaging in real-ear measurements. Paper presented at meeting of American Speech-Language-Hearing Association, Boston, MA.

Chi, C., Balmer, W., Punch, J., & Williamson, M. (November 1988). Analysis of hearing aids: Complex signal vs. pure tone. Paper presented at meeting of American Speech-Language-Hearing Association, Boston, MA.

Allan, J., Punch, J., Lasky, R., & Chertoff, K. (November 1989). Effects of stimulus condition on preferred signal-processing characteristics. Paper presented at meeting of American Speech-Language-Hearing Association, St. Louis, MO.

Durrant, J., Punch, J., & Allan, J. (November 1989). Comparison of digital noise reduction algorithm vs. binaural processing. Paper presented at meeting of American Speech-Language-Hearing Association, St. Louis, MO.

Punch, J., Allan, J., & Jenison, R. (November 1989). Comparative evaluation of three binaural hearing aid fitting strategies. Paper presented at meeting of American Speech-Language-Hearing Association, St. Louis, MO.

Punch, J., Levitt, H., & Studebaker, G. (November 1989). Paired-comparison judgments in hearing aid evaluation: A status report. Miniseminar presented at meeting of American Speech-Language-Hearing Association, St. Louis, MO.

Benedict, E., Punch, J., Lasky, R., & Chi, C. (November 1989). The sliding scale: A preference based hearing aid fitting procedure. Paper presented at meeting of American Speech-Language-Hearing Association, St. Louis, MO.

Codd, M.B., Sammeth, C.A., Ochs, M.T., & Punch, J.L. (April 25-28, 1991). Effects of reduced low-frequency amplification on speech recognition thresholds and perceived sound quality in noise. Poster presented at meeting of American Academy of Audiology, Denver, CO.

Punch, J.L. (June 21-23, 1991). Prescriptive hearing aid fitting by parameter adjustment and selection. Paper presented at International Hearing Aid Conference: Signal Processing, Fitting, and Efficacy, University of Iowa, IA.

Punch, J., & Rakerd, B. (November 1991). Loudness matching of signals in a simulated multimemory hearing aid. Paper presented at meeting of American Speech-Language-Hearing Association, Atlanta, GA.

Punch, J.L., Robinson, D. O., & Katt, D.F. (November 1992). Hearing performance standard for law enforcement officers: Aided and unaided. Poster presented at meeting of American Speech-Language-Hearing Association, San Antonio, TX.

Punch, J.L., & Robb, R. (November 1992). A DSP-based approach to clinical hearing aid research. Poster presented at meeting of American Speech-Language-Hearing Association, San Antonio, TX.

Punch, J.L., & Hooth, A.J. (November 1992). Hearing aid fitting: A validation study. Poster presented at meeting of American Speech-Language-Hearing Association, San Antonio, TX.

Punch, J., Congdon, S., Nelson-Wade, E., & O'Connor, T. (November 1992). Comparison of three clinical hearing aid fitting procedures. Poster presented at meeting of American Speech-Language-Hearing Association, San Antonio, TX.

Hamill, T., & Punch, J. (1994). Toward valid hearing aid evaluation procedures. Instructional course presented at meeting of American Academy of Audiology, Richmond, VA.

Patterson, J.P., & Punch, J. (November 1994). Launching multimedia applications: Issues in design and development. Miniseminar presented at meeting of American Speech-Language-Hearing Association, New Orleans, LA.

Punch, J., & Weinstein, B. (November 1994). Hearing handicap inventory for the elderly: A multimedia version. Miniseminar presented at meeting of American Speech-Language-Hearing Association, New Orleans, LA.

Arsenault, M., & Punch, J. (September 11-13, 1995). Speech recognition performance in noise under monaural and binaural listening conditions. Poster presented at First Biennial

Conference, Hearing Aid Research and Development, National Institutes of Health, Bethesda, MD.

Rakerd, B., Punch, J., Hooks, W., & Vander Velde, T. (November 1996). Speech loudness changes associated with a programmable hearing aid. Presented at meeting of American Speech-Language-Hearing Association, Seattle, WA.

Weinstein, B., & Punch, J. (April 2-5, 1998). www.phd.msu.edu/hearing. Presented at meeting of American Academy of Audiology, Los Angeles, CA.

Amlani, A., Punch, J., & Rakerd, B. (March 16-19, 2000). Reliability and transitivity of forced- and unforced-choice paired-comparison preferences. Student Research Forum. Presented at meeting of American Academy of Audiology, Chicago, IL.

Shogren, S, & Punch, J. (March 16-19, 2000). Test-retest reliability of the Multimedia Hearing Handicap Inventory. Student Research Forum. Presented at meeting of American Academy of Audiology, Chicago, IL.

Jarrett, A., & Punch, J. (March 16-19, 2000). Development of a Hearing Risk Inventory for Infants. Student Research Forum. Presented at meeting of American Academy of Audiology, Chicago, IL.

Amlani, A., & Punch, J. (April 4, 2003). A web-based Audibility-Index (AI) calculator. Poster presented at meeting of American Academy of Audiology, San Antonio, TX.

Amlani, A., Punch, J., & Rakerd, B. (April 5, 2003). Polar-pattern preferences in real-world environments. Presented at meeting of American Academy of Audiology, San Antonio, TX.

Joseph, A., Punch. J., & Rakerd, B. (April 5, 2003). Effects of test order & instructions on MCL-S & UCL-S. Poster presented at meeting of American Academy of Audiology, San Antonio, TX.

Punch, J. (April 5, 2003). Hearing standards for hearing-critical occupations. Roundtable presentation at meeting of American Academy of Audiology, San Antonio, TX.

Joseph, A.R., Punch, J.L., Stephenson, M.R., Paneth, N., Murphy, W.J., & Wolfe, E. (November 1, 2005). The effect of training modality on earplug attenuation and fit. Poster presented at meeting of Association of Medical Service Corps Officers of the Navy (AMSCON) and Association of Military Surgeons of the United States (AMSUS), Nashville, TN.

Joseph, A., Stephenson, M. Punch, J., & Murphy, W. (February 17, 2006). The effect of training modality on earplug attenuation. Paper presented at meeting of National Hearing Conservation Association (NHCA), Tampa, FL.

Joseph, A.R., Punch, J.L., Stephenson, M.R., & Murphy, W.J. (February 16-18, 2006). The Sound Attenuation Fit Estimator ($SAFE_{500}$). Poster presented at meeting of National Hearing Conservation Association (NHCA), Tampa, FL.

Atienza, H., Bhagwan, S., Kramer, A., Morris, A., Pabst, D., Warren, A., Williams, N., Punch, J.L., & Elfenbein, J.L. (April 2-5, 2008). Preferred MP3 listening levels: Earphones and environments. Poster presented at meeting of American Academy of Audiology, Charlotte, NC.

Callaway, S.L. & Punch, J.L. (April 2-5, 2008). Prescriptive fitting of over-the-counter hearing aids. Poster presented at meeting of American Academy of Audiology, Charlotte, NC.

Punch, J.L., James, R., & Pabst, D. (November 19-21, 2009). Wind turbines: What you can't hear can hurt you. Paper presented at meeting of American Speech-Language-Hearing Association, New Orleans, LA.

Alkhamra, R.A., Rakerd, B., Punch, J, Zwolan, T., & Elfenbein, J. (July 24 - 29, 2011). Cognitive effort and the perception of speech by adult cochlear implant users: A survey. Poster presented at Conference on Implantable Auditory Prostheses 2011, Pacific Grove, CA.

Rakerd, B., Alkhamra, R., Zwolan, T, Punch, J, & Elfenbein, J. (November 17-19, 2011). Cognitive effort and perception of speech by cochlear implant users. Paper presented at meeting of American Speech-Language-Hearing Association, San Diego, CA.

Schutte, D., Rivard, J., Dykstra Goris, E., Schmidt, S, Punch, J., & Schutte, B. (March 7-10, 2013). Examining hearing as a potential predictor of agitation in persons with dementia: feasibility of a comprehensive hearing assessment in persons with cognitive impairment, Paper presented at meeting of Midwest Nursing Research Society, Chicago, IL.

Hitt, R., Punch, J., & Smith, S.W. (March 28-30, 2013). Effects of hearing impairment on quality of life. Poster presented at D.C. Health Communication Conference, Fairfax, VA.

Rakerd, B., & Punch, J.L. (November 9-11, 2017). Toward a simplified protocol for speech audiometry. Poster presented at meeting of American Speech-Language-Hearing Association, Los Angeles, CA.

Punch, J., & Rakerd, B. (April 18-21, 2018). Simplifying speech audiometry. Poster presented at meeting of American Academy of Audiology, Nashville, TN.

REGIONAL/STATE/LOCAL LEVEL

Miyamoto, R.T., and other members of the Cochlear Implant Team (April 17, 1986). Cochlear implants as sensory aids for deaf children. Session presented at meeting of Indiana Speech and Hearing Association, Nashville, TN.

Robinson, D.O., Punch, J.L., & Katt, D.F. (March 1992). Michigan Law Enforcement Officers Training Council's hearing performance standard: Aided and unaided. Poster presented at meeting of Michigan Speech-Language-Hearing Association, Kalamazoo, MI.

Punch, J.L., & Jarrett, A.M. (March 25-27, 1993). State hearing aid licensing statutes and the audiologist. Poster presented at Meeting of Michigan Speech-Language-Hearing Association, Shanty Creek Resort, Bellaire, MI.

Weinstein, B., & Punch, J. (May 2-5, 1996). A multimedia approach to marketing hearing health care services for older adults. Presented at meeting of New York State Speech-Language-Hearing Association, Albany, NY.

Punch, J., & Weinstein, B. (March 13-15, 1997). Hearing Handicap Inventory: From milk carton to multimedia. Presented at meeting of Michigan Speech-Language-Hearing Association, Kalamazoo, MI.

Artymovich, A., Frost, T, Jeong, M, Lin, F., Patel, N., & Sweet, E. (Mentors: J. L. Elfenbein & J. Punch) (April 13, 2007). Listening to music: University students' device preferences. Poster presented at University Undergraduate Research and Arts Forum, Michigan State University.

Artymovich, A., Frost, T, Jeong, M, Lin, F., Patel, N., & Sweet, E. (Mentors: J. L. Elfenbein & J. Punch) (April 13, 2007). University students' patterns of MP3 player use: Is there hearing health risk? Poster presented at University Undergraduate Research and Arts Forum, Michigan State University.

Atienza, H., Bhagwan, S., Kramer, A., Morris, A., Pabst, D., Warren, A., & Williams, N. (Mentors: J. Punch & J. L. Elfenbein) (March 9, 2007). Output levels of the Apple iPod® for three types of earphones. Poster presented at meeting of Michigan Speech-Language-Hearing Association, Ypsilanti, MI.

Atienza, H., Bhagwan, S., Kramer, A., Morris, A., Pabst, D., Warren, A., & Williams, N. (Mentors: J. Punch & J. L. Elfenbein) (April 13, 2007). Output levels of the Apple iPod® for three types of earphones. Poster presented at University Undergraduate Research and Arts Forum, Michigan State University.

Atienza, H., Bhagwan, S., Kramer, A., Morris, A., Pabst, D., Warren, A., & Williams, N. (Mentors: J. Punch & J. L. Elfenbein) (April 13, 2007). Preferred listening levels of MP3 music with three types of earphones. Poster presented at University Undergraduate Research and Arts Forum, Michigan State University.

Atienza, H., Bhagwan, S., Kramer, A., Morris, A., Pabst, D., Warren, A., & Williams, N. (Mentors: J. Punch & J. L. Elfenbein) (April 20, 2007). Output levels of the Apple iPod® for three types of earphones. Poster presented at Pediatric and Human Development Research Day, Michigan State University.

Atienza, H., Bhagwan, S., Kramer, A., Morris, A., Pabst, D., Warren, A., & Williams, N. (Mentors: J. Punch & J. L. Elfenbein) (April 20, 2007). Preferred listening levels of MP3 music with three types of earphones. Poster presented at Pediatric and Human Development Research Day, Michigan State University.

Sanders, S., Rajasekhar, R, & Viaches, K. (Mentors: J. Punch & S. Smith) (April 2009). Development of a quality-of-life inventory for the hearing impaired. Poster presented at University Undergraduate Research and Arts Forum, Michigan State University.

Punch, J. (February 7, 2014). Wind turbine noise: Can sounds we can't hear hurt us? Lecture presented at Research Colloquium, Department of Communicative Sciences and Disorders, Michigan State University.

WORKS SUBMITTED AND IN PRESS

Punch, J., & Rakerd, B. Evaluation of a simplified protocol for speech audiometry. In press (*American Journal of Audiology*).

Punch, J.L., Hitt, R., & Smith, S.W. Hearing loss and quality of life. Submitted, under review.

MASTER'S THESES DIRECTED

D'Agostino, A.D. (1972). Effect of word number on spondee threshold in normal listeners (Master's thesis, University of Mississippi, 1972).

Guckert, S.A. (1973). The development and evaluation of a filtered speech test for use in hearing screening. (Master's thesis, University of Mississippi, 1973).

Rushing, C. (1973). The effects of single ear occlusion on sound field speech discrimination scores (Master's thesis, University of Mississippi, 1973).

Shogren, S. (1999). Test-retest reliability and clinical utility of the Multimedia Hearing Handicap Inventory (Master's thesis, Michigan State University, 1999).

Callaway, S.L. (2007). How well do over-the-counter hearing aids benefit the hearing impaired? (Master's thesis, Copenhagen University, Denmark, 2007). Thesis co-adviser, Copenhagen University: Niels Reinholt Petersen, Ph.D.

Ph.D. DISSERTATIONS DIRECTED

Amlani, A. (2003). Paired-comparison preferences for polar directivity patterns in different listening environments (Doctoral dissertation, Michigan State University, 2003).

Joseph, A. (2004). Attenuation of passive hearing protection devices as a function of group versus individual training. (Doctoral dissertation, Michigan State University, 2004).

HONORS AND AWARDS

Fellow, American Speech-Language-Hearing Association
American Men and Women of Science
Dedicated Service Award, Michigan Law Enforcement Officers Training Council, January 1993
Editor's Award, American Auditory Society, September 1987. Relative effects of low-frequency amplification on syllable recognition and speech quality, *Ear and Hearing*, 7, 57-62 (1986), with Lucille B. Beck
Who's Who in America
The National Distinguished Service Registry: Speech, Language and Hearing
Student Research Forum Award for submission to meeting of American Academy of Audiology. Sara Shogren & Jerry Punch, Test-retest reliability of the Multimedia Hearing Handicap Inventory, March 17, 2000, Chicago, IL.
Merit Recognition Award for poster presented at University Undergraduate Research and Arts Forum, Michigan State University (Authors: Atienza, H., Bhagwan, S., Kramer, A., Morris, A., Pabst, D., Warren, A., & Williams, N.; Mentors: J. Punch & J. Elfenbein), April 13, 2007. Output levels of the Apple iPod® for three types of earphones.
James Jerger Award for Excellence in Student Research for poster presented at meeting of American Academy of Audiology. Susanna L. Callaway & Jerry L. Punch, Prescriptive fitting of over-the-counter hearing aids, April 2-5, 2008, Charlotte, NC.

PROFESSIONAL MEMBERSHIPS/CREDENTIALS/AFFILIATIONS

INTERNATIONAL ORGANIZATIONS

Member, Editorial Board, *Iranian Audiology*; October 2003-present.

NATIONAL ORGANIZATIONS

Member, American Speech-Language-Hearing Association (ASHA)

Certificate of Clinical Competence (CCC) in Audiology, ASHA
Member, American Auditory Society
Fellow, American Academy of Audiology
Member, Acoustical Society of America, 1976-1991; 2000-present

STATE ORGANIZATIONS

Member, Michigan Speech-Language-Hearing Association (MSHA); early 1990s; currently a non-member
Hearing Aid Dealers License (#17000751A), State of Indiana, May 16, 1985-June 30, 1988
Hearing Aid Dealers License (#3501003452), State of Michigan, November 2001-November 2005
Audiologist License (#1601000461), State of Michigan, May 2007-December 2009

COMMITTEE APPOINTMENTS, CONSULTANTSHIPS, AND RELATED PROFESSIONAL SERVICE ACTIVITIES

INTERNATIONAL LEVEL

Member, Hearing International, September 2000-2007; Member, Committee on Management and Rehabilitation of Hearing Loss, Hearing International, September 2000-2007; Chair, Subcommittee on High-Quality, Low-Cost Hearing Aids
Invited reviewer of promotional materials of Dr. Wahab Owolawi Owolawi, King Saud University, Saudi Arabia, June 2007

NATIONAL LEVEL

Congressional Action Contact (CAC) of ASHA, July 1972-June 1973
ASHA Committee on Audiometric Evaluation, January 1978-June 1980
Chair, ASHA Task Force on Information Services, 1982-83
Ex-officio Member: ASHA Committee on Scientific Affairs, ASHA Committee on Personnel and Service Needs in Communication Disorders, ASHA Committee on Educational Technology, July 1982-July 1984
Consultant to Deafness, Speech and Hearing Publications (DSHP) Board, ASHA, October 1982-July 1984
Chair, ASHA Task Force on Personnel Supply, Demand and Utilization, 1983-1984
Chair, Forum on Allied Health Data, Spring 1983-Spring 1984
Steering Committee, American Speech-Language-Hearing Foundation Conference, "The Personal Computer as a Professional Tool," 1983-1984
Member, ASHA Task Force on Personnel Supply, Demand and Utilization, July 1984-April 1986
Editorial Consultant, *Journal of the Acoustical Society of America*, 1987-1989, June 2014-present
Reviewer of NIH-SBIR research grant proposals, Washington, DC, March 14-15, 1995
Reviewer of House Ear Institute research proposal, March 1997

Editorial Consultant, *Journal of Speech-Language-Hearing Research* (periodic)
Editorial Consultant, *Ear and Hearing* (periodic)
Editorial Consultant, *American Journal of Audiology* (periodic)
Editorial Consultant, *Iranian Audiology* (periodic)
Editorial Consultant, *International Journal of Audiology* (March 2006-present)
Editorial Consultant, *Pediatrics*, February 2013
Editorial Consultant, *International Journal of Environmental Research and Public Health*, June 2014-present

STATE LEVEL

Member, Clinic, Hospital, and Private Practice Ad Hoc Committee, Indiana Speech and Hearing Association, September 1986-October 1987
Member, Program Committee, Indiana Speech and Hearing Association, 1986-1987, 1987-1988
Member, Michigan Speech-Language-Hearing Association, 1991-1997
Member, Audiology Committee, Michigan Speech-Language-Hearing Association, 1991
Vice-President for Membership, Executive Council, Michigan Speech-Language-Hearing Association, January 1991-December 1992
Consultant, Michigan Law Enforcement Officers Training Council re hearing standards, 1991-1992; 2003.
Michigan representative for Membership Recruitment Network, American Academy of Audiology, 1992
Consultant to Department of Veterans Affairs (DVA), Audiology Section, Ann Arbor, Michigan, 1992
Consultant to Michigan Department of Social Services, Hearing and Hearing Aid Reimbursement Program, Lansing, Michigan, 1993-1995
Reviewer, Sertoma Communicative Disorders Scholarship Applications, Michigan Region, Sertoma International, 1994-95, April 1994
Participant in first MSHA-sponsored Legislative Day, May 4, 1994, contacting offices of Senators Debbie Stabenow and Fred Dillingham and Representative Lynn Jondahl
Member, Advisory Board for Occupational Noise-Induced Hearing Loss, State of Michigan, 1995-2010
Member, Audiology Advisory Panel, Michigan Department of Community Services, 1995-1996
Participant, with Nigel Paneth, M.D., in gaining approval of Michigan Chapter of American Academy of Pediatrics to implement resolution that pediatricians refer all children to audiologists for audiological and hearing aid evaluations, 1997
Consultant to Pyle, Rome, Lichten, & Eurenberg, P.C., Attorneys at Law, Boston, MA, re cases of occupational hearing loss in law enforcement officers and firefighters, January 1999-January 2002.
Consultant, SHL, Contractor with Commonwealth of Massachusetts, Evaluation of hearing standards for police officers and firefighters, May 28, 2002
Member and Chair, Wind and Health Technical Work Group, Bureau of Energy Systems, Department of Energy, Labor, and Economic Growth (DELEG), State of Michigan, March 2010-June 2011

UNIVERSITY LEVEL

Participant in CIC Predoctoral Fellows Conference, Michigan State University, November 1993
Participant in Focus Group meeting of Committee on Improving, Evaluating and Rewarding Teaching (CIERT), Michigan State University, December 1993
Member, Ad Hoc Committee on University-Wide Performance Indicators, Michigan State University, May-June 1995
Member, Clinical Services Group, Evaluating Quality Outreach Working Group, Michigan State University, February-May 1999
Faculty Tenure Committee, Michigan State University, 1993-1995
University Committee on Research in Human Subjects (UCRIHS, or Institutional Review Board), Fall Semester 2000, January 2011-September 2017
Member, Ad Hoc Working Group on Institutional Indicators for Evaluating Quality Outreach, Michigan State University, February-May, 1999
Faculty Participant in Freshman Orientation Program, Michigan State University, June 29, 1999
Member (interim), Faculty Council, Michigan State University, January-May 2005
Member (interim proxy), Academic Council, Michigan State University, January-May 2005
Member, University Committee on Academic Policy (UCAP), May 2007-May 2009
Representative, Academic Council (from University Committee on Academic Policy), August 2007-May 2008

COLLEGE/DEPARTMENTAL LEVEL

Departmental committees (Michigan State University): Audiology Faculty Position Search Committee (1993-94); Clinical Affairs Committee (1990-94); Clinical Specialist Position Search Committee (1993); Curriculum Committee (1991-92); Faculty Advisory Committee (1990-91); Graduate Studies and Admissions Committee (1990-2010, periodically serving as Chair); Neuropathology Faculty Position Search Committee (1990-91)
College committees (Michigan State University): Intracollegiate Planning Committee (1992-1994); CAS Technology Advisory Committee (2002-2003)
Revised Departmental Handbook, Fall 2003
College Reappointment, Tenure, and Promotion Committee (2006-2007)
Member, Admissions Committee, Department of Communicative Sciences and Disorders, 2008
Member, Committee to Review Health Communication Program, College of Communication Arts and Sciences, September 2008-January 2009
Member and Chair, Admissions Committee, Department of Communicative Sciences and Disorders, 2009

MEDIA EVENTS

Interview on WJLM-TV, Channel 6, Lansing, Michigan, Better Hearing and Speech Month, May 20, 1997.
Interview on WLAN-TV, Channel 6, Lansing, Michigan, Segment on Hearing Loss, February 4, 1999.

Interview with Broadcast/Photo Division of MSU's Office of University Relations, Dennis Krolik, February 5, 1999.

MSU State News, Study Finds Hearing Loss in Youth, September 15, 2005.

Detroit News, Hearing under Assault, January 3, 2006.

Wall Street Journal, Behind the Music, IPods and Hearing Loss, January 10, 2006.

Newstips provided to Melanie Lynne Trusty (via e-mail), MSU Journalism student, February 6, 2006, concerning Apple's iPod lawsuit.

Interview with MSU Focal Point on Listening with iPods, Jamie Lauren Kandel, February 22, 2006.

Not All Hearing Aids Are Created Equal, article appearing in:

MSU News, http://news.msu.edu/story/5621/, August 13, 2008.

ScienceDaily, http://www.sciencedaily.com/releases/2008/08/080813164634.htm, August 14, 2008.

CHINA ORG.CN, http://www.china.org.cn/health/2008-08/14/content_16223170.htm, August 14, 2008.

Detroit Free Press, http://www.freep.com/apps/pbcs.dll/article?AID=/20080815/NEWS06/808150346/1008/NEWS06, August 15, 2008 (appearing under title, Drugstore Hearing Aids May Do Harm).

Hearing Review Insider, http://www.hearingreview.com/insider/2008-08-28_08.asp; August 28, 2008.

(This article also appeared on numerous additional Web sites and blogs.)

Interview with Naomi Schalit, Executive Director and Senior Reporter, Maine Center for Public Interest Reporting, Hallowell, ME on topic of wind-turbine noise, July 27, 2010.

Interview with Bob Allen, Interlochen Public Radio, Interlochen, Michigan, on topic of health effects of wind-turbine noise, January 14, 2011.

Interview with Mark Bashore, Wind Turbines Pose Health Risks, Current State, WKAR, Michigan State University, February11, 2014.

Interviews on WKAR-AM Radio Call-In Show, Michigan State University:

Topic/Theme	Date
Better Hearing and Speech Month	May 1991
Audiology Topics	September 3, 1992
Audiology Topics	March 31, 1993
Better Hearing and Speech Month	May 17, 1993
Hearing Topics: Children and Adults	July 9, 1993
Hearing Aids	September 27, 1993
Audiology Topics	January 10, 1994
Audiology Topics	March 2, 1994
Better Hearing and Speech Month	May 3, 1994
Hearing Loss and Hearing Aids	July 19, 1994
Auditory Perception Disorders and Hearing Aids	December 29, 1994

Hearing, Hearing Loss, Prevention of Hearing Loss	March 30, 1995
Better Hearing and Speech Month	May 1, 1997
Better Hearing and Speech Month	May 10, 1999
Better Hearing and Speech Month	May 16, 2000

BOOK REVIEWS

Probe Microphone Measurements: Hearing Aid Selection and Assessment (1992), by H. Gustav Mueller, David B. Hawkins, & Jerry L. Northern, San Diego, CA: Singular Publishing Group, Inc. In (1992) *Ear and Hearing*, 13, 467-468.

RESEARCH PROPOSALS, GRANTS, AND CONTRACTS

Faculty Research Grant, "Investigation of Adequacy of Audiometric Calibration in Hearing Conservation Programs," University of Mississippi, 1971-72 (funded).

Faculty Research Grant, "Development and Evaluation of a Filtered Speech Screening Test," University of Mississippi, 1972-73 (funded).

Principal Investigator, Veterans Administration Contract, "The Clinical and Acoustic Parameters of Hearing Aid Effectiveness," University of Maryland, October 1976-June 1980 (funded).

Project Director, "Speech-Language Pathology and Audiology: An Educational Perspective for the Future," American Speech-Language-Hearing Association, July 1980-June 1981 (funded $222,433).

Co-author of, and consultant to, project funded by Department of Education, "Leadership Training in Computer Technology: A Tri-Level Multidisciplinary Program for Special Educators and Speech-Language Pathologists and Audiologists," ASHA National Office, January 1983-June 1984 (funded $122,875).

Co-author of, and consultant to, project funded by Department of Education, "Validation of a Model to Evaluate Microcomputer Software for Communicatively Handicapped Students," ASHA National Office, December 1983-June 1984 (funded $154,489).

Primary writer of a grant proposal funded by NINCDS-NIH, "Comparison of Sensory Aids in Deaf Children," Indiana University School of Medicine, January 1987 (funded $348,869).

Principal Investigator of project funded by NIDCD-NIH, "A DSP-Based Approach to the Study of Hearing Aid Fitting," Michigan State University, January 1991-December 1992 (funded $63,946).

Project Director, "Development of a Multimedia-Based Version of the Hearing Handicap Inventory for the Elderly," All-University Outreach Grant, Michigan State University, July 1992-December 1993 (funded $14,947).

Project Director, “Accommodation of the Communicative Needs of Hearing-Impaired Students and Faculty at Michigan State University,” Project funded (for support of Graduate Assistant) by Office of the Provost, Michigan State University, January 1995-August 1995 (funded).

Member, Project Team, Strategic Partnership Grant to Fund Hearing Research Center, Michigan State University, July 1, 1996-June 1997 (funded ~$210,000).

Lead Investigator (with Brad Rakerd and Amyn Amlani), "Intelligibility Testing of Digitized Sound Files Provided by IC Tech," Contract with IC Tech, Inc., Okemos, MI, February 6-May 31, 2001 (funded).

NIH-NIDCD Proposal, "Real-World Based Fitting of a Multimemory Hearing Aid," 2002 (unfunded).

Sponsor, NIH fellowship (1 F31 DC05429-01) to Michigan State University to support doctoral dissertation project of Amyn Amlani, "Predicting Speech Intelligibility from Directivity Index," February 2002-January 2003 (funded $25,783).

NIH-STTR Fast-Track Proposal, "High-Quality, Affordable Hearing Aids for World Markets," 2003 (unfunded).

NIH-NINCD Proposal, "Development of a Quality-of-Life (QoL) Inventory for Hearing-Impaired Adults," 2009 (unfunded).

PROGRAM SITE VISITS

Department of Audiology and Speech Sciences, Purdue University, October 19-21, 1994 (Invited by School of Liberal Arts).

Selected Audiology programs in state of Louisiana, commissioned by Louisiana Board of Regents (March 18-23, 2002; January 15, 2003); reviewed programs include Louisiana Tech University, Louisiana State University, and Louisiana State University Health Sciences Center.

ACCOMPLISHMENTS AS STAFF MEMBER OF ASHA NATIONAL OFFICE (1980-84)

Served as Project Director during first year of a federally funded three-year study of service and training needs in the profession of speech-language pathology and audiology.

Conducted study of employment and salary trends in the profession, May 1982.

Edited a comprehensive review of literature relative to incidence and prevalence of speech, language and hearing disorders entitled, "The Prevalence of Communicative Disorders: A Review of the Literature."

Co-compiled a comprehensive report on federal and private funding sources in speech, language, and hearing, "Profiles of Funding Sources."

Served as ASHA representative to Forum on Allied Health Data (FAHD), a consortium of federal, professional and private organizations devoted to establishing databases on manpower in medically related health professions; Chair of FAHD, May 1983-May 1984

Coordinated survey of graduate training programs for publication, *Guide to Graduate Education 1983.*

Coordinated development of guidelines for conduct of ASHA surveys by committees, boards, councils, and National Office staff members.

Developed proposal for comprehensive investigation of work force supply and demand in speech-language pathology and audiology profession (approved by ASHA Executive Board).

Instituted "Data Page" feature in *Asha*, a regular feature devoted to reporting data on ASHA membership, the speech-language pathology and audiology profession, and the communicatively impaired.

Coordinated development, creation, publication, and distribution of *Research Bulletin*, newsletter for member and nonmember scientists.

Conducted survey and completed co-authored report on consolidation of ASHA's scholarly publications, "National Office Report of Journals Survey."

Conducted survey and completed report on *dsh Abstracts*, Report of 1980 *dsh Abstracts* Survey

Coordinated special issue of *Asha* on science theme (December 1984); participated in interview, "*Asha* Interviews: Janis Costello, Jerry Punch, Teris Schery, Lawrence Shriberg;" and co-authored "Data Page" feature on that issue.

Developed proposal for initiating computer-based information services for use of ASHA members, and coordinated ASHA Task Force on Information Services (1982-83).

Coordinated effort to structure and initiate the Research Information Service (RIS), a computerized database containing information on research career opportunities, research technology resources, and research funding opportunities.

MISCELLANEOUS PROJECTS

Web site (http://www.msu.edu/~asc/hhi); originally developed in conjunction with Hearing Research Center, Michigan State University, September 1997; includes Web-based Hearing Handicap Inventory.

Hearing Handicap Inventory: Multimedia Version, developed in February 1998 by J.L. Punch and B.E. Weinstein. Interactive CD-ROM program designed to screen for hearing handicap, and as an outcomes-assessment measure, in young and elderly adult populations; available through MSU's Instructional Media Center.

Developed ASC 843C, Hearing Amplification I (http://vu.msu.edu/preview/asc843c/), online course offered for credit and non-credit, beginning summer 2002-fall 2006.

Developed ASC 843I, Hearing Amplification II (http://vu.msu.edu/preview/asc843i/), online course offered for credit and non-credit beginning summer 2002-spring 2006.

FORENSIC ACTIVITIES

Legal deposition, Dana Rowe v. North Reading Fire Department, Boston, MA, July 29, 1999.

Legal deposition, Boston Police Department v. Richard Dahill, Boston, MA, July 30, 1999.

Legal consultant, Montana Advocacy Program, Phil Hohenlohe, Staff Attorney, Helena, MT, 2004-2009 (no testimony given).

Legal consultant, Stephen J. Vujcevic v. Oglebay Norton Marine Services Company, LLC et al., Eastern District of Michigan, Southern Division, May 2007-December 2008.

Legal consultant, Bonfiglio v. Northridge Church, et al., Hohauser Law Firm, Rochester, Michigan, May 2008-December 2009.

Reviewed and commented on hearing standard proposed by American College of Occupational and Environmental Medicine (ACOEM), at invitation of Daniel G. Samo, MD, Medical Director, Health Promotion and Corporate Services, Northwestern Memorial Hospital, Northwestern University Feinberg School of Medicine, February-May 2009.

Legal testimony, Application of Highland Wind Farm, LLC, to construct a 102.5 MW wind electric generation facility and associated electric facilities, to be located in the Towns of Forest and Cylon, St. Croix County, Wisconsin; Hearing by Public Service Commission, Madison, Wisconsin, Docket 2535-CE-100, Towns of Forest and Cylon represented by Glenn Reynolds; Direct testimony, 8/30/12 (PSC Ref# 171133); Surrebuttal testimony, 10/3/12 (PSC Ref# 173730); Testimony re Shirley Wind, 1/15/13 (PSC Ref# 17149), Rebuttal testimony, 1/22/13 (PSC Ref# 179627).

Legal testimony on behalf of Union Neighbors United, Inc., Robert and Diane Mcconnell, and Julia F. Johnson, In the Matter of the Application of Champaign Wind LLC to Install Electricity Generating Wind Turbines in Champaign County, Ohio, Hearing by Power Siting Board, Columbus, Ohio, Case No. 12-0160-EL-BGN, Union Neighbors United, Inc. represented by Jack A. Van Kley and Christopher A. Walker; Direct testimony, 10/31/12; Oral deposition, 11/7/12; Cross examination, 11/19/12.

Legal deposition, Michigan, 7/24/13, Charles and Debby Wiltzer, and Seager Wiltzer (represented by Chester E. Kasiborski, Jr., and Susan Hlywa Topp, vs. Heritage Sustainable Energy and Heritage Stoney Corners Wind Farm LLC (represented by Robert G. Chaklos, Jr., and Kurt M. Bowden).

Legal testimony, Michigan, 12/5/13, Daubert Hearing, Charles and Debby Wiltzer, and Seager Wiltzer (represented by Chester E. Kasiborski, Jr., and Susan Hlywa Topp) vs. Heritage Sustainable Energy and Heritage Stoney Corners Wind Farm LLC (represented by Robert G. Chaklos, Jr., and Kurt M. Bowden); qualified as expert witness, 12/16/13.

Legal consultant, Iowa, Reuters v. Osceola Windpower; Abby Walleck, attorney for plaintiff, 6/30/14; Steve Hamilton currently serves as plaintiff's attorney.

Legal testimony on behalf of United Citizens for Livingston County (represented by Phillip Luetkehans) vs. Pleasant Ridge Wind Farm (Invenergy represented by Michael S. Blazer), Livingston County Zoning Board of Appeals, Livingston County, Illinois; Direct testimony, 1/21/15; cross-examination 1/22/15; additional cross-examination, 2/23/15.

Legal deposition on behalf of Daniel Brian Williams (represented by James McCandlish) vs. Invenergy LLC, Portland, Oregon, 7/2/15.

Legal testimony on behalf of Town of Somerset, Niagara County, New York, 12/22/15-1/11/16 (represented by Mark Davis) vs. Lighthouse Wind LLC.

Presentation, Wind Turbines: What's the Noise All About? Henry County, Indiana, October 15, 2016.

Letter (invited) to Board of Zoning Appeals, Warren County, Indiana, November 4, 2016.

Retained as expert witness in Falmouth Wind 1 case, Massachusetts, (plaintiffs represented by Christopher Senie), November 2016.

Expert witness in multiple cases in the state of New York (Gary Abraham, attorney for plaintiffs): (1) Citizens v. Cassadaga Wind LLC, (2) Citizens v. Lighthouse Wind LLC, (3) Citizens v. Baron Winds LLC, and (4) Combined (three-party) case involving Centerville's Concerned Citizens, Concerned Citizens of Cattaraugus County, Inc., and the Old Amish of Farmersville v. Alle-Catt Wind Energy LLC.

Expert witness, Twin Forks Wind Farm, LLC, Macon County, Illinois (Richard Porter and Jeffrey Hoskins, attorneys for plaintiffs).

Presentation and legal testimony, Bright Stalk Wind Farm, McLean County, Illinois (Phillip Luetkehans and Brian Armstrong, attorneys for plaintiffs), February 22, 2018.

CONTINUING EDUCATION ACTIVITIES
(Listed in chronological order, since 1990; does not include attendance at professional meetings or local hearing aid update meetings, at which attendance was regular prior to retirement)

- ASHA Audio Teleconference, Amplification for Difficult to Fit Clients: A Case Study Exchange, July 20, 1990.
- Van Riper Lectures in Speech Pathology and Audiology, Signal Processing Hearing Aids: Bridging the Clinical-Research Gap, Western Michigan University, Kalamazoo, MI, October 4-5, 1990.
- Introduction to the IBM Mainframe Computing System, Michigan State University, Fall 1990.
- International Hearing Aid Conference: Signal Processing, Fitting, and Efficacy, University of Iowa, Iowa City, IA, June 21-23, 1991.
- MSUnet: An Introduction to Communications and Networking, Michigan State University, Summer 1991.
- Using Multimedia for Instruction and Presentation, Michigan State University, Fall 1991
- Au.D. Audio Teleconference, Sponsored by Purdue University, October 30, 1991.
- Middle Ear Disorders in Children: An Update on Treatment and Management, ASHA Teleconference, February 26, 1993.
- Participant, Meet Michigan, guided tour of northeastern Michigan outreach programs receiving support from Michigan State University, May 10-13, 1993.
- International Hearing Aid Conference II: Signal Processing, Fitting, and Efficacy, University of Iowa, Iowa City, IA, June 24-27, 1993.
- Lilly Faculty Seminar Programs, Sheila Tobias, Conversations about Teaching and Learning: Testing Workshop, Michigan State University, January 21, 1994.
- Lilly Faculty Seminar Programs, Ann Austin, Teaching for Understanding: Building on What Students Bring to the Classroom, Michigan State University, February 11, 1994.
- Telecommunication and Community Health Care for Michigan, conference sponsored by Michigan State University, June 17-18, 1994.
- Student Information System Training Course, Michigan State University, August 16 & 18, 1994.
- Leadership Workshop for Chairs and Directors, Sponsored by Office of the Provost, Michigan State University, September 16, 1994.
- Leadership Workshop for Chairs and Directors, Sponsored by Office of the Provost, Michigan State University, October 14, 1994.
- Facilitator, Faculty retreat in Department of Audiology and Speech Sciences, October 22, 1994.
- Expanding Educational Opportunities Conference, Sponsored by ASHA, Bethesda, MD, January 28-29, 1995.
- Leadership Workshop for Chairs and Directors, Sponsored by Office of the Provost, Michigan State University, February 3, 1995.

- Leadership Workshop for Chairs and Deans, Peter Seldin and Linda Annis, Improving Teaching: What Works and What Doesn't, Sponsored by Office of the Provost, Michigan State University, February 3, 1995.
- Workshop, Sponsored by National Consortium for Universal Newborn Screening, TEOAE-Based Universal Newborn Hearing Screening, Georgetown University Medical Center, Washington, DC, November 16-18, 1995.
- Visited University College London, London, England, to establish Study Abroad semester program for MSU undergraduate students, June 24-July 6, 1996.
- Leadership Workshop for Chairs and Deans, Lou Anna K. Simon, The Continuing Dialogue on Motivating Change, Sponsored by Office of the Provost, Michigan State University, August 29, 1996.
- Leadership Workshop for Chairs and Deans, Jude West, Leadership Skills in a Changing Academic Environment, Sponsored by Office of the Provost, Michigan State University, October 2, 1996.
- Leadership Workshop for Chairs and Deans, Robert Church, Enhancing the Teaching Mission with Technology: Issues and Challenges, Sponsored by Office of the Provost, Michigan State University, November 12, 1996.
- Leadership Workshop for Chairs and Deans, Terry Curry, Faculty Performance Reviews, Sponsored by Office of the Provost, Michigan State University, February 19, 1997.
- Seminar, Internet Tools for Distance Learning, Michigan State University, April 17, 1997
- Conference, Reaching for Cultural and Social Diversity in Speech Pathology and Audiology: Assessing Cultural and Social Values, Ronald Jones, Ph.D., Co-sponsored by Department of Audiology and Speech Sciences, Michigan State University, and Speech Pathology and Audiology Program, Norfolk State University, Michigan State University, April 21, 1997.
- Conference, Preventive Ethics, Sponsored by Office of the Provost, Michigan State University, April 23, 1997.
- Graduate Program Director Workshop on Financing Graduate Education, Sponsored by the Graduate School, Michigan State University, April 30, 1997.
- Leadership Workshop for Chairs and Directors, Sponsored by Office of the Provost, Michigan State University, September 23, 1997.
- Leadership Videoconference, The Nonprofit Leader of the Future: A Seminar in Social Sector Leadership, Peter Druker, Sponsored by Office of the Provost, Michigan State University, September 25, 1997.
- Leadership Workshop for Chairs and Directors, Drug-Free Workplace, ADA, Sexual Harassment Prevention, and EAP Issues, Sponsored by Office of the Provost, Michigan State University, October 23, 1997.
- Communication and Aging: Interdisciplinary Approaches to Keep Elders Communicating, Van Riper Lectures in Speech Pathology and Audiology, Western Michigan University, October 24, 1997.
- Leadership Workshop for Chairs and Directors, Technology and Computing at the Unit Level: Issues and Opportunities, Paul Hunt, Sponsored by Office of the Provost, Michigan State University, November 11, 1997.

- Instructional Videoconference, Putting Your Course Online, Sponsored by Office of the Provost, Michigan State University, November 13, 1997
- Workshop, Using Computers in Instruction, Sponsored by Office of the Provost, Michigan State University, November 18, 1997.
- Workshop, Technology in the Classroom, Frank Tate, Sponsored by Instructional Media Center, Michigan State University, January 12, 1998.
- Workshop, Effective Teaching, Sandi Smith, Sponsored by Office of the Provost, Michigan State University, January 29, 1998.
- Workshop, Writing Faculty Performance Reviews, Terry Curry, Sponsored by Office of the Provost, Michigan State University, February 2, 1998.
- Workshop, Getting Things Done: Delegating and Accountability, Michael Polzin and Tina Riley, Sponsored by Office of the Provost, Michigan State University, February 18, 1998.
- Workshop, Getting New Faculty Started, Ann E. Austin, Sponsored by Office of the Provost, Michigan State University, October 23, 1998.
- Workshop, Faculty Course Portfolios: From Teaching to Learning, Deborah M. Langsam, Sponsored by Office of the Provost, Michigan State University, October 26, 1998.
- Workshop, Using Learning Theory to Inform Teaching Practice, Marilla Svinicki, Sponsored by Office of the Provost, Lilly Faculty Seminar Programs, Michigan State University, November 6, 1998.
- Workshop, Writing Faculty Performance Review: What We Have Learned in One Year, Terry Curry, Sponsored by Office of the Provost, Michigan State University, February 9, 1999.
- Workshop, The Chairs Plague: Dealing with Difficult People, George A. Lopez, Sponsored by Office of the Provost, Michigan State University, March 3, 1999.
- Workshop, Survival Skills for Administrators, Kristina Gunsalus, Sponsored by Office of the Provost, Michigan State University, April 28, 1999.
- Audiology Curriculum Conference, Wendell Johnson Speech and Hearing Center, University of Iowa, July 9-11, 1999.
- Participant, Parent Panel for Parent Orientation Program (POP), Michigan State University, June 29, 1999.
- Educational Seminar, Directional Microphone Usage in Hearing Aids, Unitron Industries, Inc., Grand Rapids, MI, July 8, 1999.
- Academic Leaders Program, Presented by Faculty of the Wharton School of Business and Institute for Research on Higher Education, University of Pennsylvania, Grand Rapids, MI, August 19-21, 1999.
- Leadership Workshop for Chairs and Deans, Lou Anna K. Simon, The Continuing Dialogue on Motivating Change, Sponsored by Office of the Provost, Michigan State University, September 28, 1999.
- Sexual Harassment Workshop, Sponsored by Office of the Provost, Michigan State University, October 8, 1999.
- Dreamweaver2, Levels 1 and 2 (Web Publishing Software), Seminars for Faculty on Instructional Technology, Libraries, Computing and Technology, Michigan State University, January 6, 2000.

- Virtual Universities: Online and On Target?, Video Teleconference, Sponsored by Dallas County Community College District, February 3, 2000.
- Fulbright Scholar Program, Sponsored by Michigan State University Office of International Studies and Programs, May 9, 2000.
- Blackboard II (Advanced Features), Mary Longcore, Sponsored by Office of Libraries, Computing and Technology, Michigan State University, July 2000.
- Creating a High Impact Classroom Climate, James Eison, Lilly Faculty Seminar Programs, Michigan State University, September 15, 2000.
- Copyrights and Wrongs for the Web and the Classroom, Michael Seadle, Explorations in Instructional Technology Series, Michigan State University, September 22, 2000.
- Blackboard I (October 26, 2000) & Blackboard II (November 15, 2000), Ben Goodsell, Sponsored by Office of Libraries, Computing and Technology, Michigan State University
- Cerumen-Related Hearing Aid Repairs, Offered by Michael Phillips, Phonak, Inc., East Lansing Marriott, November 10, 2000.
- Teaching Online, One-week seminar offered by William A. Draves, LERN organization (http://www.lern.org), February 5-9, 2001.
- Using Video to Enhance Lectures and Courses, WKAR Staff, Michigan State University, March 23, 2001.
- Visited Singapore to attend meeting of *Hearing International*, March 2001
- Unison Digital Systems Training Seminar, Unitron Hearing, August 19, 2002
- Angel A and B, Anne Hunt, Sponsored by Office of Libraries, Computing and Technology, Michigan State University, May 6, 2003.
- Web Page Design, Sponsored by Office of Libraries, Computing and Technology, Michigan State University, June 3 & 5, 2003.
- Web Accessibility, Sponsored by Office of Libraries, Computing and Technology, Michigan State University, June 6, 2003.
- Fireworks MX, Level 1, Sponsored by Office of Libraries, Computing and Technology, Michigan State University, June 16, 2003.
- Dreamweaver MX, Level 1, Sponsored by Office of Libraries, Computing and Technology, Michigan State University, June 17 & 19, 2003.
- Dreamweaver MX, Level 2, Sponsored by Office of Libraries, Computing and Technology, Michigan State University, July 24, 2003.
- Socrates in Cyberspace, Cecil Mackey, Explorations in Instructional Technology Series, Michigan State University, October 3, 2003.
- Grant-Writing Strategies in the Social Sciences and Education, Sponsored by The University Research Council and The Office of Faculty and Organizational Development, Kellogg Center, Michigan State University, March 30, 2004.
- Three Proven Approaches to Web-Supported Teaching, MSU Virtual University Design and Technology (vuDAT) Breakfast Series, February 28, 2005.
- Maintaining, Storing and Using Sensitive Data, Sponsored by MSU Libraries, Computing & Technology, October 25, 2005.
- Human Participant Protections Education for Research Teams, Sponsored by the National Cancer Institute, NIH, Certification awarded December 19, 2005.

- Output Levels of Personal Stereo Systems, and What Audiologists Need to Know, Brian Fligor, Live Online Videoconference Sponsored by Audiology Online eLearning, February 22, 2006.
- Constructing Knowledge through Inquiry: Teaching Students the Art of Asking Questions in the Disciplines, Laura Greene, Sponsored by Office of the Provost, Michigan State University, May 16, 2006.
- Grants.gov Workshop, Sponsored by Office of Contracts and Grants Administration, Michigan State University, August 16, 2006.
- LSE Workshop, Sponsored by Department of Epidemiology, Michigan State University, July 10, 2007.
- Developing and Operating an Online Course for Over 1000 Students per Year, Sponsored by Explorations in Instructional Technology Series, Michigan State University, November 9, 2007.
- LSE Workshop, Sponsored by Department of Epidemiology, Michigan State University, November 29, 2007.
- Hear the Difference: What Audiologists Need to Know about the Baha Implantable Hearing Solution. Workshop sponsored by Cochlear Americas, Lansing, Michigan, May 7, 2008.
- Visited University of Dakar (UCAD), Senegal, Africa, to assist in establishing a special-education program at the graduate level, March 23-June 2, 2008 (with Ida Stockman, Ph.D.).
- Completed continuing-education unit (CEU) requirements for continued certification by ASHA and licensing by state of Michigan as an audiologist (completed 3.5 CEUs; 3.0 CEUs required for certification and licensing), December 2008.
- CITI Collaborative Institutional Training Initiative (most recent update), April 1, 2009
- Data Analysis Using SPSS, Sandra Herman, Sponsored by Center for Statistical Training & Consulting, Michigan State University, October 9, 2009.
- Funding and Proposal Writing Resources, Jon Harrison, MSU Libraries, Michigan State University, September 18, 2009.
- Workshop: The Grant Process, Maria Lapinski, College of Communication Arts and Sciences, Michigan State University, October 23, 2009.
- Completed continuing-education unit (CEU) requirements for continued certification by ASHA as an audiologist, December 2011.
- IRB Retreat, Michigan State University, June 8, 2013.
- IRB Administrative Workshops, Kuali Coeus IRB Blueprint Sessions 1 and 2, Michigan State University, October 31 and November 4, 2013.
- Audiology Online, Continuing-education credits completed toward renewal of ASHA Certificate of Clinical Competence through 2017, October 2014.
- Hearing Technology: Stream On, Conference sponsored by Department of Otolaryngology, University of Michigan, East Lansing Marriott, East Lansing, Michigan, October 14, 2015.
- Audiology Online, Continuing-education credits completed toward renewal of ASHA Certificate of Clinical Competence through 2020, August 2017.

Wind Turbine Noise and Human Health: A Four-Decade History of Evidence that Wind Turbines Pose Risks*

Jerry L. Punch,[i] Richard R. James[ii]

Abstract

Many expert-review panels and some individual authors, in the U.S. and internationally, have taken the position that there is little literature to support concerns about adverse health effects (AHEs) from noise emitted by industrial wind turbines (IWTs). In this review, we systematically examine the literature that bears on some of the particular claims that are commonly made in support of the view that a causal link is non-existent. Investigation of the veracity of those claims requires that multiple topics be addressed, and the following specific topics were targeted for this review: (1) emissions of infrasound and low-frequency noise (ILFN) by IWTs, (2) the perception of ILFN by humans, (3) the evidentiary bases for establishing a causative link between IWTs and AHEs, as well as the physiological bases for such a link, (4) recommended setback distances and permissible noise levels, (5) the relationship between annoyance and health, (6) alternative causes of the reported health problems, (7) recommended methods for measuring infrasound, (8) foundations for establishing a medical diagnosis of AHEs due to IWTs, (9) research designs useful in establishing causation, (10) the role of psychological expectations as an explanation for the reported adverse effects, (11) the prevalence of AHEs in individuals exposed to IWTs, and (12) the scope and quality of literature addressing the link between IWT noise and AHEs. The reviewed evidence overwhelmingly supports the notion that acoustic emissions from IWTs is a leading cause of AHEs in a substantial segment of the population.

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Key Words: Adverse health effects, human health, industrial wind turbines, infrasound, inner ear, low-frequency noise, wind turbine noise

Introduction

Whether infrasound and low-frequency noise (ILFN) from industrial wind turbines (IWTs) is detrimental to human health is currently a highly controversial topic. Advocates of industrial-

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scale wind energy assert that there is no credible scientific evidence of a causal relationship, while many reputable professionals believe that there is sufficient scientific evidence to establish a causal link between IWTs and detrimental health effects *for a non-trivial percentage of individuals who reside in communities hosting IWTs*. The veracity of claims regarding the effects on human health is being debated on a global scale by the wind industry; individuals living near IWTs; attorneys and expert witnesses in courts of law; print and web-based media; documentary films (which currently include *Windfall*, *Wind Rush*, and *Down Wind*); and scientists and other professionals in government reports, on the Internet, and in scientific and professional papers presented at society meetings and published in peer-reviewed journals.

The debate surrounding IWTs extends to many controversial issues, including physical safety, visibility, shadow flicker, and threats to property values and wildlife. Many problems involving wind turbines, including mechanical failures, accidents, and other mishaps, have been discussed on the Internet. At least one website has extensively catalogued these incidents,[1] and the large number of incidents reported by that site is described by its webmaster as grossly underestimating the actual number of documented incidents. The most vigorous debate, however, centers on ILFN and its effects on human health.

The overall purpose of this article is to provide a systematic review of legitimate sources that bear directly and indirectly on the question of the extent to which IWT noise leads to the many health complaints that are being attributed to it. The authors accessed most articles and reports referenced in this review by employing Google, Google Scholar, and PubMed as the primary search engines. Our basic aim was to provide a comprehensive and representative—though not exhaustive—review of the literature that is relevant to many of the claims made by wind industry advocates. An exhaustive review is an elusive and impractical goal, given the large volume of directly and indirectly related work done in this area over the past several decades and the current pace of such work.

The role of evidentiary facts

Adverse impacts on people and property are among the most contentious issues that are typically the focus of legal proceedings involving IWT noise. Based on the forensic and research experiences of the authors, we believe that a resolution of the controversial aspects of this debate will require not just relevant scientific research, but rather a series of legal judgments based on the effective evaluation and interpretation of the existing research. In fact, much research and some already-rendered legal decisions show convincingly that some segments of the population suffer damaging effects from exposure to wind turbine noise (WTN). What is needed among the scientific community, local and national governmental agencies, and political leaders, is honest

discourse about methods for reducing carbon emissions in ways that do not turn some rural communities into *sacrifice zones*.[2, 3]

Many symptoms and complaints of adverse health effects (AHEs) related to IWTs have been self-reported by individuals living near wind turbines and described in published case reports. There is a group of core symptoms and complaints, however—including sleep disturbance, headache, dizziness, vertigo, and ear pressure or pain—that are remarkably common worldwide. Dr. Nina Pierpont was the first to report these core symptoms in a case series,[4] and she termed these core symptoms *Wind Turbine Syndrome*. For the sake of brevity, we will on occasion refer to Wind Turbine Syndrome as a substitute for this group of common symptoms and complaints, even though the phrase itself is currently not utilized as a medical diagnostic entity.

Numerous reviews of the literature have already been published that allege that there is no credible link between WTN emissions and AHEs. Those reviews have typically been sanctioned by state or provincial government agencies that have missions to support the development of wind energy, and which in turn appoint *expert panels* whose members hold views that regularly favor the wind industry and, therefore, may have conflicting interests. Too often, in the opinion of the authors, such reviews are biased in support of political policy decisions that promote the financial interests of wind developers, and perceived financial benefits to local communities, over the common good. None of those reviews has been specifically targeted toward describing or explaining the relationship between exposure to complex, dynamically modulated infra- and low-frequency sound from wind turbines or other industrial sources (e.g., noise-induced Sick Building Syndrome) and AHEs. Our primary objective in this article is to review the existing scientific and professional literature that is frequently overlooked in such reviews conducted by wind energy proponents. Such literature can be useful in legal proceedings in questioning and articulating the available evidence of risks to people who live in the footprint of utility-scale wind energy projects.

Some of the published reviews have been criticized for their failure to meet the standards noted by Horner,[5] who reminds us that readers should regard literature reviews with caution, and employ an audit strategy in evaluating their completeness, accuracy, and objectivity. Authors, including ourselves, have an inherent obligation to ensure that such reviews cite all known legitimate sources that serve as the basis for their views of the issues and reflect accurately the contents of all references cited.

Some courts of law in the U.S. and other countries now tend to rely heavily on testimony that adheres to the principle that proof of evidence of causation of AHEs from IWTs be based on the

peer-reviewed literature. Presumably, that practice in the U.S. stems at least partially from advocacy by the Office of Management and Budget[6] that internal and external government science documents be peer-reviewed government-wide for the purpose of increasing the quality and credibility of scientific information generated by the federal government. Peer-review standards are considered paramount in that effort.

While the peer-review process has many virtues, it also has its shortcomings, which are well known. For example, not all journals or individual reviews of submitted manuscripts are of equal quality, as specific journals and specific reviewers may have ideological or philosophical biases, which may or may not be surmised from the journals' mission statements. Nonetheless, the peer-review process is one of the most widely acknowledged ways to control the quality of published works. We contend, however, that there are other credible sources of information, even though those sources may not have been subjected to as rigorous a peer-review process as that employed by many scientific journals. Such sources include papers presented at meetings of scientific and professional societies; reports and other documents commissioned by state and local governmental agencies, especially if such documents are authored by independent researchers; legal testimony given under oath by qualified scientists and professionals; and some information available on the Internet, especially if written by professionals who have reputable track records in their disciplines. Although we will emphasize the peer-reviewed literature in this article, we will also cite some of these additional sources as authoritative. Our citing of selected non-peer-reviewed reports, with a few exceptions, is based on our familiarity with the professional reputations of the authors of those reports, normally earned through publication of a solid body of work in the peer-reviewed literature and by acceptance of their work by other professionals and peers. Typically, individuals so referenced enjoy positive national or international recognition in their respective fields of expertise.

We begin this review by calling attention to a quote from geophysicist Marcia McNutt, who once headed the U.S. Geological Survey and is now editor of the prestigious journal *Science*. McNutt has been quoted as stating: "Science is not a body of facts. Science is a method for deciding whether what we choose to believe has a basis in the laws of nature or not."[7] In fact, science consists of a variety of overlapping methodological approaches, which must be interwoven to discover answers to complex problems. That conviction has guided our attempt to re-examine the controversial topic at hand.

Review of wind industry claims and positions

Our review is organized by summarizing the past and present literature that addresses each of 12 selected statements, listed below, that encapsulate specific claims, or positions, commonly taken by advocates for the wind industry:

1. Infrasound is not an issue, as infrasound generated by wind turbines is not perceptible to humans.
2. There is nothing unique about wind turbine noise, as infrasound and low-frequency noise are commonly produced by the body and by many environmental sources.
3. There is no evidence that wind turbine noise, audible or inaudible, is the cause of adverse health effects in people, and there are no physiological mechanisms to explain how inaudible acoustic energy can be harmful.
4. Setback distances of 1,000-1,500 ft. (approximately 0.3-0.5 km) are sufficiently safe to protect humans from harm, regardless of height or other physical characteristics of the IWTs.
5. Annoyance is a nuisance, but it is not a health issue.
6. Noise cannot account for all of the complaints of people living in the vicinity of wind turbines; there must be another, unknown reason for the complaints.
7. Infrasound from wind turbines is sufficiently correlated to the A-weighted sound emissions to allow an A-weighted model to be used to predict how much infrasound is present in homes.
8. Wind Turbine Syndrome has not been accepted as a diagnostic entity by the medical profession, so medical professionals cannot diagnose or treat it.
9. Peer-reviewed epidemiological literature is the only acceptable basis for proving a causative relationship between wind turbine noise and adverse health effects.
10. The nocebo effect, a manifestation of psychological expectations, explains why people complain of adverse health effects when living near wind turbines.
11. Only relatively few people, if any, are adversely impacted by wind turbine noise, and the majority have no complaints.
12. There is no evidence in the literature to support a causative link between wind turbine noise and adverse effects.

Statement 1: Infrasound is not an issue, as infrasound generated by wind turbines is not perceptible to humans.

The argument that infrasound as a cause of AHEs is not an issue has been advanced in the published literature primarily by Dr. Geoff Leventhall,[8, 9] with support from several other researchers. Those researchers have dismissed the influence of infrasound on human health by describing it as not exceeding the thresholds of audibility, and therefore ineffectual, without noting that those thresholds were established using steady pure tones instead of the complex, dynamically modulated tones emitted by wind turbines. Leventhall claims that infrasound from wind turbines is not a problem and that it is misunderstood largely because of mischaracterization by the media and by "those with limited knowledge" (p. 29). He states that there may be noise problems associated with wind turbines, but that such problems are due to *audible* swishing sounds due to interactions of the blades with the tower. Supporters of wind energy have generally followed Leventhall's lead, although his own research has shown conclusively that exposure to modulated ILFN produced by large industrial equipment, including heating, ventilating and air-conditioning (HVAC) systems, leads to mental fatigue, lack of concentration, headaches, reduced performance, and work dissatisfaction. Indeed, there is a long history of noise-induced *Sick Building Syndrome*, stemming from investigations in the 1970s-1990s of the effects of low-frequency noise on knowledge workers (see James[10] and Schwartz[11] for reviews of that research). Leventhall[12] stated:

> "Low frequency noise causes extreme distress to a number of people who are sensitive to its effects. Such sensitivity may be a result of heightened sensory response within the whole or part of the auditory range or may be acquired. The noise levels are often low, occurring in the region of the hearing threshold, where there are considerable individual differences" (p. 4).

Later in the same document, he states:

> "There is no doubt that some humans exposed to infrasound experience abnormal ear, CNS (central nervous system), and resonance induced symptoms that are real and stressful. If this is not recognised by investigators or their treating physicians, and properly addressed with understanding and sympathy, a psychological reaction will follow and the patient's problems will be compounded. Most subjects may be reassured that there will be no serious consequences to their health from infrasound exposure and *if further exposure is avoided* (emphasis added) they may expect to become symptom free" (p. 60).

Leventhall has also stated that the ear is designed to protect us from infrasound and that, in essence, *If you can't hear it, you can't feel it.*[13, 14] The idea that ILFN from wind turbines does

not affect health was further reinforced in a 2009 white paper co-authored by Leventhall and sanctioned by the wind industry,[15] to be reviewed later.

The position that infrasound from wind turbines is not harmful to humans because it is not perceptible to the human ear also has support from Møller & Pedersen,[16] who investigated noise emissions from 48 wind turbines with electrical output capacities of between 2.3 and 3.6 MW. They stated:

> "The turbines do emit infrasound (sound below 20 Hz), but levels are low when human sensitivity to these frequencies is accounted for. Even close to the turbines, the infrasonic sound pressure level is much below the normal hearing threshold, and infrasound is thus not considered as a problem with turbines of the investigated size and construction" (pp. 3742-3743).

Evans et al[17] found that levels of infrasound measured at two residential locations near wind projects in South Australia were within the range of infrasound levels experienced in other urban and rural environments. Although Colby et al[15] and Bolin et al[18] dismiss wind turbines as a cause of AHEs, they acknowledge that turbines emit ILFN. A number of authors indicate that large turbines emit more such noise than smaller turbines (see, for example, Bolin et al[18] and Møller & Pedersen.[16]) George Kamperman (personal communication, 2009) has concluded that the amount of low-frequency noise generated by IWTs increases by 3–5 dB for every megawatt of electrical power generated.

Evidence that IWTs produce perceptible levels of infrasound, in addition to audible low-frequency noise above 20 Hz, has been available since the 1980s. In their seminal research on large-scale wind turbines, which was funded by the U.S. Department of Energy, Kelley et al[19] measured noise levels emitted by a DOE/NASA MOD-l wind turbine operating near Boone, North Carolina, in response to noise complaints. They concluded that:

> "…one of the major causal agents responsible for the annoyance of nearby residents by wind turbine noise is the excitation of highly resonant structural and air volume modes by the coherent, low frequency sound radiated by large wind turbines. Further, there is evidence that the strong resonances found in the acoustic pressure field within rooms actually measured indicates a coupling of subaudible energy to human body resonances at 5, 12, and 17-25 Hz, resulting in a sensation of whole-body vibration" (p. 120).

Those conclusions were further strengthened in a subsequent report.[20] In a second follow-up report, also funded by the Department of Energy, Kelley[21] electronically simulated three interior environments resulting from low-frequency acoustical loads radiated from both single and grouped upwind and downwind turbines. (These terms refer to the placement of the rotor and

blades with respect to the tower. With upwind designs, the more contemporary design, the airflow strikes the blades before striking the tower, and with downwind designs, the airflow strikes the tower before striking the blades.) Relatively low levels of low-frequency acoustic noise from a single, 2-MW MOD-1 wind turbine led to annoyance of residents of the surrounding community, largely through interaction with residential structures. Most importantly, Kelley found that the turbines radiated their peak sound power in the infrasonic range, typically between 1 and 10 Hz. An extensive investigation revealed that the reported annoyance was the result of a coupling of the turbine's impulsive low-frequency acoustic energy into the structures of some of the surrounding homes, and that annoyance was "frequently confined to *within the home itself*" (p. 1). Despite these early findings that IWTs generate infrasonic levels that produce acoustic energy, vibrations, and resonances that affect people in their homes, the wind industry has chosen to regard them as insignificant or only applicable to obsolete, downwind wind turbine designs.

The basis for discounting the research by Kelley and associates is predicated on the assumption that pressure changes of equal levels to wind turbines occur in natural environments and do not cause any similar complaints. The authors find that their own experiences with rapidly changing pressures have caused similar experiences. If these rather short-duration sensations were to continue over days, weeks, and months, as they do for people living near wind projects, they would likely find them to be unacceptable.

The primary argument of people who deny any effects is encapsulated by Leventhall[9] in his *Child on a Swing* example:

> "A child on a swing experiences infrasound at a level of around 110dB and frequency 0.5Hz, depending on the suspended length and the change in height during the swing" (p. 30).

The inference is that because children often swing on swings, there are no adverse sensations. That fails to acknowledge that the experience of swinging is one that elicits many visceral sensations that are pleasant to the child as long as the sensations stop when the swing stops. The example, however, misses one major point. The duration and motion of the swing provide a smooth, sinusoidal pressure change that has two high pressure points (at the top of each swing) that occur over a period of several seconds or so. This is a completely different experience to that of pressure pulses lasting 100 msec or less. If one considers a swing with a period of 3.5 sec, there is a pressure change at 1.75 sec, resulting in a frequency of 0.57 Hz. The pressure changes are approximately 120 dB peak-to-peak, or 110 dB rms. The overall G-weighted value in this example is -60 dB, with a smooth pressure change, resulting in a net 50 dBG for the child, versus

the 75 dBG experienced as a pulse for a person living near a wind turbine (calculations provided by Malcolm Swinbanks, personal communication, 2010).

The assertion that wind turbine infrasound immissions, especially when received in the bedroom of a quiet home, must be at or above the threshold of hearing to cause adverse effects has been disproved, as noted above in the works of Kelley and colleagues in the 1980s.[19, 20, 21] The significant finding of the Kelley studies is that when the intruding infrasound is dynamically modulated short-duration pulses (generally under 100 msec and as short as 4 msec), the thresholds of sound pressure levels (SPLs) for non-auditory perception are in the range of 60 to 70 dB. In the work by one of the authors of this paper (James, with Mr. Wade Bray, INCE, of Head Acoustics, GMBH), infrasound pulsations were measured from a GE 1.5-MW wind turbine with a blade-pass frequency of 1 Hz that reached a level as high as 100 dB.[22] The people living in the home 'felt' the pulsations when the crests of the pressure waves were as low as 60 dB at 1 Hz. During similar measurements, Swinbanks, who has reported that he is sensitive to infrasound pulsations, was present at the test site. His experience was that he could feel the pulsations outside the home at similar SPLs.

Subsequent to the papers by Kelley and colleagues, several other studies have also reported the thresholds for significant experiences at similar thresholds, all substantially below the threshold for audibility of steady pure tones. In many of those tests, the rms SPL of the dynamically modulated blade-pass tone and its harmonics has been as low as 40 dB when using narrow-band analysis with windows of 40 to 80 sec, providing the crest of the pressure waves are 10 to 15 dB higher than the rms levels. These studies include the works of Robert Rand, INCE, and Stephen Ambrose, Bd. Cert., INCE, in their study of homes of complainants in Falmouth, Massachusetts;[23] Walker, Hessler, Hessler, and Schomer, in their work at the Shirley Wind project in Brown County, Wisconsin, for the Wisconsin Public Health Service;[24] and most recently, Steven Cooper's study of the Cape Bridgewater project in Victoria, Australia.[25] All of these studies report similar findings, namely that perception, generally non-auditory in character, begins when the rms SPLs of the modulating tones are as low as 40 dB rms, with increasing impacts as the rms levels rise to 50, 60, and to 70 dB and higher levels. In all these studies, the dynamic modulation of the blade-pass tones produce pressure peaks that are often 10 dB or more, sometimes much more, than the rms values.

In the opinion of the authors, a paper prepared by Swinbanks for the 2015 conference on wind energy in Glasgow, Scotland, shows the impact of dynamically modulated infrasound on a sensitive individual—himself—along with high-quality measurements of the environments in which he experienced the sensations.[26] That paper shows that a highly respected acoustician and

scientist with expertise in infra- and low-frequency sound also responds to this acoustic energy in a way that is similar to the many complaints from others, both in the location of his tests and at other wind energy projects around the world. In the paper, Swinbanks reports that he was able to differentiate the pulsations in the test data from at least six separate wind turbines in a project consisting of 46 1.5-MW GE models. He also reports that he was able to perceive the effects of the pulsations in his home's basement, approximately 3 km from the nearest operating wind turbine, with the SPLs of the blade-pass frequency and harmonics summing to about 55 dB rms. At closer locations, he measured positive-going pressure peaks of 87 dB with corresponding negative-going peaks of equal level. It is worth noting that at the Glasgow conference, Swinbanks presented the paper as a poster session,[27] as he was informed by the conference moderator that time restraints prevented him from presenting his paper to conference attendees.

In the 2012 investigation of infrasound at the Shirley Wind project, where local regulations require that the Nordex 2.5-MW turbines be sited at least 1,250 ft., or 381 m, from residences, Walker et al reported infrasound levels at one of the three test homes.[24] WTN was not audible outside the residence where infrasound was greatest, supporting the position that infrasound is at the root of at least some of the complaints. The blade-pass frequency and harmonics were clearly evident from the measurements inside that one home, and the family had moved far away for a solution.

Following the Shirley Wind team study, several members of the community conducted a series of micro barometer measurements inside homes ranging from 1,280 ft. to approximately 6 mi. from the wind turbine towers. Infrasonic tones at blade-pass frequencies and harmonics were found at all test sites, including test sites at distances of several miles or more from towers under downwind conditions. Testimony to Wisconsin's Brown County Board of Health by people with homes more than 4 mi. from the nearest wind turbines reported AHEs during the times the turbines operated. In mid-October 2014, the Brown County Board of Health went on record declaring that wind turbines at the Shirley Wind site "…are a human health hazard."[28] That action, which appears to be a precedent in the U.S., meant that Duke Energy's Shirley Wind utility were forced to prove to the Board that the utility was not the cause of the health complaints documented in the study and voiced by community residents. The outcome could result in a shut-down order, but no final decision had been made in that case at the time of this writing. Other examples of legally ordered turbine shutdowns include those in Massachusetts[29, 30] and Portugal.[31]

We will return to the issue of perceptibility of infrasound later in this paper, as we describe the physiological bases for perceptibility.

Statement 2: There is nothing unique about wind turbine noise, as infrasound and low-frequency noise are commonly produced by the body and by many environmental sources.

To begin, when the spectral characteristics of IWT noise, as depicted in several papers,[24, 32, 33] are compared to the spectra of subsonic jet transport planes,[34] five different types of aircraft,[35] and road traffic noise,[36] it is clear that noise generated by wind turbines has a number of unique acoustical characteristics. These comparisons reveal dissimilarities in spectral and peak levels in both the higher and lower frequency regions, including the low-frequency and infrasonic range.

Leventhall[37] was one of the first to describe how low-frequency noise is a special noise problem, particularly to sensitive people in their homes. He indicated that annoyance to low-frequency noise increases rapidly with level, often starting just above the threshold of audibility, and that about 2.5% of the population may be 12 dB more sensitive than the average person to low-frequency noise. He also noted that the World Health Organization (WHO) places a special emphasis on low-frequency noise as an environmental problem and source of sleep disturbance, even at low levels. The WHO[38] acknowledges that a noise consisting of a large proportion of low-frequency components may considerably increase AHEs and should be limited to below 40 dBA. Cummings[39] notes that sound levels of 40 dBA trigger high levels of community pushback.

Jung et al[40] experimentally identified the characteristics of acoustic emissions from large upwind wind turbines, with emphasis on ILFN. The sound spectral density showed that the blade-passage frequency component is clearly dominant, revealing up to 6-7 harmonics that generally occupy the infrasonic frequency region of 1 to 10 Hz. They voiced a concern that the low-frequency noise of the 1.5-MW and 600-kW wind turbines in the frequency range over 30 Hz would very likely lead to psychological complaints from ordinary adults.

In responding to a bylaw to restrict wind turbine infrasound in the town of Plympton-Wyoming, Ontario, Leventhall[41] declared that “Infrasound has become the Godzilla of acoustics” (p. 2). He concluded that science does not support the conditions in the bylaw, which was largely aimed at restricting blade-passing tones, because “There is no evidence that the very low level of blade passing tones affects humans, whilst there is evidence that it does not” (p. 7). Based on the kinds of evidence just discussed, we strongly disagree.

WTN has been described as having a character that makes it far more annoying and stressful than other sources of noise at the same A-weighted level, including traffic and industrial noise.[42, 43, 44, 45] Harrison[42] concluded that IWTs cause annoyance in about 20% of residents living within a distance considered acceptable by most regulatory authorities, and that for many of the 20%, the

annoyance and sleep disturbance lead to AHEs. Thorne[46] has pointed out that human perception of noise is based primarily on sound character rather than sound level, and that wind turbines are unique sound sources that exhibit special audible and inaudible modulated and tonal characteristics. He states that sound levels of 32 dBA Leq outside a residence and/or above an individual's threshold of hearing inside the home are markers for serious AHEs, especially among susceptible individuals.

Structural and human responses to low-frequency noise, including noise from wind turbines, have been described by Hubbard.[47] Hubbard and Shephard[48] illustrated the special characteristics of WTN by explaining its sources, pathways, and receptors. Thorne[46, 49] described wind turbines and *wind farms* as a unique source of sound and noise, like no other noise source or set of noise sources. The sounds are often of low amplitude and shifting in character, making it difficult for people who have never been exposed to such sounds to understand the problems of those who complain about the sounds. Shepherd et al[50] have described WTN as having characteristics sufficiently different from other, more extensively studied, noise sources to justify the application of standards different from pre-existing noise standards.

The preponderance of evidence on this point leads to the conclusion that WTN has special acoustic characteristics that distinguish it from other industrial sounds. A primary feature is that it consists of measureable energy down to below 1 Hz.[24, 51, 52] Its sound pressure level decreases rapidly with increasing frequency from about 0.5-5 Hz. It varies in amplitude over time,[9, 49, 51, 53, 54, 55, 56, 57, 58] it tends to have an intermittent tonal quality,[49, 52, 59] and its characteristics vary with distance and direction.[52, 53] It can result in an impulsive sound,[21, 40, 49, 60] even at long distances.[52] According to Lee et al,[53] the swishing sounds of turbines can be perceived from all directions, but at long distances from a turbine, low-frequency amplitude-modulated sounds can be heard only in particular directions and when the SPL is sufficiently high. This effect may make the WTN seem more impulsive at long distances despite an overall SPL that is relatively low.

Furthermore, ILFN from any source, including IWTs, is well known to penetrate walls and other barriers (e.g., Minnesota Department of Health[55]); is typically more disruptive indoors than outdoors;[46, 47, 61, 62, 63] and is not easily masked by atmospheric sounds, including road traffic and other sources of infrasound.[63, 64, 65, 66] The perception of low-frequency noise depends on density level, modulations, bandwidth, purity of blade-pass tones and harmonics, discrete beating tones, or other time-varying properties, and can occur even at near-infrasonic frequencies if any of these factors is present; otherwise, it might pass unnoticed.[57, 67, 68] James[69] describes the

infrasound occurring when wind turbine blades rotate past the tower as a short pressure pulse that consists of a well-defined array of tonal harmonics below 10 Hz. If the pressure peaks are received at the same time, they sum in a linear manner that significantly raises the overall SPL. Often, however, there are many wind turbines rotating at similar speeds, but not synchronized in time. This can lead to another form of modulation as the wind turbine infrasound is perceived as rising and falling, intermittent, or pulsating with variable intensity.

A common argument of wind industry proponents—one that is sometimes raised in legal proceedings—is that humans themselves generate infrasound by virtue of their own heartbeat and breathing, at levels that can be substantially higher than an external noise source such as wind turbines. In a rebuttal to a formal statement to this effect by the Association of Australian Acoustical Consultants (AAAC), Salt has provided a definitive explanation of why the two sources of infrasound (internal vs. external) cannot be equated. In a letter addressed to the AAAC,[70] Salt stated:

> "Stimulation of the ear occurs not directly by pressure (which is why deep sea divers can still hear) but by induced motions of the inner ear fluids, which in turn move sensory tissues and motion-sensitive cells….when low frequency and infrasound enters the ear via the stapes, it causes fluid movements throughout the entire ear between the stapes in the vestibule, through scala vestibuli and scala tympani to the compliant round window membrane at the base of scala tympani. It is these fluid movements that drive sensory tissue movements and cause stimulation. In contrast, pressure fluctuations generated by the body, such as by heartbeat and respiration, enter the ear via the cochlear aqueduct, not through the stapes. The cochlear aqueduct enters the ear adjacent to the round window membrane in the very basal part of scala tympani, so the fluid flows are localized in this tiny region of the ear. As the rest of the ear is bounded by a bony shell which is not compliant, fluid flows in the rest of the ear are substantially lower so that displacements of sensory tissues are negligible. Infrasound generated by the body, because it enters through the aqueduct, therefore does not cause stimulation of the ear."

Statement 3: There is no evidence that wind turbine noise, audible or inaudible, is the cause of adverse health effects in people, and there are no physiological mechanisms to explain how inaudible acoustic energy can be harmful.

In fact, there is ample evidence that noise in general, and especially low-frequency noise, has long-term consequences for human health.[71, 72] For example, long-term exposure to ordinary traffic noise has been associated in a dose-dependent manner with higher risk of myocardial infarction.[73]

Two landmark reports embodying diametrically opposing perspectives with regard to the impact of WTN on health appeared almost concurrently in 2009. One was published as a book by Dr.

Nina Pierpont,[4] a Fellow of the American Academy of Pediatrics who holds an MD degree from Johns Hopkins University School of Medicine and a PhD degree in Population Biology from Princeton University. The other report was written by a panel of seven experts (three physicians, two acousticians, an audiologist, and an audiologist/hearing scientist) commissioned by the American Wind Energy Association and the Canadian Wind Energy Association. The latter report[15] is commonly referred to as the AWEA/CanWEA report, or white paper. These respective reports, more than any others, quickly became the rallying cry for so-called anti-wind and pro-wind advocacy groups in the media, in the public discourse, and in court proceedings.

In her book, Pierpont[4] coined the term *Wind Turbine Syndrome* to describe a range of symptoms reported for 38 family members (adults and children) of 10 families who lived near wind turbines. Based on telephone interviews, she treated her observations and analyses as a case-series research design. She described the syndrome as consisting of 10 classes of symptoms (enumerated below), many of which she attributed to overstimulation of the vestibular system of the inner ear by ILFN. The wind industry, in its AWEA/CanWEA report and elsewhere, has vigorously criticized her study for being non-scientific and non-peer-reviewed. In fact, Pierpont's book was critically reviewed by far more than the usual number of reviewers for a peer-reviewed journal article. While it is true that case series are prone to selection bias, and can at best suggest hypotheses, many discoveries of new phenomena begin with a case study or case series. Furthermore, an increasing body of scientific evidence supports Pierpont's observations of a relationship between WTN and AHEs. More recent laboratory research, described later in this review, suggests that a variety of health symptoms may be due to ILFN stimulation of both vestibular and cochlear components of the inner ear.

Prior to Pierpont's book,[4] Dr. Amanda Harry[74] and Dr. Robyn Phipps and colleagues[75, 76] had documented the occurrence of ill effects from IWTs by use of questionnaire-based surveys of the health complaints of people living near wind projects in Cornwall, England, and Palmerston North, New Zealand, respectively. These authors concluded that a substantial number of people living near wind turbines suffer from health problems and that the cause of the disturbances was the complexity of the noise and vibration. Harry[74] observed that the symptoms were evident for people living within a mile from the wind development and recommended that no wind turbine should be sited closer than 1.5 mi. from the nearest residents. She noted that the guidelines used at the time to site wind turbines were developed when the turbines were 20% the size of the current ones. She concluded that annoyance from noise adversely affects human well-being, and that developers are wrong when they state that WTN is not a problem. Phipps et al[75] noted that 45% of households living within 2 km of the wind farm and 20% of households living up to 8

km away reported hearing noise from the turbines. Phipps[76] reported on the negative consequences of noise that were evident in her own survey and in the works of others, warning that residents do not readily habituate to the presence of WTN.

The AWEA/CanWEA report[15] has been widely used by the wind industry as a basis for its denial of AHEs from IWTs. However, the report is the product of a hand-picked group of experts, at least some of whom were known to hold positions favorable to the report's sponsors, it was never peer-reviewed, and it shows signs of bias, such as conclusions not supported by the research referenced in the report. That white paper concluded that sound from wind turbines, including sub-audible low-frequency sound, does not pose a risk of hearing loss or any other AHE in humans, whether those health effects are described as Wind Turbine Syndrome or otherwise. It also concluded that some people may be annoyed at the presence of sound from wind turbines, including its fluctuating nature, but described annoyance as unrelated to health. Although there is indeed no evidence that IWTs causes hearing loss, the report's conclusion that ILFN does not cause AHEs, and its dismissal of annoyance as a serious entity, have been heavily criticized as erroneous. Horner et al[77] cite many specific examples of the AWEA/CanWEA report's failure to use proper documentation, concluding that it lacks scientific merit and that it is neither authoritative nor convincing. They criticized the report's conclusion that the issue of AHEs stemming from IWTs is settled and that no more research is required, a conclusion that is rarely voiced by scientists. Horner[78] has characterized the report as offering nothing new in its treatment of annoyance, as annoyance has long been known to result from the stress effects of exposure to noise, and he criticized the report for downplaying the relationship between annoyance and health. Phillips[79] has indicated that the report mischaracterized the research designs used by epidemiologists. Despite widespread denial by wind industry advocates of a causal relationship between IWTs and AHEs, the vast majority of peer-reviewed papers have shown that IWTs significantly disturb sleep in at least some residents at distances and noise levels that are typical where IWTs are installed. Furthermore, not a single well-designed scientific study has found WTN to be harmless.[80, 81]

A panel of seven independent experts was commissioned by the Massachusetts Departments of Environmental Protection and Public Health to identify any documented or potential health impacts of risks that may be associated with exposure to IWTs and to facilitate a discussion of IWTs and public health based on scientific findings. The panel generated a report[82] concluding that scientific evidence is lacking to show that WTN leads to AHEs and that a more comprehensive assessment of WTN in populated areas is needed for establishing and refining siting guidelines and for developing best practices. Closer investigation was recommended near

homes where outdoor A- and C-weighted levels differ by more than 15 dB, a strategy for detecting the presence of ILFN (e.g., Kamperman & James[83]). The Massachusetts report has been criticized as misrepresenting the evidence it cites, as well as underestimating evidence indicative of AHEs from IWTs.[84, 85] Schomer and Pamidighantam[86] have described the report as a critique of the literature relating to wind turbine acoustic emissions and health effects, and one with problems similar to those it criticizes.

Some laypersons have remarked disparagingly in the media on the factual evidence—including observations and scientific reports—that shows a relationship between IWTs and AHEs. Shahan,[87] for example, confidently states: "To date, there is no scientific evidence that anything such as 'Wind Turbine Syndrome' actually exists." A common argument of wind energy advocates is that studies show that wind turbines do not lead to AHEs, or that studies that draw such a conclusion are not sufficiently scientific to establish causation. Efforts to discredit those who take a skeptical view toward the wind industry commonly use terms such as *opponents*, *detractors*, *anti-wind activists*, or in the case of Shahan,[87] "paid anti-wind 'experts' who have a long history of directly testifying against wind energy in various court cases." Such critics casually ignore the fact that many of the industry experts, including consulting acousticians and physicians, routinely testify on behalf of the industry in such cases, sometimes for substantial fees, and those individuals are rarely described as paid pro-wind experts or activists.

Numerous researchers have reported the existence of a constellation of health symptoms, either directly mirroring or closely related to those described as Wind Turbine Syndrome by Pierpont,[4] in persons living near IWTs. Significantly, the WHO[38] states that there is sufficient evidence that nighttime noise, irrespective of its source, is related to self-reported sleep disturbance and other health problems, and that these effects can lead to a considerable burden of disease in the population.

Sleep disturbance has been identified as a major adverse impact of IWTs.[4, 18, 45, 47, 54, 57, 58, 72, 74, 76, 77, 79, 80, 81, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107] Nighttime exposure to 40-dBA low-frequency noise has been shown to affect cortisol levels, a physiological indicator of stress. Those levels, following awakening, have been found to be associated with subjective reports of lower sleep quality and mood changes.[108] Sleep is a biological necessity, and disturbed sleep is associated with a number of adverse health conditions. The WHO[71] has concluded that there is available, good-quality evidence supporting a causal association between noise and sleep disruption. Sleep disturbance has important implications for public health and may be a particular problem in children.[84, 94, 109]

Even if no other adverse effects were associated with WTN, sleep disturbance alone is a sufficient reason to site turbines at distances that do not disrupt sleep. Many rural communities have background, nighttime sound levels that do not exceed 25 dBA, and observable effects of nighttime, outdoor noise do not occur at levels of 30 dBA or lower.[71] As outdoor sound levels increase, the risk of AH1Es increases, the most vulnerable populations being the first to show their effects. Vulnerable populations include elderly persons; children, especially those younger than age six; and people with pre-existing medical conditions, especially if sleep is affected.[38, 71] According to the WHO, there is ample evidence to link AHEs with prolonged exposure to outdoor sound levels of 40 dBA or higher. It is important to note that the WHO guidelines are based largely on industrial and transportation noise research, and not on wind turbine research. Because multiple studies (covered in this review) have indicated that WTN is significantly more annoying, has higher infra- and low-frequency sound energy, and is modulating, pulsatile, and sometimes tonal, it may impact health to a greater degree than other noises. This means that noise limits in the WHO guidelines may need to be adjusted downward when applied to WTN.

Additional factors increase the probability of sleep disruption due to WTN. The noise can be heard especially well in areas with low background noise levels, which usually occur at night. Also, lower nighttime wind speeds at ground level increase the nighttime contrast between WTN and background sound levels. Using test data taken during daytime wind conditions will result in a large underestimate of nighttime WTN levels, and thus underestimate the potential for sleep disruption.[38, 58]

Researchers who have studied the impacts of ILFN in general and WTN specifically on health, including some who have reviewed and assessed the findings of other researchers, have attributed a variety of symptoms to ILFN exposure. Those symptoms have been variously described by different researchers, with varying degrees of overlap and detail. They are shown, in no particular order, in Table 1.

Clearly, in addition to annoyance, the most commonly experienced and least-contested health symptom suffered by people living near IWTs is sleep disturbance.[110] Both the United Nations Committee against Torture (CAT) and the Physicians for Human Rights[111] describe sleep deprivation as critical to human functioning. According to Physicians for Human Rights:

> “Sleep deprivation ... causes significant cognitive impairments including deficits in memory, learning, logical reasoning, complex verbal processing, and decision-making; sleep appears to play an important role in processes such as memory and insight formation” (p. 22).

Table 1. Health symptoms described by different researchers as linked to exposure to infrasound and low-frequency noise, including exposure to industrial wind turbines.

Author (Year)	Reference	Symptomatology
Pierpont (2009)	4	Sleep disturbance; headache; Visceral Vibratory Vestibular Disturbance (VVVD); dizziness, vertigo, unsteadiness; tinnitus; ear pressure or pain; external auditory canal sensation; memory and concentration deficits; irritability and anger; and fatigue and loss of motivation
Leventhall (2003) Kasprzak (2014)	12 112	Vibration of bodily structures (chest vibration), annoyance (especially in homes), perceptions of unpleasantness (pressure on the eardrum, unpleasant perception within the chest area, and a general feeling of vibration), sleep disturbance (reduced wakefulness), stress, reduced performance on demanding verbal tasks, and negative biological effects that include quantitative measurements of EEG activity, blood pressure, respiration, hormone production, and heart rate
Havas & Colling (2011)	91	Difficulty sleeping, fatigue, depression, irritability, aggressiveness, cognitive dysfunction, chest pain/pressure, headaches, joint pain, skin irritations, nausea, dizziness, tinnitus, and stress
Horner (2013) Paller et al (2013)	78 113	Headaches, nausea, tinnitus, vertigo, and worsened sleep
Jeffery et al (2013)	92	Sleep disturbance; subjective complaints such as headaches, fatigue, temporary feelings of dizziness, and nausea; objective complaints such as vomiting, insomnia, and palpitations; annoyance; and reduced quality of life (QoL)
Jeffery et al (2014)	93	Negative impacts on the physical, mental and social well-being of people
Krogh et al (2012)	96	Annoyance (regarded as an adverse health effect associated with stress), sleep disturbance, headaches, difficulty concentrating, irritability, fatigue, and a variety of more-serious ailments
Minnesota Department of Health (2009)	55	Annoyance, reduced quality of life, sleeplessness, and headache
Howe Gastmeier Chapnik Limited (2010)	114	High levels of annoyance in a non-trivial percentage of persons, with annoyance associated with sound from wind turbines expected to contribute to stress-related health impacts in some persons

Author (Year)	Reference	Symptomatology
Nissenbaum (2013)	81	Sleep disturbances/sleep deprivation and the multiple illnesses that cascade from chronic sleep disturbance, which include cardiovascular diseases mediated by chronically increased levels of stress hormones, weight changes, and metabolic disturbances (including the continuum of impaired glucose tolerance through diabetes); psychological stresses that can result in cardiovascular disease, chronic depression, anger, and other psychiatric symptomatology; headaches, auditory and vestibular system disturbances; an increased requirement for and use of prescription medication; tinnitus; and vertigo
Nissenbaum et al (2012)	97	Increased sleep disruption, reduced mental health
Thorne (2013)	49	Sleep disturbance, headache, tinnitus, ear pressure, dizziness, vertigo, nausea, visual blurring, tachycardia, irritability, problems with concentration and memory, and panic attack episodes
Pawlaczyk-Luszczyńska et al (2005)	115	Problems with vision, concentration, and continuous and selective attention (especially in persons who are highly sensitive to low-frequency noise)
Pedersen (2011)	99	Annoyance (both outdoors and indoors), statistically related to SPLs; sleep interruption, diabetes, and tinnitus (at one of three test sites); annoyance outdoors, significantly related to sleep interruption, tension, stress, irritability (at all three sites), headache (at two sites), and undue fatigue (at one site); annoyance indoors, significantly related to sleep interruption (at all three sites), and to diabetes, headache, undue fatigue, tension, stress, and irritability (at one of three sites)
Roberts & Roberts (2013)	102	Vibration or fatigue, annoyance or unpleasantness
Shepherd & Billington (2011)	103	Annoyance, which has been linked to increased levels of psychological distress, stress, difficulty falling asleep, and sleep interruption
Taylor (2013)	58	Annoyance, stress, sleep disturbance, interference with daily living, headache, irritability, difficulty concentrating, fatigue, dizziness, anxiety, and reduced QoL
Ambrose et al (2012) Rand et al (2011)	61 116	Dizziness, irritability, headache, loss of appetite, fatigue, inability to concentrate, a need to leave the home, and a preference for being outdoors (during investigations of WTN by seasoned researchers, including acousticians)
Thorne (2011)	46	Sleep disturbance, anxiety, stress, and headaches

Author (Year)	Reference	Symptomatology
Palmer (2013)	117	Negative impacts on sleep, job stability, social relationships, care giving, pursuit of hobbies, leisure, learning, and overall health (based on interviews of residents four years after living near operational wind turbines)
Castelo Branco & Alves-Pereira (2004) Castelo Branco (1999)	118 119	Vibroacoustic disease, described as occurring only after extensive exposure to high levels of infrasound

Other sources quoted by the Physicians for Human Rights[111] note that:

> "A review of the medical literature reveals numerous adverse cognitive effects of sleep deprivation including impaired language skills-communication, lack of innovation, inflexibility of thought processes, inappropriate attention to peripheral concerns or distractions, over-reliance on previous strategies, unwillingness to try out novel strategies, unreliable memory for when events occurred, change in mood including loss of empathy for colleagues, and inability to deal with surprise and the unexpected" (pp. 22-23).

Another line of reasoning is that there is a cause-effect relationship between AHEs and ILFN from wind turbines that mirrors that in motion sickness. Kennedy et al[120] made acceleration recordings during 193 standard training mission scenarios for two moving-base flight trainers. The pilots, who were of comparable age and experience in both groups, were interviewed for motion sickness symptomatology and tested for ataxia after leaving the simulators. Motion sickness incidence was high for one of the simulators, but not for the second. Ataxia scores departed slightly from expected improvements following exposure in both simulators. Spectral analyses of the motion recordings showed significant amounts of energy in the *nauseogenic range* of 0.2 Hz. The authors concluded that simulator sickness in moving-base simulations may be, at least in part, a function of exposure to infrasonic frequencies that make people seasick. Later, von Gierke and Parker[121] advanced the notion that motion sickness may involve an intermodal sensory conflict between visceral graviceptor signals and vestibular stimulation. Schomer and colleagues[52, 86] have argued that similarities with motion sickness may explain some of the health symptoms suffered by individuals living near IWTs, given that the inner ear is capable of responding to accelerations of the kind that lead to seasickness. These accelerations correspond to frequencies in the infrasonic range, around and under 1 Hz. Schomer[66] states that

some persons affected by WTN may be responding directly to acoustic factors, rather than to non-acoustic factors, as argued by Leventhall.[14]

In a rare show of cooperation between the wind industry and independent acousticians, Pacific Hydro agreed to allow acoustician Steven Cooper, a consultant for The Acoustic Group,[25] unlimited access to its Cape Bridgewater wind project in SW Victoria, which had been in operation for about six years. The company allowed Cooper to make noise measurements and independently investigate the noise complaints of six affected residents at three residences located 650-1,600 m from the nearest turbines while the company controlled the on-off cycling of turbine operation. Given Cooper's credentials as an acoustician, the study was described as an acoustical study, as opposed to a medical study. Noise levels were based on A-, G-, and Z-weighted measurements, as well as 1/3-octave band and narrow-band measurements. Participants vacated their homes at night when necessary for Cooper to perform his acoustic studies, and they provided detailed diary accounts of their observations during on-off cycles. Those accounts included severity ratings of perceptions of noise impacts, vibration impacts, and other disturbances, which were collectively labelled as *sensations*. The sensations included headache; pressure in the head, ears, or chest; ringing in the ears; heart racing; or a sensation of heaviness. Synchronization of the timing of the residents' experiences with turbine operational data revealed heightened sensations inside their dwellings during turbine operation. Sensations were not dependent on the ability to hear or see the turbines, as residents were not aware of any of the turbines' operational characteristics. Cooper found that *sensation*, and not *noise disturbance*, was the major disturbance identified. Furthermore, sensations were most related to several different operating conditions of the turbines: at start-up, when there was an increase or decrease in power output of about 20%, and when the turbines were operating at maximum power and the wind speed increased above 12 m/sec.

Based on narrow-band data, Cooper identified a unique *wind turbine signature* (WTS) in which there was an energy peak at the blade-pass frequency and first five harmonics. Shutdown testing confirmed that the WTS, which included an amplitude-modulated signal, was present when the turbines were operating, but not in a natural environment during a turbine shutdown. Participants rated sensations as proportionally more severe as increases occurred in the magnitude of the low-frequency amplitude-modulated signature. The identification of infrasound components was consistent with earlier observations of Kelley et al.[19] Based on his findings, Cooper recommended that further studies be conducted to determine a threshold level of the WTS that protects against adverse impacts, and that the signature concept be used in medical studies by

identifying energy from the operation of wind turbines, as the A-weighted scale inside homes is of no assistance in such studies.

In consideration of the above findings and observations, it is reasonable to conclude that IWTs cause AHEs and other unwanted disturbances. We next examine the physiological mechanisms that may explain how inaudible infrasound can be harmful.

In a recent paper, Berger et el[122] concluded that ILFN levels are insufficient to induce AHEs, given the levels of ILFN typically produced by wind turbines, and that guidelines for audible noise are sufficient to protect human health. Their conclusions were based on measurements of indoor infrasound levels and low-frequency noise levels at distances >500 m that were similar to background levels. While we believe the design and major conclusions of their study to be faulty, their conclusions are consistent with the position taken by Leventhall and other wind energy advocates over the past decade.

In her original description of Wind Turbine Syndrome, Pierpont[4] described a distinctive constellation of symptoms that she believed to be due to stimulation, or overstimulation, of the vestibular organs of balance as a consequence of ILFN from wind turbines. She termed these symptoms Visceral Vibratory Vestibular Disturbance (VVVD). In a follow-up report, Pierpont[100] **s**uggested that the observed symptoms of Wind Turbine Syndrome are due to air-borne or body-borne low-frequency sounds that directly stimulate the inner ear, both the cochlea, or hearing organ, and the vestibular organs of balance and motion detection. As discussed below, research by Salt and associates shows that responses in the cochlea suppress the perception of low-frequency sound but still send signals to the brain, signals whose function is, at present, mostly unknown. The physiologic response of the cochlea to WTN is also a trigger for tinnitus and the brain-cell-level reorganization that tinnitus represents. Although cochlear and vestibular organs are housed within the same bony (otic) capsule, evolutionary adaptations have led to selective activation of auditory or vestibular hair cells. In the presence of certain disorders of the inner ear, however, anatomical defects in the otic capsule can alter the functional separation of auditory and vestibular stimuli, resulting in pathological activation of vestibular reflexes in response to sound.[123] The possibility that high-level ILFN can stimulate the vestibular organs lends credibility to Pierpont's suggestion and may explain the basis for symptoms that mimic other vestibular disorders. Physiologic responses from the otolith organs generate a wide range of brain responses, including dizziness and nausea, seasickness (even without bodily movement), fear and alerting responses such as startle and wakefulness, and difficulties with visually based problem-solving.[100] One candidate for the other destination of cochlear input from the outer hair cells may be the interface between the insula and the medial surface of the transverse (Heschl's)

gyrus, where primary hearing is experienced but not recognized as sound; the latter involves adjacent secondary areas.[124]

WTN can increase alerting responses that disturb sleep, even when people do not recall being awakened. This effect is one that clearly disturbs sleep and mental well-being out to 1,400 m (4,600 ft.) from turbines, with diminishing effects out to 3 km (3 mi.), as shown in a cross-sectional study by Nissenbaum et al.[97]

Laboratory studies conducted by Salt and colleagues have provided evidence that clearly establishes the biological plausibility that infrasound can adversely affect health. That work shows that there are mechanisms in the inner ear that are capable of transducing infrasonic energy into a neural signal that can be transmitted to the brain, where the signals can lead to such symptoms as tinnitus, dizziness, pulsations, and sleep disturbance. Those studies by Salt and associates have involved laboratory experiments funded primarily by the National Institutes of Health and conducted mostly on guinea pigs, whose ears are very similar to human ears. Basically, electrodes were inserted into the inner ears to determine which structures respond to specific types of electroacoustic stimulation. Their findings help to explain why sound that is normally inaudible can result in the kinds of negative reactions reported by people who are exposed to wind turbine ILFN. Findings from their research indicate the following:

(1) The inner hair cells (IHCs) of the inner ear, which are primarily responsible for transmitting signals to the brain that are interpreted as sound, are velocity-sensitive, and thus unresponsive to infrasound. The outer hair cells (OHCs), on the other hand, are displacement-sensitive and respond to infrasonic frequencies at levels well below those that are heard (i.e., interpreted as sound). This suggests that most IWTs produce an unheard stimulation of OHCs;[56, 125, 126] specifically, at 5 Hz the OHCs can be stimulated at sound pressures 40 dB below those that stimulate the inner hair cells associated with conscious hearing.[126]

(2) Low frequencies, which are coded in the cochlear apex, require less low-frequency SPL to be amplitude modulated, when compared to higher frequencies, which are coded in the cochlear base. This means that amplitude modulation of audible sounds by wind turbine infrasound may be the basis for complaints of those living near wind turbines, including complaints such as annoyance or feelings of throbbing and rumbling sensations. It also means that infrasound from wind turbines need not be audible to annoy people, since infrasound can amplitude modulate sounds that are within the range of audibility.[54]

(3) There are several ways that infrasound could affect people, even though they cannot hear it: (a) causing amplitude modulation (pulsation) of heard sounds, (b) stimulating subconscious pathways, (c) causing endolymphatic hydrops, and (d) possibly potentiating, or exacerbating, noise-induced hearing loss.[127]

(4) Responses to infrasound reach the brain through pathways that do not involve conscious hearing but instead may produce sensations of fullness, pressure or tinnitus, or absence of sensation. Activation of subconscious pathways by infrasound could disturb sleep.[128]

(5) The presence of other, higher-pitched sounds (between 150-1,500 Hz) can suppress infrasound.[129, 130, 131] Because the ear is maximally sensitive to infrasound when higher frequency sounds are absent, this means that WTN is most disturbing to persons inside their homes at night, when background sound levels are low and higher-pitched sounds are attenuated by walls and other physical structures.

(6) A pathway exists, through the OHCs, for infrasound to reach the brain. There, parts of the brain other than auditory centers become active and the signals are perceived as something other than sound. This pathway to the brain, which also includes the vestibular mechanism of the inner ear, means that it is biologically plausible for infrasound to produce a variety of sensations, including pulsation, annoyance, stress, panic, ear pressure or fullness, unsteadiness, vertigo, nausea, tinnitus, general discomfort, memory loss, and disturbed sleep (with chronic sleep deprivation leading to blood pressure elevation and possibly changes in heart rate).

On the above grounds, Salt dismisses the common perception that *What we can't hear can't hurt us*, and has stated unequivocally that "Wind turbines can be hazardous to human health."[132]

Interestingly, Oohashi et al,[133]using non-invasive physiological measurements of brain responses, found evidence that sounds containing *high-frequency* components *above* the audible range, or ultrasound, significantly affect the brain activity of healthy human listeners. It should not be considered implausible, therefore, that infrasonic stimulation can also activate the brain.

Recent research supports the plausibility of such effects. Bauer et al,[134] using functional magnetic resonance imaging (fMRI), found a significant response down to the 8 Hz, the lowest frequency presented, to be localized within the auditory cortex. Using magnetoencephalography (MEG), significant brain responses could be detected down to a frequency of 20 Hz. The authors hypothesized that a somatosensory excitation of the auditory cortex possibly contributes at these frequencies. In a somewhat related study, He and Krahé[135] demonstrated a significant relationship between EEG reactions under different low-frequency noise exposures and

subjective annoyance. Noise sensitivity was also found to be an important factor in most of the observations. The authors of these two studies suggested that EEG, fMRI, and MEG may serve as effective physiological measures to explain negative reactions to low-frequency noise.

Kugler et al[136] measured spontaneous otoacoustic emissions (SOAEs) before and after stimulation with perceptually unobtrusive low-frequency sound (30 Hz) and found significant changes to occur; these changes were positively correlated in frequency and level to pre-exposure status and lasted for about 2 min after stimulation. SOAEs are narrow-band acoustic signals that are spontaneously emitted by the inner ear in the absence of acoustic stimulation, and they can be recorded simply and non-invasively in the ear canal with a sensitive microphone. Otoacoustic emissions, first reported by physicist David Kemp,[137] are a by-product of active biophysical amplification by OHCs in the cochlea, persisting in relatively stable form for years under normal physiological conditions. The main task of the OHCs is to detect and mechanically amplify sound waves. In acting as a cochlear amplifier, OHCs actively generate mechanical energy, which is fed back into the cochlear travelling wave to maximize the sensitivity and dynamic range of the mammalian ear. In humans, non-invasive recordings of different classes of sound-evoked otoacoustic emissions (EOAEs) allow indirect access to OHC function, but only SOAE measurements can probe the cochlea in its natural state. The presence of OAEs signals a healthy ear, and their absence or changes in their response patterns can signal pathological function. The significance of the work by Kugler et al is that it reveals OHC function to be affected by a brief exposure to very low-frequency sound that is largely imperceptible. It also reveals that measures of perception severely underestimate OHC sensitivity. The authors concluded that direct quantifications of inner ear active amplification, as measured in their study, are well suited for assessing the risk potential of low-frequency sounds. In the present context, the study provides further support for the notion that what we can't hear can potentially affect us.

Motion sickness has been mentioned in this article as being among the variety of symptoms suffered by individuals living near IWTs. Recalling the work of Kennedy et al,[120] who found evidence of motion sickness in Navy pilots subjected to acceleration during flight simulation, Schomer et al[138] stated that it is plausible that the ear responds similarly to accelerations of a moving vehicle and acoustic pressures at infrasonic frequencies under 1 Hz, in the nauseogenic range. They suggested that the AHEs experienced as a consequence of exposure to IWTs not only bear a striking resemblance to motion sickness, but that the condition may be induced by stimulation of the otolithic organs in the vestibular system of the inner ear. That type of stimulation is purportedly worse when a person is subjected to pressure changes in a closed

cavity, including inside one's home. Further, they describe the type of research needed to verify their hypothesis.

Michaud and colleagues have recently authored a series of papers[139, 140, 141, 142, 143, 144, 145] describing a cross-sectional epidemiological study conducted under the sponsorship of Health Canada, in which they investigated the prevalence of health effects or health indicators among a sample of Canadians exposed to WTN. The studies employed both self-reported and objectively measured health outcomes. The final sample, drawn from communities in Ontario and Prince Edward Island where a sufficient number of dwellings were located near wind turbine installations, included 1,238 participates (606 males, 632 females) living between 0.25 and 11.2 km from operational turbines. One participant between the ages of 18-79 years was randomly selected from each household. The reported response rate was 78.9% and did not significantly vary across sampling strata or provinces. Modelled A- and C-weighted WTN levels reached 46 dBA and 63 dBC, respectively, and the two levels were found to be highly correlated, which suggested that C-weighted values offered no additional information beyond that offered by A-weighted values. Only minor differences across strata were reported for age, employment, and type and ownership of dwelling. WTN exposure was not found to be related to hair cortisol concentrations, blood pressure, resting heart rate, or any of several measured sleep parameters (i.e., sleep latency, sleep time, rate of awakenings, sleep efficiency). Self-reported results obtained through an in-person questionnaire did not provide support for an association between increasing WTN levels and self-reported sleep disturbance, use of sleep medication, or diagnosed sleep disorders. Similarly, no significant association was found between WTN levels and self-reported migraines, tinnitus, dizziness, diabetes, hypertension, perceived stress or any measure of QoL. However, they observed statistically significant exposure-response relationships between increasing WTN levels and the prevalence of long-term high levels of annoyance toward noise, shadow-flicker, visual impacts, blinking lights, and vibrations.

The authors of the present report, along with a number of professional colleagues with acoustical or medical expertise, have carefully analyzed the reports by Michaud and colleagues and have concluded that the research protocol of the Health Canada study reflects shortcomings that severely undercut the conclusions that were drawn in the various reports. To enumerate the major flaws in the Michaud et al reports:

(1) They incorrectly concluded that AHEs were not found when sound levels were below 46 dBA by failing to benchmark their "surrogate control group" against the general population. Proper analysis, using a proper control group, would have resulted in high correlations of these symptoms with decreasing distances to, and increasing noise levels from, wind

turbines. In reports of the sound-exposure data, sound levels of 30-35 dBA were significantly associated with increases in the prevalence rates of symptoms. This indicates that the 40 dBA currently used as the permissible threshold in Ontario and other Canadian provinces is not protective of the public's health and welfare.

(2) Key health symptoms were reported primarily for non-vulnerable populations, in that younger individuals and individuals who had left their homes were excluded from participation. Those exclusions invalidate the study as a reflection of health conditions in the general population.

(3) Evidence provided by the World Health Organization[38] showing that exposure to noise from vehicles, railways, and aircraft is linked to serious physiological and psychological health effects at sound levels of 40 dBA and higher, and that lower levels are needed to protect the more vulnerable members of the population, was ignored in the Health Canada study. The finding that AHEs did not occur below 46 dBA should have been a warning sign to the researchers that their study design, their analyses, or both, were flawed.

Statement 4: Setback distances of 1,000-1,500 ft. (approximately 0.3-0.5 km) are sufficiently safe to protect humans from harm, regardless of height or other physical characteristics of the IWTs.

Many zoning ordinances that regulate IWTs specify the height of the turbine tower from its base to blade tip, plus 10% to 100%, as a setback distance that sufficiently protects residents against a catastrophic event such as a tower failure, a falling blade, or ice throw. Some ordinances specify a distance of twice the base-to-blade tip height, roughly 900 ft., while others arbitrarily specify slightly longer distances such as 1,500 ft. or 0.5 km. Most of the reported health symptoms have been observed at distances much greater than these setback distances. One can deduce, therefore, that *setbacks intended to protect physical health from mechanical or other traumatic failure of a wind turbine component are not adequate to protect general health and well-being*.

While terrain, weather patterns, number and size of turbines, and the turbine array itself can influence the ILFN emitted from IWTs, the two major factors are turbine size and distance from the receiver. Distance is the only practical means of achieving acceptable sound levels, as controlling the noise through the erection of barriers or enclosures near the source or receiver are not feasible or effective. Because infrasound is involved, closing windows, insulating buildings (including residences), and sleeping in basements are not normally helpful in attenuating the noise, and there is less likelihood that the emissions will be masked by wind at ground level.[60, 146] Noise levels must be measured by qualified personnel, and the sound level at the residence—or arguably at the property line—is the key element in protecting the health of residents.

To protect human health, a number of researchers have recommended specific distances, while others have recommended limitations on sound levels, irrespective of the distances needed to achieve those levels. Such recommendations are based on observed or reported complaints of AHEs. Though quite specific, the recommendations vary somewhat widely, as shown in Table 2.

The recommendations in Table 2 include boundaries of distance and noise levels of 0.5-2.5 mi. and 30-40 dB, respectively, that are believed by various professionals to protect human health. Although the use of maximum permissible noise levels appears to be the optimal approach for protecting the greatest number of people, the existence of multiple acoustic and environmental factors complicates our ability to recommend a single distance or noise level that protects most residents. Those factors are covered elsewhere in this review.

Table 2. Recommended minimum siting distances and maximum noise levels of industrial wind turbines, based on the protection of human health.

Author (Year)	Reference	Distance/Level
Pierpont (2009) Kamperman & James (2008)	4 83	Distance of 1.25 mi, or 2 km
Nissenbaum et al (2012)	97	Minimum distance of 0.87 mi, or 1.4 km, based on experimental conditions studied
Harry (2007)	74	Minimum distance of 1.5 km from nearest turbine
Frey & Hadden (2007)	90	2 km between family dwellings and IWTs of up to 2-MW installed capacity, with greater separation for a wind turbine greater than 2-MW installed capacity
Shepherd & Billington (2011)	103	4 km, to protect against amplitude-modulated turbine noise
Position of the National Institute of Public Health-National Institute of Hygiene on wind farms (2016)	147	A minimum distance of 2 km of wind farms from buildings
Cummings (2011)	39	Distance of ½ mi or greater; noise levels within 5-10 dB of existing background conditions; sound levels below 40 dBA, or even 30-35 dBA, as levels of 40 dBA or higher trigger large numbers of noise complaints
World Health Organization (2009)	38	Outdoor sound levels <40 dBA, with vulnerable populations expected to be most affected

Author (Year)	Reference	Distance/Level
Knopper et al (2014)	148	Sound levels <40 dBA, for non-participating receptors
Horner (2013)	78	Sound levels <30 dB
Harrison (2011)	42	Sound levels limited to 35 dBA at nighttime and 40 dBA during daytime hours; 5-dBA and 4-dBA penalties, respectively, imposed for the periodic or impulsive character of turbine noise and for uncertainty in noise prediction
Thorne (2013)	49	Sound levels <32 dB LAeq outside a residence

Statement 5: Annoyance is a nuisance, but it is not a health issue.

In the past few years, the position of the wind industry has changed from a blanket denial of any impact from noise to admitting that IWT noise is annoying to a substantial portion of exposed populations, and that annoyance from ILFN is a well-accepted phenomenon. While Bolin et al[18] and Ellenbogen et al[82] downplay the relationship between annoyance and WTN, the larger research community has documented that ILFN from wind turbines and other sources leads to annoyance.[12, 14, 15, 19, 21, 37, 38, 40, 42, 46, 49, 55, 58, 59, 63, 64, 74, 78, 80, 81, 90, 92, 94, 98, 99, 102, 118, 146, 149, 150]

Several investigators have concluded that annoyance increases in a dose-response relationship as distance from turbines is reduced.[44, 89, 146] A number of studies have concluded that noise annoyance appears to be worse when nearby residents have negative attitudes and when visual annoyance or intrusive sound characteristics are also involved.[e.g., 44, 65, 112, 146, 151] However, the annoyance from visual stimulation and the annoyance from noise may be entirely independent. The two irritants do not have to be linked. The common factor is that as one moves closer to a wind turbine, it is perceived as both larger *and* louder. One recent study,[152] which compared visual, audible noise, and combined visual-auditory representations of wind turbines, found noise sensitivity to correlate with both noise and visual annoyance. That study also demonstrated a reciprocal influence between auditory and visual stimuli, but in essentially a direction opposite that predicted by earlier studies of wind turbine visibility and noise. Interestingly, the study showed that a visual stimulus had a mitigating effect on noise annoyance, while an auditory stimulus had a disturbing effect on visual annoyance. This finding supports the idea that humans perceive the environment holistically and in context of all perceptual information. In suggesting that auditory and visual features are processed in close interaction, it forces us to question the idea that annoyance from WTN arises largely because the turbines are visible. Given our current

state of knowledge, it seems reasonable to accept that people can be annoyed by auditory and visual irritants independently, even though there may be interactions between them.

Annoyance occurs in residents living near wind turbines at lower sound levels than for transportation noise, industrial noise, or other sources.[38, 42, 43, 58, 64, 93] Perception and annoyance have been found to be associated with both urbanized and rural terrains.[149] Pedersen et al,[146] in summarizing survey data on annoyance from wind turbines in the Netherlands and Sweden, found that 25% or more of all respondents were annoyed by levels of 40-45 dBA, while about 18-20% were very annoyed by those levels. A total of 18% found outdoor levels of 35-40 dBA to be rather annoying or very annoying outdoors and 8% found those levels to be rather or very annoying indoors. For outdoor levels of 40-45 dBA, 18% and 16% were rather or very annoyed outdoors and indoors, respectively. Because such surveys tend to emphasize *noise* from wind turbines, results often reflect levels of annoyance that relate more directly to audible sounds, as opposed to infrasound.

While few would argue that noise from wind turbines annoys a substantial percentage of nearby residents, there is disagreement over whether it leads to AHEs. Colby et al[15] stated that:

> "…there is no evidence for direct physiological effects from either infrasound or low frequency sound at the levels generated from wind turbines, indoors or outside" (p. 3-8).

They reasoned, therefore, that annoyance is not a pathological entity. Their basic contention was that although wind turbines produce infrasound, it is not harmful because people can't hear it. They contended that while some people may be annoyed by the sound from wind turbines — presumably audible sound—annoyance is primarily due to the fluctuating nature of the noise and personal attitudes. In their view, it is a psychological reaction, as opposed to a direct physiological reaction to sound. As noted above, however, several investigations[44, 89, 146] have found a dose-response relationship to exist between measured or estimated sound levels and annoyance. IWT noise emissions have been found to be a mediator between exposure and sleep disturbance and psychological distress,[89] and to be directly associated with stress.[e.g., 104]

The documented health symptoms from exposure to wind turbines are often stress-related and exacerbated by sleep disorders; they appear to be mediated through both direct and indirect pathways, and the result can be serious harm to human health.[92] There is an association between WTN, stress, and well-being, and this association is a potential hindrance to psycho-physiological restitution.[58, 98] The WHO has described annoyance as a critical health effect, in that in some people it is associated with stress, sleep disturbance, and interference with daily living.[38] A range of symptoms, often described as stress responses, have been associated with

WTN in people living in the vicinity of wind projects. As Pierpont[4] and others have noted, these symptoms include headache, irritability, difficulty concentrating, fatigue, dizziness, anxiety, and sleep disturbance. Regardless of whether the perceived impacts of noise from wind projects are physiological or psychological in nature, they are considered to cause AHEs through sleep disturbance, reducing the quality of life and serving as a source of annoyance that sometimes leads to stress-related symptoms.[71] The potential of environmental noises to induce stress reactions is well known. These reactions are dependent on how the noises are interpreted in the central nervous system; medical effects such as increased blood pressure, for example, are known to result from prolonged noise exposure.[153]

Generally, models that explain the relationship between noise and health fall into two broad categories, based on pathways that are *direct* or *indirect*. Figure 1, which is a modification of a figure from Shepherd et al,[50] depicts three models, one direct and two indirect, that have been described in the contemporary literature. The first (Fig. 1a) represents a direct pathological relationship between an environmental parameter (e.g., noise level) and a target organ that affects health. For example, in this model, noise can affect both cognition and sleep, and thus directly impact health. An alternative approach (Fig. 1b) distinguishes between direct health effects and psychosomatic illness. This approach suggests that any physiological illness coinciding with the onset of WTN may be caused by a negative psychological response to the noise, and not the noise *per se*. Any anxiety or anger resulting from the presence of WTN induces stress and strain that, if maintained, can eventually lead to AHEs. Another explanation that involves an indirect pathway from sound to health effects is one that is consistent with the WHO's definition of health.[38] That model (Fig. 1c) recognizes the role of environmental moderators, or mediators, in the determination of whether a sound is (unwanted) noise, and, if so, whether or not the noise

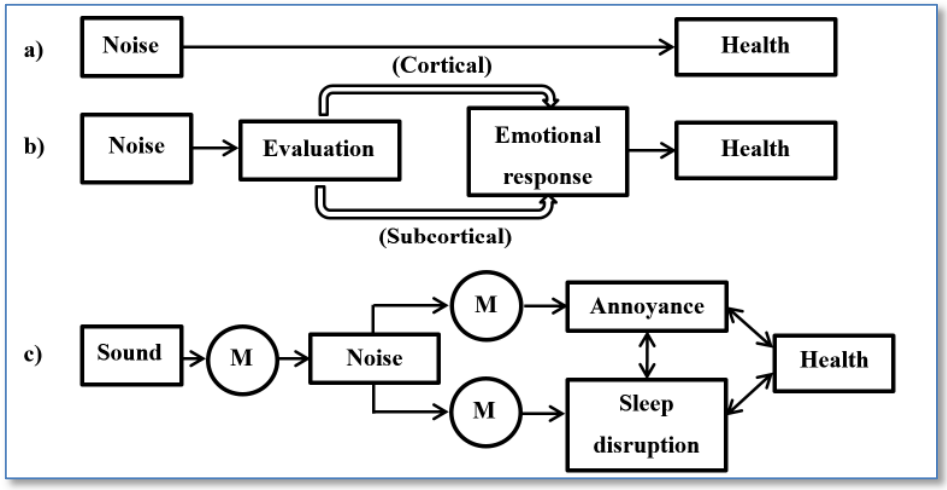


Figure 1. Three models representing the relationship between noise and health: the biomedical model (a) stipulating a direct causal relationship and indirect models (b and c) containing moderators and mediators (Adapted from original source and used with permission of first author, Daniel Shepherd).[50]

negatively impacts health. Mediators include factors such as degree of urbanization, house type, and sound level, and psychological and demographic moderators such as age, gender, education, employment status, attitudes to wind energy, noise sensitivity, and whether the individual receives a monetary return from the turbines. In this model, sleep disruption plays a major role in producing AHEs, with annoyance and sleep disruption being intervening factors between noise and AHEs for some people.

Authors of a recent study,[154] which focused on the province of Ontario, acknowledge both the link between annoyance and health and the possibility that wind projects can exacerbate psychosocial health problems through social processes such as intra-community conflict. They list socially mediated health concerns, distribution of financial benefits, lack of meaningful engagement, and failure to treat landscape concerns seriously, as the core stumbling blocks to a community's acceptance of wind energy development.

Statement 6: Noise cannot account for all of the complaints of people living in the vicinity of wind turbines; there must be another, unknown reason for the complaints.

Havas & Colling[91] have observed that wind turbines generate electromagnetic waves in the form of poor power quality (*dirty electricity*) and ground current, and speculate that these waves can adversely affect those who are electrically hypersensitive. McCallum et al[155] performed magnetic field (EMF) measurements in the proximity of 15 Vestas 1.8-MW wind turbines, two substations, various buried and overhead collector and transmission lines, and nearby homes in the vicinity of Goderich, Ontario, during high-wind, low-wind, and shut-off operational stages. They concluded that there is nothing unique to wind farms with respect to EMF emissions, finding that magnetic field levels in the vicinity of wind turbines were lower than those produced by many common household electrical devices and that levels were well within any existing regulatory guidelines with respect to human health.

Although at least a few of the health symptoms mentioned above have been self-reported by individuals who are exposed to electromagnetism, clinical trials to date suggest the link between health complaints and exposure to electromagnetism to be a purely psychological one, or a nocebo effect, in that self-described sufferers of electromagnetic hypersensitivity are unable to distinguish between exposure and non-exposure to electromagnetic fields.[156] Another review paper[157] found no convincing scientific evidence that symptoms are caused by electromagnetic fields. However, one cannot rule out that the design of the experiments upon which the review papers drew their conclusion may have missed some unique characteristic that could account for the anecdotal evidence. (See our earlier statements describing how failure to identify infrasound

pulsations as a causal factor for perception at pressure levels below those needed for audibility have led some to conclude that IWT infrasound causes no harm.) When faced with health complaints from families who live near IWTs, especially when there are repeated instances of symptoms that wax and wane with alternating sequences of exposure and non-exposure, and especially when those families have taken the drastic step of abandoning their homes, it is unreasonable to argue that noise is not the cause of the complaints. Even if other factors such as electromagnetic waves are the root cause of a given complaint, it is still the placement of turbines too close to those residents that is the most likely cause of the problem.

Unfortunately, not as much is known about the effects of electromagnetism as is known about ILFN. At this point in time, therefore, it is reasonable to conclude that more people who live near wind turbines are negatively affected by ILFN than by hypersensitivity to dirty electricity or ground current, as measurable levels of ILFN from wind turbines are highly associated with individual complaints. When Stigwood et al[57] studied and analyzed complaints at over 75 wind developments in the U.K., they found that identifying the problems was straightforward, occurrence was common (i.e., some residents reported problems in all developments), all developments generated excess amplitude modulation (AM), and AM was the cause of the vast majority of the complaints. These findings have recently been reinforced by Cooper's work[25] in Australia.

Statement 7: Infrasound from wind turbines is sufficiently correlated to the A-weighted sound emissions to allow an A-weighted model to be used to predict how much infrasound is present in homes.

This statement is not typically stated explicitly, but it is one that is inherent in the positions commonly taken by wind energy advocates and regulatory bodies through their interpretations and acceptance of research on WTN, which is based largely on A-weighted levels. As noted in many previous papers, including one of our own,[101] the continued use of the A-weighting scale in sound level meters is a major basis for misunderstandings that have led to acrimony between advocates and opponents of locating wind turbines in residential areas. The dBA scale was devised as a means to incorporate into measurements of environmental and industrial SPLs the inverse of the minimum audibility curve[158] at the 40-phon level. It is typically used, though, to specify the levels of noises that are more intense, where the audibility curve becomes considerably flattened, obviating somewhat the need for A-weighting. Use of the A-weighted scale is mandated or recommended in various national and international standards for measurements that are compared to damage-risk criteria for hearing loss and other health effects resulting from occupational or environmental noise exposure. It drastically reduces sound-level

readings in the lower frequencies, beginning at 1,000 Hz, and reduces sounds at 20 Hz by 50 dB. For WTN, the A-weighting scale is especially inappropriate because of its devaluation of the effects of ILFN. Many authors have commented on its inadequacy. For example, Pederson and Persson Waye[159] state:

> "There is… support both from experimental and field studies that intrusive sound characteristics not fully described by the equivalent A-weighted sound pressure level contribute to annoyance with wind turbine noise" (p. 4).

A number of researchers have recommended comparing C-weighted measurements to A-weighted measurements when considering the impact of sound from wind turbines.[10, 12, 37, 61, 67, 75, 76, 83, 101] According to these sources, the presence of infra- and low-frequency sound is generally indicated when the difference between levels on the two scales differs by 10-20 dB. When such differences are observed, the use of third-octave or linear-scale measurements is typically recommended (for example, see Shepherd et al[50]). Other weighting scales have been suggested for wind turbine applications, but at present, linear-scale or narrow-band measurements, used in conjunction with a conventional sound level meter (with low-frequency microphone) and micro barometer, offer the best potential for accurately and completely describing the soundscape in the vicinity of IWTs.

As noted above, Cooper[25] has suggested that A-weighted levels, measured inside homes, are not likely to be useful indicators of AHEs. That report concluded that A-weighted levels are not a valid index of protection from AHEs and recommended the further exploration of a newly developed *wind turbine signature* scale, based on the discovery of its capability to quantify the amplitude-modulated peak energy in the infrasonic frequency region. That scale was shown to be directly linked to a variety of adverse bodily sensations when nearby turbines were operating or undergoing transitions in operation.

Although A-weighted sound level measurements have been the *sine qua non* for specifying environmental and occupational noise levels for many decades, we must recognize the inherent inadequacies of applying the A-weighting scale to quantifying noise emitted by IWTs. Bray[160] goes even further by noting that people, and not electronic devices, are the ultimate analysts of data that affect their responses to sound, making the point that people's responses should be given the credence they deserve, and not be devalued when physical measurements fail to confirm them.

Statement 8: Wind Turbine Syndrome has not been accepted as a diagnostic entity by the medical profession, so medical professionals cannot diagnose or treat it.

Currently, Wind Turbine Syndrome is not included in the International Classification of Diseases (ICD) coding system, which is used globally for purposes of establishing categories for diagnosing diseases and other health conditions, and as a basis for reimbursing medical providers for diagnostic and treatment services. Yet, of the 10 symptom sets comprising Pierpont's Wind Turbine Syndrome,[4] at least seven are included as a category or subcategory in the newly revised (ICD-10) coding system. The fact that the syndrome itself is not included may be due to its relatively recent discovery, but is more likely due to the fact that the syndrome consists of symptoms that are highly variable from person to person and affect a minority of the exposed population.

Especially in legal proceedings, it is important to distinguish between the terms *differential diagnosis* and *causation assessment*. It is the latter that is most often the subject of such proceedings. Attorneys and expert witnesses often get the terms confused. Differential diagnosis refers to the identification of disorder(s) that may account for a particular complaint or symptom complex. It rarely deals with the external cause of the disorder. Causation assessment, on the other hand, typically requires an evaluation of whether potential causative agents have irritating properties; a determination of the approximate amount of exposure, or dose, of that agent, and the timing between exposure (and non-exposure) and the occurrence of symptoms; and an assessment of whether alternative potential causes of the disorder can be ruled out. These latter steps are not necessarily considered part of the diagnosis.

Notwithstanding the fact that Pierpont herself is a practicing pediatrician, a couple of recent developments would appear to increase the prospect that medical personnel will soon be able to establish Wind Turbine Syndrome, by that or a similar label, as a clinical entity caused by exposure to WTN. Dr. Robert McMurtry, a physician who is a special advisor to the Canadian Royal Commission on the Future of Health Care, and a long-time advocate for more effective public involvement in healthcare policy, recently published a set of highly specific criteria for establishing such a link. McMurtry[161] originally proposed a case definition that identifies first-, second-, and third-order criteria, as well as specified circumstances and symptoms that must be established before AHEs can be attributed to wind turbine exposure. According to those criteria, probable AHEs are present when:

(1) All four of the following first-order criteria are met: (a) The individual resides within 5 km of IWTs, (b) Health status is altered following the start-up of or initial exposure to, and during

the operation of, IWTs (a latent period of up to 6 months may be allowed), (c) Amelioration of symptoms occurs when more than 5 km from the environs of IWTs, and (d) Recurrence of symptoms occurs upon return to the environs of IWTs within 5 km.

(2) At least three of the following second-order criteria are met (occurring or worsening after the initiation of operation of IWTs): (a) Compromised quality of life, (b) Continuing sleep disruption, difficulty initiating sleep, and/or difficulty with sleep disruption, (c) Annoyance producing increased levels of stress and/or psychological distress, and (d) A preference to leave the residence temporarily or permanently for sleep restoration or well-being.

(3) At least three specified symptoms occur or worsen following the initiation of IWTs, those symptoms referred to as third-order criteria that fall within the following categories: (a) Otological and vestibular disorders, (b) Cognitive disorders, (c) Cardiovascular disorders, (d) Psychological disorders, (e) Regulatory disorders, or (f) Systemic disorders.

To be confirmed as AHEs from WTN exposure, McMurtry indicated that consideration should be given to other stressors present in the community, that sleep studies be carried out if at all possible, and that a licensed physician be able to rule out alternate explanations for AHEs. These alternate explanations include substantial barometric changes from prevailing winds, a stressful home environment, and psychological and/or mood disorders, all of which can normally be ruled out when symptoms subside or disappear when the individual leaves the vicinity of the wind turbines. Apart from these three factors, he indicates that there are very few, if any, other health conditions that can mimic those caused by exposure to wind turbines and at the same time meet the three orders of criteria outlined in his case definition. More recently, McMurtry and Krogh[162] published a revised case definition, in which the third-order criteria—which are commonly present—are not considered essential elements. In both papers, the authors acknowledged that the identification of IWTs as the cause of adverse health symptoms is a complex emerging issue that requires further study to validate the criteria. They proposed key elements that ought to be included in any model used to assess the validity of the case-definition criteria.

McCunney and colleagues[163] have challenged those case definitions as having poor specificity, leading to a substantial potential for false-positive assessments and missed diagnoses. A potential fallacy in this challenge is that the authors unnecessarily conflate the concept of case definition for medical practitioners with that of an epidemiologic research plan. The case definitions presented by McMurtry[161] and McMurtry and Krogh[162] represent guidelines for medical doctors whose individual patients are experiencing new or unusual symptoms. It is erroneous to purport that a physician’s mental process can be encapsulated into a set of equations, especially

during the earliest stages of developing a case definition. The criticisms of these early case definitions should not deter physicians from attempting to evaluate and treat patients who report AHEs after living in the vicinity of IWTs. This area may indeed benefit from further study. Our view, however, is that such criteria provide an adequate starting point for guiding medical practitioners.

Dr. Steven Rauch, an otolaryngologist at the Massachusetts Eye and Ear Infirmary and a professor at Harvard Medical School, recently declared that he believes Wind Turbine Syndrome to be a real phenomenon.[164] As reported by numerous websites and newspapers, multiple patients have sought treatment from him for AHEs stemming from consistent exposure to IWTs. Rauch compares the syndrome to migraine headaches and believes that people who suffer from migraines are among the most sensitive to the effects of WTN, and he has stated that the wind industry aims to suppress the notion of Wind Turbine Syndrome by blaming the victim.

Given these developments, it is possible that the medical profession may someday embrace Wind Turbine Syndrome—by that or another name—as a clinical entity. This prospect is encouraging, as such acceptance by the profession will facilitate efforts to protect individuals from the harmful health effects of exposure to IWTs. Even though it may be some time before such a diagnostic label is formally acknowledged as an ICD code, it is currently possible for physicians to identify many of the specific symptoms associated with wind turbine exposure and to bill for diagnosing and treating those symptoms, with or without regard for their underlying cause. Paradoxically, it is apparently the case that the most effective treatment for AHEs associated with WTN exposure is non-medical in nature; it is to recommend that patients physically remove themselves from the vicinity of IWTs.

Statement 9: Peer-reviewed epidemiological literature is the only acceptable basis for proving a causative relationship between wind turbine noise and adverse health effects.

This issue runs as a thread through virtually all the other issues addressed in this paper, as it relates to the kind of scientific evidence frequently called for, especially in legal settings, to prove that IWTs are the cause of AHEs. While personal physicians of complainants in legal cases are often considered the only expert witnesses qualified to establish *specific causation*, others can testify to *general causation*, which is the methodology by which scientists determine whether or not an agent is responsible for producing a particular disorder. In general, this requires evaluation of the scientific and medical literature to identify documented instances of health-related conditions arising from exposure to specific agents and, when available, the dose-response relationships between agents and their effects. This process is highly similar to that of

causation assessment, as explained above, and it does not necessarily require the input of a complainant's personal physician, although such input may be helpful. In legal cases involving WTN, it is critical that expert witnesses in acoustics and health be able to reconcile their positions with the reports and standards of the WHO,[165] the International Organization of Standards (ISO),[166] and the American National Standards Institute/Acoustical Society of America (ANSI/ASA)[167] that have linked low-frequency noise to symptoms of the type involved in complaints. These acoustical documents and research reports are seldom, if ever, included in literature reviews used by the industry to deny potential health risks. If challenged on the validity of the available evidence, acousticians need to be knowledgeable of the relevant acoustical standards and make sure that they are understood by all parties. In reality, the wind industry's almost universal refusal to cooperate with researchers has made it virtually impossible to conduct proper acoustical or epidemiological studies. The industry has been largely unwilling, or claims it is unable, to shut down or modify operations of its turbines for experimental purposes. To date, such a situation has rarely occurred, most notably in the case of the Cape Bridgewater study.[25]

The veracity of Statement 9 is strongly challenged by the classic address by Sir Austin Bradford Hill,[168] Professor Emeritus of Medical Statistics, University of London, to the newly founded Section of Occupational Medicine of the Royal Society of Medicine. In his essay, Hill shared his thinking about association and causal evidence surrounding environmental disease. He posited nine elements that are critical in establishing causation:

(1) *strength* (strength of observed relationships),
(2) *consistency* (consistency, or repeatability, of relationships, based on observations by different persons, in different places, under different circumstances, and at different times),
(3) *specificity* (causation is indicated if the association is limited to specific individuals and to particular sites and types of disease and there are no associations with other factors),
(4) *temporality* (there is a clear temporal relationship between outcomes and periods of exposure and non-exposure),
(5) *biological gradient* (a dose-response relationship exists),
(6) *plausibility* (causation is more likely when certain outcomes are biologically plausible, or possible, a caveat being that plausibility depends on the biologic knowledge of the day; this element is best expressed in the statement: "When you have eliminated the impossible, whatever remains, however improbable, must be the truth" (p. 10),

(7) *coherence* (the cause-and-effect interpretation of data should not seriously conflict with generally known facts of the natural history and biology of the disease),

(8) *experiment* (experimentation or semi-experimental evidence, even if only occasional, can reveal the strongest kind of evidence for causation), and

(9) *analogy* (the recognition that similar cause-effect relationships have occurred under similar conditions).

Hill states:

> "What I do not believe (is) …that we can usefully lay down some hard-and-fast rules of evidence that must be obeyed before we can accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question – is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?... No formal tests of significance can answer those questions. Such tests can, and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the 'proof' of our hypothesis" (p. 299).

Hill makes this final observation in his essay:

> "All scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time" (p. 300).

Extrapolating from Hill's essay, the totality of our knowledge gained from the available evidence must be considered when examining the link between WTN and AHEs. Fortunately, in addition to experimentation, this evidence includes simple tools that are useful, particularly if we are willing to recognize their collective value. Those tools begin, but do not end, with adverse health reporting.

Dr. Carl Phillips, a specialist in epidemiology and science-based policy making, and a former professor of public health, has stated:[169]

> "In cases of emerging and unpredictable disease risk, adverse event reports are the cornerstone of public health research. Since it is obviously not possible to study every possible exposure-disease combination using more formalized study methods, just in case an association is stumbled on, collecting reports of disease cases apparently attributable to a particular exposure is the critical first step" (p. 304).

He gives familiar examples of hazards revealed by adverse event reporting, including infectious disease outbreaks and side effects from pharmaceuticals. He points out that:

> "Pharmaceutical regulators rely heavily on clearinghouses they create for adverse event reporting about drug side effects (and often become actively concerned and even implement policy interventions based on tens of reports)" (p. 304).

Phillips indicates that the case of wind turbines and health fits the same pattern. He describes adverse event reporting as a special type of case study—sometimes denigrated as anecdotes—that generally reports on the rapid onset of a disease that appears to be related to a particular exposure. He advocates self-reporting of adverse events as a highly useful approach in studying the health effects of wind turbines. In addition, he advocates the use of case-crossover experiments as useful and well-accepted sources of epidemiologic information, stating that they are intuitively recognized by both experts and laypersons seeking to assess whether an exposure is causing specifiable outcomes.

Other forms of evidence, all considered scientific, have been or can be used to determine the impacts of WTN on health. These include case studies, case-series studies, and other pre-experimental, quasi-experimental, true experimental, correlational analysis, and single-subject designs. Single-subject designs, like the case-crossover design used by epidemiologists, can also be applied across multiple individuals to reveal relationships between specific interventions and changes in outcomes in individuals or groups. In both designs, subjects serve as their own controls while crossing over from one treatment to another (A vs. B) during the course of the experimental trial. Both are flexible designs and useful in studying events that are infrequent or sporadic. Numerous individuals living near IWTs have experienced health symptoms that have waxed and waned during repeated cycles of exposure (A) and non-exposure (B), which indicates that the wind industry has unwittingly engaged individuals and families worldwide in a series of quasi-empirical studies for many years, without obtaining informed consent from un-enrolled subjects, typically by downplaying any concerns about potential health impacts. The outcomes from these experiments offer some of the strongest evidence available that there is a causative link between WTN and AHEs in some individuals.

According to the WHO,[170] epidemiology is "the study of the distribution and determinants of health-related states or events (including disease), and the application of this study to the control of diseases and other health problems. Although the randomized clinical trial (RCT) is generally considered the *gold standard* of designs for establishing causation, various methods can be used to carry out epidemiological investigations: surveillance and descriptive studies can be used to study distribution; analytical studies are used to study determinants." Epidemiology uses a

systematic approach to study the differences in disease distribution in subgroups and allows for the study of causal and preventive factors.[171] Descriptive epidemiological studies describe the occurrence of outcomes, and analytical studies reveal associative linkages between exposure and outcomes. Descriptive studies include primarily case reports and case-series studies. Analytic designs include experimental studies such as community trials and randomized controlled clinical trials, and observational studies, in which observations can be made retrospectively, concurrently, or prospectively. Observational studies include those in which either grouped (i.e., ecologic) or individual data are collected, the latter normally favored by the scientific community. Those designs involving individual data include cross-sectional, cohort, case-control, and case-crossover studies. Although epidemiological studies rely on statistical analyses of relationships between exposure to specific agents and AHEs in relatively large samples of the population, they are not aimed at revealing the cause of a disease or disorder in specific individuals. A cogent summary of research designs used in *evidence-based medicine* can be found online.[172]

Cross-sectional studies survey exposures and disease status at a single point in time in a cross-section of the population. They measure prevalence, not incidence, of a disease process, and have the disadvantage of difficulty in establishing the temporal sequence of exposure and effect. Also, rare and quickly emerging events may be difficult to detect. Their major advantage is that data can be collected at the same time on all participants, which means the study can be completed in a relatively short time. Notably, several cross-sectional investigations of the effects of WTN exposure have been reported.[44, 97, 98, 99, 104, 149] These studies serve as major contributions to the scientific literature on the subject.

Cohort studies involve an observational design in which a sample of the population is followed to discover new events.[75] They compare individuals with a known risk factor or exposure with others without the risk factor or exposure and aim to determine whether there is a difference in the risk, or incidence, of a disease over time. They tend to be the strongest observational design, especially when the data are collected prospectively, as opposed to retrospectively. Compared to the cross-sectional design, cohort studies tend to require more time, which partially explains the paucity of such studies involving wind turbine exposure.

Case-control designs compare exposures in diseased cases vs. healthy controls from the same general population. Specific disease states must be known prior to initiation, and exposure data must be collected retrospectively. This design can be applied to cases of IWT exposure, despite the fact that it requires the cooperation of affected and unaffected segments of the same population, a circumstance made difficult by attempts on the part of energy companies to

maintain confidentiality and privacy as a means to facilitate wind turbine development in areas involving both participants and non-participants.

In case-crossover studies, which are a special type of case-control design, the *case* and *control* components reside in the same individual. This design is especially useful in investigating triggers of a disease process within an individual. In the behavioral sciences, it is commonly referred to as a *single-subject design*, as already described. The case component signifies the hazard period, which is the time period before the disease or event onset (e.g., exposure to IWTs), and the control component signifies a specified time interval other than the hazard period, namely the non-exposure interval. As already mentioned, wind companies themselves have unwittingly subjected residents to the basic conditions of this design, and results clearly suggest that exposure to WTN leads to a variety of health complaints in some individuals and families. Phillips[79] argues that:

> "A case-crossover study is one of the most compelling sources of epidemiologic data. It consists of observing whether someone's outcomes change as their exposure status changes. This is often not possible because the outcomes only happen a single time as a result of long-term exposure (e.g., cancer) or the exposure cannot be changed. But the observed effects of turbine exposure lend themselves perfectly to such studies because the exposure is transient and the effects, while not instantaneous in their manifestation or dissipation, are generally transient over a period of days or weeks at most. Thus, unlike a case of a lifelong exposure or non-transient disease, where we can only make one observation about disease and outcome per person, the effects of turbines allow multiple observations by the same person, including experimental interventions" (p. 305-306).

Turning to experimental designs, the clinical trial is considered the ideal design to test hypotheses of causation. In a clinical trial, the investigator has control of the exposure to an extent similar to a laboratory experiment. The subjects generally are randomly assigned to one of at least two groups, an experimental and a control group. The experimental group receives the treatment (i.e., exposure in the case of wind turbines) and the control group does not; instead, it usually is subjected to a condition that simulates a generic treatment of some type, and the purpose and procedures of the control condition are explained only after the experiment ends.

A fully developed clinical trial of residents who live near wind turbines has never been conducted, and the reasons are fairly clear if we consider the circumstances surrounding such a trial. In a rigorous trial done to establish the link between AHEs and WTN, the investigator would randomly assign hundreds of people selected from the general population—including adults and children, elderly adults, and chronically ill adults—to either an experimental or a control group. Randomization would control for pre-experimental biases toward or against wind

energy, as well as for other factors that could confound the outcome. The experimental group would be required to spend a significant period (day and night for weeks or months) in homes located between approximately 1,000 ft. and several miles from the nearest wind turbine. The control group would be required to take up residence several miles or more away from the nearest wind turbine, where they would presumably be free from any effects due to extraneous noise or infrasound. Homeowners who leave their homes, as well as research participants occupying those homes, would have to adjust to new residences and modify their work and school activities, eating patterns, and overall lifestyles. Participants in both groups and at least some of the homeowners who vacate their homes for the experiment would have to be reimbursed for their participation, as well as for the costs incurred as a result of their participation, and the research staff would also have to be paid. To maintain some control across sites, the average age and health status within each group should be equivalent, and data would have to be gathered regarding such factors as turbine size, wind speed and other weather conditions, length of time the turbines were operating, terrain, the exact distance of each participating family from the nearest turbines, and actual noise levels present outside and inside the homes. Scientifically rigorous methods for measuring low-frequency noise and infrasound would have to be agreed upon and used. Although self-report via a survey technique could be part of the experimental design, medical examinations and physiological measurements, including sleep studies, should also be incorporated into the research protocol.

While possible, it is not practical to expect such a study design, in its ideal form, to be implemented. Aside from the difficulty of recruiting and enrolling enough families in enough geographic areas to form statistically strong samples, legitimate ethical questions should be raised regarding the exposure of individuals, especially children and other vulnerable individuals, to potentially hazardous conditions. One might conjecture, however, that consent to participate in such a study could be gained from fully informed adults because the effects of WTN are widely believed to be reversible when a period of non-exposure follows a period of exposure.

Statement 10: The nocebo effect, a manifestation of psychological expectations, explains why people complain of adverse health effects when living near wind turbines.

This statement is the core position of some of the most outspoken critics of the view that IWTs cause AHEs. Any discussion of this statement should begin with an acknowledgment that human behavior and beliefs are highly variable and are often driven by psychological and emotional influences, and not just by observations, logic, intellectual knowledge, or cognitive thought processes. It is not surprising, therefore, that some have adopted the view that negative reactions

to wind turbines are based primarily or solely on psychological expectations. Our analysis of the limited literature on the topic leads us to state unequivocally that it is lacking in scientific rigor. Even if the results were as described, the existing studies and observations do not support a conclusion that psychological forces are the only or even primary explanation for most of the negative reactions toward IWTs. Here, we will critically review four papers, all supporting a psychological explanation for the negative reactions.

Chapman et al[173] tested four hypotheses relevant to psychogenic explanations of the variable timing and distribution of health and noise complaints about wind farms in Australia. They obtained records from the wind companies of complaints about noise or degraded health from residents living near 51 wind projects operating between 1993 and 2013 and corroborated those records with complaints documented by three government public agencies, news media records, and court affidavits. Complaints were expressed as proportions of estimated populations residing within 5 km of a wind project. The authors concluded that historical and geographical variations in complaints were consistent with psychogenic hypotheses expressing health problems as "communicated diseases," with nocebo effects likely to play an important role in the etiology of complaints.

Nocebo effects are commonly described as being the opposite of placebo effects. While the *placebo effect* usually refers to a positive reaction to an inert substance—the placebo—the *nocebo effect* refers to a negative reaction to an inert substance—the nocebo. Both effects are psychogenic, but known to exert powerful influences on human physiology, behavior, and attitudes. Essentially, Chapman and his supporters believe that psychogenic reasons are the basis for health complaints about wind turbines, which they believe to be harmless.

Our major criticism of the work of Chapman et al is that wind companies typically engage in practices that discourage local residents to complain. These companies require participating residents to sign contracts before turbines are constructed and before the residents can receive compensation for leasing their land, and they often request non-participating residents to sign contracts prior to initiating a project. Those contracts, which are binding, often include gag clauses that effectively limit resident complaints. The contracts have often stipulated not only that residents refrain from voicing negative views of the wind project, but also that they support the development of future projects. Such conditions create an atmosphere in which is it is highly unlikely that the records of wind companies, governments, courts, or the media will sufficiently reflect all of the complaints that residents have and would voice under less-restrictive circumstances. We argue that the only way to gather accurate data on such complaints is through a survey of either an adequate sample of residents living near multiple wind projects or all such

residents, where residents are free of restrictions by the wind companies. Such data would allow a valid determination of the proportion of residents who experience adverse effects. Whether that proportion is large or small, we could all act on the basis of factual evidence, as opposed to incomplete observations.

Another shortcoming of the study by Chapman et al,[173] which is less well documented but a factor observed in legal cases in which the present authors have been involved, is that residents near IWT projects tend to be delayed in their responses to AHEs. Many of them believe their health problems to be linked to other causes before suspecting that the turbines are the cause. Some or most of these individuals were supporters of wind projects prior to experiencing such problems, as Phipps et al[75] noted in New Zealand. The delay factor would mean that the types of records used by Chapman et al would not likely reflect the reactions of many affected residents.

Crichton and colleagues conducted two laboratory investigations, each of which has bolstered the argument that negative reactions to audible and inaudible WTN can be explained by psychological expectations. Crichton et al[174] conducted what they described as a sham-controlled double-blind provocation study, in which participants were exposed to 10 min of infrasound and 10 min of sham infrasound. Fifty-four participants were randomized to high- or low-expectancy groups and presented with audio-visual information, using material from the Internet that was designed to invoke either high or low expectations that exposure to infrasound causes specified symptoms. High-expectancy participants reported significant increases, from pre-exposure baseline assessment, in the number and intensity of symptoms experienced during exposure to both infrasound and sham infrasound. There were no symptomatic changes in the low-expectancy group. Healthy volunteers, when given information about the expected physiological effect of infrasound, reported symptoms that aligned with that information, during exposure to both infrasound and sham infrasound. According to the authors, results suggest that psychological expectations are sufficient to explain the link between wind turbine exposure and health complaints.

Punch[175] has criticized that study as methodologically weak, on the following grounds:

(1) Subjects were never exposed to infrasound that adequately represented that to which residents near wind turbine projects are subjected. It is extremely unlikely that the employed studio woofer was capable of producing a 5-Hz stimulus; the authors did not describe or show a graph of the output spectrum. Even if a true infrasound stimulus was produced by their equipment, 40 dB (presumably SPL) was not sufficient to represent the level of

infrasound commonly produced by IWTs. Even if a sufficient stimulus had been produced to represent wind turbine infrasound, a 10-min exposure would have been meaningless in representing the duration of exposure that is likely necessary to produce any substantial health symptoms.

(2) In effect, subjects were exposed to two sham conditions. If they had been exposed to infrasound that adequately mimicked infrasound from IWTs (preferably actual IWT infrasound), subjects in both the high- and low-expectancy groups would have had a physical stimulus (in the infrasound condition) that could have overridden, or at least moderated, their psychological reactions.

(3) The design limited the study's external validity, the ability to generalize the results to other populations and situations. Most of the individuals who have reported AHEs from WTN, some of whom have abandoned their homes, are not people who were adequately warned of potential health effects prior to their exposures. In fact, most of them were likely told by the wind company to expect no harmful effects. Again, many individuals who report AHEs were advocates of wind energy prior to being exposed. Because the major premise underlying the study is that people complain of WTN based primarily on expectancies that align with prior information, the study is based on a false premise. Also, the recruitment of university students does not represent the type of subjects who are apt to complain about WTN. This population is probably the least vulnerable to the effects of WTN in that few, if any, were very young, very old, likely to have chronic health conditions, or disabled. Also, they are more likely to exhibit a response bias because they are less likely than prospective residents of a wind project to believe that they might be harmed by participating in an experiment. Furthermore, the extensive use of pretesting introduced reactive or interactive effects that could have affected post-test behaviors and ratings. Finally, the use of a laboratory setting and short exposure times, as opposed to a real-life setting in which wind turbine blades are turning at night and the subjects are inside a home, introduced situational effects that limit the ability to generalize the data. The authors admit this shortcoming in their statement:

> "… exposure to infrasound in a listening room purpose (sic) built for sound experiments may not be directly comparable to exposure to infrasound from a wind farm" (p. 4).

(4) This was an experiment whose outcomes could have been predicted, given the conditions employed. Aside from the fact that the outcome had virtually nothing to do with the real-world conditions of exposure to infrasound from wind turbines, none of the factors that influence how expectations can affect perceptions through top-down, or cognitive- based, processing, as opposed to bottom-up, or stimulus-based, processing, were controlled or even

discussed. (Interested readers should refer to Williams[176] for examples of the effects of top-down processing and for a discussion of how such experiments might be improved.)

In a second laboratory study by Crichton and colleagues,[177] similar in design to the first, the authors investigated whether positive expectations can produce a reduction in symptoms and improvements in reported health. Sixty participants were randomized to either positive or negative expectations and subsequently exposed to audible wind turbine sound and infrasound. According to the authors,

> "Participants were … exposed to infrasound (9Hz, 50.4dB) and audible wind farm sound (43dB), which had been recorded 1 km from a wind farm, during two 7-minute listening sessions. Both groups were made aware they were listening to the sound of a wind farm, and were being exposed to sound containing both audible and sub audible components and that the sound was at the same level during both sessions" (p. 2).

Prior to exposure, negative-expectation participants watched a DVD incorporating TV footage about health effects said to be caused by infrasound produced by wind turbines. In contrast, positive-expectation participants viewed a DVD that:

> "…framed wind turbine sound as containing infrasound, sub audible sound created by natural phenomena such as ocean waves and the wind, which had been reported to have positive effects and therapeutic benefits on health" (p. 2).

The authors described the results as indicating that during exposure to audible wind turbine sound and infrasound, symptoms and mood were strongly influenced by subject expectations. Negative-expectation participants experienced a significant increase in symptoms and a significant deterioration in mood, while positive-expectation participants reported a significant decrease in symptoms and a significant improvement in mood. The authors concluded that if expectations about infrasound are framed in more neutral or benign ways, then it is likely that reports of symptoms or negative effects could be nullified.

That second investigation by Crichton and colleagues has some of the same methodological weaknesses as the first, particularly with respect to the use of what was described as experimental infrasound. Again, recordings of WTN were used, and no description of the recording instrumentation was provided, leading us to assume that the instrumentation may have been incapable of accurately reproducing infrasound, and thus its true effects. All participants were informed of the purpose of the study, which was:

> "…to investigate the effect of sound below the threshold of human hearing (infrasound) on the experience of physical sensations and mood" (p. 2).

Preferably, the purpose should have been divulged only after the data were gathered because the description of sounds as those that humans cannot hear would presumably have established a mind-set, or bias, in both groups that the sound would have little impact. That preconception could have confounded any reactions to the different DVD messages. Another criticism of the study is that wind companies frame their turbines in the best possible light, so positive expectations have already been established in the minds of most wind-project participants and non-participants. Despite neutral or positive framing that has sometimes included assurance that the turbine sounds would be no louder than that of a refrigerator (see, for example, Chen & Narins[178]), the consequences of living near IWTs are catastrophic for some residents.

Tonin et al[179] repeated the experimental work of Crichton and her colleagues by using specially modified headphones to produce infrasound, as opposed to the loudspeaker system used in the previous studies, and exposed participants to 23 min of infrasound, as opposed to the 10-min exposures in the Crichton studies. Similar results were reported, suggesting that the simulated infrasound had no statistically significant effect on the symptoms reported by volunteers, while the prior expectations the volunteers had about the effect of infrasound had a statistically significant influence on the symptoms reported, thereby supporting the nocebo effect hypothesis. Some of the same criticisms of the Crichton et al study[174] levelled by Punch[175] also apply to the Tonin et al study, as participants were not being stimulated by sufficient durations or peak levels of infrasound exposure to which residents living near IWTs are exposed, and participants were effectively exposed to two sham conditions, denying them any opportunity to experience realistic infrasonic stimuli that could have overridden or moderated their psychological reactions based on expectancy.

In a related study, Taylor et al[180] assessed the effect of negatively oriented personality (NOP) traits (Neuroticism, Negative Affectivity and Frustration Intolerance) on the relationship between both actual and perceived noise on “medically unexplained non-specific symptoms (NSS)” (p. 338), presumably their euphemism for Pierpont’s Wind Turbine Syndrome.[4] Households within 500 m of 8 0.6-kW micro turbine installations and within 1 km of 4 5-kW small wind turbines in two U.K. cities were surveyed, and 138 questionnaires were completed and returned for analysis. Turbine noise level for each household was also calculated. There was no evidence for an effect of calculated noise on NSS. A statistically significant relationship was found between perceived noise and NSS for individuals high in NOP traits.

That study is similar in concept to those performed by Crichton and colleagues,[174, 177] with virtually the same conclusion—that the link between wind turbines and AHEs has a psychological origin. The study can be criticized on several grounds:

(1) Only smaller wind turbines were investigated; there is virtually no literature demonstrating that such turbines produce noise levels of any consequence to humans. The fact that no relationship was found between "calculated actual noise" from the turbines and participants' attitudes toward wind turbines was thus predictable because the noise levels were either too low to affect attitudes differentially or were completely inaudible.

(2) The authors state:

> "Actual noise turbine level for each household was also calculated" (p. 338).

Calculated levels (from noise maps) are not necessarily actual levels, so this procedure was, at a minimum, mischaracterized.

(3) It should not be surprising to find that individuals with negatively oriented personalities respond negatively to WTN, as they would likely respond negatively to almost any stimulus. However, the findings, as acknowledged by the authors, resulted from reports of participants' retrospective perceptions of noise from turbines and symptoms at the same point in time, possibly resulting in common-method variance and retrospective bias. Also, although the authors reported a statistically significant relationship between NSS and negatively oriented personality, the reported variance explained by those relationships was quite low. That finding suggests that a meaningful (i.e., clinical) significance was not established, in which case one might reasonably question whether symptom reporting in the study was actually linked to negative personality type.

(4) Among other possible confounders, individual differences are likely to have complicated the authors' analyses (see Williams[176] for an explanation).

To conclude this section, we believe that while psychological expectations conceivably can influence perceptions of the effects of WTN on health status, no scientific studies have yet convincingly shown that psychological forces are the major driver of such perceptions. Based on the bulk of literature covered in this review, those drivers are the physical stimuli themselves and the internal physiological reactions they induce.

Statement 11: Only relatively few people, if any, are adversely impacted by wind turbine noise, and the majority have no complaints.

As indicated earlier, most of the studies that have documented specified percentages of the population adversely affected by WTN have been those focusing on annoyance, as opposed to health. While the exact percentage of people whose health is affected by WTN has not been accurately determined, countless reports worldwide suggest that the acoustic energy emitted by

IWTs is harmful to the health of substantial numbers of people. As already noted, Phipps et al[75] found that 45% of households living as far away as 4 km from a wind project and 20% of households living up to 8 km away reported hearing turbine noise. Those figures take into account only the audible noise, of course, and not the inaudible infrasound, and they do not account for any documented adverse impacts.

Estimates of the percentage of people adversely affected by WTN should not be based solely on questionnaire surveys of populations known to be experiencing health problems, due to selection bias. Such surveys can be helpful in arriving at rough estimates of AHEs, however, but only if those surveys also report estimates of the total population from which the affected sample is drawn. The main value of surveys that include only affected individuals (e.g., Harry[74]; Pierpont[4]; The Acoustic Group[25]) is that they strongly suggest that substantial numbers of people living near wind turbines suffer health symptoms. For example, Harry[74] reported that 81% of her 42 survey respondents had health complaints, 76% had visited a doctor regarding those complaints, and 73% reported a reduced quality of life. In a somewhat more representative survey of residents living within 15 km of a wind turbine project—most of whom lived within 3 km—Phipps[76] found that 42 of 614 households who responded to a questionnaire (6.8%) reported occasional sleep disturbance, another 21 (3.4%) reported frequent sleep disturbance, and an additional 5 (0.8%) reported sleep disturbance most of the time due to WTN. Eleven percent of households, therefore, reported suffering at least occasional sleep disruption due to the wind turbines. Fifteen percent of respondents to that survey reported that they had suffered at least occasional reductions in their quality of life since the turbines became operational.

Despite the lack of definitive scientific evidence, we cannot ignore the numerous accounts of such effects reported worldwide on the Internet, in legal proceedings, and in news accounts. Krogh et al[96] have reviewed studies that document such incidents, many of which have involved the abandonment of homes. In a 2010 report commissioned by the Ontario Ministry of the Environment, the engineering firm of Howe Gastmeier Chapnik Limited,[112] despite its general conclusion that Ontario IWTs do not pose a risk to human health, stated:

> "The audible sound from wind turbines, at the levels experienced at typical receptor distances in Ontario, is nonetheless expected to result in a non-trivial percentage of persons being highly annoyed …. research has shown that annoyance associated with sound from wind turbines can be expected to contribute to stress related health impacts in some persons" (p. 39).

In conclusion, we should recall that Phillips[169] advocates self-reporting of adverse events as a critical element in the study of the health effects of wind turbines. As stated earlier, he has noted

the importance of case-crossover experiments as useful and well-accepted sources of epidemiologic information. Numerous households around the world have been subjected to this type of quasi-experiment by the wind industry. It is unfortunate that an accurate count of these incidents has never been tallied formally and scientifically. Although that task must be left to future research, we should regard complaints of AHEs from individuals living near wind turbine installations seriously, when they occur, and the wind industry must act responsibly by siting its turbines at distances from residents that protect health and quality of life.

It is widely accepted that the industry has warned that tighter siting restrictions will destroy its prospects for growth. Such growth, however, should not continue in areas where there are probable and potential risks to human health. There are regions of the U.S. and other countries where turbines can operate safely, presumably without such risks. Some examples of those sites are illustrated in Figure 2.

Statement 12: There is no evidence in the literature to support a causative link between wind turbine noise and adverse health effects.

The above review has been aimed specifically at addressing this point, which is often cited as factual by wind industry advocates in the literature and in legal proceedings. Namely, there is an abundant literature, much of it peer-reviewed and authored by highly reputable researchers, indicating that audible and inaudible noise emitted by IWTs adversely impacts the health and well-being of substantial numbers of people who are regularly exposed to wind turbines. It is clear that the literature reviews and papers claiming no AHEs fail to include important studies, international standards, guidance from the WHO, and research conducted on wind turbine noise and other sources of infra- and low-frequency sound. Whether this is through oversight or calculation, only reports that cite scientifically credible references should be considered legitimate sources of information. Our review has shown that it is unacceptable simply to state that the literature contains little or no evidence of a causal link between WTN and AHEs. At a minimum, those effects have been shown to be regularly correlated to living in proximity to IWTs, and there is sufficient evidence that those effects are highly associated with objective measurements of audible noise and infrasound.

Although sleep disturbance and its associated impacts on health and quality of life appear to be the most salient consequences of IWT noise, varying health effects that are unrelated to sleep have also been widely and consistently reported by different investigators. While not everyone who is exposed to IWTs suffers AHEs, it is incumbent on governmental officials and the wind industry to take seriously the health implications of their decisions to locate wind turbines near

residential and other populations, especially vulnerable populations, that are or likely to be negatively affected.



Figure 2. Photographic images of sites illustrating onshore landscapes where industrial wind turbines expose humans to minimal health risks due to large setback distances. Note that homes are not seen in the photos. (Source: https://images.search.yahoo.com/search/images?p=wind+turbine+images+california&fr=tightropetb&imgurl=http%3A%2F%2Fwww.freefoto.com%2Fimages%2F39%2F01%2F39_01_1---Wind-Turbine-Generators--Palm-Springs--California_web.jpg#id=36&iurl=http%3A%2F%2Fmedia-cdn.tripadvisor.com%2Fmedia%2Fphoto-s%2F01%2F70%2Ff9%2Fbb%2Ftehachepi-area-california.jpg&action=close).

Conclusion

We have discussed in this paper various elements of acoustics, sound perception, sound measurement, and psychological reactions, and the role these factors play in support of the view that a general-causative link exists between human health and ILFN emitted by IWTs. The available evidence warrants the following conclusions:

(1) Large wind turbines generate infrasound, which is not normally experienced as sound by most human listeners. Some people, however, experience it in the form of pathological

symptoms such as headache, dizziness, nausea, or motion sickness, which appear to be caused by the excitation of resonances inside closed structures and the human body itself.

(2) WTN has unique acoustic characteristics when compared to other environmental noises. These characteristics include low-amplitude, amplitude-modulated, intermittent occurrences of tones that mirror the peak energy of the blade-pass frequency and the first several harmonics. The coupling mechanisms in the inner ear prevent internally generated sound, but not externally generated sound, from being perceived, which means that perception of wind turbine infrasound is far more disturbing than infrasound generated within the human body.

(3) There is voluminous evidence, ranging from anecdotal accounts from around the world to peer-reviewed scientific research, that audible and inaudible low-frequency noise and infrasound from IWTs lead to complaints ranging from annoyance to AHEs in a substantial percentage of the population. Although sleep disturbance is the most common problem cited, a variety of other health problems has been reported by numerous reputable sources. Recent research is largely consistent with Pierpont's original description of Wind Turbine Syndrome. Research on humans and lower animals has shown that it is biologically plausible that inner ear mechanisms, in conjunction with the brain, can process acoustic energy in ways that result in pathological perceptions that are not interpreted as sound. Both balance and hearing mechanisms appear to be involved in evoking these perceptions. The findings that infrasonic stimuli can amplitude modulate higher frequencies in the audible region, and that infrasound may be more perceptible when higher frequencies are absent, are especially compelling in suggesting that what we can't hear can hurt us.

(4) To prevent AHEs, scientists have recommended that distances separating turbines and residences be 0.5-2.5 mi., and 1.25 mi. (2 km) or more has been commonly recommended. Clearly, the short siting distances used by the industry for physical safety do not protect against AHEs. Alternatively, researchers have recommended sound levels typically ranging from 30-40 dBA for safeguarding health, which is consistent with the recommendation of nighttime noise levels by the WHO.

(5) Annoyance is a health issue for many people living near IWTs, which is consistent with both the WHO's definition of health and contemporary models of the relationships among annoyance, stress, and health.

(6) The scientific evidence regarding factors other than amplitude-modulated ILFN as an explanation for most of the health complaints near IWTs, including electromagnetic fields (dirty electricity), is weak; the preponderance of research suggests that ILFN is the most viable explanation for such complaints.

(7) The A-weighted decibel scale, which effectively excludes infrasound and substantial amounts of low-frequency noise, is inadequate to predict the level of outdoor or indoor infrasound, to reveal correlations to infrasound, or to show a definitive relationship with AHEs. Achievement of these goals requires the development of new measurement methods.

(8) Even though Wind Turbine Syndrome is not currently included in the ICD coding system, that system includes most of the acknowledged symptoms of the syndrome. Medical professionals, therefore, have the necessary tools to evaluate and treat it, and that process has already begun on a limited scale.

(9) While some epidemiologically solid research has been done in the area of IWTs and AHEs, evidence from other sources cannot be ignored. Hill noted the nature of such sources in 1965, and Phillips, in 2011, described the importance of other kinds of evidence, including adverse event reports, in establishing a causative relationship. One of the strongest types of evidence is the case-crossover experimental design, which the wind industry has unwittingly imposed for years on multiple families, many of whom have abandoned their homes to escape IWT noise exposure.

(10) While psychological expectations and the power of suggestion conceivably can influence perceptions of the effects of WTN on health status, no scientifically valid studies have yet convincingly shown that psychological forces are the major driver of such perceptions.

(11) Accurate estimates of the percentage of people who are affected by IWTs exist only for annoyance, not AHEs. Multiple reports, however, emphasize the relationships that exist between annoyance, stress, health, and quality of life, and indicate that a non-trivial percentage of people who live near IWTs experience AHEs. Those reports are consistent with thousands of reports worldwide. Although it seems reasonable to conclude that noise from IWTs does not cause AHEs in the majority of exposed populations, and that accurate estimates of AHEs are yet to be established, it is also clear that considerable numbers of people are affected and that they deserve to be heard and protected from adverse health impacts.

(12) The available literature, which includes research reported by scientists and other reputable professionals in peer-reviewed journals, government documents, print and web-based media, and in scientific and professional papers presented at society meetings, is sufficient to establish a general causal link between a variety of commonly observed AHEs and noise emitted by IWTs.

Based on all the evidence presented, our fundamental view is that the controversy surrounding AHEs should not be polarized into two groups consisting of either *pro-wind* or *anti-wind*

factions, but rather one in which there is room for a third, *pro-health*, perspective. Essentially, the pro-wind view is that IWTs should be installed wherever feasible, that definitive scientific research is lacking to indicate that turbines cause AHEs, and that if you can't hear it, you can't feel it. The anti-wind view is that IWTs should not be installed anywhere because wind is not an economically viable source of renewable energy, that all government subsidies and development efforts should end, and that what we can't hear can hurt us. A pro-health view is that there is enough anecdotal and scientific evidence to indicate that ILFN from IWTs causes annoyance, sleep disturbance, stress, and a variety of other AHEs to warrant siting the turbines at distances sufficient to avoid such harmful effects, which, without proper siting, occur in a substantial percentage of the population. That view holds that what we can't hear can hurt some of us, and that the precautionary principle must be followed in siting IWTs if such health risks are to be avoided. Industrial-scale wind turbines should not be located near people's homes, educational and recreational facilities, and workplaces. It is our belief that the bulk of the available evidence justifies a pro-health perspective. It is unacceptable to consider people living near wind turbines as collateral damage while this debate continues.

Further scientific investigations of the dose-response relationship between IWT noise and specific health effects in exposed individuals are sorely needed. However, people should be protected by conservative siting guidelines that recognize the concerns raised in this review. Hopefully, such research can and will be planned and executed by independent researchers with the full cooperation of the wind industry. The major objective of such research should be to reveal directions for the industry in balancing the energy needs of society with the need to protect public health.

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References

[1] Caithness Windfarm Information Forum. Summary of wind turbine accident data to 30 June 2016. Available from: http://www.caithnesswindfarms.co.uk/AccidentStatistics.htm. [Last accessed on 2016 July 5].

[2] Hedges C, Sacco, J. *Days of Destruction, Days of Revolt*. New York: Nation Books; 2012.

[3] Krogh C, Horner B, Morris J, May M, Papadopoulos G, Watts AC. Trading off human health: wind turbine noise and government policy. Presentation at 5th International Meeting on Wind Turbine Noise, August 2013. Available from: http://waubrafoundation.org.au/wp-content/uploads/2014/02/Wind-Turbine-Noise-Conference-2013-Policy-paper.pdf. [Last accessed on 2016 July 5].

[4] Pierpont N. *Wind turbine syndrome: a report on a natural experiment*. Santa Fe (NM): K-Selected Books, 2009.

[5] Horner B. Open Letter Audit: National Health and Medical Research Council Public Statement (2010) and Rapid Review (2010). Available from: http://docs.wind-watch.org/Horner_NHMRC-references-audit_July-31-2012.pdf. [Last accessed on 2016 July 5].

[6] Office of Management and Budget: Final Information Quality Bulletin for Peer Review, December 16, 2004. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=0CB4QFjAA&url=http%3A%2F%2Fwww.cio.noaa.gov%2Fservices_programs%2Fpdfs%2FOMB_Peer_Review_Bulletin_m05-03.pdf&ei=zxo8VI2PLevesASY64LQBA&usg=AFQjCNHRv2lLaSO_W6HyH05x9E90yQynDQ&sig2=jewV1g8YLe-Yrqz3IZ6rxg&bvm=bv.77161500,d.cGE&cad=rja. [Last accessed on 2016 July 20].

[7] Achenbach J. Why do many reasonable people doubt science? *National Geographic*, 2015. Available from: http://ngm.nationalgeographic.com/2015/03/science-doubters/achenbach-text. [Last accessed on 2016 July 5].

[8] Leventhall G. How the "mythology" of infrasound and low frequency noise related to wind turbines might have developed. Presentation at First International Meeting on Wind Turbine Noise: Perspectives for Control, October 2005. Available from: https://www.researchgate.net/publication/237542296_First_International_Meeting_on_Wind_Turbine_Noise_Perspectives_for_Control_Berlin_17th_and_18th_October_2005_How_the_mythology_of_infrasound_and_low_frequency_noise_related_to_wind_turbines_might_have_developed. [Last accessed on 2016 July 5].

[9] Leventhall G. Infrasound from wind turbines—fact, fiction or deception. *Canadian Acoustics* 2006; 34:29-36.

[10] James RR. Wind turbine infra and low-frequency sound: warning signs that were not heard. *Bulletin of Science, Technology & Society* 2012;32:108-127.

[11] Schwartz S. Linking noise and vibration to Sick Building Syndrome in office buildings. Air & Waste Management Association. March 2008:26-28. Available from: http://pubs.awma.org/gsearch/em/2008/3/schwartz.pdf. [Last accessed on 2016 July 5].

[12] Leventhall G. A review of published research on low frequency noise and its effects, May 2003. Available from: http://docs.wind-watch.org/leventhall-defra-lowfreqnoise.pdf. [Last accessed on 2016 July 20].

[13] Broner N. The effects of low frequency noise on people—a review. *Journal of Sound and Vibration* 1978;58:483-500.

[14] Leventhall G. Concerns about infrasound from wind turbines. *Acoustics Today* 2013;9:30-38.

[15] Colby WD, Dobie R, Leventhall G, Lipscomb DM, McCunney RJ, Seilo MT, et al. Wind turbine sound and health effects: an expert panel review. Report prepared for Prepared for American Wind Energy Association and Canadian Wind Energy Association, December 2009. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0CB4QFjAA&url=http%3A%2F%2Fcanwea.ca%2Fpdf%2Ftalkwind%2FWind_Turbine_Sound_and_Health_Effects.pdf&ei=Ja5TVIvDJ4WjyATUq4HQBA&usg=AFQjCNGYQ3-rkSZttsOVpuyhzeGsk2o4lQ&sig2=kZ_liEZ8jzUkFsT4bE7dbg&bvm=bv.78677474,d.aW. [Last accessed on 2016 July 5].

[16] Møller H, Pedersen CS. Low-frequency noise from large wind turbines. *Journal of the Acoustical Society of America* 2011;129:3727-3744.

[17] Evans T, Cooper J, Lenchine V. Infrasound levels near windfarms and in other environments. Environment Protection Authority, South Australia, January 2013. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=7&cad=rja&uact=8&ved=0CD8QFjAG&url=http%3A%2F%2Fwww.epa.sa.gov.au%2Fxstd_files%2FNoise%2FReport%2Finfrasound.pdf&ei=wIJeU_n_AfOFyQG26YCoDQ&usg=AFQjCNG-V01kqE5hz7j0Jajwv8nFac0C4A&sig2=fj0z4hblVm_TOTTKxs-0nA&bvm=bv.65397613,d.aWc. [Last accessed on 2016 July 20].

[18] Bolin K, Bluhm G, Eriksson G, Nilsson ME. Infrasound and low frequency noise from wind turbines: exposure and health effects. *Environmental Research Letters* 2011;6:1-12.

[19] Kelley ND, Hemphill RR, McKenna ME. A methodology for assessment of wind turbine noise generation. *Journal of Solar Energy Engineering* 1982;104:112-120.

[20] Kelley ND, McKenna HE, Hemphill RR, Etter CL, Garrelts RL, Linn NC. Acoustic noise associated with the MOD-1 wind turbine: its source, impact, and control. Report prepared for the U. S. Department of Energy, Contract No. DE-AC02-83CH-10093, February 1985. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0CB4QFjAA&url=http%3A%2F%2Fwww.nrel.gov%2Fdocs%2Flegosti%2Fold%2F1166.pdf&ei=uK9TVIL3L82P7AajoIHQDQ&usg=AFQjCNGgDEPZV0hd1gkxw4hGcqWROWkufQ&si

g2=abXK4ET8bDhYWbdG7UsKaA&bvm=bv.78677474,d.ZGU. [Last accessed on 2016 July 20].

[21] Kelley ND. A proposed metric for assessing the potential of community annoyance from wind turbine low-frequency noise emissions. Presentation at Windpower'87 Conference and Exposition, November 1987. Available from: http://www.nrel.gov/docs/legosti/old/3261.pdf. [Last accessed on 2016 July 20].

[22] Bray W, James R. Dynamic measurements of wind turbine acoustic signals, employing sound quality engineering methods considering the time and frequency sensitivities of human perception. Presentation at Noise-Con 2011 conference, July 2011. Available from: http://waubrafoundation.org.au/wp-content/uploads/2013/07/Bray-James-11-07-25-Dynamic-measurement-of-wind-turbine-noise.pdf. [Last accessed on 2016 July 20].

[23] Rand RW, Ambrose SE, Krogh CME. Occupational health and industrial wind turbines: a case study. *Bulletin of Science, Technology & Society* 2011;31:329-362.

[24] Walker B, Hessler GF, Hessler DM, Rand R, Schomer PD. A cooperative measurement survey and analysis of low frequency and infrasound at the Shirley Wind Farm in Brown County, Wisconsin. Report Number 122412-1, December 24, 2012. Available from: https://docs.wind-watch.org/Shirley-LFN-infrasound.pdf. [Last accessed on 2016 July 5].

[25] The Acoustic Group. The results of an acoustic program: Cape Bridgewater Wind Farm. Report prepared for Energy Pacific (Vic), Melbourne, Vic 3000, January 2015. Available from: http://www.pacifichydro.com.au/english/our-communities/communities/cape-bridgewater-acoustic-study-report/?language=en. [Last accessed on 2016 July 20].

[26] Swinbanks M. Direct experience of low-frequency noise and infrasound within a windfarm community. Paper prepared for presentation at 6th International Meeting on Wind Turbine Noise, April 2015. Available from: https://docs.wind-watch.org/WTN2015-Swinbanks.pdf. [Last accessed on 2016 July 5].

[27] Swinbanks M. Direct experience of low frequency noise and infrasound within a windfarm community. Presentation at 6th International Meeting on Wind Turbine Noise, April 2015. Available from: http://docs.wind-watch.org/WTN2015-Swinbanks-poster.pdf. [Last accessed on 2016, July 20].

[28] Artz K. Health threat from Wisconsin wind farm affirmed, October 29, 2014. Available from: http://news.heartland.org/newspaper-article/2014/10/28/health-threat-wisconsin-wind-farm-affirmed. [Last accessed on 2016 July 20].

[29] Abel D. State wants turbine shut over noise. *Boston Globe*, May 16, 2012. Available from: https://www.bostonglobe.com/metro/2012/05/15/massachusetts-officials-recommend-falmouth-shut-down-turbines/mCPNKvUoq1IrdPClRygmWN/story.html. [Last accessed on 2016 September 10.]

[30] Anon, Kingston wind turbine shut down indefinitely; financial problems reported to the Town, June 19, 2016. Available from: http://www.windaction.org/posts/45258-kingston-wind-turbines-shut-down-indefinately#.V8x_tDXlS-E. [Last accessed on 2016 September 4.]

[31] Castelo Branco NAA, Alves-Pereira M, Pimenta AM, Ferreira JR. Low frequency noise-induced pathology: contributions provided by the Portuguese wind turbine case. Presentation at EuroNoise 2015 conference. Available from: http://docs.wind-watch.org/Euronoise2015-000602.pdf. [Last accessed on 2016 September 11.]

[32] Leloudas G, Zhu WJ, Sørensen JN, Shen WZ, Hjort S. Prediction and reduction of noise from a 2.3 MW wind turbine. Journal of Physics: Conference Series 75, 2007. Available from: http://iopscience.iop.org/article/10.1088/1742-6596/75/1/012083/pdf. [Last accessed on 2016 July 20].

[33] Rogers AL. Wind turbine noise, infrasound and noise perception. January 18, 2006. Available from: http://www.google.com/url?sa=t&source=web&cd=1&ved=0CBoQFjAA&url=http%3A%2F%2Fwww.windpoweringamerica.gov%2Fpdfs%2Fworkshops%2Fmwwg_turbine_noise.pdf&rct=j&q=frequency%20spectrum%20wind%20turbine%20noise&ei=u_fLTdu9OunRiALiotmUCA&usg=AFQjCNFWWT5fFWS6aO3XYlpvqkevckXbmw&sig2=1C0uNL4d19vHkmbvE4V8Lg&cad=rja. [Last accessed on 2016 July 20].

[34] Powell CA, Preisser JS. NASA's subsonic jet transport noise reduction research. Available from: http://hdl.handle.net/2060/20040086700. [Last accessed on 2016 July 8].

[35] Hubbard HH, Maglieri DJ. A brief review of air transport noise. *Journal of Sound and Vibration* 1975;43:159-172.

[36] Cermak GW, Cornillon PC. Multidimensional analyses about judgments about traffic noise. *Journal of the Acoustical Society of America* 1976;59:1412-1420.

[37] Leventhall HG. Low frequency noise and annoyance. *Noise & Health* 2004;6:59-72.

[38] World Health Organization. Night Noise Guidelines for Europe. World Health Organization, 2009. Available from: http://www.euro.who.int/__data/assets/pdf_file/0017/43316/E92845.pdf. [Last accessed on 2016 July 20].

[39] Cummings, J. Looking for wind industry leadership in reducing noise impacts. February 23, 2011. Available from: http://www.renewableenergyworld.com/rea/news/article/2011/02/looking-for-wind-industry-leadership-in-reducing-noise-impacts. [Last accessed on 2016 July 20].

[40] Jung SS, Cheung W-S, Cheong C, Shin S-H. Experimental identification of the acoustic emission characteristics of large wind turbines, with emphasis on infrasound and low frequency noise. *Journal of the Korean Physical Society* 2008;53:1897-1905.

[41] Leventhall G. On the overlap region between wind turbine infrasound and infrasound from other sources and its relation to criteria. Presentation at 6th International Conference on Wind

Turbine Noise, April 2015. Available (for purchase) from: http://www.proceedings.com/26771.html. [Last accessed on 2016 July 15.]

[42] Harrison JP. Wind turbine noise. *Bulletin of Science, Technology & Society* 2011;31:256-261.

[43] Janssen SA, Vos H, Eisses AR, Pedersen E. Predicting annoyance by wind turbine noise. Presentation at 2010 Internoise conference, June 2010. Available from: http://docs.wind-watch.org/Janssen_Predicting-annoyance.pdf. [Last accessed on 2016 July 20].

[44] Pedersen E, Persson Waye KP. Perception and annoyance due to wind turbine noise—a dose–response relationship. *Journal of the Acoustical Society of America* 2004;116:3460-3470.

[45] Van Renterghem T, Bockstael A, De Weirt V, Botteldooren D. Annoyance, detection and recognition of wind turbine noise. *Science of the Total Environment* 2013;456-7:333–345.

[46] Thorne B. The problems with "noise numbers" for wind farm noise assessment. *Bulletin of Science, Technology & Society* 2011;31:262-290.

[47] Hubbard HH. Noise induced house vibrations and human perception. *Noise Control Engineering Journal* 1982;19:49-55.

[48] Hubbard HH, Shepherd KP. Wind turbine acoustics. NASA Technical Paper 3057. December 1990. Available from: http://hdl.handle.net/2060/19910007366. [Last accessed on 2016 July 8].

[49] Thorne B. Wind farm noise and human perception: a review. April 2013. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=2&cad=rja&uact=8&ved=0CC8QFjAB&url=http%3A%2F%2Fdocs.wind-watch.org%2FNMS-Wind-Farm-Noise-Human-Perception-April-2013.pdf&ei=vcprU_3KOZCFyQG-p4HYBA&usg=AFQjCNGJS5ylvcUMNXbqjy7lML7kMd_H7w&sig2=Ls0JpH4uFoSdfxYnlhz6Vg&bvm=bv.66330100,d.aWc. [Last accessed on 2016 July 8].

[50] Shepherd D, Hanning C, Thorne B. Noise: windfarms. In: Jorgensen SE, editor. *Encyclopedia of Environmental Management*. New York: Taylor & Francis Online; 2012. pp. 1-17.

[51] Schomer P. Can wind turbine sound that is below the threshold of hearing be heard? Presentation at ICA 2013 Conference, June 2013. Available from: http://www.ica2013montreal.org/Proceedings/mss/040063_1.pdf. [Last accessed on 2016 July 20].

[52] Schomer PD, Erdreich J, Boyle J, Pamidighantam P. A proposed theory to explain some adverse physiological effects of the infrasonic emissions at some wind farm sites. Presentation at 5th International Conference on Wind Turbine Noise, August 2013. Available from: http://docs.wind-watch.org/Schomer-et-al-physiologic-effects-infrasound-wind.pdf. [Last accessed on 2016 July 20].

[53] Lee S, Lee S, Lee, S. Numerical modeling of wind turbine aerodynamic noise in the time domain. *Journal of the Acoustical Society of America* 2013;133:94-100.

[54] Lichtenhan JT, Salt AN. Amplitude modulation of audible sounds by non-audible sounds: understanding the effects of wind turbine noise. Presentation at 2013 ICA conference, June 2013. *Journal of the Acoustical Society of America* 2013;133:3419. Available from: http://oto2.wustl.edu/cochlea/LichtenhanAMASA.pdf. [Last accessed on 2016 July 20].

[55] Minnesota Department of Health. Public health impacts of wind turbines. May 22, 2009. Available from: http://www.health.state.mn.us/divs/eh/hazardous/topics/windturbines.pdf. [Last accessed on 2016 July 20].

[56] Salt AN. Does the infrasound from wind turbines affect the inner ear? Presentation at Association for Research in Otolaryngology conference, February 2011. Available from: https://c.ymcdn.com/sites/aro.site-ym.com/resource/resmgr/Abstract_Archives/2011_Abstract_Book.pdf. Pp. 137-138. [Last accessed on 2016 July 20].

[57] Stigwood M, Large S, Stigwood D. Audible amplitude modulation: results of field measurements and investigations compared to psycho-acoustical assessment and theoretical research. Presentation in Denver, Colorado, August 2013. Available from: http://www.masenv.co.uk/uploads/WTN13_Stigwood_Presentation.pdf. [Last accessed on 2016 July 20].

[58] Taylor S. Report on the health impacts of wind farms: Shetland 2013 Summary. July 15, 2013. Available from: http://www.shb.scot.nhs.uk/board/publichealth/documents/Summary_Report_on_Health_Impacts_Wind_Farms.pdf. [Last accessed on 2016 July 20].

[59] Bray WR. Measurement and sound quality issues concerning low-frequency noise. Presentation at Noise-Con 2007 conference (Abstract). Available from: http://www.head-acoustics.de/downloads/messen/Noise-Con_2007_Measurement_and_SQ_WBray.pdf. [Last accessed on 2016 July 20].

[60] van den Berg GP. Effects on the wind profile at night on wind turbine sound. *Journal of Sound and Vibration* 2004;277:955-970.

[61] Ambrose SE, Rand RW, Krogh ME. Wind turbine acoustic investigation: infrasound and low-frequency noise – a case study. *Bulletin of Science, Technology & Society* 2012;32:128-141.

[62] Vasudevan RN, Gordon CG. Experimental study of annoyance due to low frequency environmental noise. *Applied Acoustics* 1977;10:57-69.

[63] Frey BJ, Hadden PJ. Wind turbines and proximity to homes: the impact of wind turbine noise on health. January 2012. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=2&cad=rja&uact=8&ved=0CC8QFjAB&url=http%3A%2F%2Fdocs.wind-watch.org%2FFrey_Hadden_WT_noise_health_01Jan2012.pdf&ei=op9eU4aJCsjK2wXeyIDQCA&usg=AFQjCNHGxv6iq3RAc_CXS0KtVqKxdedzNw&sig2=hMEop9T4B7eFyX97B_36Fw&bvm=bv.65397613,d.b2U. [Last accessed on 2016 July 20].

[64] Farboud A, Crunkhorn R, Trinidade A. Wind turbine syndrome: fact or fiction? *Journal of Laryngology & Otology* 2013;127:222-226.

[65] Pedersen E, van den Berg F. Why is wind turbine noise poorly masked by road traffic noise? Internoise 2010 conference, June 2010. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=2&cad=rja&uact=8&ved=0CCQQFjAB&url=http%3A%2F%2Fwww.diva-portal.org%2Fsmash%2Fget%2Fdiva2%3A332207%2FFULLTEXT01.pdf&ei=sLJTVJeaNNCWyATQkIGoBA&usg=AFQjCNEyiBjgIJpCsq3ShXZAMUPdSP4rzQ&sig2=n3ZJBFNLUzT4dMjib-gghw&bvm=bv.78677474,d.aWw. [Last accessed on 2016 July 20].

[66] Schomer PD. Comments on recently published article, "Concerns about infrasound from wind turbines." *Acoustics Today* 2013;9:7-9.

[67] Moorhouse A, Waddington D, Adams M. Proposed criteria for the assessment of low frequency noise disturbance. Report (NANR45) prepared for DEFRA, February 2005. Available from: http://webarchive.nationalarchives.gov.uk/20130402151656/http://archive.defra.gov.uk/environment/quality/noise/research/lowfrequency/documents/nanr45-criteria.pdf. [Last accessed on 2016 July 20].

[68] Swinbanks MA. Numerical simulation of infrasound perception, with reference to prior reported laboratory effects. Presentation at 2012 Internoise conference, August 2012. Available from: http://docs.wind-watch.org/Internoise2012-Swinbanks.pdf. [Last accessed on 2016 July 20].

[69] James RR. Untitled. ND. Available from: http://www.windturbinesyndrome.com/wp-content/uploads/2014/07/How-Blade-pass-causes-infrasound-pulsations.pdf. [Last accessed on 2016 July 8].

[70] Salt AN. Letter to Chairman of Association of Australian Acoustical Consultants. September 18, 2013. Available from: https://s3.amazonaws.com/windaction/attachments/1998/Warpenius.pdf. [Last accessed on 2016 September 5].

[71] Berglund B, Lindvall T, Schwela DH (eds.), Guidelines for Community Noise. World Health Organization, April 1999. Available from: https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=0CB8QFjAAahUKEwjsqc_CnJPHAhXEi5IKHeJxANk&url=http%3A%2F%2Fwhqlibdoc.who.int%2Fhq%2F1999%2Fa68672.pdf&ei=RK3CVez3A8SXygTi44HIDQ&usg=AFQjCNHq0SRjmXAXPKGPvQC88NtlGaJJBQ&bvm=bv.99261572,d.aWw&cad=rja. [Last accessed on 2016 July 20].

[72] Ising H, Kruppa B. Health effects caused by noise: evidence in the literature from the past 25 years. *Noise & Health* 2004;6:5-13.

[73] Sørensen M, Andersen ZJ, Nordsborg RB, Jensen SS, Lillelund KG, Beelen R., et al. Road traffic noise and incident myocardial infarction: a prospective cohort study. *PLoS ONE* 2012;7(6);1-7.

[74] Harry A. Wind turbines, noise and health. February 2007. Available from: http://docs.wind-watch.org/wtnoise_health_2007_a_harry.pdf. [Last accessed on 2016 July 20].

[75] Phipps R, Amati M, McCoard S, Fisher R. Visual and noise effects reported by residents living close to Manawatu Wind Farms: preliminary survey results. ND. Available from: http://docs.wind-watch.org/phipps-visualnoiseeffects.pdf. [Last accessed on 2016 July 8].

[76] Phipps R. Evidence of Dr. Robyn Phipps, in the matter of Moturimu Wind Farm application, Palmerston North. March 2007. Available from: http://docs.wind-watch.org/phipps-moturimutestimony.pdf. [Last accessed on 2016 July 20].

[77] Horner B, James RR, Jeffery RD, McMurtry RY, Nissenbaum MA, White DL. Wind energy industry acknowledgement of adverse health effects: part 1 conclusion and executive summary. January 2010. Available from: http://co.wasco.or.us/planning/Energy/BOCC/New_Comments/after_2-15/swaim1_SWV_Critique_CANAWEA.pdf. [Last accessed on 2016 July 20].

[78] Horner B. Open letter to Prime Minister of Canada and others to request formally that representatives of CanWEA, the Government of Canada, Health Canada, and the Government of Ontario provide Canadians immediate and full disclosure of the health effects “conclusively demonstrated” from exposure to wind turbine noise. March 21, 2013. Available from: https://mothersagainstturbines.files.wordpress.com/2013/09/request-stop-health-canada-experiment-on-canadians-march-21-2013.pdf. [Last accessed on 2016 July 20].

[79] Phillips CV. An analysis of the epidemiology and related evidence on the health effects of wind turbines on local residents. Public Service Commission of Wisconsin, Docket no. 1-AC-231. July 3, 2010. Available from: http://docs.wind-watch.org/Phillips-wind-turbines-and-health.pdf. [Last accessed on 2016 July 8].

[80] Hanning C. Wind turbine noise, sleep and health. July 2010. Available from: http://www.acousticecology.org/wind/winddocs/health/Hanning%202010_Wind%20turbine%20noise%20sleep%20and%20health%20November%202010.pdf. [Last accessed on 2016 July 20].

[81] Nissenbaum MA. Industrial wind turbines, human variability, and adverse health effects. NOCOEM Reporter, *New England College of Occupational and Environmental Medicine*. Fall 2013;2(38). Available from: http://www.necoem.org/documents/V2_38e.pdf. [Last accessed on 2016 July 20].

[82] Ellenbogen JM, Grace S, Heiger-Bernays WJ, Manwell JF, Mills DA, Sullivan KA, et al. Wind Turbine Health Impact Study. Report prepared for Massachusetts Department of Environmental Protection. January 2012. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0CCMQFjAA&url=http%3A%2F%2Fwww.mass.gov%2Feea%2Fdocs%2Fdep%2Fenergy%2Fwind%2Fturbine-impact-study.pdf&ei=wSw0VMjCDoz_yQTRpILoAQ&usg=AFQjCNEdk-4087qFlCDXRnPB2mXjV5MU1Q&bvm=bv.76943099,d.aWw. [Last accessed on 2016 July 20].

[83] Kamperman GW, James RR. The "how to" guide to criteria for siting wind turbines to prevent health risks from sound. October 28, 2008. Available from: http://www.windturbinesyndrome.com/wp-content/uploads/2008/11/kamperman-james-10-28-08.pdf. [Last accessed on 2016 July 20].

[84] Hanning C. Comments on wind turbine health impact study: report of independent expert panel. ND. Available from: http://docs.wind-watch.org/MassDEP-wind-health-1-Hanning.pdf. [Last accessed on 2016 July 26].

[85] Hartman RS. Statement of Dr. Raymond S. Hartman presented to the zoning board of Charlestown, Rhode Island, Critique of the Massachusetts Department of Environmental Planning (DEP) "Wind Turbine Health Impact Study, Report of Independent Expert Panel," January 2012. Available from: https://www.wind-watch.org/docviewer.php?doc=Hartman-Mass-DEP-study-RI-130605.pdf. [Last accessed on 2016 July 20].

[86] Schomer P, Pamidighantam P. A critical analysis of Wind Turbine Health Impact Study: report of independent expert panel. Presentation at 166th meeting of Acoustical Society of America, December 2013. Available from: http://scitation.aip.org/deliver/fulltext/asa/journal/poma/20/1/1.4870639.pdf?itemId=/content/asa/journal/poma/20/1/10.1121/1.4870639&mimeType=pdf&containerItemId=content/asa/journal/poma. [Last accessed on 2016 July 20].

[87] Shahan, Z. Wind Turbine Syndrome… not! Available from: http://cleantechnica.com/2012/12/27/wind-turbine-syndrome-not/. December 27, 2012. [Last accessed on 2016 July 20].

[88] Hanning CD, Evans A. Wind turbine noise. *British Medical Journal* 2012;344:1-2.

[89] Bakker RH, Pedersen E, van den Berg GP, Stewart RE, Lok W, Bouma J. Impact of wind turbine sound on annoyance, self-reported sleep disturbance and psychological distress. *Science of the Total Environment* 2012;425:42–51.

[90] Frey BJ, Hadden PJ. Noise radiation from wind turbines installed near homes: effects on health. February 2007. Available from: http://waubrafoundation.org.au/wp-content/uploads/2014/06/Frey-Hadden-Noise-Radiation-fro-Wind-Turbines-Installed-Near-Homes.pdf. [Last accessed on 2016 July 20].

[91] Havas M, Colling D. Wind turbines make waves: why some residents near wind turbines become ill. *Bulletin of Science, Technology & Society* 2011;31:414–426.

[92] Jeffery RD, Krogh C, Horner B. Adverse health effects of industrial wind turbines. *Canadian Family Physician* 2013;59:473-475.

[93] Jeffery RD, Krogh CME, Horner B. Industrial wind turbines and adverse health effects. *Canadian Journal of Rural Medicine* 2014;19:21-26.

[94] Krogh C. Risk of harm to children and industrial wind turbines: health and social-economic impacts in Canada, Health Canada Wind Turbine Noise and Health Study. December 27, 2012.

Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0CB0QFjAA&url=http%3A%2F%2Fwepat.files.wordpress.com%2F2013%2F05%2Fhealth-canada_risks-to-children-december-27-2012-final.pdf&ei=Lp2tU862N4W2yAT6kYDwDQ&usg=AFQjCNGeq2a21hWue4rFj3f48l7L8nvQ5g&sig2=fTwIBMrIj6KwvfzeNnVtQQ&bvm=bv.69837884,d.aWw. [Last accessed on 2016 July 20].

[95] Krogh CME, Gillis L, Kouwen N, Aramini J. WindVOiCe, a self-reporting survey: adverse health effects, industrial wind turbines, and the need for vigilance monitoring. *Bulletin of Science, Technology & Society* 2011;31:334-335.

[96] Krogh CME, Jeffery RD, Aramini J, Horner B. Wind turbines can harm humans: a case study. Presentation at 2012 Internoise conference, August 2012. Available from: http://docs.wind-watch.org/Krogh et al. - Wind Turbines - Risk of Harm.pdf. [Last accessed on 2016 July 20].

[97] Nissenbaum MA, Aramini JJ, Hanning CD. Effects of industrial wind turbine noise on sleep and health. *Noise & Health*, 2012;14:237-243.

[98] Pedersen, E. Effects of wind turbine noise on humans. Presentation at Third International Meeting on Wind Turbine Noise, June 2009. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0CB4QFjAA&url=http%3A%2F%2Fwww.diva-portal.org%2Fsmash%2Fget%2Fdiva2%3A240110%2FFULLTEXT01.pdf&ei=crJTVOWIFpP3yQTomYCIBw&usg=AFQjCNGWc9olvAaMXMa-If7gi3yK9DAHUA&sig2=UBo9Smw5E7w3xJFYx01hbg&bvm=bv.78677474,d.aWw. [Last accessed on 2016 July 20].

[99] Pedersen E. Health aspects associated with wind turbine noise—results from three field studies. *Noise Control Engineering Journal* 2011;59:47-53.

[100] Pierpont N. Wind Turbine Syndrome and the brain. Keynote address at First International Symposium on the Global Wind Industry and Adverse Health Effects: Loss of Social Justice?, November 15, 2010. Available from: http://www.windturbinesyndrome.com/img/WTSbrain-color.pdf. [Last accessed on 2016 July 20].

[101] Punch J, James R, Pabst D. Wind turbine noise: what audiologists should know. *Audiology Today* 2010:20-31.

[102] Roberts JD, Roberts MA. Wind turbines: is there a human health risk? *Journal of Environmental Health* 2013;75: 8-13, 16-17.

[103] Shepherd D, Billington R. Mitigating the acoustic impacts of modern technologies: acoustic, health, and psychosocial factors informing wind farm placement. *Bulletin of Science, Technology & Society* 2011;31:389-398.

[104] Shepherd D, McBride D, Welch D, Dirks KN, Hill EM. Evaluating the impact of wind turbine noise on health-related quality of life. *Noise & Health* 2011;13:333-339.

[105] Timmerman NS. Wind turbine noise. *Acoustics Today* 2013;9:22-29.

[106] Abbasi M, Monnazzam MR, Zakerian SA, Yousefzadeh A. Effect of wind turbine noise on workers' sleep disorder: a case study of Manjil Wind Farm in Northern Iran. *Fluctuation and Noise Letters* 2015;14(2):1-15.

[107] Abbasi M, Monazzam MR, Akbarzadeh A, Zakerian SA, Ebrahimi MH, Impact of wind turbine sound on general health, sleep disturbance and annoyance of workers: a pilot-study in Manjil wind farm, Iran. *Journal of Environmental Health Science & Engineering* 2015,13:71-79.

[108] Persson Waye KP, Clow A, Edwards S, Hucklebridge F, Rylander R. Effects of low frequency noise on the cortisol response to awakening and subjective sleep quality. *Life Sciences* 2003:72:863-875.

[109] Bronzaft AL. The noise from wind turbines: potential adverse impacts on children's wellbeing. *Bulletin of Science, Technology & Society* 2011;31:291-295.

[110] Fiumicelli D. Wind farm noise dose response. *Acoustics Bulletin* November/December 2011:26-35.

[111] Physicians for Human Rights. Leave no marks: enhanced interrogation techniques and the risk of criminality. August 2007. Available from: https://s3.amazonaws.com/PHR_Reports/leave-no-marks.pdf. [Last accessed on 2016 July 20].

[112] Kasprzak C. The influence of infrasound noise from wind turbines on EEG signal patterns in humans. *Acoustic and Biomedical Engineering* 2014;125:A-20-A23.

[113] Paller C, Bigelow P, Majowicz S, Law J, Christidis T. Wind turbine noise, sleep quality, and symptoms of inner ear problems. Presentation at Symposia of the Ontario Research Chairs in Public Policy. ND. Available from: http://docs.wind-watch.org/Paller_Poster_15Oct2013.pdf. [Last accessed on 2016 July 20].

[114] Howe Gastmeier Chapnik Limited. Low frequency noise and infrasound associated with wind turbine generator systems: a literature review. December 10, 2010. Ontario Ministry of the Environment RFP No. OSS-078696. Available from: https://dr6j45jk9xcmk.cloudfront.net/documents/1160/88-noise-and-wind-turbine-generator-systems-en.pdf. [Last accessed on 2016 July 20].

[115] Pawlaczyk-Luszczyńska M, Dudarewicz A, Waszkowska M, Szymczak W, Śliwińska-Kowalska M. The impact of low frequency noise on human mental performance. International *Journal of Occupational Medicine and Health* 2005;18(2):185-198.

[116] Rand RW, Ambrose SE, Krogh ME. Occupational health and industrial wind turbines: a case study. *Bulletin of Science, Technology & Society* 2011;31:359-362.

[117] Palmer W. Wind turbine sound prediction – the consequence of getting it wrong. Presentation at ICA 2013 meeting of Acoustical Society of America, June 2013. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=3&ved=0CDUQFjAC&u

rl=http%3A%2F%2Fs3.amazonaws.com%2Fwindaction%2Fattachments%2F1791%2FPMA040066_Palmer.pdf&ei=E12RUsyLFIS2rQHp-oDYAQ&usg=AFQjCNGQ45VRuryLVl9x2-T81J8z3qdukQ&bvm=bv.56988011,d.aWM. [Last accessed on 2016 July 20].

[118] Castelo Branco NAA, Alves-Pereira M. Vibroacoustic disease. *Noise & Health* 2004;6(23):3–20.

[119] Castelo Branco NA. The clinical stages of vibroacoustic disease. *Aviation, Space, and Environmental Medicine* 1999;70(3, Pt 2):A32-39.

[120] Kennedy RS, Allgood GO, Van Hoy BW, Lilienthal MG. Motion sickness symptoms and postural changes following flights in motion-based flight trainers. *Journal of Low Frequency Noise and Vibration* 1987;6(4):147-153.

[121] von Gierke HE, Parker DE. Differences in otolith and abdominal viscera graviceptor dynamics: implications for motion sickness and perceived body position. *Aviation, Space, and Environmental Medicine* 1994;65:747-751.

[122] Berger RG, Ashtiani P, Ollson CA, Whitfield Aslund M, McCallum LC, Leventhall G, et al. Health-based audible noise guidelines account for infrasound and low-frequency noise produced by wind turbines. *Frontiers in Public Health* 2015;3:1-14.

[123] Carey J, Amin N. Evolutionary changes in the cochlea and labyrinth: solving the problem of sound transmission to the balance organs of the inner ear. *The Anatomical Record* Part A, 2006;288A:482-90.

[124] Persinger MA. Infrasound, human health, and adaptation: an integrative overview of recondite hazards in a complex environment. *Natural Hazards* 2013;70:501-525.

[125] Salt AN. Infrasound: your ears “hear” it but they don't tell your brain. Presentation at Symposium on Adverse Health Effects of Industrial Wind Turbines. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0CCcQFjAA&url=http%3A%2F%2Foto2.wustl.edu%2Fcochlea%2FWTPicton_salt_final.pdf&ei=H1hqU7XUA4iiyATMpoGoBQ&usg=AFQjCNHzz_0mBFhOP1GKgv4b0ncCC_iW7g&sig2=UxKESjK4Yr-lulkP7o2xmQ&bvm=bv.66111022,d.aWc. [Last accessed on 2016 July 20].

[126] Salt AA, Hullar TE. Responses of the ear to low-frequency sounds, infrasound and wind turbines. *Hearing Research* 2010;268:12-21.

[127] Salt AN, Lichtenhan JT. How does wind turbine noise affect people? *Acoustics Today* 2014;10:20-28.

[128] Salt AN, Kaltenbach JA. Infrasound from wind turbines could affect humans. *Bulletin of Science, Technology & Society* 2011;31:296-302.

[129] Salt AN, Lichtenhan JT. Responses of the inner ear to infrasound. Presentation at Fourth International Meeting on Wind Turbine Noise, April 2011. Available from: http://oto2.wustl.edu/cochlea/saltlichtenhan.pdf. [Last accessed on 2016 July 20].

[130] Salt AN, Lichtenhan JT. Perception-based protection from low-frequency sounds may not be enough. Presentation at 2012 Internoise conference, August 2012. Available from: http://oto2.wustl.edu/cochlea/saltlichtenhaninternoise2012.pdf. [Last accessed on 2016 July 20].

[131] Salt AN, Lichtenhan JT, Gill RM, Hartsock JJ. Large endolymphatic potentials from low-frequency and infrasonic tones in the guinea pig. *Journal of the Acoustical Society of America* 2013;133:1561-1571.

[132] Salt AN. Wind Turbines can be hazardous to human health. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&ved=0CCcQFjAA&url=http%3A%2F%2Foto2.wustl.edu%2Fcochlea%2Fwind.html&ei=PSl6U-fkMIedqAbW1YLIAw&usg=AFQjCNE7m2g3uKmdL4OgN4es_-gymFbe-g&sig2=a8SmsvCVRQ2lb_TC4Idjfw&bvm=bv.66917471,d.b2k&cad=rja. [Last accessed on 2016 July 20].

[133] Oohashi T, Nishina E, Honda M, Yonekura Y, Fuwamoto Y, Kawai N., et al. Inaudible high-frequency sounds affect brain activity: hypersonic effect. *Journal of Neurophysiology* 2000; 83:3548-3558.

[134] Bauer M, Sander-Thömmes T, Ihlenfeld A, Kühn S, Kühler R, Koch C. Investigation of perception at infrasound frequencies by functional magnetic resonance imaging (fMRI) and magnetoencephalography (MEG). Presentation at 22nd International Congress on Sound and Vibration. Available from: https://stopthesethings.files.wordpress.com/2015/07/full_paper_koch-v2-1.pdf. [Last accessed on 2016 July 14.]

[135] He M, Krahé D. EEG reaction under low frequency noise exposure. Presentation at 22nd International Congress on Sound and Vibration. Available from: http://waubrafoundation.org.au/wp-content/uploads/2016/02/He-Krahe-EEG-Reaction-Under-Low-Frequency-Noise-Exposure.pdf. [Last accessed on 2016 July 15.]

[136] Kugler K, Wiegrebe L, Grothe B, Kössl M, Gürkov R, Krause E, et al. Low-frequency sound affects active micromechanics in the human inner ear. *Royal Society Open Science* October 2014. Available from: http://dx.doi.org/10.1098/rsos.140166. [Last accessed on 2016 July 20].

[137] Kemp DT. Stimulated acoustic emissions from within the human auditory system. *Journal of the Acoustical Society of America* 1978;64:1386-1391.

[138] Schomer PD, Erdreich J, Pamidighantam PK, Boyle JH. A theory to explain some physiological effects of the infrasonic emissions at some wind farm sites. *Journal of the Acoustical Society of America* 2015;137,1356-1365.

[139] Michaud DS, Feder K, Keith SE, Voicescu SA, Marro L, Than J, et al. Effects of wind turbine noise on self-reported and objective measures of sleep. *Sleep* 2016;39(1):97-109.

[140] Michaud D. Health and well-being related to wind turbine noise exposure: Summary of results. *Journal of the Acoustical Society of America* 2015;137:2368.

[141] Michaud DS, Feder K, Keith SE, Voicescu SA, Marro L, Than J, et al. Exposure to wind turbine noise: perceptual responses and reported health effects. *Journal of the Acoustical Society of America* 2016;139:1443-1454.

[142] Michaud DS, Keith SE, Feder K, Voicescu SA, Marro L, Than J, et al. Personal and situational variables associated with wind turbine noise annoyance *Journal of the Acoustical Society of America* 2016;139:1455-1466.

[143] Michaud DS, Feder K, Keith SE, Voicescu SA, Marro L, Than J, et al. Self-reported and measured stress related responses associated with exposure to wind turbine noise. *Journal of the Acoustical Society of America* 2016;139:1467-1479.

[144] Voicescu SA, Michaud DS, Feder K, Marro L, Than J, Guay M, et al. Estimating annoyance to calculated wind turbine shadow flicker is improved when variables associated with wind turbine noise exposure are considered. *Journal of the Acoustical Society of America* 2016;139:1480-1492.

[145] Michaud DS. An evaluation of how nightly variations in wind turbine noise levels influence wrist actigraphy measured sleep patterns. *Journal of the Acoustical Society of America* 2016;139:2150.

[146] Pedersen E, van den Berg F, Bakker R, Bouma J. Response to noise from modern wind farms in The Netherlands. *Journal of the Acoustical Society of America* 2009;126:634-643.

[147] Position of the National Institute of Public Health – National Institute of Hygiene on wind farms. March 8, 2016. Available from: http://en.friends-against-wind.org/health/position-of-the-polish-public-health-institute-on-wind-farms. [Last accessed on 2016 July 14.]

[148] Knopper LD, Ollson CA, McCallum LC, Whitfield Aslund ML, Berger RG, Souweine K, McDaniel M. Wind turbines and human health. *Frontiers in Public Health* 2014;19.

[149] Pedersen E, Persson Waye KP. Wind turbine noise, annoyance and self-reported health and wellbeing in different living environments. *Occupational and Environmental Medicine* 2007;64(7):480-486.

[150] Tempest W. Loudness and annoyance due to low frequency sound. *Acustica* 1973;29: 205-209.

[151] Bastasch M, van Dam J, Søndergaard B, Rogers A. Wind turbine noise: an overview. *Canadian Acoustics* 2006;34:7-15.

[152] Ruotolo F, Senese VP, Ruggiero G, Maffei L, Masullo M, Iachin T. Individual reactions to a multisensory immersive virtual environment: the impact of a wind farm on individuals. *Cognitive Processing* 2012; Suppl 1, S319-23.

[153] Rylander R. Physiological aspects of noise-induced stress and annoyance. *Journal of Sound and Vibration* 2004;277:471–478.

[154] Fast S, Mabee W, Baxter J, Christidis T, Driver L, Hill S, et al. Lessons learned from Ontario wind energy disputes. *Nature Energy* 2016. Available from: http://www.nature.com/articles/nenergy201528. [Last accessed on 2016 July 18.]

[155] McCallum LC, Whitfield Aslund ML, Knopper LD, Ferguson GM, Ollson CA. Measuring electromagnetic fields (EMF) around wind turbines in Canada: is there a human health concern? *Environmental Health* 2014;13:1-6.

[156] Röösli M. Radiofrequency electromagnetic field exposure and non-specific symptoms of ill health: a systematic review. *Environmental Research* 2008;107(2):277–287.

[157] Rubin GJ, Das Munshi J, Wessely S. Electromagnetic hypersensitivity: a systematic review of provocation studies. *Psychosomatic Medicine* 2005;67(2):224–232.

[158] Fletcher H, Munson WA. Loudness, its definition, measurement and calculation. *Journal of the Acoustical Society of America* 1933;5:82-108.

[159] Pedersen E, Persson Waye KP. Wind turbines—low level noise sources interfering with restoration? *Environmental Research Letters* 2008;3:1-5.

[160] Bray W. Relevance and applicability of the soundscape concept to physiological or behavioral effects caused by noise at very low frequencies which may not be audible. Presentation at 164th meeting of Acoustical Society of America, October 2012. Available from: http://acoustics.org/pressroom/httpdocs/164th/Bray_2aNS6.html. [Last accessed on 2016 July 20].

[161] McMurtry RY. Toward a case definition of adverse health effects in the environs of industrial wind turbines: facilitating a clinical diagnosis. *Bulletin of Science, Technology & Society* 2011; 31:316-320.

[162] McMurtry RY, Krogh CME, Diagnostic criteria for adverse health effects in the environs of wind turbines. *Journal of the Royal Society of Medicine Open* 2014;5:1–5.

[163] McCunney RJ, Morfeld P, Colby WD, Mundt KA. Wind turbines and health: An examination of a proposed case definition. *Noise & Health* 2015;17:175-181.

[164] Halperin A. Big wind is better than big oil, but just as bad at P.R. June 15, 2014. Available from: https://newrepublic.com/article/118138/big-wind-needs-address-wind-turbine-syndrome. [Last accessed on 2016 July 20].

[165] Berglund B, Hassmen P, Soames Job RF. Sources and effects of low-frequency noise. *Journal of the Acoustical Society of America* 1996;99:2985-3002.

[166] ISO 1996-1:2016. Acoustics—Description, measurement and assessment of environmental noise—Part 1: Basic quantities and assessment procedures. Available from: http://www.iso.org/iso/home/store/catalogue_ics/catalogue_detail_ics.htm?csnumber=59765. [Last accessed on 2016 July 11].

[167]ANSI/ASA S12.9—2005/Part 4 (R2015). Quantities and procedures for description and measurement of environment sound—Part 4: Noise assessment and prediction of long-term community response. Available from: http://webstore.ansi.org/RecordDetail.aspx?sku=ANSI%2fASA+S12.9-2005%2fPart+4+(R2015). [Last accessed on 2016 July 11].

[168] Hill AB. The environment and disease: association or causation? *Proceedings of the Royal Society of Medicine* 1965;58:295-300.

[169] Phillips CV. Properly interpreting the epidemiologic evidence about the health effects of industrial wind turbines on nearby residents. *Bulletin of Science, Technology & Society* 2011;31:303-315.

[170] World Health Organization Health topics: epidemiology. Available from: http://www.who.int/topics/epidemiology/en/. [Last accessed on 2016 July 20].

[171] Songer T. Basic epidemiology: study designs in epidemiologic research. Available from: http://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=2&ved=0CCsQFjAB&url=http%3A%2F%2Fwww.pitt.edu%2F~super7%2F19011-20001%2F19101.ppt&ei=LRibU4PAPMyiyATpioGwDQ&usg=AFQjCNG5R1wNMj3ipEws4rzLaG7kHvH0CQ&sig2=T1hDZwtnlN7BGvsvpCIfgQ&bvm=bv.68911936,d.aWw&cad=rja. [Last accessed on 2016 July 20].

[172] University of Ottawa. Society, the individual and medicine: study designs. Available from: http://www.med.uottawa.ca/sim/data/Study_Designs_e.htm. [Last accessed on 2016 July 20].

[173] Chapman S, St. George A, Waller K, Cakic V. The pattern of complaints about Australian wind farms does not match the establishment and distribution of turbines: support for the psychogenic, 'Communicated Disease' hypothesis. *PLoS ONE* 2013;8(10). Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3797792/. [Last accessed on 2016 July 20].

[174] Crichton F, Dodd G, Schmid G, Gamble G, Petrie KJ. Can expectations produce symptoms from infrasound associated with wind turbines? *Health Psychology* 2013;33:360-364.

[175] Punch J. Review of Crichton et al. (Can expectations produce symptoms from infrasound associated with wind turbines?). March 28, 2013. Available from: http://www.wind-watch.org/documents/review-of-crichton-et-al-can-expectations-produce-symptoms-from-infrasound-associated-with-wind-turbines/. [Last accessed on 2016 July 20].

[176] Williams T. The effects of expectations on perception: experimental design issues and further evidence. Working paper series, Federal Reserve Bank of Boston, No. 07-14. Available from: http://www.bostonfed.org/economic/wp/wp2007/wp0714.pdf. [Last accessed on 2016 July 20].

[177] Crichton F, Dodd G, Schmid G, Gamble G, Cundy T, Petrie KJ. The power of positive and negative expectations to influence reported symptoms and mood during exposure to wind farm sound. *Health Psychology* 2013; 33:1588-1592.

[178] Chen HA, Narins P. Wind turbines and ghost stories: the effects of infrasound on the human auditory system. *Acoustics Today* 2012;8:51-56.

[179] Tonin R, Brett J, Colagiuri B. The effect of infrasound and negative expectations to adverse pathological symptoms from wind farms. *Journal of Low Frequency Noise, Vibration and Active Control* 2016;35(1);77–90.

[180] Taylor J, Eastwick C, Wilson R, Lawrence C. The influence of negative oriented personality traits on the effects of wind turbine noise. *Personality and Individual Differences* 2013;54:338–343.

BEFORE THE PUBLIC UTILITIES COMMISSION

OF THE STATE OF SOUTH DAKOTA

IN THE MATTER OF THE APPLICATION BY PREVAILING WIND PARK, LLC FOR A PERMIT OF A WIND ENERGY FACILITY IN BON HOMME COUNTY, CHARLES MIX COUNTY AND HUTCHINSON COUNTY, SOUTH DAKOTA, FOR THE PREVAILING WIND PARK PROJECT

EL18-026

PREFILED TESTIMONY OF PROF. MARIANA ALVES-PEREIRA

ON BEHALF OF INTERVENORS

EXHIBIT
Int. I-3

I. ACADEMIC AND PROFESSIONAL BACKGROUND

Q: Please state your name, address and academic affiliation.

A: My name is Mariana Alves-Pereira. My mailing address is Rua do Viveiro, 402, 1E, Estoril, 2765-294 Portugal. I am currently affiliated with Lusófona University, in Lisbon Portugal.

Q: Please describe your academic qualifications and research expertise.

A: I hold a B.S. in Physics from the State University of New York at Stony Brook, a M.Sc. in Biomedical Engineering from Drexel University in Philadelphia, and a Ph.D. in Environmental Sciences from the New University of Lisbon. I am an expert in the field of the biological responses to exposures to infrasound and low frequency noise (ILFN).

In 1988, before attending undergraduate studies in the U.S., I was employed by the Portuguese Air Force where I worked as a technical translator. I soon became involved with the onsite biomedical research team (founded in 1980 and led by Col. Nuno A. A. Castelo Branco, MD, pathologist) that was studying the 'non-auditory' effects of occupational noise exposure. Thus began my interest and involvement in this matter. Since then, I published my first peer-reviewed paper in 1999 and the latest in 2017. Please see:

- *Exhibit 1: Curriculum Vitae 2018 – Mariana Alves-Pereira*
- *Exhibit 1a: Annex to Curriculum Vitae - List of Publications*

Research into the 'health-effects' of ILFN exposure is (necessarily) a multidisciplinary topic, requiring familiarity with a) the physics of acoustics, acoustical propagation and measurement, b) biomechanical properties of mammalian tissue, c) cellular architecture and cellular biology, d) histological and ultra-structural features of tissue and cellular organization in health and in disease, e) non-invasive and invasive clinical evaluations, f) laboratorial animal studies and studies among human populations, and g) patient anamnesis. Over the past thirty years, I have been involved in all these aspects in order to gain a deeper understanding into the biological response to ILFN exposure.

II. PURPOSE OF TESTIMONY

Q: What is the purpose of your testimony?

A: The purpose of my testimony is to provide the South Dakota Public Utilities Commission with scientific information regarding the health-effects associated with ILFN exposure.

Q: What documents related to this case have you reviewed in preparation of your testimony?

A: For the purposes of providing expert testimony at these hearings, I have reviewed the following documents:

- Applicant's burden of proof, SDCL § 49-41B-22
- Prevailing Wind Park Sound Study, prepared by Burns & McDonnell, dated 30MAY18. Docket #: Appendix M
- Chris Howell Direct Testimony, dated 30MAY18, SD PUC Docket #: EL 18-026
- Dr. Mark Roberts Supplemental Direct Testimony, dated 10AUG18, SD PUC Docket #: EL-18-026
- Bon Homme County, Article 17 – Wind Energy Systems
- David M. Hessler Direct Testimony, dated 04MAY18, Docket #: EL18-003
- I have also been provided with the PUC's final decisions on the Crocker Project and the Dakota Range Project.

Q: Please provide a brief summary of your opinion on these matters.

A: When 'something' is suspected of being an agent of disease, then, for a scientist, the priority is to figure out a way to *quantify* said agent of disease.

Once the agent of disease is quantified, dose-response relationships can be sought. These relate a certain amount of the agent of disease to a particular health-endpoint(s).

These health-related endpoints must be in the form of useful *scientific* data, i.e., capable of being clinically-corroborated.

The above assertions are in compliance with the Scientific Method (which, among other aspects, requires that parameters under study be of an *objective* nature), and with its corollary, that of Evidence-based Medicine (which, among other aspects, requires that medical endpoints be *objective* in nature, i.e., capable of being clinically corroborated).

When it comes to studying the health effects of ILFN exposure, however, these fundamental axioms of the Scientific Method and Evidence-based Medicine are somehow forgotten, or deemed not applicable.

Specifically:

1. The dBA metric *does not* quantify ILFN, hence, the dBA unit is not useful for establishing, denying or predicting *de facto* health effects on humans and animal populations exposed to anthropogenic (human-made) or to natural ILFN.

2. Annoyance *is not* an objective parameter and hence, in accordance with the axioms of Evidence-based Medicine, cannot be used to ascertain *de facto* health effects.

3. It is often put forth that people living in ILFN-contaminated homes (whatever the source), and who attribute their deteriorating health to anthropogenic sources of ILFN are, in realty, suffering from a 'nocebo effect', i.e., effects that have no real organic basis and are of a psychosomatic origin. In accordance with the axioms of Evidence-based Medicine and, even more fundamentally, the Scientific Method, *psychosomatic illnesses must also be clinically corroborated*; their proposed existence based on mere assertions is not scientifically valid.

III. CONTEXTUALIZATION OF THE MATTER – THE dBA METRIC

Q: Why do you claim that the dBA metric does not quantify ILFN?

A: Complex mathematical concepts are sometimes best understood with the aid of images. For this purpose I am including the image below (line 100, Figs 1-3) showing that when the dBA metric is used to measure the acoustic energy at 10 Hz (classically considered to be within the infrasound range), the difference between what is measured and what is actually present in the environment is 70 dB.

This image is separately attached to this Testimony as

- *Exhibit 2 – Pictorial explanation of the dBA metric*

The dBA curve was developed in the 1920's for the purposes of improving telephone acoustics, for which ILFN was considered irrelevant.

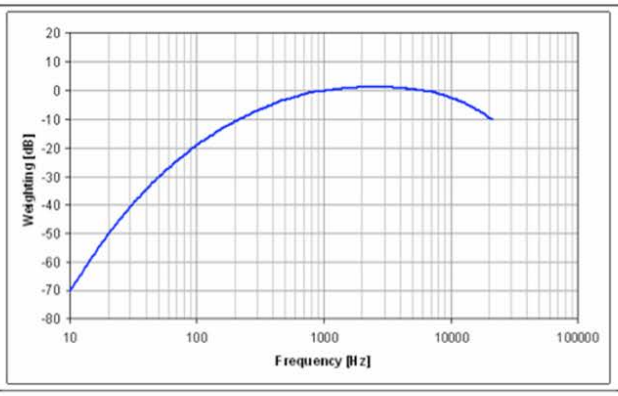


Fig. 1
Frequency response curve used by the dBA metric.

The dBA began its development in the 1920's, to improve telephone acoustics.

With decreasing frequency, there is an increasing difference between what the dBA measures and what is actually present in the environment.

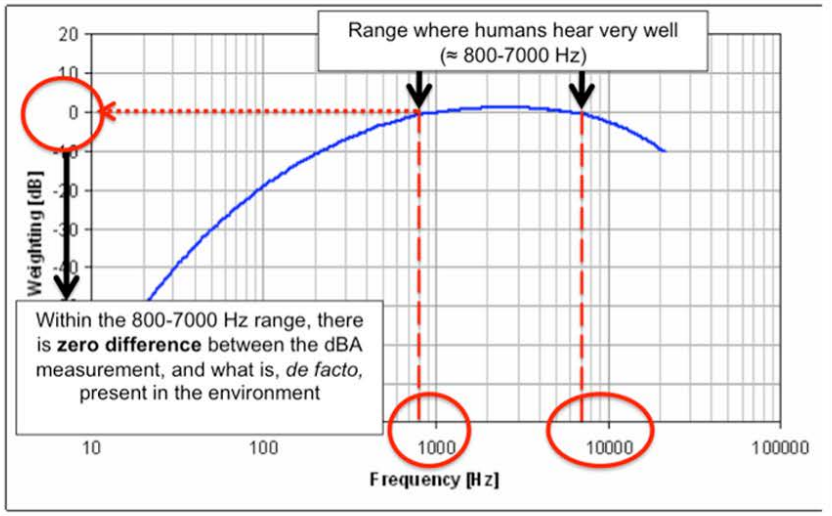


Fig. 2
The dBA metric is a good methodology to protect the human hearing function.

For forensic purposes, occupational deafness is checked at 4000 Hz.

The dBA assumes: "what you can't hear won't hurt you."

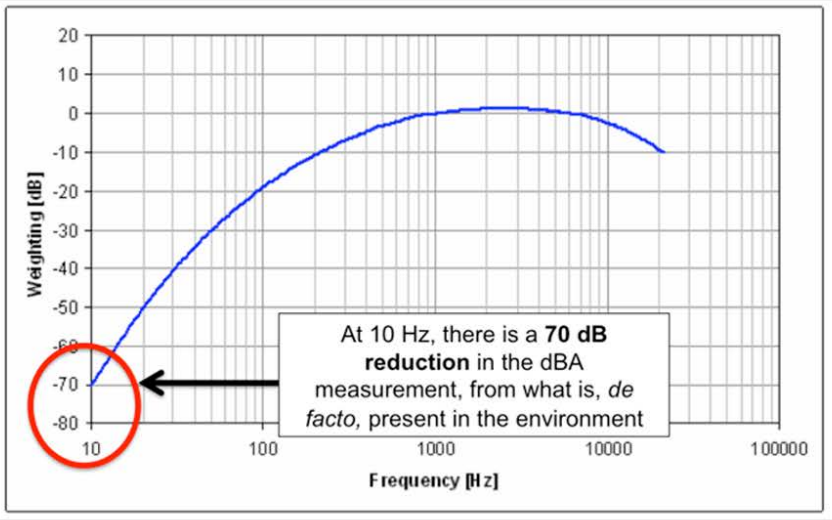


Fig. 3
At 10 Hz, the dBA metric will reduce the numerical value of its measurement by 70 dB.

At 10 Hz, the dBA metric *does not quantify* the acoustical energy that is present in the environment.

Image source: Dirac Dirac Delta Science & Engineering Encyclopedia (2018) A-Weighting. http://www.diracdelta.co.uk/science/source/a/w/aweighting/source.html#.W45nwy2ZORs

Q: If the dBA unit is so inadequate to quantify ILFN, why, then, do the vast majority of studies from Governmental Agencies and Research Centers use the dBA metric?

A: For several reasons:

1. The purpose of measurement is oftentimes not scientific, but legal, i.e., to ascertain compliance with existing regulations. Indeed, since ILFN is not regulated, why measure it at all? And since the vast majority of regulations are expressed in dBA units, why measure with anything else? Even if ILFN *is* quantified, science has not yet determined the appropriate dose-response relationships for human exposures. Therefore its numerical quantification is deemed as unnecessary by many professional acousticians.

2. "What you can't hear won't hurt you." Since the dBA curve describes the human *auditory* threshold, and since noise exposure is (erroneously) assumed to only affect humans via the ear, the dBA metric is therefore (erroneously) considered sufficient to establish, deny or predict health-effects of noise exposure on human populations. Most medical schools do not cover the topic of ILFN as an agent of disease. Health-complaints due to noise exposure are, classically, dealt with by otolaryngologists (ear-nose and throat specialists), who are taught that the human auditory threshold, represented by the dBA curve, is the only issue of concern.

3. There are larger issues that are beyond my area of expertise: conceding that the dBA unit is insufficient to protect populations against 'noise' would necessarily imply a massive shift in the way we protect people from this agent of disease; it would potentially open the floodgates for lawsuits related to occupational exposures (similar to those seen with occupational deafness and asbestosis) and/or class actions due to environmental exposures.

4. Because that is how it has always been done…

IV. CONTEXTUALIZATION OF THE MATTER – HEALTH EFFECTS

Q: What are 'self-reported' health complaints and what is their scientific validity?

A: Any health condition usually begins with health complaints that, when reported by the individual, are, by definition, considered as 'self-reported'. When these complaints are 'self-reported' to a medical doctor, they become part of the *anamnesis*, i.e., the documentation of the patient's medical history and current complaints that is partially obtained through information that is 'self-reported' by the patient.

For self-reported complaints to acquire any type of scientific validity, the intervention of a medical doctor is required. Based on the patient's self-reported complaints (called *symptoms*) and based on medical doctors' trained observations (called *signs*), a hypothesis will be put forth as to the cause of the medically observed *signs* that occur in conjunction with the patient's ('self-reported') *symptoms*.

Self-reported symptoms become scientifically-valid data when the results of relevant and pertinent medical diagnostic tests become available. These would then have the attributes of 'objective, and clinically-corroborated' parameters, a *sine qua non* condition to be considered scientifically valid.

Q: Can you provide an example?

A: Yes, I can. There is a group of individuals (US citizens) that began having cognitive and behavioral complaints such as: "difficulty remembering, mental fog, difficulty concentrating, feeling slowed, irritability, feeling more emotional." They also self-reported: "balance problems, dizziness and nausea," in addition to increased "visual problems, sound sensitivity," sleep disorders and headaches. All these self-reported symptoms are very similar to those made by families living in ILFN-contaminated homes (whatever the source).

Based on these self-reported complaints, this particular group of individuals received a series of pertinent medical diagnostic tests that provided scientifically valid medical data, i.e., data that was clinically-corroborated. These included: neuropsychological testing, posture and balance studies, oculomotor evaluations, audiometric assessments, and brain MRI. It was concluded that: "These individuals appeared to have sustained injury to widespread brain networks without an associated history of head trauma."

The hypothesis that this "constellation of acute and persistent signs and symptoms" could be of a psychosomatic etiology (i.e., a nocebo effect) does not appear to have been placed.

The above information refers to a peer-reviewed paper, published in JAMA (Journal of the American Medical Association) in 2018, and is included in this Testimony as:

- *Exhibit 3: 2018-JAMA-Swanson et al*

Exhibit 3 serves as an example of the pertinent medical diagnostic tests that should be prescribed when people 'self-report' specific health complaints, i.e., when they manifest certain types of symptoms.

Q: Why are some people affected and others not within the same household?

A: There are two exposure-linked factors (other factors are co-existent) that profoundly condition the onset of symptoms among families living in ILFN-contaminated homes (whatever the source):

1. *Prior ILFN exposure histories*, i.e., the overall, life-time exposure the individual may already have had, before being exposed to (anthropogenic) ILFN in the home.

2. *Residential time exposure patterns*, i.e., how much time is spent in the ILFN-contaminated home (homemakers vs. working outside the home, and sleep time).

3. *Individual susceptibility factors*, i.e., genetic make-up, diet, lifestyle, etc

This information, crucial to any health-related study on ILFN exposure, is generally not taken into account. There appears to be an (erroneous) expectation that once ILFN contamination begins in a dwelling, all family members will manifest symptoms within the same time span. This would only be true if *all* members of the family had the exact same prior noise exposure histories, *and, simultaneously,* the same schedules in terms of remaining within the contaminated home.

When this information is not taken into account in large-scale studies, the subsequent statistical analysis is likely to return inconclusive results.

V. INDUSTRIAL WIND TURBINES

Q: When was your group first contacted about an ILFN-contaminated home caused by the proximity of wind turbines?

A: The first family to contact our group specifically because of wind turbines was Canadian, back in the early 2000's.

Q: And since then, how many have contacted you, specifically because of wind turbines?

A: Hundreds.

Q: Do you conduct field-research?

A: Yes, I do.

Q: What kind of data are you and your group collecting?

A: We are collecting acoustical data in a manner that allows us to quantify ILFN, and we are conducting extensive interviews among the complaining populations, taking into account prior noise exposure histories and time exposure patterns within and around the residence.

Q: Have you published any peer-reviewed results of this field data?

A: Not very many, as of yet, because we have only had a new measuring equipment since 2016. This new acoustical measuring equipment allows us not only to *quantify* ILFN, but also provides information on the time profile of the acoustical event, an important parameter when studying health effects.

Last year we published a peer-reviewed paper on the acoustics of industrial wind turbines in a mink farm in Denmark. This year, in a more informal publication, *Engineers Ireland,* we also published some results from our recent fieldwork in Ireland. Both these papers are submitted with this Testimony as:

- *Exhibit 4: 2017- SJAEM-Alves Pereira et al.*
- *Exhibit 5: 2018-Engineers Ireland- Alves-Pereira et al.*

In both papers, the wind turbine acoustic signature was clearly identified within the animal sheds of the Danish mink farm, and within a home in Ireland that has been abandoned. The youngest child of this Irish family has been formally diagnosed with epilepsy, while the oldest child has been formally diagnosed with post-traumatic stress disorder. The owner of the Danish mink farm has also been formally diagnosed with post-traumatic stress disorder.

We currently have several other papers undergoing the peer-review process.

Q: What is a safe distance between wind turbines and homes?

A: Science does not yet have the numerical values for that. We have picked up wind turbine acoustic signatures in homes 12 km (7.4 miles) away from the nearest industrial wind turbine. We are currently analyzing data from 8 homes around the same wind development, that distance from 1 km (0.62 miles) to 23 km (14.2 miles) to the nearest industrial wind turbine. Results are not yet available. Safe distances have not yet been established for the ILFN generated by wind turbines.

Q: What is a 'wind turbine acoustic signature'?

A: All things have acoustic signatures, such as (for example) road vehicles, aircraft, trains, etc. An acoustic signature is the periodic and identifiable characteristic of the acoustic output associated with any machinery. The acoustic signature of an industrial wind turbine is associated with the number of times a blade passes a given point, per second – this is called the *blade pass frequency* or *fundamental frequency.*

The image shown in line 257 (Figs 4,5) represents an industrial wind turbine acoustic signature with a blade pass frequency of 0.65 Hz. The 'signature' appears as consecutive peaks, interspaced by a mathematical sequence (harmonic series), and which is due to the periodic rotation of the blades.

There are *two noteworthy features* that are pertinent to the matter at hand:

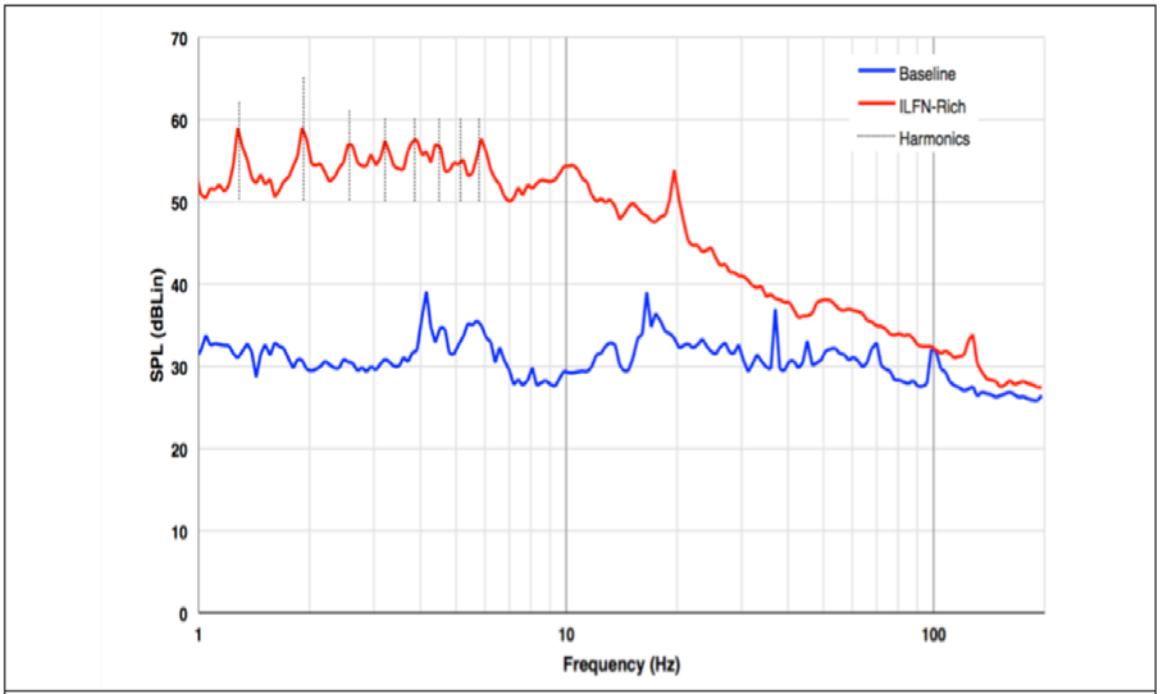


Fig 4. The blue curve represents the acoustical environment when industrial wind turbines (IWT) are not rotating. The red curve represents the same acoustical environment while IWT were rotating. (IWT rotation confirmed with video footage during measurements.) The dotted lines (Harmonics) represent peaks of acoustical energy that fall on a mathematical sequence, and hence, cannot be attributed to wind.

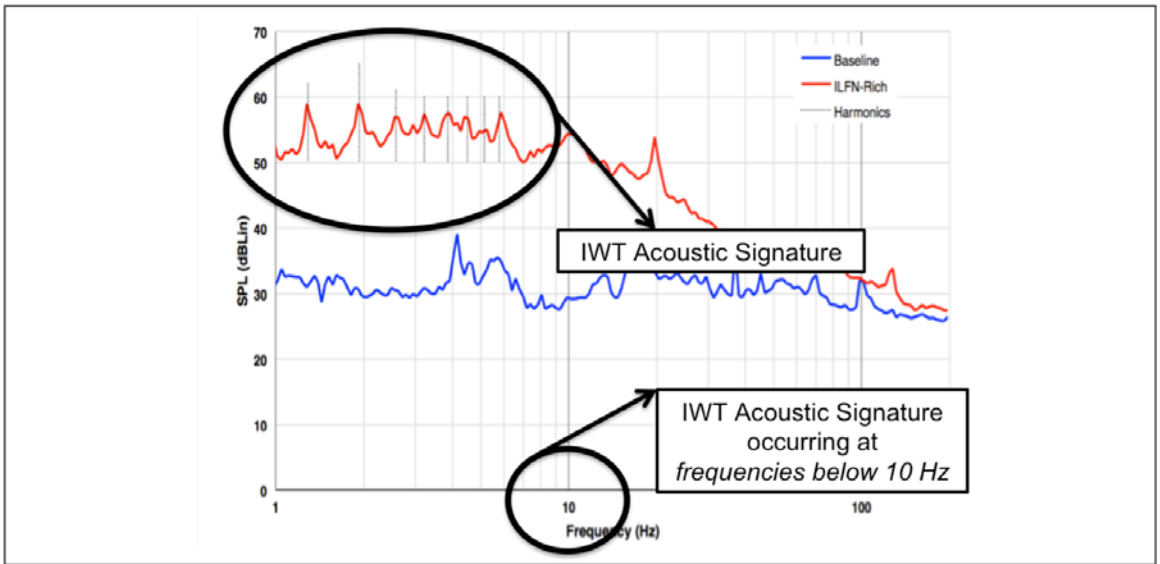


Fig 5. The industrial wind turbine acoustic signature occurs at frequencies below 10 Hz, hence, the dBA metric is unsuitable to characterize this type of acoustical environment.

Image Source: Alves-Pereira M, Bakker HHC. *Occupational and residential exposures to infrasound and low frequency noise in aerospace professionals: Flawed assumptions, inappropriate quantification of acoustic environments, and the inability to determine dose-response.* Scientific J Aerosp Eng Mech 2017, 1(2):83-98. (Figure 5)

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1. The fact that the wind turbine acoustic signature coincides with a mathematical sequence (harmonic series) means that blowing wind is not the cause.

2. The fact that the wind turbine acoustic signature occurs in a frequency range below 10 Hz means that the dBA metric is unsuitable for characterizing these types of acoustical environments.

The image shown below is taken from Exhibit 4, Figure 5.

VI. GUIDELINES AND REGULATIONS

Q: What is your opinion on the noise requirements as stated in Article 17 – Wind Energy Systems?

A: They are antiquated, as are many other regulations on this matter, for the reasons explained above. A single numerical value (45 dBA) is entirely insufficient to characterize the type of acoustical pollution that is generated by industrial wind turbines. In fact, for a rural area, 45 dBA is quite high given that 'normal background noise' in many rural areas around the world is around 25-35 dBA, or lower.

Q: Do you have any comment on the Applicant's burden of proof, as set forth in SDCL § 49-41B-22?

A: Yes. I would like to point out that the way in which this paragraph is worded implies that even if the applicant complies "with all applicable laws and rules," it must *also* assure that the project "will not substantially impair the health, safety or welfare of the inhabitants." This means that the possibility of health, safety and welfare impairment is acknowledged *even when* compliance "with all applicable laws and rules" is met. I find this to be an extraordinarily 'open-minded' position by implicitly putting forth the notion that "applicable laws and rules" on their own will not necessarily prevent impairment of inhabitant "health, safety or welfare". Indeed, this is the situation in which we, as a worldwide collective, find ourselves: current laws and rules are insufficient to protect human populations.

By applying the dBA standard, developed to protect hearing, in any and all cases regardless of the ILFN content of the environment, applicable rules and laws are being complied with, but the health of human population is not fully protected.

VII. COMMENTARY ON TESTIMONY OFFERED BY OTHER EXPERTS

Q: You have had the opportunity to read several testimonies provided by other witnesses.

A: Yes

Q: Do you have any specific comments on the information provided by Dr. Mark Roberts?

A: Yes. I would merely like to clarify some aspects to which Dr. Roberts makes reference.

1. Agents of disease are classified as:

a) biological (viruses, bacteria, etc),

b) chemical (carbon monoxide, pepper spray. etc)

c) physical (electromagnetic radiation, noise, etc)

d) psychosocial (sexual harassment, bullying, etc)

Industrial wind turbines in and of themselves are not considered agents of disease by the medical sciences; it is the ILFN that they generate that is the agent of disease.

While this may seem to be a question of semantics, indeed it is not, as it can lead to crucial flaws in the designs of studies that claim to investigate ILFN-induced health effects.

By assuming that industrial wind turbines are an agent of disease, then studies comparing people who live near wind turbines with those who do not, will appear to be scientifically valid – they are not! The agent of disease is ILFN. To conduct a proper study, people living near industrial wind turbines have to be compared with people who do not live *near any major source of anthropogenic ILFN*. Otherwise, one runs the risk of

comparing people who live near industrial wind turbines with those who live near airports or large volume roadways. Under these circumstances, it is likely that little to no difference will be identified between both populations, and the typical ensuing conclusion is that there is no problem associated with industrial wind turbines.

This type of study design flaw has happened before, in the 2001 Vieques Heart Study conducted by the Agency Disease Registry and Toxic Substances. Please see:

- *Exhibit 6: 2013-Vieques Final Report*

2. In line 524 it is stated that I am the primary researcher in vibroacoustic disease. I am not. The primary researcher has always been Col. Nuno A. A. Castelo Branco, MD, who has just recently retired.

3. In lines 526-528 it is stated that I personally have "not reconciled the difference in the intensity of the low frequency sound she as studied in aircraft maintenance workers and the low intensity of sound produced by wind turbines." Perhaps merely reflecting the witness' unfamiliarity with this physical agent of disease, this is, nevertheless, untrue.

The following was published in 2007 (eleven years ago):

- *Exhibit 7: 2017-PBMB-Alves-Pereira et al.*

"Not all ILFN-exposed workers have [the standard 8hr/day, 5 days/week] exposure schedule. For example, ship machinist can spend 3 weeks onboard ship (i.e., exposed to substantial ILFN-rich environments) and 2 weeks at home (i.e., presumably not in ILFN-rich environments) (Arnot, 2003). Other professional activities exist where the ILFN-exposure time pattern is not the standard 8-h/day exposure, such as with submarine and oil rig operators, astronauts, and environmental exposures in residential areas, where exposure can be continuous over long periods of time, and exists during sleeping hours. In these cases, the evolution of signs and symptoms could be greatly accelerated. For examples, in the case of a Dublin homemaker, epileptic seizures consistent with [vibroacoustic disease] developed after 3 years of residence within an ILFN-infested home (Monteiro et al., 2004). If the ILFN exposure is environmental and /or leisurely, the standard 8h/day model is also not applicable."

The references cited in this quote are offered in this Testimony as

- *Exhibit 8: 2003-Institute of Acoustics UK, Arnot*
- *Exhibit 9: 2004-Internoise-Monteiro et al.*

VII-A. TIME EXPOSURE PROFILES

Q: How does exposure time relate to the onset of symptoms?

The time over which the person is exposed to the agent of disease makes a difference to the evolution of symptoms. Because laypersons are more familiar with radiation, lets us take the example of the common chest x-ray. Receiving a chest x-ray once or twice a year, given its relatively low-dose of radiation, is not considered harmful to humans. It is considered that whatever damage is done during that very brief period of time where x-ray exposure occurred, the body will recuperate. However, receiving 10 chest x-rays per day for a year, might indeed begin to pose a problem in terms of health effects. It is the same with ILFN.

Occupational ILFN exposures (although usually implying exposures to higher levels of acoustical energy than in residences) *cease at the end of the workday*. The workers are afforded a recovery period during which, at the cellular level, the body restores itself from the whole-body mechanical insult.

When anthropogenic ILFN is contaminating a home (even with lower levels than in occupational environments) the body can be exposed 24/7 (particularly in homemakers). Moreover, and perhaps more worrisome, families in ILFN-contaminated homes *are sleeping* while enveloped within an environment that is bombarding their bodies with mechanical agents of disease.

VII-B ILFN – A PHYSICAL AGENT OF DISEASE

Q: You mentioned *mechanical* agent of disease and *physical* agent of disease when referring to ILFN. What, if any, is the difference?

A: 'Noise' in general is classified as a *physical* agent of disease because it is composed of airborne pressure waves. Pressure means a force impacting on a particular area (like the punch of a boxer on the jaw of his/her opponent). The World Health Organization classifies noise as "inanimate mechanical forces" (ICD-10, items W42 and W43). When one is exposed to acoustical phenomena, one becomes enveloped by airborne pressure waves that 'beat on' the biological organism. When the airborne pressure waves have specific 'beating' attributes, then they 'beat on' the auditory hair cells, and we call it sound. With ILFN, these airborne pressure waves do not necessarily induce the perception of sound. This is particularly true in the early stages of residential exposure.

VII-C CELLULAR ARCHITECTURE IN MAMMALS

Q: What happens at the cellular level when these airborne pressure waves bombard the body?

A: Cells respond to biochemical signaling and mechanical signaling. Cellular communication that is made through mechanical signals is called mechanotransduction. When a

mechanical force impacts a cell or group of cells, depending on the attributes of the mechanical force, the cells can be made to stretch, elongate or spread out. When the mechanical force impacting the cell is periodic and continuous, the cells have no time to recover back to their initial (and equilibrium) positions. Damage is cumulative and not instant.

Metaphorically, this is similar to the boxer, who endures 10-12 rounds but then, the cumulative amount of mechanically induced damage (the punches) finally catches up and the boxer is knocked-out. Also similar to ILFN-induced pathology, if the boxer is afforded an appropriate recovery time, he or she can be back in the ring. However, in the hypothetical scenario that the boxer keeps getting punched, even outside the ring, then his/her recovery time will necessarily require a much longer period of time.

Q: Do you have any specific comments on the information provided by Mr. David M. Hessler?

A: Yes. I would like to applaud Mr. Hessler's candid testimony, which I will use as an example for further clarifying for the PUC this complex subject of 'noise'-induced health effects.

In lines 11-12, page 7: “I heard nothing but complete silence, I felt nothing and I could not understand what these people were complaining about;” and in lines 14-16, page 7, quoting Mr. Steven Cooper, a well-known and highly respected acoustician from Australia: “on my first experience the noise was extremely low, could not be detected inside the dwelling and I didn’t understand why these residents would be so vocal and genuinely distressed from the turbines.” These very candid observations are commonly encountered among acousticians when they begin to deal with ILFN issues. Their inability to consciously perceive anything (initially) could stem from their reduced prior exposure to this type of agent of disease and hence their reduced ‘sensitivity.’

Based on Mr. Cooper’s recent and exciting experiments (lines 15-22, page 6 to lines 1-2, page 7), Mr. Hessler has now become convinced that “a minority of people do have a sensitivity to minute pressure pulsations associated with the blade passing frequency which is typically extremely low; less than 1 Hz. The question is: how small or large is this minority?” (lines 2-5, page 8).

If, indeed, the effects of ILFN exposure are cumulative, this questionable minority will certainly have a tendency to grow. Please understand that industrial wind turbines are not the only sources of residential ILFN-contamination. Many people around the world are suffering (*the same*) health effects due to residential ILFN that is unrelated to the

existence of industrial wind turbines. Some of these cases never get documented in scientific journals, however the following Exhibits provide examples of such cases:

- *Exhibit 6: 2013-Vieques Final Report*
- *Exhibit 9: 2004-Internoise-Monteiro et al. (case no. 4)*
- *Exhibit 10: 2004-Internoise-Castelo Branco et al.*

The development of symptoms and conscious perceptions occurs *over time*. Perhaps, in the near future, the 'minority group' will be composed of those who, after living within ILFN-contaminated residences for over one-to-two years, have not developed any clinically-corroborated health endpoints.

Q: Do you have any further information that might be useful for the PUC regarding the Prevailing Wind Park Project?

A: Installing industrial wind turbines as a serpentine throughout residential areas, is not a good idea *if* the health of human (and animal) populations is of any concern.

It is fully recognized that industrial wind turbines are being 'sold' as providing 'green' energy, and this has instigated a worldwide impetus to install these types industrial complexes. Our group has as much against industrial wind turbines as we have against airports, other transportation systems, manufacturing plants, and etc. They are all acknowledged as an integral part of a modern technological society, as we know it. However, protection of public health, the precautionary principle and ethical

considerations preclude us from serpentining airports, transport systems and manufacturing plants among residential areas.

Appropriate zoning laws for industrial wind turbines should be considered. However, in the absence of zoning laws based on scientific information, then the governmental agencies responsible for Public Health should step in to conduct *appropriately designed* epidemiological studies. Ideally, this would study relevant health endpoints *before* and *after* installation of the industrial wind turbines. It would also include the quantification of ILFN *before* and *after* the installations of the industrial wind turbines, with the same wind speed and wind direction, and evaluated *inside* the affected homes.

Q: Does this conclude your Direct Testimony?

A: Yes.

Dated this 10th day of September of 2018



Prof. Mariana Alves-Pereira

Curriculum Vitae



Personal information

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Nationality Portugal

Academic Background

Date 2010

Title of qualification awarded **Doctoral degree in Environmental Sciences**

Name and type of organisation providing education Universidade Nova de Lisboa, Caparica, Portugal

Date 2000

Title of qualification awarded **Masters degree in Biomedical Engineering**

Name and type of organisation providing education Drexel University Philadelphia, PA, USA

Date 1995

Title of qualification awarded **Bachelors degree in Physics**

Name and type of organisation providing education State University of New York Stony Brook, NY, USA

Dates 1990

Title of qualification awarded **12th year High School Diploma**

Name and type of organisation providing education Algés Secondary School Lisbon, Portugal

Dates 1988

Title of qualification awarded **10th-11th years High School – Area of Technological Sciences**

Name and type of organisation providing education Fontes Pereira de Melo Secondary School Porto, Portugal

Scientific Awards

2006 - Prevent More Live Better Scientific Research Award
Diagnosis of Vibroacoustic Disease for Legal & Forensic Purposes.
Attributed by the Instituto de Segurança Higiene e Saúde no Trabalho (Portuguese Governmental Institute for Safety, Hygiene & Health in the Workplace).

2005 - Thomé Villar/Boehringer Ingelheim Research Award
Participation of the Central Airways in Vibroacoustic Disease.
Attributed by the Portuguese Lung Society (Sociedade Portuguesa de Pneumologia).

1999 - Young Investigator Award Finalist
Pericardial Thickening in Commercial Airline Flight Crew.
Attributed by the Space Medicine Branch of the Aerospace Medical Association (USA).

EXHIBIT Int. I-3(a)

Expertise

Acoustics	Infrasound and low frequency noise (ILFN) exposure. Conducted extensive acoustical measurements and analyses in 1/3- and 1/36-octave band, dB Linear, within industrial, urban and residential areas.
Clinical Medicine	Design, implementation and data analyses of clinical studies pertaining to ILFN-exposed populations, including gathering of patient medical and noise exposure histories.
Cellular Biology	Interpretation of light and electron microscopy imaging comparing ILFN-exposed vs. non-exposed cell and tissues of both human and animal models. Analyses of the biological responses to ILFN exposure based on principles of materials and structural engineering and on cytoskeleton dynamics.
Bioengineering	Analysis and interpretation of the response of actin- and tubulin-based structures to ILFN exposures. First to associate cellular and tissue tensegrity architecture to better understand the biological response observed in ILFN-exposed specimens.
International Expert Witness & Consultant	-- Vibroacoustic Disease -- Effects of ILFN on occupationally- or environmentally-exposed human populations.

Teaching Experience

Associate Professor since 2010
Adjunct Professor since 2005.
Assistant Professor since 2002

Dates	Since 2009
Main activities and responsibilities	Coordinating/ Teaching: Physics (Occupational Safety & Health Program) Epidemiology (Occupational Safety & Health Program) Workplace Safety & Health (Safety & Security Program)
Name and address of employer	School of Economic Sciences & Organizations Universidade Lusófona Campo Grande 376, 1749-024 Lisbon, Portugal
Type of business or sector	Higher education

Dates	2007-2013
Main activities and responsibilities	Teaching: Biophysics Lab (Pharmaceutical Sciences Program)
Name and address of employer	School of Health Sciences and Technologies Universidade Lusófona Campo Grande 376, 1749-024 Lisbon, Portugal
Type of business or sector	Higher education

Dates	2005-2007
Main activities and responsibilities	Teaching: Physics (Nursing Program), Coordinating/ Teaching: Biomaterials (Pre-Bologna Radiology Program) Non-ionizing Radiation (Pre-Bologna Radiology Program) Experimental Statistics (Pre-Bologna Radiology Program)
Name and address of employer	Ribeiro Sanches School of Health Sciences Universidade Lusófona Rua dos Telhais aos Olivais, 8-8ª, 1900-693 Lisbon, Portugal
Type of business or sector	Higher education

Dates	2002-2005
Main activities and responsibilities	Coordinating/ Teaching: Acoustical Pollution (Environmental Engineering Program)
Name and address of employer	Department of Environmental Sciences & Engineering Universidade Nova de Lisboa Quinta da Torre, 2829-516 Caparica, Portugal
Type of business or sector	Higher education

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Work Experience

Dates	1988-2013
Occupation or position held	**Senior researcher, Assistant Coordinator for the Vibroacoustic Disease Project**
Main activities and responsibilities	Measure low frequency noise; Patient interviews; Interpreting electron microscopy imaging; Data analyses; Preparation of scientific papers for publication; Oral presentations at international scientific meetings and conferences; Organization of scientific conferences; Expert court witness.
Name and address of employer	Centro da Performance Humana, Estrada Nacional 10, Edificio Cinema, 1º Piso, 2615 Alverca, Portugal
Type of business or sector	Biomedical research in occupational medicine (non-for-profit enterprise)

Dates	2010-2012
Occupation or position held	**Administrator**
Main activities and responsibilities	Liaison between NASA officials and European Governmental officials and private enterprises; Procure projects, actions and activities related to sustainable energy and environmental issues of mutual interest to NASA; Preparation of status reports; Oral presentations of ongoing projects at NASA/C3P Annual Technical Workshops.
Name and address of employer	Center for Pollution Prevention (C3P)
Type of business or sector	Ministry of Environment (non-for-profit enterprise)

Dates	2002-2005
Occupation or position held	**Fellowship recipient**
Main activities and responsibilities	Write, enact and develop research project funded by Portuguese Government (POCTI/FCT): "Low frequency noise in public transportation systems in the Greater Lisbon area"
Name and address of employer	IMAR – Instituto do Mar, Pólo da Faculdade de Ciênicias e Tecnologia, Universidade Nova de Lisboa, Quinta da Torre, 2829-516 Caparica, Portugal
Type of business or sector	Higher education / Research

Dates	Feb-Jul 2000
Occupation or position held	**Associate researcher**
Main activities and responsibilities	PCR studies investigating the genetic expression of ubiquitin and ciclogenase-2 in cells exposed to cadmium.
Name and address of employer	Department of Biological Sciences, Hunter College, City University of New York
Type of business or sector	Higher education / Research

Dates	1991-1995
Occupation or position held	**Librarian**
Main activities and responsibilities	Catalogue new books and scientific journals; Manage book lending; Assist with inquiries.
Name and address of employer	Math/Physics Library State University of New York, Stony Brook, NY, USA
Type of business or sector	Academic

Dates	1988-1990
Occupation or position held	**Technical translator**
Main activities and responsibilities	Quality control inspection manuals for the Lockheed C-130 Hercules, P-3P Orion, and Aerospatiale SA-330 Puma aircraft; Avionics, fuel systems and ground support equipment manuals; Scientific research papers developed by the Medical Division Research Team.
Name and address of employer	OGMA-Indústria Aeronautica de Portugal Parque Aeronautico de Alverca, 2615-173 Alverca, Portugal
Type of business or sector	Aeronautical industry

009413

Dates	1982-1985
Occupation or position held	**Summer-hire** (Commercial Section, US Air Force Section, US Information Services)
Main activities and responsibilities	Administrative tasks; Written and simultaneous translations; Public relations. In 1985, collaborated in the organization of President Ronald Reagan's official visit to Lisbon.
Name and address of employer	United States Embassy Av. das Forças Armadas, 1600-081 Lisbon, Portugal

Training Programs

Dates	2001
Title of qualification awarded	**Epidemiological Surveillance Technician**
Name and type of organisation providing education	Institute of Preventive Medicine, School of Medicine, University of Lisbon
Dates	1990
Title of qualification awarded	**C-130 Hercules: Specialization in Engine, Propellers and Auxiliary Power Unit.**
Name and type of organisation providing education	Lockheed Martin Official Training Center OGMA-Indústria Aeronautica de Portugal Parque Aeronautico de Alverca, 2615-173 Alverca, Portugal
Dates	1987
Title of qualification awarded	**Computer programmer** (DOS, DBase III, Cobol, WordStar)
Name and type of organisation providing education	Instituto de Tecnologia Avançada para a Educação Porto, Portugal

Languages

Native speaker: **English/Portuguese**

Other languages: **French, Spanish**

Relevant publications

Alves-Pereira M, Bakker HHC (2017) *Occupational and residential exposures to infrasound and low frequency noise in aerospace professionals: Flawed assumptions, inappropriate quantification of acoustic environments, and the inability to determine dose-response.* Scientific Journal on Aerospace Engineering and Mechanics, 1(2):83-98.

Alves-Pereira M, Castelo Branco NAA (2007) *Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling.* Progress Biophysics & Molecular Biology, 93: 256-279.

Alves-Pereira M, Joanaz de Melo J, Castelo Branco, NAA (2005) *Pericardial biomechanical adaptation to low frequency noise stress.* In: A. Méndez-Vilas (ed.) Recent Advances in Multidisciplinary Applied Physics. Elsevier: London, 2005: 363-7. (ISBN: 978-0-08-044648-6)

Alves-Pereira M (1999) *Noise-induced extra aural pathology. A review and commentary.* Aviation, Space and Environmental Medicine, 70 (3, Suppl.): A7-A21.

(Annex I: Complete List of Publications)

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Mariana Alves-Pereira, Ph.D.

Curriculum Vitae

ANNEX I

List of Publications

January 2018

EXHIBIT
Int. I-3(b)

Scientific Publications

Mariana Alves-Pereira, Ph.D.

2018

Alves-Pereira M, Bakker H, Rapley B, Summers R. **Residential environments rich in infrasound and low frequency components: Why measuring inside the home is important.** (In preparation for submission)

Alves-Pereira M, Bakker H, Rapley B, Summers R. **Infrasound and low frequency noise: shall we measure it properly?** Engineers Ireland e-journal, 23 January, 2018. http://www.engineersjournal.ie/2018/01/23/ilfn-infrasound-low-frequency-noise-turbine-health/

2017

Alves-Pereira M, Bakker HHC. **Occupational and residential exposures to infrasound and low frequency noise in aerospace professionals: Flawed assumptions, inappropriate quantification of acoustic environments, and the inability to determine dose-response.** Scientific J Aerosp Eng Mech 2017, 1(2):83-98.

Bakker HHC, Alves-Pereira M, Summers SR. **Citizen Science Initiative: Acoustical Characterisation of Human Environments.** Proceedings International Conference Biological Effects of Noise (ICBEN 2017), Zurich, Switzerland, No. 3653, 12 pages.

Bakker HHC, Rapley BI, Summers SR, Alves-Pereira M, Dickinson PJ. **An Affordable Recording Instrument for the Acoustical Characterisation of Human Environments**. Proceedings International Conference Biological Effects of Noise (ICBEN 2017), Zurich, Switzerland (Paper No. 3654).

Rapley BI, Alves-Pereira M, Bakker H. **The inadequacy of the A-frequency weighting for the assessment of adverse effects on human populations**. Proceedings International Conference Biological Effects of Noise (ICBEN 2017), Zurich, Switzerland (Paper No. 3873).

Rapley BI, Bakker HHC, Alves-Pereira M, Summers SR. **Case Report: Cross-sensitisation to infrasound and low frequency noise.** Proceedings International Conference Biological Effects of Noise (ICBEN 2017), Zurich, Switzerland (Paper No. 3872).

Alves-Pereira M, HHC Bakker. **New methodologies for acoustical assessment: infrasound and low frequency noise.** In: Corticeiro Neves M, Leal A, Camarada M, Silva M, Onofre C, *et al* (Eds), Vertentes e Desafios da Segurança 2017, Associação Vertentes e Desafios da Segurança, Lisboa, 2017, pp. 235-44. (ISBN: 978-989-20-7943-1) (Portuguese/English)

2015

Castelo Branco NAA, Alves-Pereira M, Martinho Pimenta A, Reis Ferreira J. **Clinical protocol for evaluating pathology induced by low frequency noise exposure**. EuroNoise 2015, Maastricht, The Netherlands, 31 May-3 Jun, 6 pages.

009416

Castelo Branco NAA, Alves-Pereira M, Martinho Pimenta A, Reis Ferreira J. **Low frequency noise-induced pathology: contributions provided by the Portuguese wind turbine case.** EuroNoise 2015, Maastricht, The Netherlands, 31 May-3 Jun, 5 pages.

2014

Alves-Pereira M, Castelo Branco NAA. **Letter to the Editor re: "How the factoid of wind turbines causing vibroacoustic disease came to be 'irrefutably' demonstrated".** Australian & New Zealand Journal Public Health 2014; 38(2): 191-192.

Mendes AP, Bonança I, Jorge A, Alves-Pereira M, Castelo Branco NAA, Caetano M, Oliveira N, Graça A, Santos C, Ferraria R. **Voice acoustic profile of males exposed to occupational infrasound and low-frequency noise.** Laryngology & Voice 2014; 4(1): 12-20.

2012

Mendes AP, Graça A, Jorge A, Alves-Pereira M, Castelo Branco NAA, Freitas A, Laranjeira M, Bonança I. **The effects of ILFN-exposure on voice acoustic parameters of commercial cabin crewmembers.** Laryngology & Voice 2012; 2(2): 70-80.

Alves-Pereira M. **Review of the wind turbine health study: Report of independent expert panel**, commissioned by the Massachusetts Department of Environmental Protection & Massachusetts Department of Public Health. March 2012.
http://docs.wind-watch.org/MassDEP-wind-health-2-Alves_Pereira.pdf

2010

Alves-Pereira M. **Infrasound and low frequency noise: Quantification in several rural and urban environments.** Revista Lusófona de Ciências e Tecnologias da Saúde 2010 7(1): 91-108. (English/Portuguese)
http://revistas.ulusofona.pt/index.php/revistasaude/article/view/1237/1003

Castelo Branco NAA, Costa e Curto T, Mendes Jorge L, Cavaco Faísca J, Amaral Dias L, Oliveira P, Martins dos Santos J, Alves-Pereira M. **Family with wind turbines in close proximity to home: follow-up of the case presented in 2007.** Proceedings of the 14th International Meeting on Low Frequency Noise, Vibration and Its Control. Aalborg, Denmark, 9-11 June, 2010, 31-40.

2009

Alves-Pereira M, Castelo Branco NAA. **Infrasound and low frequency noise dose responses: contributions**. Revista Lusófona de Ciências e Tecnologias da Saúde 2009 6(1): 31-44. (English/Portuguese)
http://revistas.ulusofona.pt/index.php/revistasaude/article/view/725/605

Alves-Pereira M., Castelo Branco NAA. **Understanding the Biological Responses Elicited by Low Frequency Noise Exposure: Contributions to Vibroacoustic Disease Research** (No.

009417

079R-Oral presentation). Second International Fascia Research Congress, Amsterdam, The Netherlands, 27-30 October, 2009.

Castelo Brnaco NAA, Alves-Pereira M. **Production of Functional Collagen Units in the Absence of Inflammatory Processes as a Response to Low Frequency Noise Exposure** (No. J_075-Oral presentation). Second International Fascia Research Congress, Amsterdam, The Netherlands, 27-30 October, 2009.

Alves-Pereira M, Castelo Branco NAA. **Low Frequency Noise Exposure Destroys Tubulin- and Actin-based Structures** (No. 077-Poster presentation). Second International Fascia Research Congress, Amsterdam, The Netherlands, 27-30 October, 2009.

Oliveira P, Martins dos Santos J, Mendes JJ, Alves-Pereira M, Castelo Branco NAA. **Perivasculo-Ductal Connective Tissue in the Parotid Gland of Wistar Rats Exposed to Low Frequency Noise** (J_092-Poster presentation). Second International Fascia Research Congress, Amsterdam, The Netherlands, 27-30 October, 2009.

2008

Mendes AP, Graça A, Santos CP, Galvão A, Carvalho RO, Sousa MJ, Alves-Pereira M, Castelo Branco NAA. **Voice acoustic analyses in airline cabin crewmembers.** Proceedings Internoise2008. Shangai, China, 2008; No. IN-08-595, 13 pages.

Mendes AP, Graça A, Santos CP, Galvão A, Carvalho RO, Sousa MJ, Alves-Pereira M, Castelo Branco NAA. **Voice acoustic analyses in commercial airline pilots.** Proceedings Internoise2008. Shangai, China, 2008; No. IN-08-591 , 11 pages.

2007

Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling.** Progress Biophysics & Molecular Biology 2007 93: 256-279.

Alves-Pereira M, Castelo Branco NAA. **On the impact of infrasound and low frequency noise on public health: 2 cases of residential exposure.** Revista Lusófona de Ciências e Tecnologias da Saúde 2007; 4(2): 186-200. (Bilingual)
http://revistas.ulusofona.pt/index.php/revistasaude/article/view/670/564

Castelo Branco NAA, Reis Ferreira J, Alves-Pereira M. (2007). **Respiratory pathology in vibroacoustic disease: 25 years of research.** Revista Portuguesa Pneumologia 2007; XIII (1): 129-135.

Alves-Pereira M, Castelo Branco, NAA. **The scientific arguments against vibroacoustic disease.** Proceedings Internoise2007. Istanbul, Turkey, 2007; No. IN-07-505, 7 pages. (ISBN: 80-01-03055-5)

Alves-Pereira M, Castelo Branco, NAA. **Public health and noise exposure: the importance of low frequency noise.** Proceedings Internoise2007. Istanbul, Turkey, 2007; No. IN-07-137, 10 pages. (ISBN: 80-01-03055-5)

009418

Alves-Pereira M, Castelo Branco, NAA. **Infrasound and low frequency noise dose responses: contributions.** Proceedings Internoise2007. Istanbul, Turkey, 2007; No. IN-07-443, 10 pages. (ISBN: 80-01-03055-5)

Alves-Pereira M, Castelo Branco, N.A.A. **In-home wind turbine noise is conducive to vibroacoustic disease.** Proceedings of the Second International Meeting on Wind Turbine Noise, Lyon, France, Sep 20-21, Paper No. 3, 11 pages.

Castelo Branco, NAA, Monteiro M, Reis Ferreira R, Mendes CP, Alves-Pereira M. **Diagnosing vibroacoustic disease for legal purposes.** Proceedings Internoise2007. Istanbul, Turkey, 2007; No. IN-07-506, 8 pages. (ISBN: 80-01-03055-5)

Castelo Branco, NAA, Monteiro M, Reis Ferreira R, Monteiro E, Alves-Pereira M. **Bronchoscopy in vibroacoustic disease I – Pink Lesions.** Proceedings Internoise2007. Istanbul, Turkey, 2007; No. IN-07-507, 7 pages. (ISBN: 80-01-03055-5)

2006

Castelo Branco NAA, Alves-Pereira M. **A doença vibroacústica.** Revista Segurança 2006; XLI (171-Supl Março/Abril):1-20.

Mendes A, Alves-Pereira M, Castelo Branco NAA. **Voice acoustic patterns of patients diagnosed with vibroacoustic disease.** Revista Portuguesa Pneumologia 2006; XII (4): 375-382.

Reis Ferreira J, Mendes CP, Alves-Pereira M, Castelo Branco NAA. **Respiratory squamous cell carcinomas in vibroacoustic disease.** Revista Portuguesa Pneumologia 2006; XII (5): 539-544.

Reis Ferreira J, Albuquerque e Sousa J, Foreid P, Antunes M, Cardoso S, Alves-Pereira M, Castelo Branco NAA. **Abnormal respiratory drive in vibroacoustic disease.** Revista Portuguesa Pneumologia 2006; XII (4): 369-374.

Reis Ferreira J, Monteiro MB, Tavares F, Serrano I, Monteiro E, Mendes CP, Alves-Pereira M, Castelo Branco NAA. **Involvement of central airways in vibroacoustic disease – Thomé Villar/Boehringer Ingelheim Award 2004.** Revista Portuguesa Pneumologia 2006; XII (2): 93-105.

Da Fonseca J, Martins dos Santos J, Castelo Branco NAA, Alves-Pereira M, Grande N, Oliveira P, Martins AP. **Noise-induced gastric lesions: A light and scanning electron microscopy study of the alterations of the rat gastric mucosa induced by low frequency noise.** Central European Journal of Public Health 2006; 14 (1): 35-38.

Alves-Pereira M, Castelo Branco NAA. **Biotensigridade – O novo modelo da célula e a doença vibroacústica.** [**Biotensegrity – the new cell model and vibroacoustic disease**]. XXII Congresso de Pneumologia/IV Congresso Luso-Brasileiro de Pneumologia, Centro de Congressos do Estoril, 8-10 Dezembro 2006. Revista Portuguesa de Pneumologia; XII (6-Suplemento): S110. (Abstract) (Portuguese)

009419

Monteiro E, Alves-Pereira M, Castelo Branco NAA. **As células em escova e a doença vibroacústica.** [**Brush cells and vibroacoustic disease**]. XXII Congresso de Pneumologia/IV Congresso Luso-Brasileiro de Pneumologia, Centro de Congressos do Estoril, 8-10 Dezembro 2006. Revista Portuguesa de Pneumologia; XII (6-Suplemento): S111. (Abstract) (Portuguese)

Castelo Branco NAA, Monteiro M, Reis Ferreira J, Alves-Pereira M. **O aparelho respiratório na doença vibroacústica.** [**The respiratory tract in vibroacoustic disase**]. XXII Congresso de Pneumologia/IV Congresso Luso-Brasileiro de Pneumologia, Centro de Congressos do Estoril, 8-10 Dezembro 2006. Revista Portuguesa de Pneumologia; XII (6-Suplemento): S109. (Abstract) (Portuguese)

Castelo Branco NAA, Monteiro M, Tavares F, Monteiro E, Reis Ferreira J, Alves-Pereira M. **Aspectos morfológicos da basal sub-epitelial respiratória em doentes com a doença vibroacústica.** [**Morphological aspects of the basal subepithelium in vibroacoustic disease patients**]. XXII Congresso de Pneumologia/IV Congresso Luso-Brasileiro de Pneumologia, Centro de Congressos do Estoril, 8-10 Dezembro 2006. Revista Portuguesa de Pneumologia; XII (6-Suplemento): S112. (Abstract) (Portuguese)

2005

Alves-Pereira M, Joanaz de Melo J, Castelo Branco, NAA. **Pericardial biomechanical adaptation to low frequency noise stress.** IN: A. Méndez-Vilas (ed.) Recent Advances in Multidisciplicnary Applied Physics. Elsevier: London, 2005: 363-7. (ISBN: 978-0-08-044648-6)

Alves-Pereira M, Joanaz de Melo J, Castelo Branco, NAA. **Actin- and tubulin-based structures under low frequency noise stress.** IN: A. Méndez-Vilas (ed.) Recent Advances in Multidisciplinary Applied Physics. Elsevier: London, 2005: 955-79 (ISBN: 978-0-08-044648-6)

Alves-Pereira M, Joanaz de Melo J, Castelo Branco, NAA. **Low frequency noise exposure and biological tissue: reinforcement of structural integrity?** IN: A. Méndez-Vilas (ed.) Recent Advances in Multidisciplinary Applied Physics. Elsevier: London, 2005: 961-6 (ISBN: 978-0-08-044648-6)

Da Fonseca J, Martins dos Santos J, Castelo Branco NAA, Alves-Pereira M, Grande NR, Oliveira P. **Noise-induced duodenal lesions.** European Journal of Anatomy 2005; 9(1): 29-33.

Alves-Pereira M, Marques MC, Castelo Branco NAA. **Biological mechanisms and targets of low frequency noise exposure.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 526 (9 pages).

Alves-Pereira M, Fragata JI, Monteiro E, Sousa Silva D, Castelo Branco NAA. **The pericardium in vibroacoustic disease III – A new structure.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 569 (8 pages).

Alves-Pereira M, Motylewski J, Kotlicka E, Castelo Branco NAA. **Low frequency noise legislation.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 582 (8 pages).

Alves-Pereira M. **Low frequency noise exposure as a confounding factor in biomedical science.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 583 (8 pages).

009420

Alves-Pereira M. **...And again low frequency noise – A possible solution.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 584 (8 pages).

Castelo Branco NAA, Marques MC, Alves-Pereira M. **Physiopathology of vibroacoustic disease**. Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 563 (6 pages).

Cunha Ribeiro L, Crespo FF, Freire I, Afonso H, Castelo Branco MSN, Marques MC, Alves-Pereira M, Castelo Branco NAA. **Hemostasis and coagulation changes in vibroacoustic disease.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 564 (8 pages).

Castelo Branco NAA, Reis Ferreira J, Marques MC, Alves-Pereira M. **Auto-immune disorders in vibroacoustic disease**. Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 565 (6 pages).

Araujo A, Carranca J, Alves-Pereira M, Castelo Branco NAA. **Echocardiography in vibroacoustic disease**. Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 567 (9 pages).

Castelo Branco NAA, Fragata JI, Martins AP, Monteiro E, Alves-Pereira M. **The pericardium in vibroacoustic disease I – morphological features.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 568 (9 pages).

Castelo Branco NAA, Fragata JI, Marques MC, Monteiro E, Alves-Pereira M. **The pericardium in vibroacoustic disease II – cellular death pathways.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 570 (8 pages).

Reis Ferreira J, Mendes CP, Alves-Pereira M, Castelo Branco NAA. **Respiratory pathology in vibroacoustic disease - Current findings.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 571 (7 pages).

Monteiro MB, Reis Ferreira J, Mendes CP, Serrano I, Tavares F, Alves-Pereira M, Castelo Branco NAA. **Respiratory pathology in vibroacoustic disease – Specific morphological changes.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 572 (9 pages).

Foreid P, Martinho Pimenta AJF, Alves-Pereira M, Castelo Branco NAA. **Neuropsychological issues in vibroacoustic disease - Current findings.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 573 (10 pages).

Foreid P, Martinho Pimenta AJF, Alves-Pereira M, Castelo Branco NAA. **Neurological issues in vibroacoustic disease – Epilepsy.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 574 (9 pages).

Reis Ferreira J, Albuquerque e Sousa J, Foreid P, Antunes M, Alves-Pereira M, Castelo Branco NAA. **Neurological issues in vibroacoustic disease - Respiratory control.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 575 (6 pages).

009421

Foreid P, Martinho Pimenta AJF, Alves-Pereira M, Castelo Branco NAA. **Behavioural and psychological changes in vibroacoustic disease.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 576 (9 pages).

Da Fonseca J, Mirones J, Martins dos Santos J, Oliveira O, Alves-Pereira M, Castelo Branco NAA. **Gastrointestinal problems in vibroacoustic disease.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 577 (8 pages).

Martins dos Santos J, Albuquerque e Sousa J, Marques MC, Monteiro E, Alves-Pereira M, Castelo Branco NAA. **Urinary system – Current findings and ongoing studies.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 578 (8 pages).

Casetlo Branco NAA, Alves-Pereira M. **Personal experiences of vibroacoustic disease patients with occupational or environmental exposures.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 579 (7 pages).

Castelo Branco NAA, Alves-Pereira M, Araújo A, Reis Ferreira J, Joanaz de Melo. **Environmental vibroacoustic disease – An example of environmental low frequency noise exposure.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 580 (8 pages).

Castelo Branco NAA, Rodriguez E, Alves-Pereira M. **Forensic features in vibroacoustic disease studies.** Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 581 (9 pages).

2004

Castelo Branco NAA, Alves-Pereira M. **Vibroacoustic disease.** Noise & Health 2004; 6(23): 3-20.

Castelo Branco NAA, Monteiro E, Costa e Silva A, Martins dos Santos J, Reis Ferreira J, Alves-Pereira M. **The lung parenchyma in low frequency noise exposed Wistar rats.** Revista Portuguesa Pneumologia 2004; X (1): 77-85.

Martins dos Santos J, Grande NR, Castelo Branco NAA, Zagalo C, Oliveira P, Alves-Pereira M. **Lymphatic lesions in vibroacoustic disease.** European Journal Lymphology 2004; 12(40): 17-20.

Alves-Pereira M, Joanaz de Melo J, Marques MC, Castelo Branco NAA. **Vibroacoustic disease – the response of biological tissue to low frequency noise.** Proceedings 11th International Meeting on Low Frequency Noise and Vibration and its Control. Maastricht, Holland, 2004; 295-308.

Alves-Pereira M, Joanaz de Melo J, Motylewski J, Kotlicka E, Castelo Branco NAA. **Biomedical research, legislation and the low frequency noise contaminant.** Proceedings 11th International Congress on Sound & Vibration. St. Petersburg, Russia, 2004; 1729-1736.

009422

Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Public transportation and low frequency noise: a health hazard?** Proceedings 11th International Congress on Sound & Vibration. St. Petersburg, Russia, 2004; 1761-1766.

Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Low frequency noise in subways.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 641, 5 pages. (ISBN: 80-01-03055-5)

Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Low frequency noise in trams.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 642, 5 pages. (ISBN: 80-01-03055-5)

Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Low frequency noise in trains.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 643, 5 pages. (ISBN: 80-01-03055-5)

Alves-Pereira M, Joanaz de Melo J, Motylewski J, Kotlicka E, Castelo Branco NAA. **Biomedical research and the low frequency noise contaminant.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 644, 7 pages. (ISBN: 80-01-03055-5)

Castelo Branco NAA, Alves-Pereira M. **Vibroacoustic disease- what is known to date.** Proceedings 11th International Meeting on Low Frequency Noise and Vibration and its Control. Maastricht, Holland, 2004; 269-284.

Araujo A, Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Environmentally-induced vibroacoustic disease in a suburban family.** Proceedings 11th International Congress on Sound & Vibration. St. Petersburg, Russia, 2004; 1767-1774.

Castelo Branco NAA, Alves-Pereira M. **Vibroacoustic disease- current concepts.** Proceedings 11th International Congress on Sound & Vibration. St. Petersburg, Russia, 2004; 1775-1782.

Castelo Branco NAA, Alves-Pereira M. **Vibroacoustic disease - update of current knowledge.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 613, 8 pages. (ISBN: 80-01-03055-5)

Araujo A, Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Vibroacoustic disease in a ten-year-old male.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 634, 7 pages. (ISBN: 80-01-03055-5)

Foreid P, Martinho Pimenta AJF, Alves-Pereira M, Castelo Branco NAA. **Neurophysiological aspects of vibroacoustic disease.** Proceedings 11th International Congress on Sound & Vibration. St. Petersburg, Russia, 2004; 1737-1744.

Reis Ferreira J, Mendes CP, Antunes M, Alves-Pereira M, Castelo Branco NAA. **Loss of the neurological control of breathing in vibroacoustic disease patients.** Proceedings 11th International Congress on Sound & Vibration. St. Petersburg, Russia, 2004; 1745-1752.

Castelo Branco NAA, Fragata JI, Martins AP, Monteiro E, Alves-Pereira M. **Pericardial cellular death in vibroacoustic disease.** Proceedings 11th International Congress on Sound & Vibration. St. Petersburg, Russia, 2004; 1753-1760.

009423

Mendes CP, Reis Ferreira J, Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic disease and respiratory pathology I - Tumours.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 636, 5 pages. (ISBN: 80-01-03055-5)

Reis Ferreira J, Albuquerque e Sousa J, Mendes CP, Antunes M, Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic disease and respiratory pathology II – PCO_2 response.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 637, 6 pages. (ISBN: 80-01-03055-5)

Monteiro M, Reis Ferreira J, Mendes CP, Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic disease and respiratory pathology III – Tracheal & bronchial lesions.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 638, 5 pages. (ISBN: 80-01-03055-5)

Reis Ferreira J, Albuquerque e Sousa J, Mendes CP, Antunes M, Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic disease and respiratory pathology IV – Lung & pleura in a lung cancer patient.** Proceedings Internoise2004. Prague, Czech Republic, 2004; No. 639, 4 pages. (ISBN: 80-01-03055-5)

Reis Ferreira J, Mendes CP, Monteiro M, Marques MC, Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic disease - associated respiratory pathology.** Proceedings 11th International Meeting on Low Frequency Noise and Vibration and its Control. Maastricht, Holland, 2004; 285-294.

2003

Alves-Pereira M, Reis Ferreira J, Joanaz de Melo J, Motylewski J, Kotlicka E, Castelo Branco NAA. **Noise and the respiratory system.** Revista Portuguesa Pneumologia 2003; IX: 367-79.

Castelo Branco NAA, Monteiro E, Costa e Silva A, Reis Ferreira J, Alves-Pereira M. **Respiratory epithelia in Wistar rats**. Revista Portuguesa Pneumologia 2003; IX: 381-88

Castelo Branco NAA, Monteiro E, Costa e Silva A, Reis Ferreira J, Alves-Pereira M. **Respiratory epithelia in Wistar rats born in low frequency noise plus varying amount of additional exposure.** Revista Portuguesa Pneumologia 2003; IX (6): 481-492.

Castelo Branco NAA, Gomes-Ferreira P, Monteiro E, Costa e Silva A, Reis Ferreira J, Alves-Pereira M. **Respiratory epithelia in Wistar rats after 48 hours of continuous exposure to low frequency noise.** Revista Portuguesa Pneumologia 2003; IX (6): 474-79.

Alves-Pereira M, Joanaz de Melo, J, Motylewski J, Kotlicka E, Castelo Branco NAA. **Vibroacoustic disease II: The biological and acoustical basis of low frequency noise induced pathology**. Institute of Acoustics (U.K.) 2003; 25(Pt 2): 72-78.

Alves-Pereira M, Castelo Branco NAA. **Ciliated cells, cochlear cilia and low frequency noise.** Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 366-367. (ISBN: 90-807990-1-7)

Alves-Pereira M, Joanaz de Melo J, Motylewski J, Kotlicka E, Castelo Branco NAA. **Occupational exposure to low frequency noise**. Proceedings 8th International Conference on

009424

Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 28-29. (ISBN: 90-807990-1-7)

Alves-Pereira M, Joanaz de Melo J, Motylewski J, Kotlicka E. Castelo Branco NAA. **Legislation hinders low frequency noise research**. Proceedings Scuola Superiore. G. Reiss Romoli (SSGRRw), L'Aquila, Italy, 2003; No. 103: 7 pages.

Castelo Branco NAA, Martinho Pimenta AJ, Reis Ferreira J, Alves-Pereira M. **Monitoring vibroacoustic disease.** Proceedings Scuola Superiore. G. Reiss Romoli (SSGRRw), L'Aquila, Italy, 2003; No. 102: 5 pages.

Araujo A, Pais F, Lopo Tuna JMA, Marvão JH, Alves-Pereira M, Castelo Branco NAA. **Audio- and echocardiograms in vibroacoustic disease**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 115-117. (ISBN: 90-807990-1-7)

Castelo Branco NAA, Barreira R, Crespo FF, Freire I, Afonso H, Castelo Branco MSN, Alves-Pereira M. **Hemostasis & Coagulation changes in airline pilots**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 118-199. (ISBN: 90-807990-1-7)

Castelo Branco NAA, Monteiro E, Alves-Pereira M. **The effects of low frequency noise on rat trachea**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 378-379. (ISBN: 90-807990-1-7)

Castelo Branco NAA, Monteiro E, Alves-Pereira M. **In utero low frequency noise exposure in rats – I**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 372-373. (ISBN: 90-807990-1-7)

Castelo Branco NAA, Monteiro E, Alves-Pereira M. **In utero low frequency noise exposure in rats – II**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 374-375. (ISBN: 90-807990-1-7)

Castelo Branco NAA, Fragata JI, Martins AP, Monteiro E, Alves-Pereira M. **Pericardial cellular death in vibroacoustic disease**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 376-377. (ISBN: 90-807990-1-7)

Castelo Branco NAA, Fragata JI, Monteiro E, Alves-Pereira M. **Pericardial features in vibroacoustic disease patients**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 380-381. (ISBN: 90-807990-1-7)

Castelo Branco NAA, Monteiro E, Martins dos Santos J, Alves-Pereira M. **Low frequency noise and intra-cellular edema**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 378-379. (ISBN: 90-807990-1-7)

Lousã N, Monteiro E, Alves-Pereira M, Castelo Branco NAA. **Rat cochlea exposed to low frequency noise**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 43-45. (ISBN: 90-807990-1-7)

009425

Reis Ferreira J, Mendes CP, Castelo Branco NAA, Monteiro E, Alves-Pereira M. **The human lung and pleura in vibroacoustic disease**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 386-387. (ISBN: 90-807990-1-7)

Reis Ferreira J, Mendes CP, Castelo Branco NAA, Monteiro E, Alves-Pereira M. **The human trachea in vibroacoustic disease**. Proceedings 8th International Conf. Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 388-389. (ISBN: 90-807990-1-7)

Reis Ferreira J, Mendes CP, Antunes M, Martinho Pimenta J, Monteiro E, Alves-Pereira M, Castelo Branco NAA. **Diagnosis of vibroacoustic disease – preliminary report**. Proceedings 8th International Conference on Noise as Public Health Problem (ICBEN). Rotterdam, Holland, 29 June-3 July, 2003: 112-114. (ISBN: 90-807990-1-7)

Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Pericardial biomechanical adaptation to low frequency noise stress**. Proceedings First International Meeting on Applied Physics, Badajoz, Spain, 2003; No. 354. (Abstract)

Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Actin and tubluin-based structures under low frequency noise stress**. Proceedings First International Meeting on Applied Physics, Badajoz, Spain, 2003; No. 355. (Abstract)

Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. **Low frequency noise exposure and biological tissue: reinforcement of structural integrity?** Proceedings First International Meeting on Applied Physics, Badajoz, Spain, 2003; No. 356 (Abstract)

Castelo Branco NAA, Fragata JI, Martins AP, Monteiro E, Alves-Pereira M. **Pericardial morphology in vibroacoustic disease.** Chest 2003; 124 (4): 213-4S. (Abstract)

Mendes CP, Reis Ferreira J, Martins AP, Monteiro E, Alves-Pereira M. **Morphological changes in the respiratory system of vibroacoustic disease patients.** Chest 2003; 124 (4): 213. (Abstract)

Reis Ferreira, J, Mendes CP, Antunes M, Alves-Pereira M, Castelo Branco NAA. **Respiratory drive in vibroacoustic disease.** Chest 2003; 124 (4): 214S. (Abstract)

Castelo Branco NAA, Reis Ferreira J, Monteiro E, Alves-Pereira M. **A traqueia humana na doença vibroacústica. [Human trachea in vibroacoustic disease]**. XL Reunião da Sociedade Anatómica Portuguesa. Escola de Medicina da Universidade Nova de Lisboa, 23 de Maio, 2003: No. 42 (Livro de Resumos). (Portuguese)

Castelo Branco NAA, Monteiro E, Martins dos Santos J, Alves-Pereira M. **Aspectos do glomerulo renal em ratos expostos a ruído de baixa frequência. [Aspects of kidney glomeruli in rats exposed to low frequency noise]**. XL Reunião da Sociedade Anatómica Portuguesa. Escola de Medicina da Universidade Nova de Lisboa, 23 de Maio, 2003: No. 43 (Livro de Resumos). (Portuguese)

Castelo Branco NAA, Reis Ferreira J, Monteiro E, Alves-Pereira M. **O pulmão e a pleura na doença vibroacústica. [The lung and pleura in vibroacoustic disease]**. XL Reunião da Sociedade Anatómica Portuguesa. Escola de Medicina da Universidade Nova de Lisboa, 23 de Maio, 2003: No. 44 (Livro de Resumos). (Portuguese)

009426

Castelo Branco NAA, Fragata J, Martins AP, Monteiro E, Alves-Pereira M. **A morfologia do pericárdio na doença vibroacústica. [Pericardial morphology in vibroacoustic disease]**. XL Reunião da Sociedade Anatómica Portuguesa. Escola de Medicina da Universidade Nova de Lisboa, 23 de Maio, 2003: No. 45 (Livro de Resumos). (In Portuguese)

Castelo Branco NAA, Fragata J, Martins AP, Monteiro E, Alves-Pereira M. **A morte celular no pericárdio de doentes com a doença vibroacústica. [Pericardial cellular death in vibroacoustic disease patients]**. XL Reunião da Sociedade Anatómica Portuguesa. Escola de Medicina da Universidade Nova de Lisboa, 23 de Maio, 2003: No. 46. (Livro de Resumos) (Portuguese)

Castelo Branco NAA, Monteiro E, Martins dos Santos J, Alves-Pereira M. **Aspectos do parênquima pulmonar em ratos expostos a ruído de baixa frequência. [Aspects of lung parenchyma in rats exposed to low frequency noise]**. XL Reunião da Sociedade Anatómica Portuguesa. Escola de Medicina da Universidade Nova de Lisboa, 23 de Maio, 2003: No. 48 (Livro de Resumos). (Portuguese)

2002

Castelo Branco NAA, Alves-Pereira M, Martins dos Santos J, Monteiro E. **SEM and TEM study of rat respiratory epithelia exposed to low frequency noise.** In: Science and Technology Education in Microscopy: An Overview, A. Mendez-Vilas (Ed.), Formatex: Badajoz, Spain, 2003; Vol. II: 505-33. (ISBN: 84-607-6699-3)

Castelo Branco NAA & Colleagues. **Response to Letter to the Editor "Vibroacoustic Disease".** Aviations, Space and Environmental Medicine, 2002; 73(8): 829-30.

Alves-Pereira M, Castelo Branco NAA. **Low frequency noise and vibroacoustic disease: ignored in the workplace.** American Industrial Hygiene Conference. June 3-6, 2002, San Diego, California, USA.

2001

Alves-Pereira M, Castelo Branco MSNA, Motylewski J, Pedrosa A, Castelo Branco NAA. **Airflow-induced infrasound in commercial aircraft.** Proc Internoise2001, The Hague, Holland, 2001: 1011-14. (ISBN: 9080655422)

Alves-Pereira M, Motylewski J, Castelo Branco NAA. **Low frequency noise onboard commercial aircraft**. Proceedings 8th International Congress Sound & Vibration, July 2001, Hong Kong, P.R. China: 525-32.

Araujo A, Pais F, Lopo Tuna JMC, Alves-Pereira M, Castelo Branco NAA. **Echocardiography in noise-exposed flight crew**. Proc Internoise2001, The Hague, Holland, 2001: 1007-10. (ISBN: 9080655422)

Torres R, Tirado G, Roman A, Ramirez R, Colon H, Araujo A, Pais F, Lopo Tuna JMC, Castelo Branco MSNAA, Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic disease induced by long-term exposure to sonic booms**. Proc Internoise2001, The Hague, Holland, 2001: 1095-98. (ISBN: 9080655422)

009427

Pedrosa A, Alves-Pereira M, Castelo Branco MSNAA, Motylewski J, Castelo Branco NAA. **Infrasound in the cockpits and cabins of commercial aircraft**. Aviation, Space and Environmental Medicine 2001; 72(3): 253. (Abstract)

Alves-Pereira M, Castelo Branco NAA. **...And again low frequency noise**. Aviation, Space and Environmental Medicine 2001; 72(3): 254 (Abstract).

2000

Barreira R, Crespo FF, Freire I, Afonso H, Castelo Branco MSA, Alves-Pereira M, Castelo Branco NAA. **Hemostasis and coagulation changes in commercial airline pilots.** Aviation, Space and Environmental Medicine 2000; 71(3): 319. (Abstract)

Motylewski J, Alves-Pereira M, Castelo Branco NAA. **Infrasound analysis onboard commercial airline cockpits**. Aviation, Space and Environmental Medicine 2000; 71(3): 343. (Abstract)

Lousã N, Sousa Pereira A, Alves-Pereira M, Monteiro E, Grande NR, Castelo Branco NAA. **Morphological changes in the cochlear cilia of Wistar rats exposed to low frequency noise.** Aviation, Space and Environmental Medicine 2000; 71(3): 301. (Abstract)

1999

Alves-Pereira M. **Noise-induced extra aural pathology. A review and commentary.** Aviation, Space and Environmental Medicine 1999; 70 (3, Suppl.): A7-A21.

Castelo Branco NAA, Rodriguez E, Alves-Pereira M, Jones DR. **Forensic aspects of vibroacoustic disease**. Aviation, Space and Environmental Medicine 1999; 70(3, Suppl.): A145-51.

Alves-Pereira M, Castelo Branco NAA. **Vibroacoustic Disease: The need for a new attitude towards noise**. Proceedings International Conference on Public Participation & Information Technologies, 1999; October 20-22, Lisbon: 340-7.
http://www.citidep.pt/papers/articles/alvesper.htm

Alves-Pereira M, Motylewski J, Araújo A, Pais F, Lopo Tuna JMC, Delgado J, Lewin PA, Nowicki A, Castelo Branco NAA. **Pericardial thickening in commercial airline flight crew**. Aviation, Space and Environmental Medicine 1999; 70(4): 412. (Abstract)

Águas AP, Alves-Pereira M, Monteiro E, Sousa Pereira A, Grande NR, Castelo Branco NAA. **Features of cellular death in pericardia of vibroacoustic disease patients**. Aviation, Space and Environmental Medicine 1999; 70(4): 413. (Abstract)

Delgado J, Motylewski J, Alves-Pereira M, Nowicki A, Lewin PA, Castelo Branco NAA. **The pilots' dilemma: cosmic radiation above 30 000 ft and infrasound below 30 000 ft.** Aviation, Space and Environmental Medicine 1999; 70(4): 413. (Abstract)

009428

Motylewski J, Alves-Pereira M, Delgado J, Nowicki A, Lewin PA, Castelo Branco NAA. **Noise assessment in commercial airliners**. Aviation, Space and Environmental Medicine 1999; 70(4): 413. (Abstract)

1998

Alves-Pereira M. **Noise assessment and noise-induced extra-aural pathology.** Proc IberoAmer Inter Meet Acoustics 1998; Lisbon, Portugal, In: Bento Coelho JL, Fradique J eds., Acústica 98. Soc. Port. Acústica: Lisbon, 1998: 323-26.

Alves-Pereira M, Castelo Branco N, Águas AP, Sousa Pereira A, Monteiro E, Grande NR. **Adaptação da estrutura do pericárdio à exposição ocupacional a ruído inteso de baixa frequência.** [**Pericardial adaptation to occupational low frequency noise exposure**]. XXXIII Reunião Anual da Sociedade Portuguesa de Microscopia Electrónica e Biologia Celular. Porto, 9-11 de Dezembro, 1998. (Portuguese)

Águas AP, Alves-Pereira M, Monteiro E, Sousa Pereira A, Grande NR, Castelo Branco NAA. Aspectos da morte celular no pericárdio de doentes com a doença vibroacústica. [**Aspects of cellular death in the pericardium of vibroacoustic disease patients**]. XXXIII Reunião Anual da Sociedade Portuguesa de Microscopia Electrónica e Biologia Celular. Porto, 9-11 de Dezembro, 1998.

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Pictorial explanation for **why the dBA metric is not useful** for the quantification of infrasound and low frequency noise.

(by Mariana Alves-Pereira, 2018)

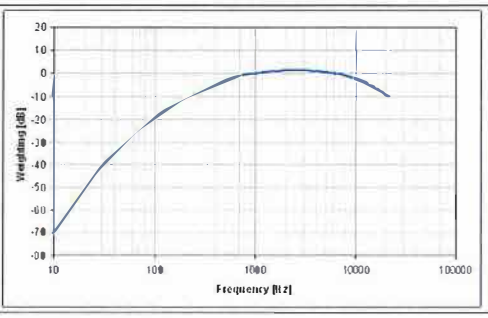


Fig. 1
Frequency response curve used by the dBA metric.

The dBA began its development in the 1920's, to improve telephone acoustics.

With decreasing frequency, there is an increasing difference between what the dBA measures and what is actually present in the environment.

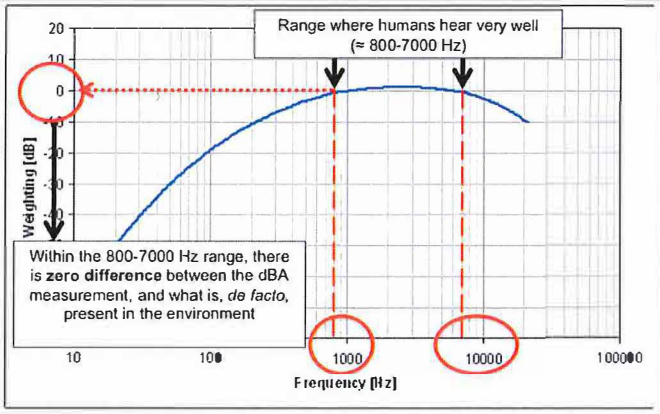


Fig. 2
The dBA metric is a good methodology to protect the human hearing function.

For forensic purposes, occupational deafness is checked at 4000 Hz.

The dBA assumes: "what you can't hear won't hurt you."

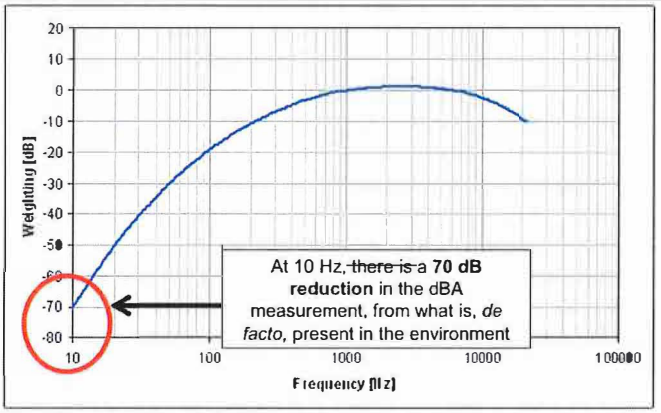


Fig. 3
At 10 Hz, the dBA metric will reduce the numerical value of its measurement by 70 dB.

At 10 Hz, the dBA metric *does not quantify* the acoustical energy that is present in the environment.

Image source: Dirac Dirac Delta Science & Engineering Encyclopedia (2018) A-Weighting. http://www.diracdelta.co.uk/science/source/a/w/aweighting/source.html#.W45nwy2ZORs

EXHIBIT Int. I-3(c)

JAMA | Preliminary Communication

Neurological Manifestations Among US Government Personnel Reporting Directional Audible and Sensory Phenomena in Havana, Cuba

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Author Audio Interview
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Supplemental content

IMPORTANCE From late 2016 through August 2017, US government personnel serving on diplomatic assignment in Havana, Cuba, reported neurological symptoms associated with exposure to auditory and sensory phenomena.

OBJECTIVE To describe the neurological manifestations that followed exposure to an unknown energy source associated with auditory and sensory phenomena.

DESIGN, SETTING, AND PARTICIPANTS Preliminary results from a retrospective case series of US government personnel in Havana, Cuba. Following reported exposure to auditory and sensory phenomena in their homes or hotel rooms, the individuals reported a similar constellation of neurological symptoms resembling brain injury. These individuals were referred to an academic brain injury center for multidisciplinary evaluation and treatment.

EXPOSURES Report of experiencing audible and sensory phenomena emanating from a distinct direction (directional phenomena) associated with an undetermined source, while serving on US government assignments in Havana, Cuba, since 2016.

MAIN OUTCOMES AND MEASURES Descriptions of the exposures and symptoms were obtained from medical record review of multidisciplinary clinical interviews and examinations. Additional objective assessments included clinical tests of vestibular (dynamic and static balance, vestibulo-ocular reflex testing, caloric testing), oculomotor (measurement of convergence, saccadic, and smooth pursuit eye movements), cognitive (comprehensive neuropsychological battery), and audiometric (pure tone and speech audiometry) functioning. Neuroimaging was also obtained.

RESULTS Of 24 individuals with suspected exposure identified by the US Department of State, 21 completed multidisciplinary evaluation an average of 203 days after exposure. Persistent symptoms (>3 months after exposure) were reported by these individuals including cognitive (n = 17, 81%), balance (n = 15, 71%), visual (n = 18, 86%), and auditory (n = 15, 68%) dysfunction, sleep impairment (n = 18, 86%), and headaches (n = 16, 76%). Objective findings included cognitive (n = 16, 76%), vestibular (n = 17, 81%), and oculomotor (n = 15, 71%) abnormalities. Moderate to severe sensorineural hearing loss was identified in 3 individuals. Pharmacologic intervention was required for persistent sleep dysfunction (n = 15, 71%) and headache (n = 12, 57%). Fourteen individuals (67%) were held from work at the time of multidisciplinary evaluation. Of those, 7 began graduated return to work with restrictions in place, home exercise programs, and higher-level work-focused cognitive rehabilitation.

CONCLUSIONS AND RELEVANCE In this preliminary report of a retrospective case series, persistent cognitive, vestibular, and oculomotor dysfunction, as well as sleep impairment and headaches, were observed among US government personnel in Havana, Cuba, associated with reports of directional audible and/or sensory phenomena of unclear origin. These individuals appeared to have sustained injury to widespread brain networks without an associated history of head trauma.

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EXHIBIT
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In late 2016, US government personnel serving in Havana, Cuba, began presenting to their embassy medical unit after experiencing unusual auditory and/or sensory stimuli of variable intensity and character, with associated onset of varied neurological manifestations. Initial signs and symptoms pointed toward injury of the auditory system, leading to the establishment of a triage program at the University of Miami centered around otolaryngology evaluation. Eighty embassy community members underwent initial evaluation between February and April 2017, and 16 individuals were identified with similar exposure history and a constellation of neurological signs and symptoms commonly seen following mild traumatic brain injury, also referred to as concussion.[1] Exposures continued with time and 8 additional individuals were identified who had similar findings. The US Department of State, Bureau of Medical Services, subsequently convened an expert panel in July 2017, which came to consensus that the triage findings were most likely related to neurotrauma from a nonnatural source and recommended that further investigation into this novel cluster of findings was necessary.

The University of Pennsylvania's Center for Brain Injury and Repair was subsequently selected to coordinate multidisciplinary clinical evaluation, treatment, and rehabilitation of individuals identified during initial triage and additional patients with exposure. The purpose of this preliminary communication is to describe preliminary findings from 21 patients who were exposed to the same nonnatural source.[1]

Key Points

Question Are there neurological manifestations associated with reports of audible and sensory phenomena among US government personnel in Havana, Cuba?

Findings In this case series of 21 individuals exposed to directional audible and sensory phenomena, a constellation of acute and persistent signs and symptoms were identified, in the absence of an associated history of blunt head trauma. Following exposure, patients experienced cognitive, vestibular, and oculomotor dysfunction, along with auditory symptoms, sleep abnormalities, and headache.

Meaning The unique circumstances of these patients and the consistency of the clinical manifestations raised concern for a novel mechanism of a possible acquired brain injury from a directional exposure of undetermined etiology.

Methods

Design

This retrospective study was approved by the institutional review board of the University of Pennsylvania's Perelman School of Medicine, which waived the need for informed consent. The participants signed general consent forms for treatment permitting use of their data in research. Because of security and confidentiality considerations, individual-level demographic data cannot be reported.

Clinical Approach

The US Department of State directly referred individuals with suspected exposure to the University of Pennsylvania for comprehensive evaluation and treatment. A multidisciplinary team was convened consisting of physical medicine and rehabilitation, occupational medicine, neurology, neuroradiology, and neurosurgery. Each specialist independently obtained clinical histories and conducted comprehensive assessments. Reported signs and symptoms were extracted from these interviews.

Based on individual clinical indication, additional referrals were made to vestibular physical therapy, neuro-optometry, neuropsychology, occupational therapy, speech therapy, audiology, otorhinolaryngology, and sleep medicine for focused evaluation and treatment. Patients were referred to the University of Pennsylvania for clinical care, as opposed to enrollment in a structured research study. The **Box** shows an abbreviated list of objective measures used during clinical assessments and supplements for additional information.[2-29]

Cognitive, Neurobehavioral, and Mood Evaluations

When clinically indicated, comprehensive neuropsychological assessments were conducted by experienced neuropsychologists, who were not blinded to patient status. Neuropsychological test batteries included assessment of the following domains: (1) auditory attention, (2) auditory and visual working memory, (3) auditory and visual memory, (4) visual-spatial perception, (5) visual-motor construction, (6) motor function, (7) language function, (8) executive function, (9) processing speed, (10) academic achievement, (11) reasoning, (12) mood functioning, and (13) effort (Box and eAppendix in the Supplement). Following neuropsychological testing, individuals with cognitive deficits were referred for cognitive rehabilitation with occupational therapy, speech therapy, or both, depending on the individual clinical indication. Cognitive rehabilitation was intentionally not started prior to completion of neuropsychological testing to avoid affecting results.

Balance and Vestibular Evaluations

Clinical evaluations identifying balance abnormalities prompted referral to vestibular physical therapy. Focused vestibular evaluation included expert clinical assessment and the use of validated measures of static and dynamic balance (Box).[15-21] Also per clinical indications, patients were referred to audiology for comprehensive evaluation of the peripheral vestibular system, including caloric reflex testing.[23,24] Individuals confirmed to have a unilateral peripheral vestibulopathy (ie, relative vestibular reduction of ≥25% on caloric reflex testing) underwent magnetic resonance imaging (MRI) of the head with and without gadolinium contrast, with focus on the internal auditory canals in addition to the MRI sequences detailed.

Oculomotor Evaluations

Individuals found on clinical evaluation to have abnormalities of oculomotor function were referred to neuro-optometry for further evaluation and treatment. Oculomotor function was

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quantified using the following standard optometric clinical measures (Box).[25,26] Vergence testing included step vergence with prism bar, vergence facility with prisms, and near point of convergence. Accommodative testing in nonpresbyopic patients included amplitude of accommodation, accommodative facility with plus and minus lenses, and accommodative lag. Pursuit and saccadic testing was done qualitatively to assess accuracy of tracking eye movements and whether symptoms were provoked as with Vestibular/Ocular Motor Screening.[29] Saccadic speed and accuracy were quantified using the Developmental Eye Movement test,[26,28] a timed visual-verbal test. Diagnoses of accommodative, vergence, and/or saccadic/pursuit dysfunction were made using standardized criteria, in conjunction with symptomatic reporting,[26] which were quantified using the Convergence Insufficiency Symptoms Survey.[27]

Auditory Evaluations

Audiometry evaluations were performed prior to referral for care at the University of Pennsylvania. However, when patients had balance function testing as described here, comprehensive audiology evaluation included both pure tone and speech audiometry.

Imaging Evaluations

Initial conventional MRI sequences were acquired at 3T on a Siemens Magnetom Prisma^{fit} scanner, and included high-resolution sagittal 3-dimensional MP-RAGE, T2 SPACE and FLAIR SPACE, coronal 2-dimensional T2-weighted imaging, axial 2-dimensional diffusion-weighted imaging, and axial T2* gradient echo. Resulting images were clinically interpreted by neuroradiology clinicians.

Box. Examples of Standardized Measures Used in Clinical Assessments[a]

Cognitive
- Boston Diagnostic Aphasia Examination[2]
- California Verbal Learning Test–2nd Edition[3]
- Grooved Pegboard[4]
- Test of Memory Malingering[5]
- Trail Making Test, Parts A and B[6]
- Wechsler Adult Intelligence Scale–IV[7]
- Wechsler Memory Scale–IV[8]

Mood
- Beck Depression Inventory (2nd edition)[9]
- Beck Anxiety Inventory[10,11]
- Frontal Systems Behavior Scale[12]
- Post-Traumatic Stress Disorder Checklist[13,14]

Balance and vestibular
- Functional Gait Assessment[15]
- Activities-Specific Balance Confidence[16]
- Balance Error Scoring System[17]
- Clinical Test of Sensory Organization and Balance[18,19]
- Dizziness Handicap Index[20]
- Computerized Dynamic Posturography[21,22]
- Caloric reflex test[23,24]

Vision and oculomotor
- Formal Evaluation of Vergence and Accommodation[25,26]
- Convergence Insufficiency Symptoms Survey[27]
- Developmental Eye Movement Test[26,28]
- Vestibular/Ocular Motor Screening[29]

[a] Measures were used based on clinical indications; therefore, every patient did not complete all measures in this abbreviated list.

Results

There were 21 individuals evaluated (11 women and 10 men, with a mean age of 43 years). Multidisciplinary evaluations began an average of 203 days (range, 3-331 days; median, 189 days; interquartile range, 125 days) following exposure (**Table 1**).

Exposure

For 18 of the 21 individuals (86%), there were reports of hearing a novel, localized sound at the onset of symptoms in their homes and hotel rooms (**Table 2**). Affected individuals described the sounds as directional, intensely loud, and with pure and sustained tonality. Of the patients, high-pitched sound was reported by 16 (76%), although 2 (10%) noted a low-pitched sound. Words used to describe the sound include "buzzing," "grinding metal," "piercing squeals," and "humming."

The sounds were often associated with pressurelike (n = 9, 43%) or vibratory (n = 3, 14%) sensory stimuli, which were also experienced by 2 of the 3 patients who did not hear a sound. The sensory stimuli were likened to air "baffling" inside a moving car with the windows partially rolled down.

Both the sound and sensory stimuli were often described as directional in that the individuals perceived a distinct direction from which the sensation emanated (hereafter referred to as *directional phenomena*). Further, the directional phenomena appeared to be localized to a precise area, as individuals (n = 12, 57%) noted that after changing location, the sensation disappeared and the associated symptoms reduced. Five individuals (24%) reported covering their head and/or ears, although doing so did not result in attenuation of the directional phenomena.

Accurately determining the dose and duration of exposure has been difficult because of the limitations of patient recall. Some patients were awakened by sounds and were unsure of the start of the event. The shortest reported event involved two 10-second pulses reported as a single exposure episode, whereas other patients reported that they perceived sound continuously for longer than 30 minutes. Owing to security concerns, further details of potential dosage cannot be provided.

Of the affected individuals, 20 (95%) reported immediate onset of neurological symptoms associated with directional phenomena (eTable 1 in the Supplement). One individual awoke from sleep with acute symptoms (including headache, unilateral ear pain, and hearing changes) but did not perceive directional phenomena. From days to weeks after exposure, individuals reported that they experienced the onset

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Table 1. Demographics of Patients Evaluated at the University of Pennsylvania[a]

	Men (n = 10)	Women (n = 11)	Total (N = 21)
Age, mean (SD), y	39 (7)	47 (8)	43 (8)
Time from exposure to evaluation, mean (SD), d	229 (98)	180 (85)	203 (93)

[a] Potentially identifying information intentionally omitted for security and privacy concerns.

Table 2. Exposure Descriptions of the Directional Phenomena

	Associated Sound			Associated Sensory Stimuli				Duration >3 mo		
Patient No.	Reported	High Pitch	Low Pitch	Reported	Pressure	Vibration	Movement Attenuation[a]	Persistent Symptoms	Objective Findings	Required Treatment
1	X	X					X	X	X	X
2	X	X					X	X	X	X
3	X	X					X			
4	X		X	X		X		X	X	X
5	X	X		X	X			X	X	X
6	X		X	X	X		X	X	X	X
7				X	X		X	X		
8	X	X					X	X	X	X
9	X	X		X		X		X	X	X
10	X	X		X		X		X	X	X
11	X	X		X	X			X	X	X
12	X	X		X	X			X	X	X
13	X	X					X	X	X	X
14	X	X					X	X	X	X
15	X	X		X	X		X	X	X	X
16	X	X					X	X	X	X
17	X	X		X	X		X	X	X	X
18								X	X	X
19	X	X		X	X			X		
20				X	X		X	X	X	X
21	X	X						X	X	X
No. (%)	18 (86)	16 (76)	2 (10)	12 (57)	9 (43)	3 (14)	12 (57)	20 (95)	18 (86)	18 (86)

[a] Patients reported attenuation of sound, pressure, or vibration when moving to a different location.

of additional cognitive, neurobehavioral/mood, and physical symptoms. Twenty individuals (95%) reported that they experienced persistent (>3 months) symptoms, and 18 individuals (86%) exhibited objective clinical manifestations in 6 predominant domains (**Table 3**).

Cognitive, Neurobehavioral, and Mood Findings

Persistent cognitive manifestations were reported by 17 individuals (81%). Subjective symptoms included memory problems (n = 16, 76%), feeling mentally foggy (n = 16, 76%), impaired concentration (n = 15, 71%), and feeling cognitively slowed (n = 14, 67%) (Table 3). In addition, they reported neurobehavioral difficulties including irritability (n = 14, 67%), nervousness (n = 12, 57%), feeling more emotional (n = 11, 52%), and sadness (n = 5, 24%). For at least 6 individuals (29%), a clear change in work performance was noted by supervisors and colleagues (eTable 1 in the Supplement). Individuals also reported a "good day-bad day" pattern where significant cognitive or physical exertion would be followed by exacerbation of their symptoms for several days. Cognitive symptoms, as well as disequilibrium and headache, reportedly were also frequently exacerbated by cardiovascular exercise.

Multidisciplinary evaluations raised concern for cognitive impairment in 16 individuals (76%). Prior to referral, 4 of these individuals underwent neuropsychological evaluation (data not shown as generated outside of the University of Pennsylvania). Repetition of comprehensive neuropsychological testing is precluded within 1 year due of practice effects when material is presented within this timeframe. With previous exposure to material, the individual may score higher on a repeated neuropsychological evaluation within 1 year. Neuropsychological assessments were performed on 10 individuals after referral. Of those, interpretation was ongoing in 4 at the time of this publication. Per their preference, 2 individuals did not complete neuropsychological testing.

For the 6 individuals with complete neuropsychological testing data and analysis at the University of Pennsylvania, all had significant areas of cognitive weakness and/or impairment (eTables 2, 3, and 4 in the Supplement). Impairments were found in executive function (n = 6), motor function (n = 5), auditory and visual memory (n = 4), visual-spatial perception and visual-motor construction (n = 4), auditory attention and working memory (n = 3), language (n = 3), processing speed (n = 4), and reasoning (n = 1). All individuals

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Table 3. Prevalence of Persistent Symptoms and Objective Findings[a]

	Subjective		Objective	
Domain	Symptom	No. (%)	Finding	No. (%)
Cognitive and behavioral	Combined	17 (81)	Neuropsychological testing indicated	16 (76)[b]
	Difficulty remembering	16 (76)	Neuropsychological testing performed at Penn	10 (48)
	Mental fog	16 (76)	Neuropsychological testing outside Penn	4 (19)
	Difficulty concentrating	15 (71)	Neuropsychological testing not yet performed	2 (10)
	Feeling slowed	14 (67)	Cognitive rehabilitation	13 (62)[b]
	Irritability	14 (67)		
	Feeling more emotional	11 (52)		
Balance and vestibular	Combined	15 (71)	Vestibular physical therapy referral	17 (81)
	Balance problems	14 (67)	Static postural stability	16 (76)
	Dizziness	13 (62)	Dynamic balance	16 (76)
	Nausea	7 (33)	VOR dysfunction	15 (71)
			Unilateral caloric impairment	4 (31)[c]
			Vestibular rehabilitation	17 (81)
Vision and oculomotor	Combined	18 (86)	Neuro-optometry referral	15 (71)
	Visual problems	16 (76)	Convergence insufficiency	11 (52)
	Light sensitivity	13 (62)	Smooth pursuit dysfunction	11 (52)
	Difficulty reading	12 (57)	Saccadic dysfunction	10 (47)
	Eye strain	11 (52)	Neuro-optometric rehabilitation	14 (67)
Auditory	Combined	15 (68)	Audiology referral	13 (62)
	Sound sensitivity	14 (67)	Moderate to severe SNHL	3 (23)[c]
	Tinnitus	12 (57)	Hearing aid provided	3 (14)
	Hearing reduction	9 (43)		
	Ear pressure	8 (38)		
Sleep	Combined	18 (86)	Pharmacological intervention	15 (71)
	Drowsiness or fatigue	16 (76)		
	Decreased sleep duration	15 (71)		
	Trouble falling asleep	14 (67)		
Headache	Combined	16 (76)	Pharmacological intervention	12 (57)
	With cognitive tasks	13 (62)		
	With therapy	11 (52)		
	Due to photophobia	9 (43)		
	Due to phonophobia	6 (29)		
Overall	Combined subjective	20 (95)	Combined objective	18 (86)

Abbreviations: Penn, University of Pennsylvania; SNHL, sensorineural hearing loss; VOR, vestibulo-ocular reflex.

[a] Persistent defined as presence more than 3 months after exposure.

[b] Neuropsychological characterization ongoing. Start of cognitive rehabilitation held until neuropsychological testing performed.

[c] Of 13 patients tested thus far during persistent symptom evaluation.

demonstrated a high level of effort during testing and had intact cognitive domains including visual working memory and academic achievement.

Neurobehavioral function was evaluated using the Frontal System Behavior Scale, a self-report measure of frontal lobe dysfunction. Specifically, comparing before and after exposure retrospectively via patient recall and self-report, individuals noted apathy (n = 5), executive dysfunction (n = 4), and disinhibition (n = 2). Two individuals met criteria for posttraumatic stress disorder and endorsed severe levels of anger on the Brief Mood Survey, 1 of whom also endorsed moderate to severe levels of depression and anxiety.

Balance and Vestibular Findings

Individuals described acute nausea (n = 7, 33%) and dizziness (n = 5, 24%) during exposure, which continued to progress in the subacute and persistent stages (acute stage = during or hours following exposure; subacute stage = days to weeks after exposure patient recall]; and persistent stage = more than 3 months after exposure). Specifically, more than 3 months after exposure, individuals reported a higher prevalence of dizziness (n = 13, 62%) and nausea (n = 7, 33%), in addition to general balance problems (n = 14, 67%) (Table 3). These symptoms were exacerbated by walking quickly, tasks involving head movements, complex visual environments, or in some cases while simply standing still. Balance symptoms were also worsened with eyes closed or in low light conditions.

Clinical examinations raised concern for balance impairment in 17 patients (81%), prompting referral to vestibular physical therapy. Focused vestibular evaluations demonstrated impairments in static postural stability (n = 16, 76%), dynamic

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balance (n = 16, 76%), and the vestibulo-ocular reflex (n = 15, 71%) (eTables 5, 6, and 7 in the Supplement). Patients with the most severe balance impairments on clinical evaluation underwent caloric reflex testing, which demonstrated peripheral vestibular dysfunction in 4 of 13 patients evaluated. MRI findings focusing on the internal auditory canals on these 4 patients were normal. Taken together, these balance symptoms and evaluation findings are consistent with central and, in some cases, peripheral vestibular abnormalities.

Oculomotor Findings

Of the individuals with persistent symptoms, 16 (76%) reported visual problems (Table 3). Light sensitivity (n = 13, 62%) and difficulty reading (n = 12, 57%) were also frequently reported. Eye strain (n = 11, 52%) was experienced particularly with reading and was associated with headaches, disequilibrium, and nausea.

Clinical examinations raised concern for oculomotor dysfunction in 15 individuals (71%), prompting referral to neuro-optometry. The most common findings confirmed on focused oculomotor evaluation were convergence insufficiency (n = 11, 52%), abnormal smooth pursuits (n = 11, 52%), and saccadic dysfunction (n = 10, 47%) (eTables 8 and 9 in the Supplement). Similar to vestibular testing that provoked symptoms, oculomotor examination elicited headache and disequilibrium.

Auditory Findings

At the onset of the directional phenomena, affected individuals reported hearing a loud sound (n = 18, 86%), associated with ear pain (n = 7,33%) and tinnitus (n = 6, 29%). Within days to weeks following exposure, individuals continued to report tinnitus (n = 12, 57%) and ear pain (n = 5, 24%), with the addition of a change in hearing (n = 7, 33%) and sensitivity to noise (n = 5, 24%). More than 3 months after exposure, sound sensitivity was the most common auditory concern (n = 14, 67%), followed by tinnitus (n = 12, 57%) and ear pressure (n = 8, 38%).

While 9 individuals (43%) reported persistent hearing reduction, pure tone audiometry, including pure tone average and word identification, revealed moderate to severe sensorineural hearing loss in 3 individuals (23%) (eTable 10 in the Supplement), who were fitted with hearing aids. For 2 individuals, the moderate to severe sensorineural hearing loss was unilateral and corresponded with the side of peripheral vestibular dysfunction on caloric testing. Otoscopy and tympanometry findings were unremarkable.

Sleep

Individuals commonly reported issues with sleep (n = 18, 86%), including reduced sleep duration (n = 15, 71%) and difficulty falling asleep (n = 14, 67%). In addition, individuals experienced significant daytime fatigue (n = 16, 76%). Most individuals required pharmacological intervention to improve subjective report of sleep architecture (n = 15, 71%) (eTable 11 in the Supplement).

Headaches

At the initiation of directional phenomena exposure, 8 individuals (38%) reported immediate onset of headache, while 5 (24%) reported intense head pressure. In the days to weeks following exposure, 17 individuals (81%) developed headaches, with 16 (76%) experiencing persistent headaches longer than 3 months after exposure (Table 3).

In the persistent stage, headaches were reported to be exacerbated or associated with cognitive tasks (n = 13, 62%), rehabilitative therapies (n = 11, 52%), photophobia (n = 9, 43%), and phonophobia (n = 6, 29%). Patients with antecedent headaches were able to differentiate the character of these headaches from that of their standard headaches. Headaches were generally reported to improve with medications (n = 12, 57%) and appropriate therapies for oculomotor and vestibular impairments (eTable 11 in the Supplement).

Imaging

MRI neuroimaging was obtained in all 21 patients. Most patients had conventional imaging findings, which were within normal limits, at most showing a few small nonspecific T2-bright foci in the white matter (n =9, 43%). There were 3 patients with multiple T2-bright white matter foci, which were more than expected for age, 2 mild in degree, and 1 with moderate changes. The pattern of conventional imaging findings in these cases was nonspecific with regard to the exposure/insult experienced, and the findings could perhaps be attributed to other preexisting disease processes or risk factors. Advanced structural and functional neuroimaging studies are ongoing.

Rehabilitation and Return to Work

Individualized rehabilitation programs were developed, which included combinations of neuro-optometric rehabilitation (n = 14, 67%), vestibular physical therapy (n = 17, 81%), and cognitive rehabilitation with speech pathology and/or occupational therapy (n = 13, 62%). The most symptomatic patients (n = 14, 67%) requiring multiple therapies did not return to work.

Vestibular physical therapy sessions focused on balance retraining, static and dynamic posture control with substitution via visual and somatosensory systems, gaze stabilization exercises, habituation, smooth pursuits, and saccadic eye movement exercises. Patients treated with vestibular rehabilitation have demonstrated a positive response with improved balance and reduction of disequilibrium.

Formal neuro-optometric rehabilitation, including manipulation of disparity vergence and accommodative amplitude and latency, has been used to treat ocular motor deficits. Rehabilitation for abnormal smooth pursuit and saccadic dysfunction was coordinated between neuro-optometric rehabilitation, vestibular physical therapy, and occupational therapy. Vestibular physical therapy focused on oculomotor function with the body in motion and occupational therapy emphasized functional tasks such as visual scanning in a simulated work environment.

Following comprehensive neuropsychological testing, a formal cognitive rehabilitation program was initiated in the form of occupational therapy and/or speech therapy.

Early return to work with intensive cognitive loading led to an exacerbation of neurocognitive, vestibular, and visual

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symptoms in 7 individuals (33%). Individualized return to work plans were designed to reintegrate individuals using a stepwise process and appropriate work modifications.

Discussion

Preliminary findings are described of a case series of individuals stationed in Havana, Cuba, nearly all of whom reported directional audible and/or sensory phenomena that was followed by the development of a consistent cluster of neurological signs and symptoms. The clinical manifestations may represent a novel clinical entity, which appears to have resulted from a widespread brain network dysfunction (ie, cognitive, oculomotor, and central vestibular) as seen in mild traumatic brain injury, or concussion,[30] as well as injury to the peripheral vestibular system in some cases. It is currently unclear if or how the noise is related to the reported symptoms. In particular, sound in the audible range (20 Hz-20 000 Hz) is not known to cause persistent injury to the central nervous system and therefore the described sounds may have been associated with another form of exposure.

Cognitive symptoms, including difficulty remembering (n=16, 76%) and feeling cognitively slowed (n=14, 67%) were the most problematic for individuals in this series more than 3 months after exposure, with neuropsychological testing identifying impairments in at least 1 cognitive domain in all 6 patients who completed neuropsychological evaluation to date (eTables 2, 3, and 4 in the Supplement). Cognitive difficulties interfered with these patients' ability to multitask, process information quickly with accurate recall, solve problems, and perform rapid decision making. Compared with vestibular and oculomotor impairments, cognitive impairments are often the slowest to improve following acquired brain injury, which was observed in this series. Therefore, extended cognitive rehabilitation with emphasis on return to work was used. In addition, it is not uncommon for patients with neurological injury resulting in cognitive impairment to have mood disturbances such as depression, anxiety, and/or posttraumatic stress disorder. Mood dysfunction can directly result from acquired brain injury or develop in response to the precipitating event and novel deficits.[31-33]

The presence of subjective neurological symptoms presenting in a cohesive community has raised concerns for collective delusional disorders, including mass psychogenic illness. However, neurological examination and cognitive screens did not reveal evidence of malingering, and objective testing and behavioral observations during cognitive testing indicated high levels of effort and motivation. Several of the objective manifestations consistently found in this cohort (such as oculomotor and vestibular testing abnormalities) could not have been consciously or unconsciously manipulated. Furthermore, mass psychogenic illness is often associated with transient, benign symptoms with rapid onset and recovery often beginning with older individuals.[34,35] In contrast, the Havana cohort experienced persisting disability of a significant nature and are broadly distributed in age. Rather than seeking time away from the workplace, the patients were largely determined to continue to work or return to full duty, even when encouraged by health care professionals to take sick leave.

While not systematically excluded, viral etiologies, chemical etiologies, or both associated with acute onset of persistent neurological impairment and peripheral vestibulopathy with the directional nature of exposure descriptions are not readily apparent. No other manifestations of viral illness, such as preceding fever, were identified. It is unlikely a chemical agent could produce these neurological manifestations in the absence of other organ involvement, particularly given that some individuals developed symptoms within 24 hours of arriving in Havana.

There are important considerations in this investigation. In particular, the anatomic substrates causing the symptoms have not yet been identified. This may represent a significant challenge because even the designation of "concussion," is not yet a true diagnosis, as no definitions include the underlying cause. Nonetheless, there is an emerging consensus that concussion, or mild traumatic brain injury, is a type of brain network disorder, based on classic symptoms (eg, slowed processing speed and memory dysfunction) as well as changes in the white matter tracts and consecutive connectivity, as detected with advanced neuroimaging studies.[30,36]

Beyond the absence of blunt head trauma, there were additional notable differences between the manifestations observed in the Havana cohort and characteristic acute and persistent symptoms of concussion. For example, individuals experienced unilateral ear pain and tinnitus after exposure, and some were later detected to have a unilateral peripheral vestibulopathy (along with central vestibular dysfunction), a finding uncommon in concussion. Further, studies have reported that while most individuals following concussion have a relatively rapid full recovery, at least 15% are thought to experience characteristic persisting symptoms.[37,38] In contrast to classic concussions, most patients referred following suspected exposure in Havana exhibited significant impairment that persisted for months with no significant improvement in multiple cases until rehabilitation was initiated.

For practicing clinicians, if a patient presents reporting a similar potential exposure and symptoms similar to those observed in mild traumatic brain injury, in addition to a thorough history, objective evaluation should include screening assessments of vestibular, oculomotor, and cognitive functioning. Based on findings of this assessment, appropriate referrals to subspecialists should be considered including neurorehabilitation physiatry, vestibular physical therapy, neuro-optometry, neuropsychology, and audiology.

Limitations

This study has several limitations. First, due to the sensitive nature of this publication, certain details typically reported in a case series of exposure were omitted, including specifics about geography, relationships between individuals, and individual demographics. Second, because these patients' first evaluation was elsewhere, each patient did not undergo each of the tests described. In particular, neuropsychological characterization was incomplete at the time of publication.

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Preliminary results were presented given the importance and strong public interest in this case series. Third, the rehabilitative course of this Havana cohort may not be representative because this represents a referral population. There may be additional individuals exposed while in Havana, Cuba, who have not been identified due to subtler manifestations that either resolved spontaneously or did not prompt presentation for medical treatment. Therefore, the actual number of individuals exposed is unknown, and the relative "dose" of exposure that causes acute and chronic symptoms remains unclear.

Conclusions

In this preliminary report of a retrospective case series, persistent cognitive, vestibular, and oculomotor dysfunction, as well as sleep impairment and headaches, were observed among US government personnel in Havana, Cuba, associated with reports of directional audible and/or sensory phenomena of unclear origin. These individuals appeared to have sustained injury to widespread brain networks without an associated history of head trauma.

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REFERENCES

1. US Senate Committee on Foreign Relations, Subcommittee on Western Hemisphere, Transnational Crime, Civilian Security, Democracy, Human Rights, and Global Women's Issues. Attacks on US diplomats in Cuba. https://www.foreign.senate.gov/hearings/attacks-on-us-diplomats-in-cuba-response-and-oversight-010918. Published January 9, 2018. Accessed February 8, 2018.

2. Goodglass H, Kaplan E, Barresi B. *Boston Diagnostic Aphasia Examination*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001.

3. Delis D, Kramer J, Kaplan E, Ober B. *The California Verbal Learning Test: CVLT-II*. 2nd ed. San Antonio, TX: Psychological Corp; 2000.

4. Brown SG, Roy EA, Rohr LE, Snider BR, Bryden PJ. Preference and performance measures of handedness. *Brain Cogn*. 2004;55(2):283-285.

5. Tombaugh TN. *Test of Memory Malingering*. North Tonawanda, NY: Multi-Health Systems; 1996.

6. Reitan R. The validity of the Trail Making Test as an indicator of organic brain damage. *Percept Mot Skills*. 1958;8:271-276.

7. Wechsler D. *WAIS-IV Technical Manual*. New York, NY: Psychological Corp; 2008.

8. Chlebowski C. *Wechsler Memory Scale All Versions*. New York, NY: Springer; 2011.

9. Beck AT, Steer RA, Brown GK. *Beck Depression Inventory Manual*. 2nd ed. San Antonio, TX: Psychological Corp; 1996.

10. Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: psychometric properties. *J Consult Clin Psychol*. 1988;56(6):893-897.

11. Beck AT, Steer RA. *Beck Anxiety Inventory Manual*. San Antonio, TX: Psychological Corp; 1993.

12. Grace J, Malloy PF. *The Frontal Systems Behavior Scale (FrSBe)*. Odessa, FL: Psychological Assessment Resources; 2002.

13. Weathers FW, Huska JA, Keane TM. *PCL-C for DSM-IV*. Boston, MA: National Center for PTSD–Behavioral Science Division; 1991.

14. McCutchan PK, Freed MC, Low EC, Belsher BE, Engel CC. Rescaling the Post-Traumatic Stress Disorder Checklist for use in primary care. *Mil Med*. 2016;181(9):1002-1006.

15. Wrisley DM, Marchetti GF, Kuharsky DK, Whitney SL. Reliability, internal consistency, and validity of data obtained with the Functional Gait Assessment. *Phys Ther*. 2004;84(10):906-918.

16. Powell LE, Myers AM. The Activities-Specific Balance Confidence (ABC) Scale. *J Gerontol A Biol Sci Med Sci*. 1995;50A(1):M28-M34.

17. Bell DR, Guskiewicz KM, Clark MA, Padua DA. Systematic review of the Balance Error Scoring System. *Sports Health*. 2011;3(3):287-295.

18. Horn LB, Rice T, Stoskus JL, Lambert KH, Dannenbaum E, Scherer MR. Measurement characteristics and clinical utility of the Clinical Test of Sensory Interaction on Balance (CTSIB) and Modified CTSIB in individuals with vestibular dysfunction. *Arch Phys Med Rehabil*. 2015;96(9):1747-1748.

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009439

19. Shumway-Cook A, Horak FB. Assessing the influence of sensory interaction of balance: suggestion from the field. *Phys Ther*. 1986;66(10):1548-1550.

20. Jacobson GP, Newman CW. The development of the Dizziness Handicap Inventory. *Arch Otolaryngol Head Neck Surg*. 1990;116(4):424-427.

21. Alahmari KA, Marchetti GF, Sparto PJ, Furman JM, Whitney SL. Estimating postural control with the balance rehabilitation unit: measurement consistency, accuracy, validity, and comparison with dynamic posturography. *Arch Phys Med Rehabil*. 2014;95(1):65-73.

22. Nashner LM. Computerized dynamic posturography. In: Jacobson GP, Newman CW, Kartush JM, eds. *Handbook of Balance Function Testing*. St Louis, MO: Mosby Yearbook; 1993:280-304.

23. Barin K. Interpretation and usefulness of caloric testing. In: Jacobson GP, Shepard NT, eds. *Balance Function Assessment and Management*. San Diego, CA: Plural Publishing; 2008:229-249.

24. Shepard N, Telian S. *Practical Management of the Balance Disorder Patient*. San Diego, CA: Singular Publishing; 1996.

25. Scheiman M, Wick B. *Clinical Management of Binocular Vision: Heterophoric, Accommodative and Eye Movement Disorders*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014.

26. Gallaway M, Scheiman M, Mitchell GL. Vision therapy for post-concussion vision disorders. *Optom Vis Sci*. 2017;94(1):68-73.

27. Rouse M, Borsting E, Mitchell GL, et al; Convergence Insufficiency Treatment Trial (CITT) Investigator Group. Validity of the Convergence Insufficiency Symptom Survey: a confirmatory study [published correction appears in *Optom Vis Sci*. 2009;86(6):786]. *Optom Vis Sci*. 2009;86(4):357-363.

28. Garzia RP, Richman JE, Nicholson SB, Gaines CS. A new visual-verbal saccade test: the Development Eye Movement test (DEM). *J Am Optom Assoc*. 1990;61(2):124-135.

29. Mucha A, Collins MW, Elbin RJ, et al. A brief Vestibular/Ocular Motor Screening (VOMS) assessment to evaluate concussions: preliminary findings. *Am J Sports Med*. 2014;42(10):2479-2486.

30. Johnson VE, Stewart W, Smith DH. Axonal pathology in traumatic brain injury. *Exp Neurol*. 2013;246:35-43.

31. Alderfer BS, Arciniegas DB, Silver JM. Treatment of depression following traumatic brain injury. *J Head Trauma Rehabil*. 2005;20(6):544-562.

32. Bryant R. Post-traumatic stress disorder vs traumatic brain injury. *Dialogues Clin Neurosci*. 2011;13(3):251-262.

33. Jorge RE, Arciniegas DB. Mood disorders after TBI. *Psychiatr Clin North Am*. 2014;37(1):13-29.

34. Jones TF, Craig AS, Hoy D, et al. Mass psychogenic illness attributed to toxic exposure at a high school. *N Engl J Med*. 2000;342(2):96-100.

35. Weir E. Mass sociogenic illness. *CMAJ*. 2005;172(1):36.

36. Shenton ME, Hamoda HM, Schneiderman JS, et al. A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. *Brain Imaging Behav*. 2012;6(2):137-192.

37. McInnes K, Friesen CL, MacKenzie DE, Westwood DA, Boe SG. Mild traumatic brain injury (mTBI) and chronic cognitive impairment: a scoping review. *PLoS One*. 2017;12(4):e0174847.

38. Bigler ED. Neuropsychology and clinical neuroscience of persistent post-concussive syndrome. *J Int Neuropsychol Soc*. 2008;14(1):1-22.

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Occupational and Residential Exposures to Infrasound and Low Frequency Noise in Aerospace Professionals: Flawed Assumptions, Inappropriate Quantification of Acoustic Environments, and the Inability to Determine Dose-Response Values

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Abstract

Professionals within the aerospace industry are often required to remain within acoustic environments characterized by a predominance of low frequency and infrasound components. Safety-and-health-in-the-workplace officials are mindful of the threat these extreme environments can pose to the hearing function. Noise-exposed professionals are, therefore, frequently provided with a plethora of ear protection devices to shield this vital human sense. The vast majority of noise-protection guidelines and regulations, however, are inappropriate to protect aerospace professionals against acoustic environments rich in Infrasound and Low Frequency Noise (ILFN) because they are based on the flawed premises that ILFN only affects humans through the aural pathway, and that dynamics are unimportant. Consequently, the numerical values needed to estimate potential harm to people (dose-response levels) are not routinely obtained or assessed. The goals of this paper are to inform a) Aerospace professionals who are consistently exposed to acoustic environments rich in ILFN, about how this agent of disease is being incorrectly evaluated leading to improper worker protection; and b) Noise control and health professionals who work within the aerospace industry, about new methodologies in acoustical evaluations pertinent to infrasound and low frequency noise dose-response values. New sources of ILFN are increasingly present in the vicinity of residential environments, quite possibly eliminating biological recovery periods for noise-exposed aerospace workers. This paper details the inadequacy of the use of the dBA metric and 1/3-octave-band analysis when protecting workers (and the public) against excessive ILFN exposures. The complexities associated with acoustical evaluation in conjunction with objective and pertinent medical outcomes are discussed, and the need for narrowband analyses in routine evaluation procedures is emphasized.

Keywords

Infrasound, Low frequency noise, dBA, Narrow-band analysis, Occupational exposure, Environmental exposure, Pathology

Introduction

In a widely read paper published by the World Health Organization (WHO), *Burden of disease from environmental noise* [1], the long-term effects of excessive noise exposure were shown to be worrisome, at best. Historically, noise exposures were deemed to be detrimental to the hearing function, i.e., people exposed to loud noise were more likely to become deaf, or hearing impaired. Consequently, only a portion of the acoustical spectrum was focused upon - the one containing the frequencies responsible for hearing loss.

This restricted segment of the acoustical spectrum, called 'the audible portion', ranges from 20 Hz to 20 kHz. Within this wide range of frequencies, though, not all of them are equally responsible for deafness or

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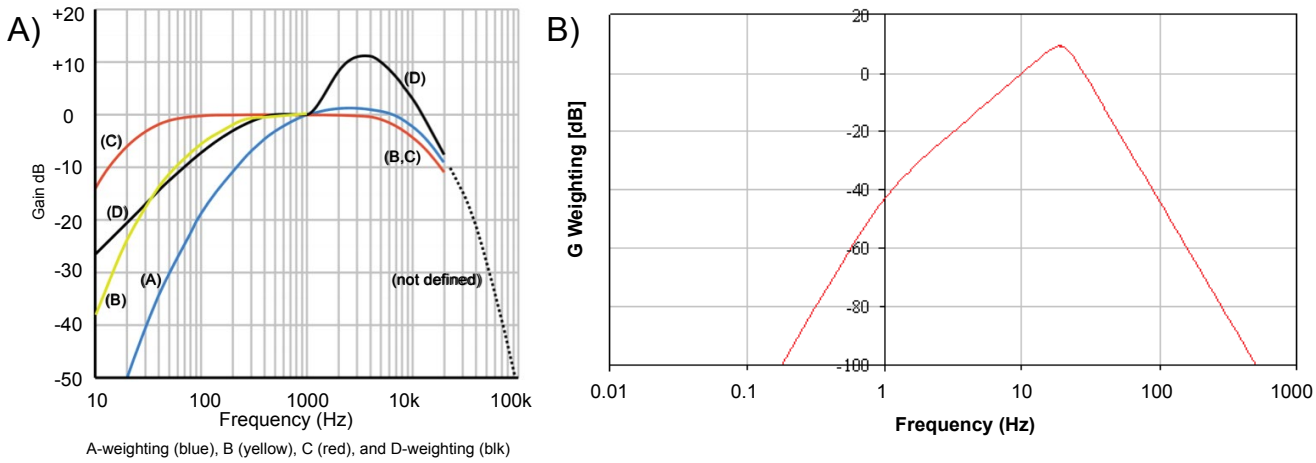


Figure 1: Acoustic Weighting Curves A) Weighting curves for A-weighting (A), B-weighting (B), C-weighting (C) and D-weighting (D) [40]. B) G-weighting Curve [41].

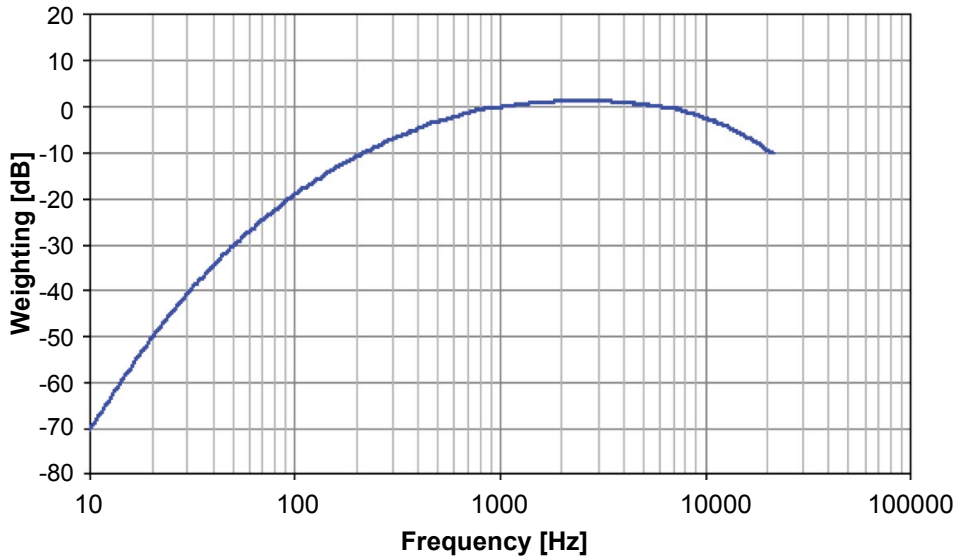


Figure 2: Frequency Response Curve for the A-weighting Network at 10 Hz, well within the ILFN region, the error is approximately a 70 dB reduction, i.e., what is measured is 70 dB less than what is actually present in the environment [42].

hearing loss. Deafness due to excessive noise exposure is predominantly a consequence of loss of hearing function within the 250-8000 Hz range, i.e., deafness at frequencies above 8000 Hz or below 250 Hz is not as relevant for speech intelligibility as are those contained within the 250-8000 Hz range. Health-related acoustical evaluations were thus urged to focus on this particular range of frequencies. This required measuring instrumentation that would eliminate all the frequencies considered irrelevant to human hearing impairment, i.e., all extending beyond the 250-8000 Hz range. Infrasound and Low Frequency Noise (ILFN) (< 200 Hz) was, therefore, deemed irrelevant for the purposes of protecting human health.

The human ear responds to sound non-linearly, both in terms of frequency and sound pressure level. Hence the development of filters (with specific frequency-weighting curves) designed to simulate the non-linear sensitivity of human hearing. Measuring sound levels with one of these filters would then better represent the human perception of the sound. Figure 1 shows examples of A-, B-, C- and D-weighting curves (Figure 1A), and the more recent G-weighting curve, developed for ILFN-rich environments (Figure 1B).

Under the dBA metric, acoustical energy contained in other portions of the spectrum are de-emphasized (< 250 Hz and > 8000 Hz) or deemed irrelevant for evaluation (< 20 Hz, or 'infrasound', and > 20 kHz, or 'ultrasound'). Regulations have been guided by these principles, leading to the ubiquitous capture of information within the restricted segment of the acoustical spectrum (20 Hz - 20

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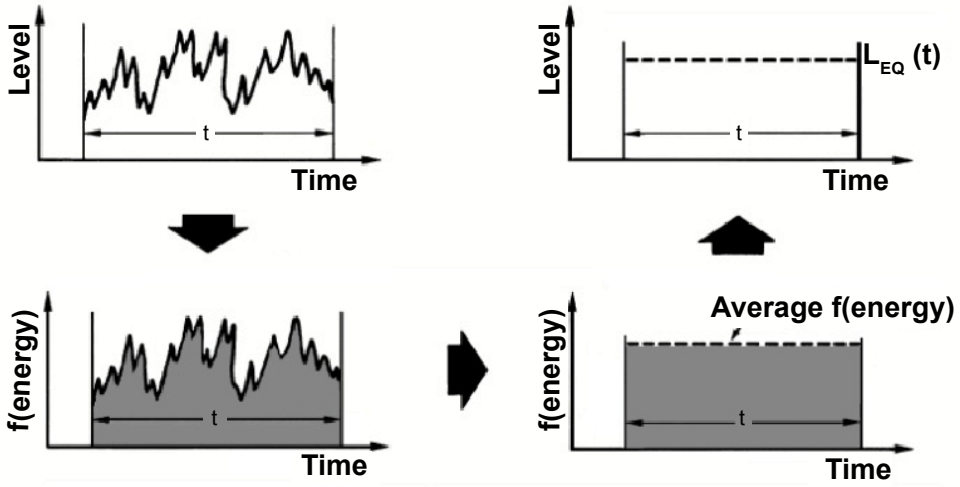


Figure 3: Mathematical treatment applied to a time-varying signal. The resulting, constant Leq value represents the same amount of energy (area in grey under the curve) contained in the original time-varying signal [43].

kHz), and with the A-weighting being applied. Figure 2 shows the frequency response curve for the A-weighting filter, which is relatively flat between 800 - 8000 Hz. When measuring frequencies < 200 Hz with an incorporated A-weighting network, the numerical value obtained for the sound pressure level presents with a 10 to 70 dB error. While the A-weighting system seems to yield good results for hearing protection, it is clearly inadequate for assessing the amount of acoustical energy present in the ILFN ranges, because these lower frequency components are discounted; for example, a reduction of about 70 dB at 10 Hz (Figure 2).

Two additional difficulties exist when evaluating acoustic environments: a) They are constantly varying in time (seconds); and b) They do so continuously across the entire acoustical spectrum. Hence, to enable analyses, both time and frequency parameters need to be segmented. To transform a time-varying signal into some manageable number, the concept of time-averaged sound level was developed (Leq). Figure 3 sketches the mathematical treatment given to a time-varying signal, such as an airborne pressure wave.

Occupational and health issues consider long term exposures in LAeq (A-weighted, time-averaged sound level), and allow short testing samples as representative of an 8-hour day exposure. Many environmental regulations, on the other hand, mandate 10-minute time averages of LAeq, so that values are not skewed by intermittent loud sounds. Either way, information on the dynamic, time-dependent portion of the acoustic environment is diluted when the signal has significant variations in time and is segmented into 10 min or 1 hour windows.

The 'audible' frequency spectrum was segmented into 'octave' and '1/3-octave' bands, facilitating further analyses for noise control evaluations. Although data resolution was improved with the 1/3-octave bands analyses, this methodology still only provides a crude resolution of the acoustic environment. Figure 4A compares octave band and 1/3-octave band measurements. In the pre-digital era, investigations of finer resolution were very involved, complex and beyond the means of general acoustic investigations.

When no weighting network is incorporated into the acoustic signal capture, measurements are un-weighted (unfiltered), and the metric written as dBLin (linear) to distinguish it from other forms of deciBel. Figure 4B shows the difference between the overall dBA and dBLin levels in an environment where ILFN components are predominant. While the overall dBA level reflects what a human would hear, the dBLin level reflects the acoustical energy to which the body is exposed.

There are further limitations related to accuracy when the frequency spectrum is segmented into 1/3-octaves. This method ignores the exact problem that impulsive sounds (perceived as irritating or not) will be invisible in such LAeq measures. For example, a single gunshot would have little, if any, impact upon a 10 minute dBA-level average but would hardly fail to wake a sleeper. In terms of urban population health, the inappropriateness of using the dBA metric to assess ILFN-rich environments was recognized almost two decades ago by the WHO: "When prominent low-frequency components are present, measures based on A-weighting are inappropriate. However, the difference between dBC (or dBLin) and dBA will give crude information about the presence of low-frequency components in noise" [2].

It is not uncommon to address the maximum dBC level or dBLin level in order to 'squeeze out' more acoustical information from measurements obtained with leg-

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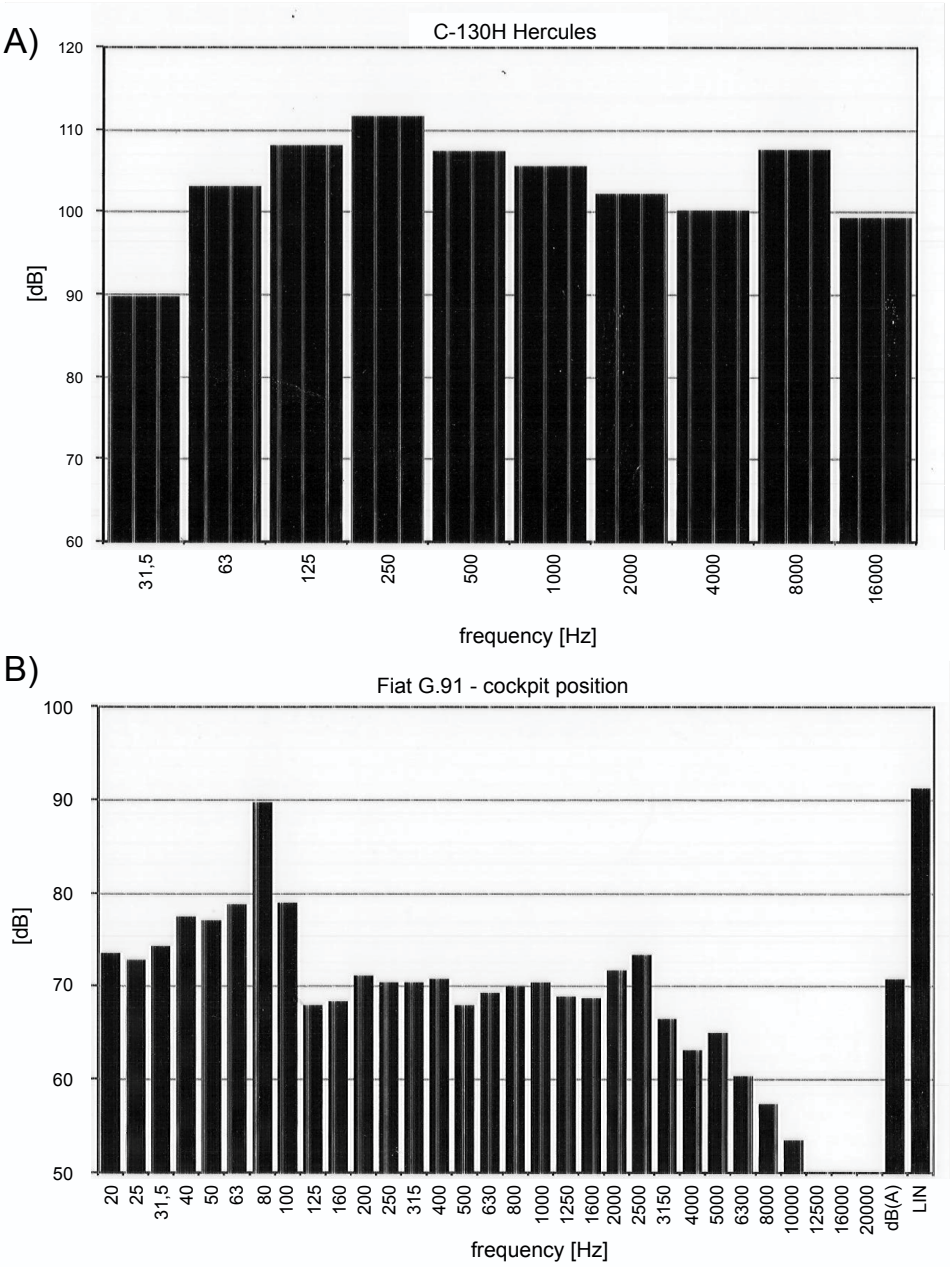


Figure 4: A) Octave band frequency distribution analysis radiated by a C-130 Hercules at normal power setting [44]; B) 1/3-octave band frequency distribution analysis inside the Fiat G-91 cockpit. The two bars on the right indicate the dBA and dBLin levels, respectively. The much lower dBA level when compared to the dBLin level reflects the numerical reduction imposed by the A-weighting Network [45].

islated methodologies. However, a more detailed numerical characterization of acoustic environments within the ILFN range is needed, in order to establish dose-response values for human protection.

Interface between acoustical phenomena and the human body

The WHO publication *International Classification of Diseases* (ICD-10 2016) [3], dedicates Chapter 20 to "External causes of morbidity and mortality", and block W20-W49 refers to "Exposure to inanimate mechanical forces":

W42 - Exposure to noise Incl.: Sound waves, Supersonic waves

W43 - Exposure to vibration Incl.: Infrasound waves [3].

Airborne acoustical phenomena interact with the human body when mechanical coupling occurs between the oncoming mechanical force and a particular tissue or tissue system of the human body. This brings the issue of ILFN health effects into the field of materials engineering. Biological tissues are viscoelastic materials, i.e., they

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possess the properties of creep, relaxation and hysteresis[a]. Moreover, they feature anisotropy, i.e., equal forces applied in different directions yield different results. Biological material, particularly when considering whole-body effects, cannot be modeled as a simple Hookean elastic[a]; and neither can its response when immersed in an ILFN-rich environment.

Prof. Donald Ingber (Wyss Institute, Harvard University), proposed decades ago that animal cells were constructed in accordance with principles of tensegrity [4-6], i.e., architectures consisting of elements providing continuous tension, and elements providing discontinuous compression [7]. By modeling the cell as a tensegrity structure instead of the prior, elastic continuum model, it was possible to begin to understand cellular mechanotransduction, i.e., inter- and intra-cellular communication established via mechanical signals, as opposed to biochemical signals [8,9 for example]. Mechanotransduction and cellular tensegrity architectures are essential to understanding the specific structural changes in ILFN-exposed cells, as seen through light and electron microscopy [10-12]. These mechanically-induced cellular effects are not accounted for under current noise protection legislation, guidelines and procedures, as they do not respond to acoustical energy via the aural-perception pathway.

Mechanical coupling between airborne pressure waves and the human body is known to occur at the ear; the design of which is an engineering marvel. Mechanical coupling between airborne pressure waves and other regions of the body are acknowledged to exist only if the acoustic energy is at sufficient amplitude (i.e., if the event is perceivable by human senses), otherwise, effects are (perhaps erroneously) considered to be irrelevant or non-existent.

The response of biological tissue to ILFN is frequency-dependent. An early example is a study conducted in 1969 (within the scope of the Soviet and US space programs), where dogs were exposed to ILFN-rich environments at sound pressure levels ranging from 105 to 155 dB. This induced multiple hemorrhages in the lung tissue that "never exceeded 3 mm in diameter" [13]. Increasing the dB-level of the environment did not increase the size of the hemorrhagic areas, but rather, their number. In the early 1990's, Professors Nekhoroshev and Glinchikov (St. Petersburg Academy of Sciences, Russia) exposed laboratory animals to infrasound (8-16 Hz) at 120-140 dB, for 3 hr daily, for 1, 5, 10, 15, 25, and 40 days, and found different morphofuntional changes in the cellular structures of myocardium and liver tissues when compared to controls [14,15]. These changes varied with frequency, exposure time and exposure level. For the past three decades, another team led by pathologist Col. Castelo Branco (Portuguese Air Force), has systematically studied both workers and animal models exposed to ILFN. As a result, the clinical evolution of the signs and symptoms consistently observed in aeronautical technicians was established in 1999 [16], supported by numerous collateral studies in human populations and animal models in subsequent years [17-19]. Since then, this ILFN-induced pathology, (termed Vibroacoustic Disease - VAD), has also been identified (as per objective clinical testing) in residential settings [20-22].

Certain, narrow frequency ranges can elicit a specific response from one type of tissue and not another, located adjacently, because each type of material has its own creep, relaxation and hysteresis coefficients[a]. Each tissue type also possesses its own mechanical resonance frequency. Hence, at the electron microscopy level of ILFN-exposed tissue, the re-organization of inter- and intra-cellular architectures seem to reflect a mechanical reinforcement required to maintain structural integrity [23,24].

In Workplace Safety, vibration is considered as that which is transmitted into the human body through solid-to-solid contact, i.e., contact with a chain-saw (hand-arm vibration) or a vibrating platform (whole-body vibration). To measure vibration, accelerometers are used instead of microphones. With ILFN, impacts to the human body occur via a different interface: air-to-solid, or rather, air-to-composite viscoelastic material. Modeling human response to ILFN exposure based on solid-to-solid interfaces has not, therefore, proven very successful.

ILFN dose-response values - the need for narrow-band analyses

Dose-responses for ILFN exposures must be frequency dependent if they are to properly protect hu-

[a]Hookean Elastic vs. Viscoelastic Material. 1) In a Hookean (or purely elastic) material, total deformation depends on total load, and no further deformation occurs even if load is maintained. In viscoelastic materials, however, when stress is applied and maintained, they may continue to further deform, even though stress load remains unaltered. This property is called creep. 2) In a purely elastic material, the strain within the material is constant throughout the application of the load; it does not vary with time, only with the amount of applied stress. In viscoelastic materials, when stress is applied and maintained, strain can decrease with time. This property is called stress relaxation. 3) Consider repetitive or cyclical loads on materials. In purely elastic materials, periodic loads will not alter the stress-strain curve. The pathway taken by the material to deform is exactly the same pathway it takes to return to its original, equilibrium position. In viscoelastic materials, however, the return to equilibrium may be different than the pathway used to get to the point of deformation. (The word pathway is here loosely used, and is meant to encompass all spatial, temporal and energetic components of these types of movements.) This property is called hysteresis.

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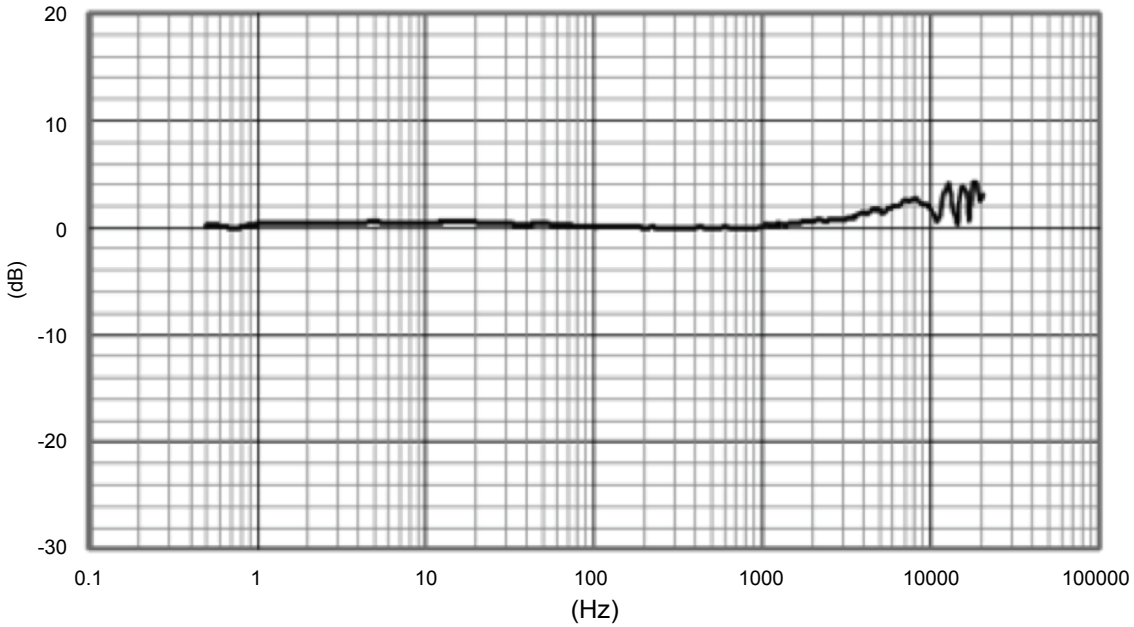


Figure 5: Frequency response curve for the microphones used by the SAM Scribe Mk1 system [29].

mans from excessive exposure to ILFN-rich environments. The determination of dose-responses for ILFN exposure has been scientifically impossible to achieve, however, because the ILFN in itself is not being quantified during routine acoustical evaluations. This is the point where the ILFN-rich occupational and environmental exposures meet. Both require precise ILFN measurements if human populations are to be properly protected from this agent of disease, whether at work or in the home.

For decades, the biomedical world has been in dire need for proper scientific instrumentation to objectively quantify ILFN-rich acoustic environments. Given that current instrumentation discards much of the information that characterizes an acoustic environment (at the behest of regulations) precluding any real scientific analysis, it becomes obvious that current general-sound-level measurement instrumentation is not suited to requirements.

The limitations of dBA methodology and 1/3-octave segmentation with respect to ILFN, can be resolved by the application of analyses using no weighting and by a proper signal capturing of the acoustic environment. This would allow a more precise identification of events that occur within frequency bands that are narrower than the 1/3-octave segmentation, and of periodic signals that occur in the time domain. Narrow band analyses can provide information that would permit the identification of discrete signals (e.g., tones or harmonics) forming acoustic signatures that are not evident with the dBA-1/3-octave methodology [25,26]. This is shown in the next sections.

Material and Methods

Instrumentation

The equipment used for acoustic capture was a SAM Scribe Full Spectrum (FS) system (Model: Mk1, Atkinson & Rapley, Palmerston North, New Zealand) [27,28]. It consists of a two-channel device that can measure at sampling rates up to 44.1 kHz, and that delivers data streams via USB to a Windows notebook computer, storing it as uncompressed wav files to hard disk. GPS information is also stored as metadata in the files, and this includes a digital signature. The system can accurately record from 0.1-1000 Hz, as per the manufacturer frequency response of the two electrets condenser microphones (custom-made Model No.: EM246ASS'Y, Primo Co, Ltd, Tokyo, Japan), Figure 5 [29].

The SAM Scribe FS unit has two switches: one enables a 1 Hz high-pass filter to remove unwanted 'micro-barom' events[b] below 1 Hz, and the other enables a + 20 dB gain boost. All measurements were conducted with the 1 Hz filter enabled and with the 20 dB gain boost disabled. All measurements are reported from 1-800 Hz, and were captured with a sampling rate of 11.025 kHz.

Measurement methodology

[b]A 'microbarom' is a transient change in air pressure caused by events such as shutting a door. This can cause the microphone to saturate, causing clipping, and, in any case, is not generally part of the acoustic environment being studied and should not, therefore be included in the analysis.

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All measurements were recorded to uncompressed wav file including the required reference calibration tone prior to and after measurements. Calibration tones were produced with a Type I calibrator (part of the SAM Scribe system) at 1000 Hz/94 dB. Calibration of the system rests on the manufacturer's frequency-response curve for the Primo microphone capsule (Figure 5) as well as comparison calibrations between 6.3 Hz and 1000 Hz of the full system against a Larsen-Davis 831 sound level meter with a current National Association of Testing Authorities (Australia) calibration certificate. The manufacturer's frequency response curve shows that the microphone capsule is very close to linear over the 1-1000 Hz range used in this study.

Wind-shields were always placed on both microphones during measurements. Microphones were attached to tripods at approximately 1.5 m above the ground. After microphone positioning and initial calibration, at least three 10 min segments of data were captured at each location. Both microphones were placed at the same location, along the same axis, approximately 20 m apart from each other, as limited by cable distance (5 m + 15 m). One microphone (red) was always placed at approximately 22 m from the shed entrance, and the second microphone (blue) was placed at approximately 42 m from the shed entrance (see below).

Measurements were performed on a rotating basis between locations, and on different days - 16, 30, and 31 December, 2016. This acoustical evaluation is part of an international, citizen-based research effort into the health effects caused by excessive exposure to ILFN, and to which the authors contribute [30]. Within this context field-sites, such as this one, become available due to the efforts of citizen scientists.

Selection of locations

Data was gathered at a farm where the residential home is in the vicinity of the animal sheds (Figure 6). This preliminary data was selected for presentation due to: a) Its pedagogical strength for clarifying the difference between the type of data obtained through narrow-band analyses and legislated methodologies; b) The relative location of the home within the ILFN-rich acoustic environment (Figure 6A); and c) The possibility of gathering clues that may contribute to an explanation of different animal behavior in the presence of anthropogenic ILFN, depending on animal-shed location (Figure 6B).

The anthropogenic sources of ILFN in question are four, 3-MW Industrial Wind Turbines (IWT), 150 m in total height (hub height + ½ blade diameter) [31]. Figure 6A shows the relative positions of the home and animal sheds to the IWTs.

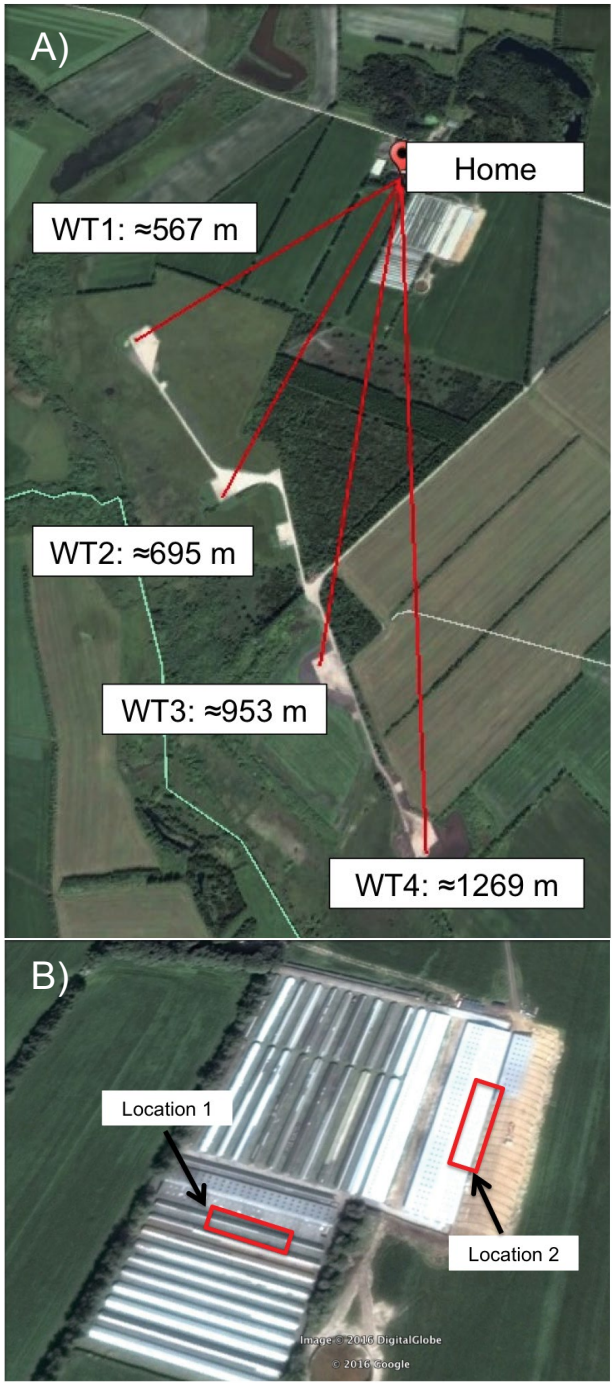


Figure 6: Images reconstructed from Google Earth A) Relative positioning of each of the 4 WT to the residential home and adjacent animal sheds; B) Definition of Location 1 and Location 2.

Locations 1 and 2 were suggested by the property owner as two locations where animal pups responded differently when IWT were rotating. Figure 7 shows the different construction types of the animal sheds in Location 1 and Location 2. Location 1 is a shed of older, wooden construction, with an interior space approximately 2.5 m in height and 75 m in length (Figure 7A). Location 2 is a more modern shed, mostly made of metal,

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Figure 7: Animal shed structures have different constructions styles A) Animal shed of Location 1; B) Animal shed of Location 2.

Table 1: Date and time of acoustical measurements per location.

	Date	Time
Location 1	16 Dec	14:34 - 15:10
	30 Dec	13:25 - 14:02
	30 Dec	17:01 - 18:05
	30 Dec	22:59 - 00-16
	31 Dec	03:28 - 04:37
	31 Dec	08:21 - 09:22
Location 2	16 Dec	15:30 - 16:10
	30 Dec	11:16 - 12:06
	30 Dec	14:11 - 15:28
	30 Dec	20:32 - 21:42
	31 Dec	00:25 - 01:41
	31 Dec	04:40 - 05:58

with an interior space approximately 7.0 m in height, and 110 m in length (Figure 7B). Both contain water-supply systems for the animals that may produce specific and identifiable signatures within the acoustic environment. Since IWT rotation began, the property-owner has opted to maintain his breeding male animals in Location 1 rather than in Location 2. Table 1 tallies the measurements conducted at Location 1 and Location 2 in 2016.

Wind speed

In the case of this anthropogenic ILFN source (IWT), acoustical emissions output depend on the wind speed. (IWT power output graphs concurrent with these measurement sessions are unavailable).

Weather data was obtained from the Danish Meteorological Institute, corresponding to the monitoring tower closest to the farm, approximately 35 km away. Air pressure values were unavailable. Although the lack of *in loco* weather monitoring equipment led to imprecise numerical values, the goal of this data is not to relate specific wind speeds to specific IWT acoustical emissions, but rather to compare ILFN components under two different IWT regimens, at two different locations. Weather data is given in Table 2.

Table 2: Weather data and IWT operation.

	16 Dec 2016 - Baseline	30 Dec 2016 - ILFN-rich
Wind speed (m/s)	0.5 - 1.5	4.0 - 6.5
Wind direction	south-southwest	southwest
Temperature (°c)	0 - 1	6 - 8
Rel. humidity (%)	90 - 100	90 - 95
Precipitation (mm)	0	< 0.5
IWT rotation	no	yes
Hour of video footage	14:38, 14:42	11:20, 13:30, 15:40

Table 3: Definition of the 10 min measurement segments selected for scrutiny.

Location	IWT Rotation	Location Classification	Date	Time
1	No	*Baseline*	16 Dec	14:40
	Yes	*ILFN-rich*	30 Dec	17:30
2	No	*Baseline*	16 Dec	15:50
	Yes	*ILFN-rich*	30 Dec	11:20

Results

Acoustic data was processed in Matlab (The MathWorks, USA) using narrow-band filters complying with the ANSI® S1.11-2004 and IEC 61260:1995 standards, as well as FFTs.

Table 3 shows the measurement segments selected for scrutiny.

Figure 8 and Figure 9 show the traditional 1/3-octave spectra and the total dBA and dBLin levels, in Locations 1 and 2 respectively, under baseline conditions. Figure 10 and Figure 11 show the same numerical data but represented as narrow-band analyses.

Similarly, Figure 12 and Figure 13 show the traditional 1/3-octave spectra and the total dBA and dBLin levels, in Locations 1 and 2 respectively, under ILFN-rich conditions. Figure 14 and Figure 15 show the same numerical data but represented as narrow-band analyses.

For ease of presentation herein, only the results corresponding to the blue microphone (placed at approximately 42 m from the entrance of the sheds) are presented.

Preliminary analysis

16 - 20 Hz:

Baseline: Both Location 1 and Location 2 exhibit a continuous acoustical phenomenon with strong tonal characteristics occurring at around 16 Hz (Figure 10 and Figure 11). This could be associated with the water systems installed in the shed to feed the animals. Water could distinctly be heard running in the background in Location 1, but less so in Location 2.

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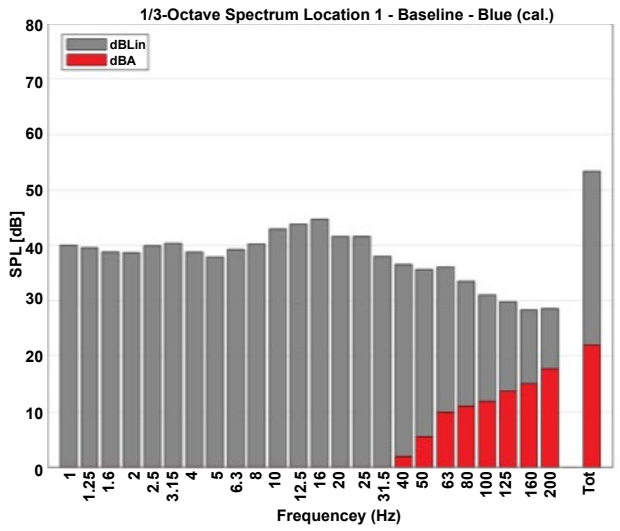


Figure 8: Location 1, Older Shed, at 14:40 on Dec 16. Representative data over a 10-min interval and analyzed between 1-200 Hz.

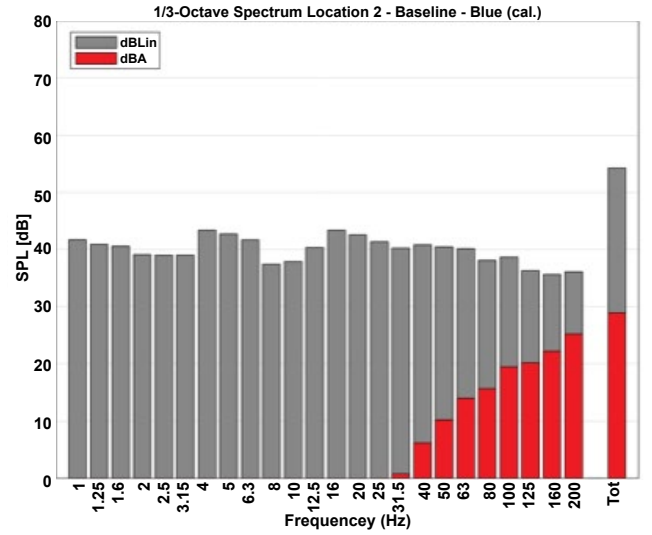


Figure 9: Location 2. Newer Shed, at 15:50, on Dec 16. Representative data over a 10-min interval and analyzed between 1-200 Hz.

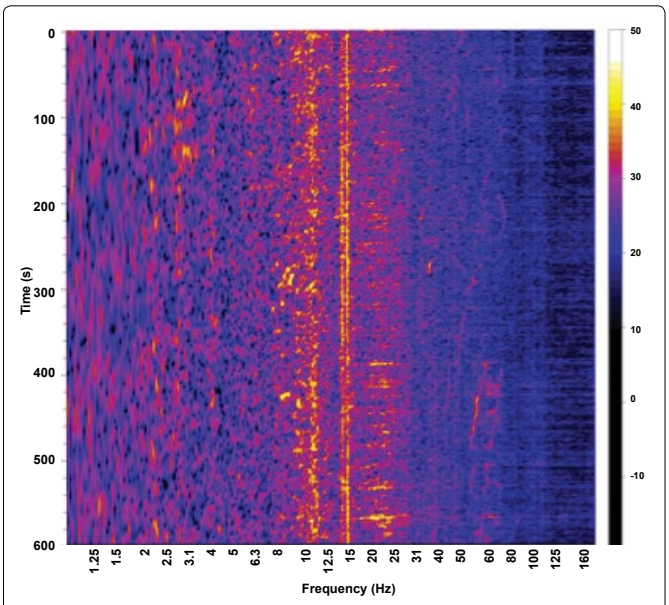


Figure 10: Location 1, Older Shed, at 14:40 on Dec 16. Representative data over a 10-min interval and analyzed between 1-200 Hz.

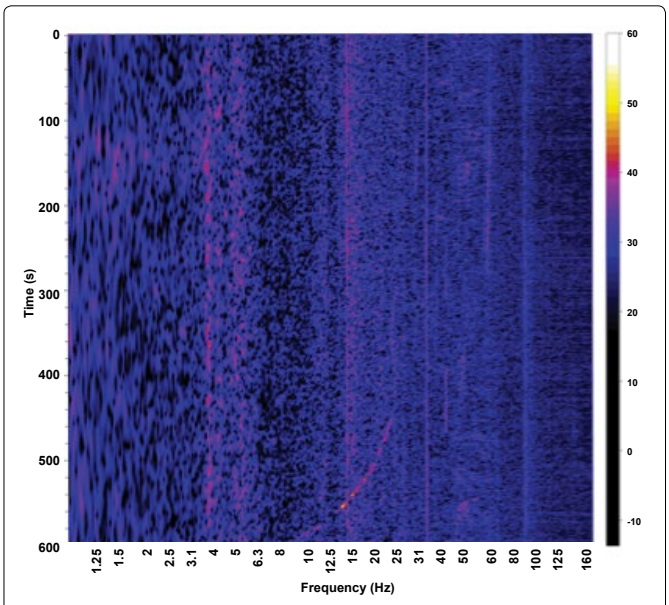


Figure 11: Location 2. Newer Shed, at 15:50, on Dec 16. Representative data over a 10-min interval and analyzed between 1-200 Hz.

ILFN-rich: In Location 1, the continuous acoustical phenomenon, seen at around 16 Hz in the baseline (Figure 10), is still visible in Figure 14, but is no longer distinguishable from other acoustical events in Figure 15 (baseline, Figure 11). The apparent constancy of this acoustical feature in at least three of the four situations seems to suggest some permanent equipment, and hence the animal water supply system is a good contender.

Concurrent with the existence of IWT rotation, a new acoustical phenomenon, not present in either baseline, appears at 20 Hz and in both locations. The non-continuous coloring of this line shows that pressure level was not continuous. Further analysis (Figure 16 and Figure 17) shows that this 20 Hz phenomenon was equally prominent in Location 2 (newer shed) as in Location 1 (older shed).

10-12.5 Hz:

Baseline: Given the continuous nature of the acoustical event that occurs between 10-12.5 Hz (Figure 10), this might also be associated with the operational water systems, or other equipment necessary to maintain the animals.

ILFN-rich: Below 12.5 Hz, both locations see an increase in their acoustical energy.

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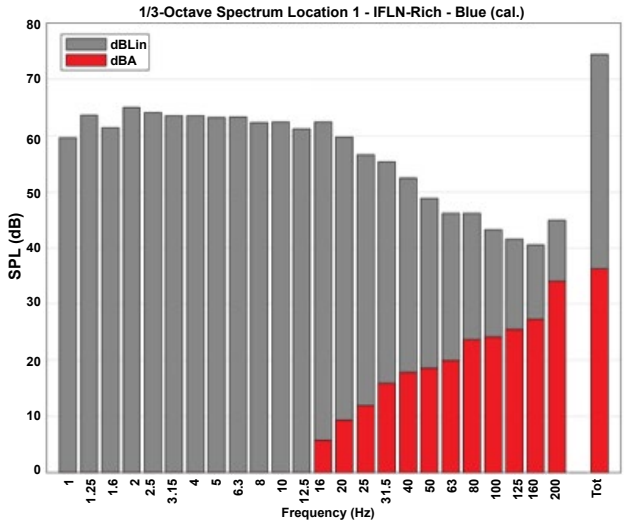


Figure 12: Location 1, Older Shed, at 17:30 on Dec 30. Representative data over a 10-min interval and analyzed between 1-200 Hz.

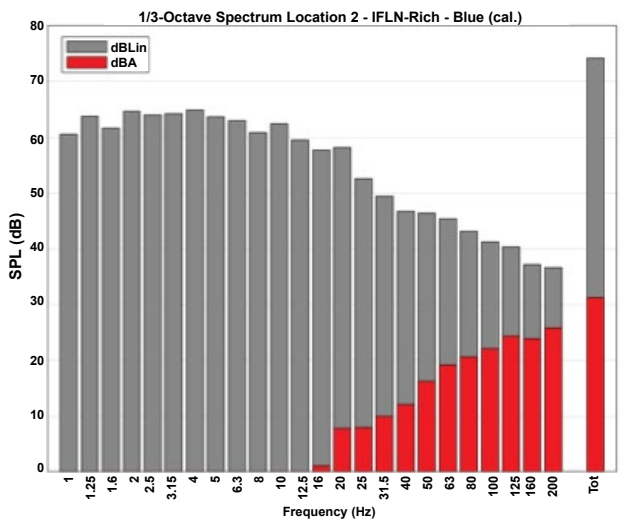


Figure 13: Location 2. Newer Shed, at 11:20, on Dec 30. Representative data over a 10-min interval and analyzed between 1-200 Hz.

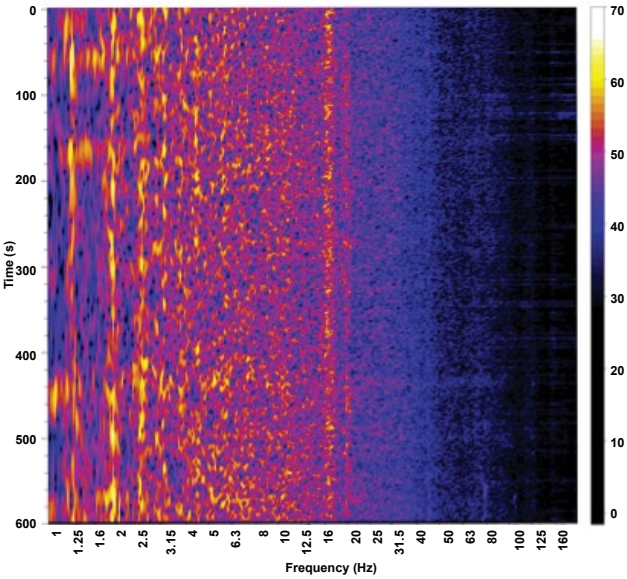


Figure 14: Location 1, Older Shed, at 17:30 on Dec 30. Representative data over a 10-min interval and analyzed between 1-200 Hz.

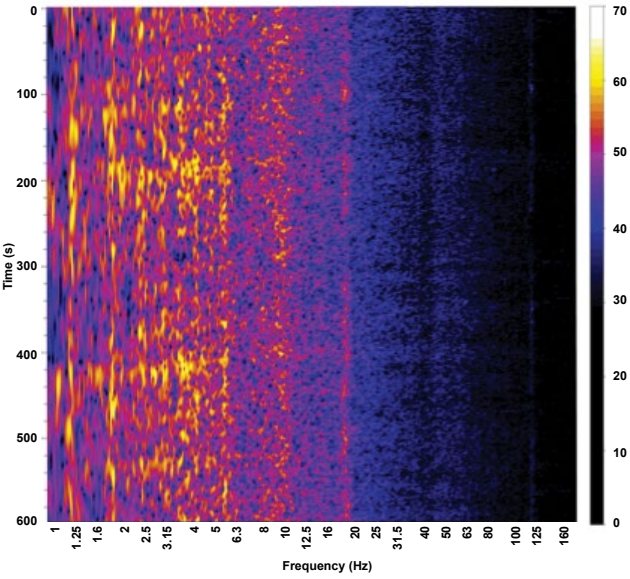


Figure 15: Location 2. Newer Shed, at 11:20, on Dec 30. Representative data over a 10-min interval and analyzed between 1-200 Hz.

< 5 Hz:

Baseline: In Location 2, the continuous phenomena occurring between 4-6.3 Hz could be related to the newer water supply system. 'Spurts' of acoustical energy are visible at the lowest frequency ranges (≤ 2 Hz), in both locations but seemingly more prominently in Location 1 (Figure 10). The source of these 'spurts' is, as yet, unknown.

ILFN-rich: The continuous acoustical phenomena occurring at 4-6.3 Hz in Location 2 during baseline (Figure 11) is no longer visually distinguishable in Figure 15. The 'spurts' of acoustical energy identified at ≤ 2 Hz in both Locations during baseline, are only vaguely visible in the ILFN-rich situation. In this lowest range of frequencies, both locations still seem to exhibit acoustical phenomena in spurts but, now, some of them contain more acoustical energy (Figure 14 and Figure 15).

dBA methodology:

Baseline: Location 2 exhibits a higher total dBA level (44.9 dBA) (Figure 9 and Table 4) than Location 1 (38.6 dBA) (Figure 8 and Table 5). While this seems to be opposite of what is seen in the respective sonograms (Figure

10 and Figure 11), it must be recalled that dBA emphasizes acoustical phenomena that occur above 200 Hz. Indeed, as that region is approached, Location 2 exhibits more acoustical energy than Location 1. This can also be seen in Figure 8, where Location 1 exhibits a dip in the pressure levels starting at approximately 50 Hz, and that is not present in Location 2 (Figure 9). Similarly, the corresponding sonogram

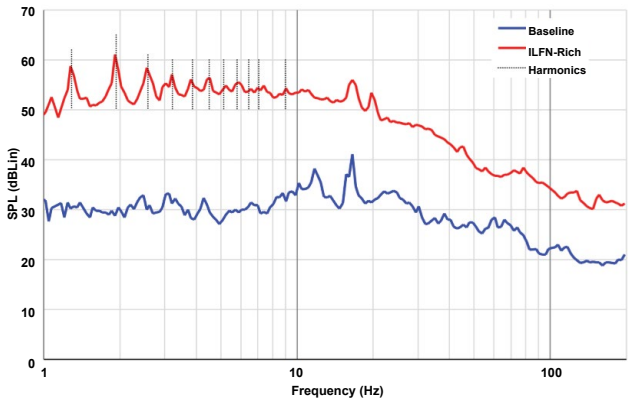


Figure 16: Narrow-band spectra comparing Location 1 - 14:40 on Dec 16 (Baseline) with Location 1 - 17:30 on Dec 30 (ILFN-rich). Representative data over a 10-min interval and analyzed between 1-200 Hz.

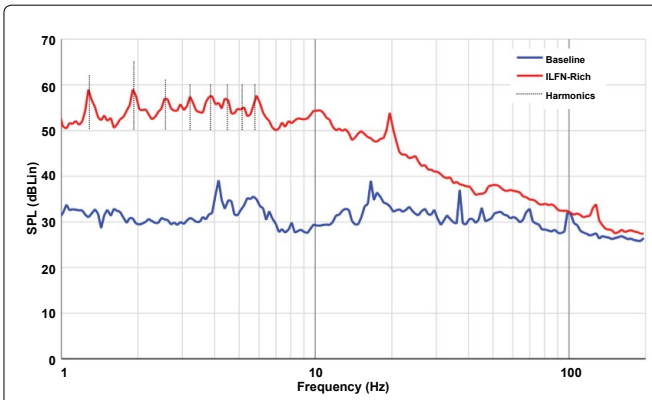


Figure 17: Narrow-band spectra comparing Location 2 - 15:50, on Dec 16 (Baseline) with Location 2 - 11:20, on Dec 30 (ILFN-rich). Representative data over a 10-min interval and analyzed between 1-200 Hz.

Table 4: Data tables for 1/3-Octave-Band and dBA analyses. 1/3-Octave-Band Analysis of location 2 (New Shed), baseline, at 15:50, on Dec 16 (Figure 9).

Frequency (Hz)	SPL (dBLin)	SPL (dBA)
1.0	41.9	-106.7
1.3	41	-99.6
1.6	40.7	-91.9
2.0	39.1	-85.5
2.5	39.1	-77.6
3.2	39.1	-69.7
4.0	43.3	-57.5
5.0	42.8	-50.3
6.3	41.8	-43.6
7.9	37.4	-40.4
10.0	37.8	-32.6
12.6	40.3	-23.1
15.8	43.3	-13.3
20.0	42.6	-7.9
25.1	41.4	-3.4
31.6	40.2	0.8
39.8	40.9	6.2
50.1	40.5	10.2
63.1	40.2	14
79.4	38.1	15.6
100.0	38.6	19.5
125.9	36.2	20.1
158.5	35.5	22.2
199.5	36	25.1
251.2	38.4	29.8
316.2	41.7	35.1
398.1	43.8	38.9
501.2	41	37.8
631.0	38.8	36.9
794.3	39.4	38.5
Overall	55.3	44.9

Table 5: Data tables for 1/3-Octave-Band and dBA analyses. 1/3-Octave-Band Analysis of location 1 (Old Shed), baseline, at 14:40, on Dec 16 (Figure 8).

Frequency (Hz)	SPL (dBLin)	SPL (dBA)
1.0	39.9	-108.7
1.3	39.6	-101
1.6	38.7	-93.9
2.0	38.7	-85.9
2.5	39.9	-76.8
3.2	40.4	-68.4
4.0	38.8	-62.1
5.0	37.8	-55.2
6.3	39.2	-46.1
7.9	40.2	-37.6
10.0	42.9	-27.5
12.6	43.8	-19.6
15.8	44.7	-12
20.0	41.6	-8.9
25.1	41.7	-3
31.6	38	-1.4
39.8	36.6	2
50.1	35.6	5.4
63.1	36.2	10
79.4	33.5	11
100.0	31	11.9
125.9	29.8	13.7
158.5	28.4	15.1
199.5	28.6	17.8
251.2	29	20.4
316.2	33.7	27.1
398.1	38.6	33.8
501.2	37.4	34.1
631.0	30.8	28.9
794.3	29.5	28.7
Overall	53.6	38.6

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(Figure 10) presents with several black areas in the >125 Hz region, while Location 2 has none (Figure 11). The fact that the total dBLin level is higher in Location 2 than in Location 1 (55.3 dBLin vs. 53.6 dBLin, Table 4 and Table 5, respectively), attests to the fact that more acoustical energy exists in Location 2 than in Location 1. This would seem to indicate that the elevated pressure levels occurring within the 10-25 Hz range in Location 1 (Figure 10, yellow areas) do not outweigh the evenly distributed pressure levels (> 125 Hz, in blue) shown in Location 2 (Figure 11).

ILFN-rich: In terms of 1/3-octave and dBA analyses, contrary to baseline, Location 1 now exhibits a higher total dBA level (53.4 dBA) (Figure 12 and Table 6) than Location 2 (44.9 dBA) (Figure 13 and Table 4), although the total dBLin values of both Locations are nearly identical (74.4 and 74.2 dB, Table 6 and Table 7, respectively).

Narrow-band-spectra methodology: Regarding the narrow-band spectra (Figure 16) a first peak can be seen at approximately 1.3 Hz with further peaks at 1.9, 2.6, 3.2, 3.8, 4.5, 5.2, 5.9 and 9 Hz. The blade passing frequency of an IWT consists of the number of times the blades rotate past the vertical tower structure, per second. Through video footage during the ILFN-rich measurements, that number was identified and the blade passing frequency was calculated as 0.65 Hz. These peaks correspond to a harmonic series with a fundamental frequency close to 0.65 Hz, and thus constitute an integral part of the IWT acoustical signature.

In Location 2, harmonics are not as prominent (Figure 17) as in Location 1, but peaks can still be seen at 1.3, 1.9, 2.6, 3.2, 3.9, 4.4, 5.2 and 5.9 Hz - again, acting as a signature of an IWT with a blade-pass frequency of about 0.65 Hz. An FFT of this frequency range (Figure 18) also shows these peaks.

Discussion

Large-scale public health epidemiological studies of residential ILFN contamination

Several exploratory studies have been conducted by governmental agencies [32-34] regarding the health effects of residential ILFN exposure. Many base their acoustical data on models (as per the dBA-1/3-octave methodology) rather than real, *in loco*, field measurements. Moreover, the unawareness of the importance of prior ILFN exposure histories when assessing health endpoints among study and control populations, predictably leads to statistically inconclusive results. As a consequence, many of the more classical groups of scientists who continue to defend the archaic notion "what you can't hear won't hurt you" feel justified in so doing. In the most recent French survey on the topic, results confirmed that: "wind turbines are sources of infra sounds and low-frequency sounds. However, the hearing thresholds for infra sounds and low frequencies of up to 50 Hz were not exceeded" [34]. Within the context of VAD studies, residential exposures have been investigat-

Table 6: Data tables for 1/3-Octave-Band and dBA analyses. 1/3-Octave-Band analysis of location 1 (Old Shed), ILFN-rich, at 17:30, on Dec 30 (Figure 12).

Frequency (Hz)	SPL (dBLin)	SPL (dBA)
1.0	59.4	-89.1
1.3	63.1	-77.4
1.6	60.8	-71.8
2.0	64.9	-59.7
2.5	64	-52.7
3.2	63.4	-45.4
4.0	63.5	-37.4
5.0	63.3	-29.8
6.3	63.3	-22.1
7.9	62.4	-15.4
10.0	62.5	-8
12.6	61.2	-2.1
15.8	62.3	5.6
20.0	59.8	9.3
25.1	56.6	11.9
31.6	55.4	15.9
39.8	52.5	17.9
50.1	48.8	18.6
63.1	46.2	20
79.4	46.2	23.7
100.0	43.3	24.2
125.9	41.6	25.5
158.5	40.7	27.3
199.5	45.1	34.2
251.2	49.8	41.2
316.2	49.1	42.5
398.1	50.7	45.9
501.2	49.2	46
631.0	49.9	48
794.3	46.8	46
Overall	74.4	53.4

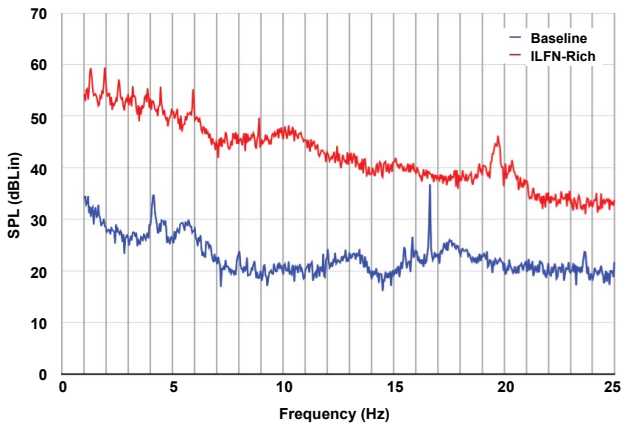


Figure 18: FFT power spectrum comparing Location 2 - 15:50, on Dec 16 (Baseline) with Location 2 - 11:20, on Dec 30 (ILFN-rich). Representative data over a 10-min interval and analyzed between 1-25 Hz.

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ed using the same clinical endpoints as those that were found relevant for ILFN-exposed aeronautical technicians [20-22]. These document the accelerated onset of symptoms among families exposed to residential ILFN (generated by a grain terminal [21] and by IWTs [22]) when compared to occupational exposures.

Narrow-band analysis methodology vs. dBA methodology data for IWT-generated ILFN

The benefit of narrow-band analysis becomes obvious by the identification of discrete peaks in the presence of IWT rotation, and that are absent when IWT are not rotating (Figure 17 and Figure 18). These peaks have been identified as the harmonics of a fundamental frequency, whose value precisely coincides with that of the IWT blade-pass frequency (as verified by video footage). This type of information is impossible to obtain with the dBA-1/3-octave methodology.

As many acousticians would quickly point out, the increased acoustical energy in the shed caused by rotating IWT is indistinguishable from that caused by the wind (Figure 10 vs. Figure 14 and Figure 11 vs. Figure 15). Blowing wind outside will increase the acoustical energy in the ILFN range within practically any structure (whether or not it is heard by humans). Identifying specific acoustic signatures associated with IWT operation (as demonstrated in Figure 16, Figure 17 and Figure 18), can help pin-point the contributions made by the blowing wind (structure resonance), and differentiate those from anthropogenic ILFN. But this cannot be accomplished with the dBA-1/3-octave methodology, as shown in Figure 8, Figure 9, Figure 12 and Figure 13.

Narrow-band analysis can also provide information that may in the future be relevant for dose-response values, and which the dBA-1/3-octave methodology cannot. For example, in the specific examples shown for the ILFN-rich locations (Figure 14 and Figure 15), Location 2 has similar acoustical energy at 20 Hz as Location 1. And yet, it is in Location 1 that the animal owner prefers to keep his breeding males. This may suggest that the frequencies that are more important for understanding the abnormal animal behavior (as reported by the owner) are not within the 20 Hz region.

Concomitant occupational and residential ILFN exposures in aerospace workers

In the summer of 2015, the home near the animal-sheds was abandoned by the family. IWTs had begun rotating in September 2013, and the ensuing health deterioration of family members demanded their removal. To this day, however, the property owner must return there everyday to care for the animals that are his livelihood. As in other ILFN-contaminated residences [35], this farmer's health continues to deteriorate considerably, having most recently been diagnosed with Post-Traumatic Stress Disorder.

Table 7: Data tables for 1/3-Octave-Band and dBA analyses. 1/3-Octave-Band analysis of location 2 (New Shed), ILFN-rich, at 11:20, on Dec 30 (Figure 13).

Frequency (Hz)	SPL (dBLin)	SPL (dBA)
1.0	60.8	-87.8
1.3	63.9	-76.7
1.6	61.8	-70.8
2.0	64.7	-60
2.5	63.9	-52.8
3.2	64.3	-44.5
4.0	65	-35.8
5.0	63.6	-29.4
6.3	63.1	-22.3
7.9	60.8	-17
10.0	62.4	-8
12.6	59.4	-4
15.8	57.6	1
20.0	58.3	7.8
25.1	52.6	7.9
31.6	49.4	9.9
39.8	46.6	12
50.1	46.4	16.2
63.1	45.3	19.1
79.4	43.1	20.6
100.0	41.2	22.1
125.9	40.4	24.3
158.5	37.1	23.8
199.5	36.5	25.6
251.2	37.8	29.1
316.2	40.7	34.1
398.1	42.9	38.1
501.2	40.7	37.5
631.0	37.5	35.6
794.3	37.5	36.6
Overall	74.2	44.9

Aerospace workers who are exposed to occupational ILFN greatly benefit from the recovery periods encountered in their homes (presumably absent of anthropogenic ILFN) [16,19,36]. A growing segment of the world population, however, particularly from rural and suburban areas, has been confronted with ILFN-contamination in their homes. Some of these families include aerospace professionals (and other occupationally exposed ILFN workers), who are now exposed to ILFN both at work and at home.

The ongoing citizen-based research effort into ILFN-induced pathology to which these authors contribute [30] includes providing proper acoustical evaluations to participating citizens' properties, and also providing pertinent medical diagnostic tests, including the established VAD diagnostic tests [16,21,37]. Within this context, initial steps include obtaining personal and medical histories from each participating citizen. Histories are ob-

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tained through one-on-one interviews that can last over 2 hours. Some of the citizens already interviewed include aerospace workers who are (or were) occupationally exposed to ILFN environments. Of these, some were already sleeping in ILFN-rich homes (due to anthropogenic sources), while others were expected to have sources of anthropogenic ILFN constructed near (< 3 km) their residential areas. Clinical information revealed in the interviews and corroborated by accompanying medical documentation, has been reiterating the association between symptom gravity and overall ILFN exposure.

People exposed to ILFN at work and at home see an acceleration of the onset of ILFN-induced pathology when compared to individuals who 'only' have ILFN exposure at work (assuming similar fetal, childhood and adolescent ILFN exposures). Individuals who are only exposed to ILFN-rich environments in the home see an accelerated onset of symptoms when compared to those who are only exposed to occupational ILFN-rich environments [20-22]. The reasons for this are twofold: on the one hand, when ILFN-exposed workers leave their place of employment, they undergo a biological respite from the agent of disease; on the other hand, ILFN-rich environments in the home are usually synonymous with sleeping in an anthropogenic ILFN-rich environment. Biological processes that only occur during sleep time are now occurring in the presence of an agent of disease [38].

Limitations of this study

When laboratorial studies are conducted using airborne acoustical phenomena, environmental parameters can be controlled with more or less ease. In real environments, however, an acoustic environment will not be homogeneous over any significant area or any significant time. Outdoors the environment may be reasonably homogeneous over tens of meters (apart from the interference effects from multiple sound sources creating 'heightened noise zones' possibly only a few meters across [39]). However, indoor environments can vary significantly over distances less than a meter. Furthermore, the acoustical differences between the two locations may have been due to time-wise changes between recording times.

Conclusions

This report highlights the difference in acoustical information gathered with two distinct methods of analysis: one sanctioned by current legislation and guidelines, and focused on protecting hearing impairment (dBA-1/3-octave methodology); the other, sanctioned by the bio-physical sciences and focused on protecting whole-body health (narrow-band methodology). The latter provides important information on the temporal and frequency profiles that are crucial for understanding how ILFN affects human health (considering both immediate and long-term effects).

The archaic notion of "what you can't hear won't hurt you" is reflected in the dBA-1/3-octave methodology, i.e., the hearing sensory pathway is the only one through which ILFN can adversely affect humans. This position is incompatible with the Scientific Method and with evidence-based medicine when quantifying a potential agent of disease, such as ILFN.

The clinical evolution of ILFN-induced pathology greatly depends on exposure-time patterns. Individuals, who work in ILFN-rich environments and simultaneously live in ILFN-rich homes, may see an accelerated onset of specific symptoms when compared with individuals who only live in the ILFN-rich home, with no prior or current history of occupational ILFN exposure. Therefore, the increasing number of ILFN-rich acoustic environments within rural residential dwellings poses a serious problem for ILFN-exposed aerospace workers, as their biological recovery periods (that occur when away from the ILFN-rich environment) may be greatly reduced, or even become non-existent.

In order to protect populations from excessive and harmful ILFN exposure, serious epidemiological studies under the auspices of 'Public Health' must be undertaken. An important step in that direction is taken here, showing the importance of departure from the established guidelines and legislation in order to obtain a scientifically useful quantification of the agent of disease under scrutiny.

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Due to his efforts in the creation of the SAM Scribe system, author HHCB has a financial interest in the SAM Scribe system.

Disclaimer

The authors of this paper:

1) Do not harbor anti-technology sentiments;

2) Consider industrial activities to be important to modern technological societies;

3) Have scrutinized the data under one and only on agenda - pure scientific inquiry;

4) Are not producing a report arguing against industrial complexes;

5) Are not producing an environmental noise assessment report focused on wind turbines;

6) Provided all acoustical evaluations and analyses pro bono.

References

1. World Health Organization (2011) Burden of disease from environmental noise.
2. World Health Organization (1999) Guidelines for community noise. In: Berglund B, Lindvall T, Schwela DH, World Health Organization, Geneva.
3. World Health Organization (2016) International Classification of Diseases. ICD-10 Version 2016.
4. Ingber DE (1993) Cellular tensegrity: defining new rules of biological design that governs the cytoskeleton. J Cell Sci 104: 613-627.
5. Ingber DE (2003) Mechanobiology and diseases of mechanotransduction. Ann Med 35: 564-577.
6. Ingber DE (2004) Mechanochemical basis of cell and tissue regulation. Mech Chem Biosyst 1: 53-68.
7. Motro R (2003) Tensegrity - Structural systems for the future. Hermes Science Publishing Limited, London, UK.
8. Sun Z, Guo SS, Fässler R (2016) Integrin-mediated mechanotransduction. J Cell Biol 216: 445-456.
9. Xu GK, Li B, Feng XQ, et al. (2016) A tensegrity model of cell reorientation on cyclically stretched substrates. Biophys J 111: 1478-1486.
10. Castelo Branco NA, Águas AP, Sousa Pereira A, et al. (1999) The human pericardium in vibroacoustic disease. Aviat Sp Environ Med 70: 54-62.
11. Castelo Branco NA, Gomes-Ferreira P, Monteiro E, et al. (2003) Respiratory epithelia in Wistar rats after 48 hours of continuous exposure to low frequency noise. Rev Port Pneumol 9: 473-479.
12. Alves-Pereira M, Joanaz de Melo J, Castelo Branco NA (2005) Pericardial biomechanical adaptation to low frequency noise stress. In: A Méndez-Vilas, Recent Advances in Multidisciplicnary Applied Physics. Elsevier, London, UK, 363-367.
13. Ponomar'kov VI, Tysik Ayu, Kudryavtseva VI, et al. (1969) Biological action of intense wide-band noise on animals. In: Problems of Space Biology. NASA TT F-529, 307-309.
14. Nekhoroshev AS, Glinchikov VV (1992) Morphological research on the liver structures of experimental animals under the action of infrasound. Aviakosm Ekolog Med 26: 56-59.
15. Nekhoroshev AS, Glinchikov VV (1991) Effect of infrasound on morphofunctional changes in myocardium exposed to infrasound. Gig Sanit 12: 56-58.
16. Castelo Branco NA (1999) Clinical stages of vibroacoustic disese. Aviat Space Environ Med 70: 32-39.
17. Castelo Branco NA, Alves-Pereira M (1999) Vibroacoustic disease. Aviat Space Environ Med 70: 1-153.
18. Castelo Branco NA, Alves-Pereira M (2004) Vibroacoustic disease. Noise Health 6: 3-20.
19. Alves-Pereira M, Castelo Branco NA (2007) Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling. Prog Biophys Mol Biol 93: 256-279.
20. Torres R, Tirado G, Roman A, et al. (2001) Vibroacoustic disease induced by long-term exposure to sonic booms. Proceedings Internoise 2001, The Hague, Holland, 1095-1098.
21. Araujo A, Alves-Pereira M, Joanaz de Melo J, et al. (2004) Vibroacoustic disease in a ten-year-old male. Proceedings Internoise 2004, Prague, Czech Republic.
22. Alves-Pereira M, Castelo Branco NA (2007) In-home wind turbine noise is conducive to vibroacoustic disease. Wind Turbine Noise, Lyon, France.
23. Alves-Pereira M, Joanaz de Melo J, Castelo Branco NA (2005) Actin- and tubulin-based structures under low frequency noise stress. In: A Méndez-Vilas, Recent Advances in Multidisciplinary Applied Physics. Elsevier, London, UK, 955-979.
24. Alves-Pereira M, Joanaz de Melo J, Castelo Branco NA (2005) Low frequency noise exposure and biological tissue: reinforcement of structural integrity? In: A Méndez-Vilas, Recent Advances in Multidisciplinary Applied Physics. Elsevier, London, UK, 961-966.
25. Cooper S (2014) The Results of an Acoustic Testing Program Cape Bridgewater Wind Farm. Energy Pacific (Vic) Pty Ltd, Melbourne, Australia.
26. Cooper S (2015) Soundscape of a wind farm, Cape Bridgewater experience. Proceedings of the 170th Meeting of the Acoustical Society of America, Jacksonville, FL, USA.
27. www.smart-technologies.co.nz
28. Bakker H, Rapley B, Summers R, et al. (2017) An affordable recording instrument for the acoustical characterisation of human environments. Proceedings International Conference Biological Effects of Noise, Zurich, Switzerland.
29. http://www.primo.com.sg/japan-low-freq-micro
30. Bakker HHC, Alves-Pereira M, Summers SR (2017) Citizen Science Initiative: Acoustical Characterisation of Human Environments. Proceedings International Conference Biological Effects of Noise, Zurich, Switzerland.
31. http://vindinfo.dk/english.aspx
32. Health Canada (2014) Wind turbine noise and health study.
33. National Health and Medical Research Council of Australia (2013) Systematic review of the human health effects of wind farms.
34. French Agency for Food, Environmental and Occupational Health & Safety (ANSES) (2017) Exposure to low-frequency sound and infrasounds from wind farms: improving information for local residents and monitoring noise exposure.
35. Castelo Branco NA, Alves-Pereira M, Martinho Pimenta A, et al. (2015) Low frequency noise-induced pathology: contributions provided by the Portuguese wind turbine case. Euronoise 2015, Maastricht, The Netherlands.
36. Castelo Branco NA, Rodriguez E, Alves-Pereira M, et al. (1999) Vibroacoustic disease: some forensic aspects. Aviat Sp Environ Med 70: 145-151.
37. Castelo Branco NA, Alves-Pereira M, Martinho Pimenta A, et al. (2015) Clinical protocol for evaluating pathology induced by low frequency noise exposure.
38. Nissenbaum MA, Aramini JJ, Hanning CD (2012) Effects of industrial wind turbine noise on sleep and health. Noise Health 14: 237-243.
39. Rapley BI, Bakker HHC (2010) Sound, Noise, Flicker and the

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Human Perception of Wind Farm Activity. In: Atkinson, Rapley Consulting, Palmerston North, New Zealand, 235-236.

40. https://commons.wikimedia.org/wiki/File:Acoustic_weighting_curves_(1).svg
41. Dirac Delta Science & Engineering Encyclopedia (2017) G-weighted overall level.
42. Dirac Delta Science & Engineering Encyclopedia (2017) A-Weighting.
43. https://www.fhwa.dot.gov/environment/noise/regulations_and_guidance/analysis_and_abatement_guidance/polguide01.cfm
44. Bento Coelho JL, Ferreira A, Serrano J, et al. (1999) Noise assessment during aircraft run-up procedures. Aviat Space Environ Med 70: 22-26.
45. Bento Coelho JL, Ferreira A, Serrano J, et al. (1994) Noise exposure in the aeronautical industry. Internoise 31: 741-744.

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Infrasound and Low Frequency Noise – Shall we measure it properly?

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On the *Engineers Ireland* website, a search on "infrasound" or "low frequency noise" yields zero results. A search on "noise" however, yields 39 results. Why is it that infrasound and low frequency noise (ILFN) is still such a taboo subject? While it is improbable that this particular question will be answered here, an exposé of ILFN will be provided with a brief historical account of how and why ILFN was ultimately deemed irrelevant for human health concerns.

Infrasound and Low Frequency Noise (ILFN) are airborne pressure waves that occur at frequencies ≤ 200 Hz. These may, or may not, be felt or heard by human beings. In order to clarify concepts, in this report the following definitions are used: *acoustic phenomena:* airborne pressure waves that may or may not be perceived by humans; *sound:* acoustic phenomena that can be captured and perceived by the human ear; *noise*: sound that is deemed undesirable; *vibration:* implies a solid-to-solid transmission of energy.

Harvey Fletcher, the Telephone and the deciBel

In the early part of the 20th-century, Harvey Fletcher of the Western Electrics Laboratories of AT&T, was tasked with improving the quality of reception in the telephone. To generate the sounds in a telephone earpiece, he used an a.c., voltage and had some of his colleagues rate the loudness of the sound received compared to the quietest tone heard. The company was already using a logarithmic scale to describe the power in an electrical cable and it made sense to rate the loudness of the sounds also on a logarithmic scale related to the quietest voltage that could just be heard. Initially he called this metric a "sensation unit" but later to commemorate their founder Alexander Graham Bell, they renamed it the "Bel." A tenth of a Bel became known as the deciBel, corrupted to decibel, which has stuck with the scientific community to this day.

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Fletcher-Munson Equal Loudness Curves and the dBA metric

To address the problem of industrial noise in the early 20th century, measurement was essential, as was a metric. At that time, researchers were critically aware that the readings on a sound level meter did not represent how loud or intense the sound was with respect to the subject's perception of hearing. From a biomedical perspective, this concept of perception is subjective, and changes between individuals and over timescales from minutes to decades. These serious constraints notwithstanding, it was acknowledged that some average measure of loudness would have some value for medicine and public health.

Harvey continued his research with Wilden Munsen, one of his team, by varying the frequency of the electricity to give pure tones, to which it is understood twenty-three of his colleagues listened to different levels of loudness, again through a simple telephone earpiece. (It is assumed they all had good hearing). They were then asked to score the sounds for equal loudness to that generated by an alternating current at 1000 cycles per second. The level of the sound of course depended on the voltage applied, which could be measured. It is important to note two significant constraints here: The sounds were 'pure' sine waves, which are not common in nature, and the headphones enclosed the ear of the subject. This is a very unnatural way to listen to a very unnatural sound.

The numerical results of this study are known as the Fletcher-Munsen Curves (Fig 1). The (logarithmic) units of these curves are known as "phons," and the inverse of the 40 phon curve forms the basis of the A-frequency weighting scale used everywhere today (Fig 2).

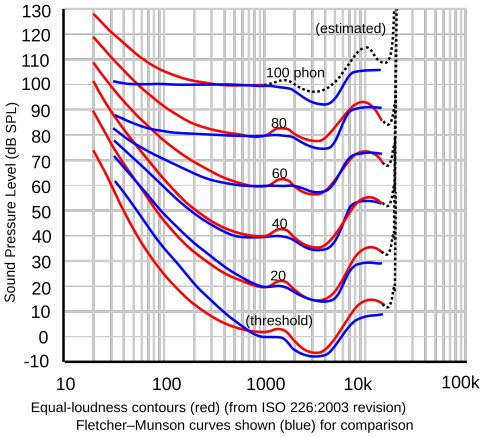


Figure 1. Fletcher Munson Curves [2]

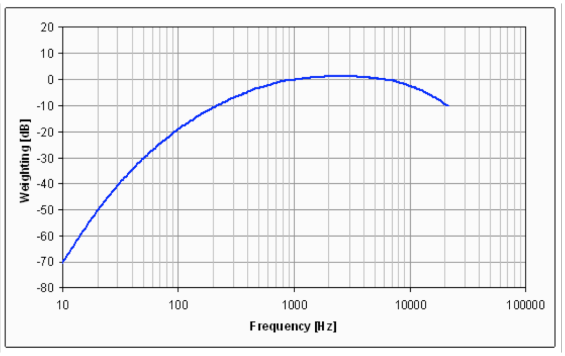


Figure 2. A-weighting frequency response curve [3]

The minimum pressure required for humans to perceive sound at 1000 Hz is considered to be 20 micropascal, or an intensity of 10^{-12} watts per square meter. This corresponds to 0 phon on Figure 1, and 0 dBA in Figure 2. For all its shortcomings, the A-Weighting has endured for decades and has become the *de facto* standard for environmental noise

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measurement. But is the A-weighting sufficient for all circumstances? The answer is an emphatic "No." It relates to the perception of loudness, which heavily discounts all frequencies below 1000 Hz and ends at 20 Hz. This 20-Hz limit was a consequence of equipment limitations of the 1920s and 30s, but has remained as the lower limit of human hearing to this day. The assumption that harm from excessive noise exposure is directly related to the perception of loudness has also remained to this day. Observe in Fig 2 that, at 10 Hz, there is a 70-dB difference between what is measured and what is, *de facto*, present in the environment. In other words, three-and-a-half orders of magnitude of energy are discounted at this frequency. The implications for public health are considerable, and within this line of reasoning, any event below 20 Hz becomes of no consequence whatsoever, and more so because it is not implicated in the classical effects of excessive noise exposure: hearing loss.

There are also issues of time and frequency resolution. Acoustic phenomena are time varying events. A 10-min average of acoustic events can hide more than it reveals. Similarly, segmenting frequencies into octave or 1/3-octave bands for analysis can also hide much that needs to be seen. Today, affordable and highly portable equipment can record acoustical environments, and allow for post-analysis in sub-second time increments and 1/36-octave resolution. Waveform analysis from the sound file directly can achieve an even better resolution.

Preliminary Results from Field Studies Conducted in Ireland

The following results, recently obtained in field-studies conducted in Ireland (Jul-Nov 2017), show why such resolution is needed to understand ILFN-rich environments. The classical metric (in dBA, 10-min averages and 1/3-octave bands) will be contrasted with what is needed for human health-related concerns (in dB with no frequency weighting, and resolutions of 0.2s and 1/36-octave bands), and not merely compliance with regulations.

Equipment and Methods

Acoustical environments were recorded with a SAM Scribe FS recording system, a 2-channel recorder with sampling rates up to 44.1 kHz at 16-bit resolution and linear response down to almost 0.1 Hz [4-6]. Recordings were saved as uncompressed WAV files including the 1000 Hz/94 dB reference calibration tone prior to and after measurements. Windshields were placed on both microphones during the entire measurement sessions. Microphones were attached to tripods at approximately 1.5 m above the ground.

Location

Five homes located around the same industrial wind turbine (IWT) development have been the object of study. The data presented here refers to Home 1 (Fig 3). Table 1 shows the dates and times of all recordings that have been made to date in this Home. The recordings selected for analysis and presentation herein were chosen on their educational value.

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Table 1.
Dates and times of recordings.

Home No.	Date	Time	Blue Channel	Red Channel
	04 Jul	04:05 – 06:48		
1	05 Jul	15:33 – 17:50	In child's bedroom-1	In child's bedroom-2
	10 Oct	17:40 – 18:43		

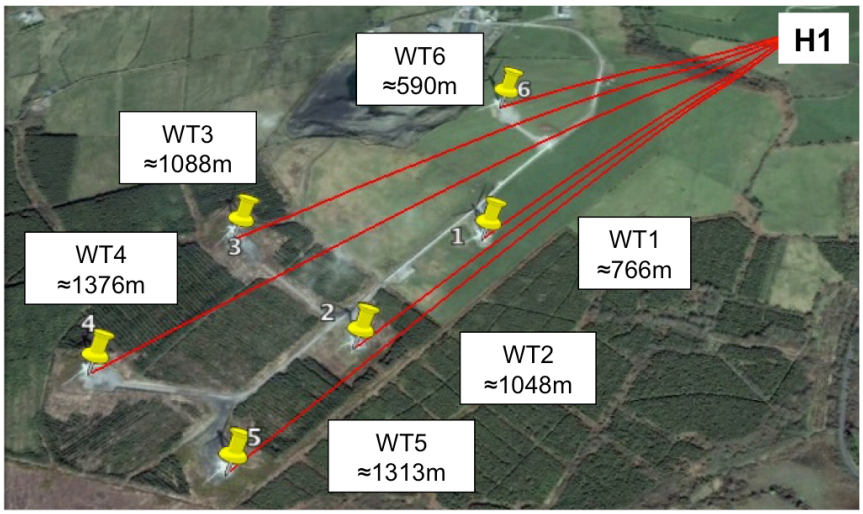


Figure 3. Reconstruction using a Google Earth Image and showing the relative position of Home 1 and each of the six industrial wind turbines.

Results

The information classically obtained with the dBA metric, 1/3-octave bands and 10-min averaging (on October 10th, 2017, at 18:30) is given in Figures 4 and 5. Weather conditions obtained from Met Éireann for the closest weather tower at this time were as follows: Air temperature: 14ºC, Precipitation: 0.1 mm, Mean Sea-Level Pressure: 1006.0 hPa, Wind Speed: 5.1 m/s (10 kt), Wind Direction: Southwest (200º az).

The values obtained for the sound pressure level and 1/3-octave bands are seen in Figures 4 and 5. The overall dBA metric (red bars labelled "Tot") reflects the sound that humans would hear if they were present in this environment. The sound pressure level in dBLin metric (grey bars labelled "Tot"), reflect the amount of acoustic energy to which humans are concomitantly exposed. The growing discrepancy between the two can be seen as the frequency falls below 1000 Hz.

Figure 6 shows the sonogram corresponding to the same 10-min period. This visual representation of time- and frequency-varying acoustic events provides much more information than the classical approach (Figs 4 and 5). Here short-term events can be

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seen in the region of 20-50 Hz (Fig 6). Tonal components can be seen at 10 Hz and 20 Hz that are not steady in amplitude and may be amplitude modulated, i.e., where the amplitude of the pressure is not continuous and varies periodically with time. The 10-min averages, used in almost all legislation, hide these variations and are representative only of tonal components that are essentially unvarying over the 10-min period in question.

The periodogram (Fig 7) over the same 10 minutes shows that there are distinct tonal components that form a harmonic series. When IWTs are the source of ILFN, the rotating blades generate repeated pressure waves as each blade replaces the previous one at any position. A harmonic series is formed with the "blade pass frequency" as the fundamental frequency (0.8 Hz here). These harmonics constitute what is called the *wind turbine signature* [7], which is impossible to identify using the classical dBA, 1/3-octave, 10-min averaging methodology.

Final Thoughts

Health concerns associated with excessive exposure to ILFN in the workplace have been around since the industrial boom in the 1960s [8]. In recent years, however, residential neighbourhoods have also begun to be flooded with ILFN [9-14]. The Family living in Home 1, for example, has abandoned their residence due to severe health deterioration in all family members. *Accredited acousticians cannot ascertain compliance levels for ILFN because there are none* - the vast majority of regulations worldwide do not cover this part of the acoustic spectrum. Nevertheless, Public Health Officials and Agencies should fulfil their job descriptions by becoming aware of the limitations of current noise guidelines and regulations. Alternatives exist to gather the acoustic information relevant to the protection of human populations, in both occupational and residential settings. Noise regulations and guidelines need urgent updating in order to appropriately reflect ILFN levels that are dangerous to human health.

Home 1 – A-weighting, 1/3 octave bands (0.5 Hz – 4000 Hz), 10–min average - Red Channel

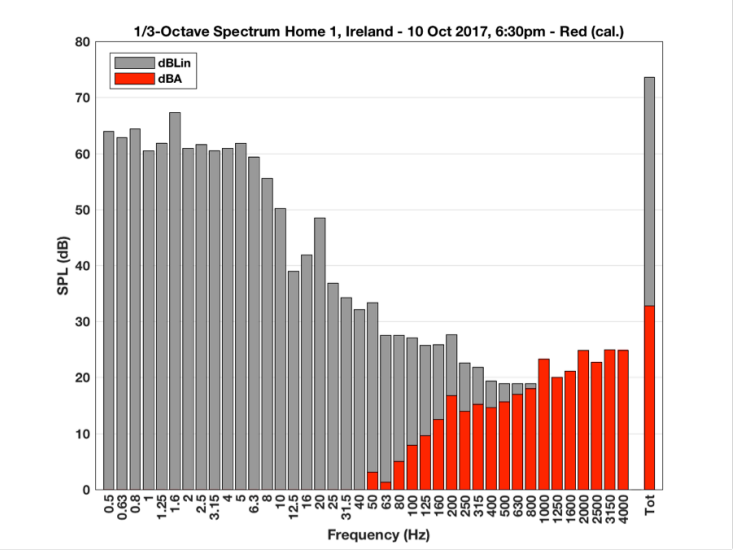


Figure 4. Data covers a 10-min interval analyzed between 0.5–4000 Hz, in 1/3-octave bands, as recorded in Home 1, on 10 Oct 2017, at 18:30 (red microphone, i.e. inside child's bedroom-2).
The red bars are A-weighted values, while the gray bars indicate the acoustic energy that is, *de facto* present, in dBLin. In this environment, the human being would perceive through the ear an overall A-weighted pressure-level of approximately 34 dBA (Tot - red bar), while being concomitantly exposed to an overall acoustic pressure-level of approximately 74 dBLin (Tot – grey bar).

Home 1 – A-weighting, 1/3 octave bands (0.5 Hz – 1000 Hz), 10–min average - Red Channel

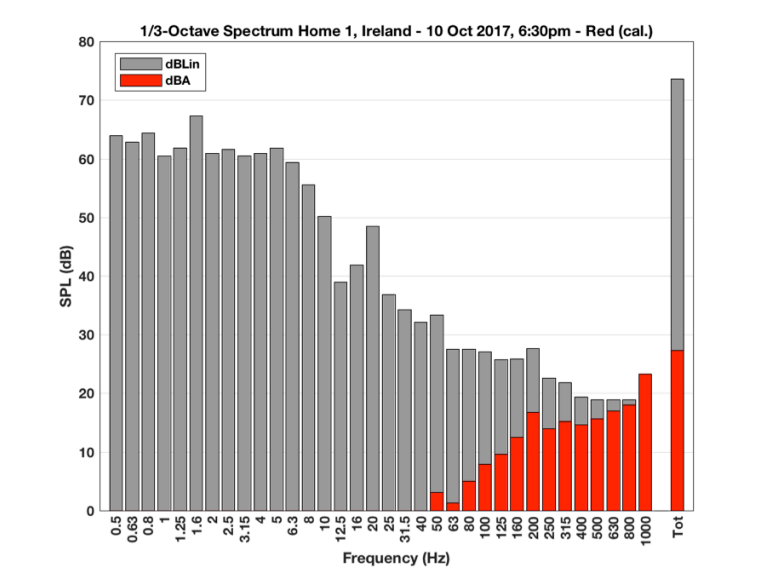


Figure 5. Data covers a 10-min interval analyzed between 0.5–1000 Hz, in 1/3-octave bands, as recorded in Home 1, on 10 Oct 2017, at 18:30 (red microphone, i.e. inside child's bedroom-2). The red bars are A-weighted values, while the gray bars indicate the acoustic energy that is, *de facto* present, in dBLin. In this environment, the human being would perceive through the ear an overall A-weighted pressure-level of approximately 26 dBA (Tot - red bar), while being simultaneously exposed to an overall acoustic pressure-level of approximately 74 dBLin (Tot – grey bar).

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Home 1 – No weighting, 1/36 octave bands (0.5 Hz – 1000 Hz), 0.2 s average - Red Channel

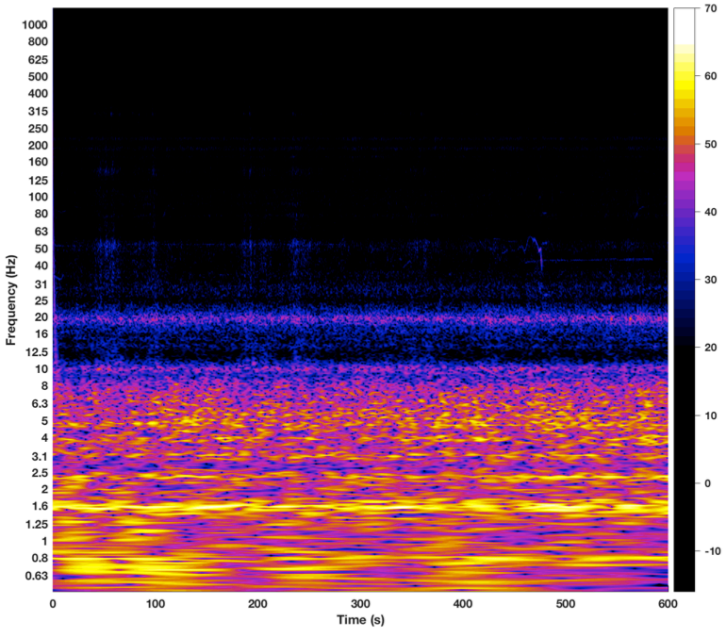


Figure 6. Sonogram that covers the same 10-min interval (600 s) as in Figures 4 and 5 showing time-varying features. The colour-coded bar on the right indicates sound pressure level values in dB Linear (no weighting). The horizontal line seen at 20 Hz is not a continuous tone because over the 600 s, its pressure level (colour-coded data) varies. A strong (yellow) acoustic phenomenon can be seen to exist at 1.6 Hz and also at 0.8 Hz.

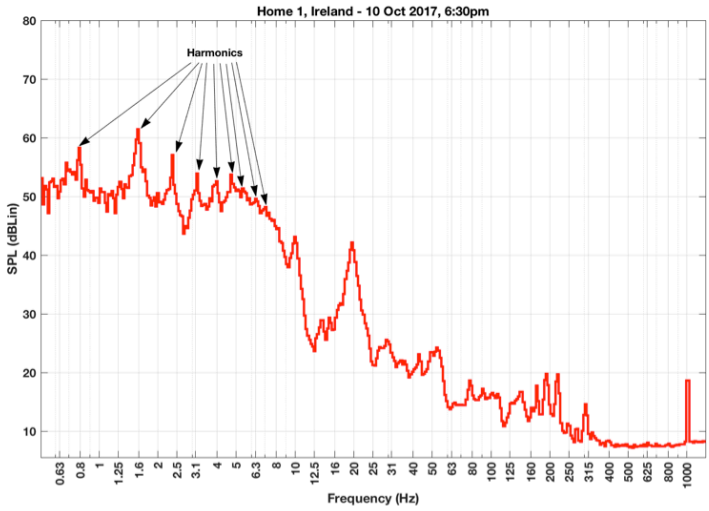


Figure 7. Periodogram covering the same 10-min interval (600 s) as in Figs 4-6, and analyzed between 0.5–1250 Hz. The blade pass frequency of the IWT is 0.8 Hz. Harmonics of this fundamental frequency are shown in the figure. Each frequency band composing the harmonic series has a well-defined peak, e.g., the horizontal line seen in Figure 7 at 20 Hz is represented here as a peak at 20 Hz.

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References

[1] Dickinson P (2006). Changes and challenges in environmental noise measurement. Acoustics Australia, 34 (3), 125-129.

[2] Wikicommons (2017). Fletcher-Munson Curves. https://commons.wikimedia.org/wiki/File:Lindos4.svg

[3] Dirac (2017). Dirac Delta Science & Engineering Encyclopedia, A-Weighting. http://diracdelta.co.uk/wp/noise-and-vibration/a-weighting/

[4] Atkinson & Rapley Consulting Ltd (2017). Specification sheet for the SAM Scribe FS Mk 1. www.smart-technologies.co.nz

[5] Primo Co, Ltd. (Tokyo, Japan) (2017). Specification sheet for the electret condenser microphone, custom-made, model EM246ASS'Y. http://www.primo.com.sg/japan-low-freq-micro

[6] Bakker HHC, Rapley BI, Summers SR, Alves-Pereira M, Dickinson PJ (2017). An affordable recording instrument for the acoustical characterisation of human environments. ICBEN 2017, Zurich, Switzerland, No. 3654, 12 pages.

[7] Cooper S (2014). The Results of an Acoustic Testing Program Cape Bridgewater Wind Farm. Prepared for Energy Pacific (Vic) Pty Ltd, Melbourne, Australia. http://www.pacifichydro.com.au/files/2015/01/Cape-Bridgewater-Acoustic-Report.pdf

[8] Alves-Pereira M (1999). Noise-induced extra aural pathology. A review and commentary. Aviation, Space and Environmental Medicine, 70 (3, Suppl.): A7-A21.

[9] Torres R, Tirado G, Roman A, Ramirez R, Colon H, Araujo A, Pais F, Lopo Tuna JMC, Castelo Branco MSNAA, Alves-Pereira M, Castelo Branco NAA (2001). Vibroacoustic disease induced by long-term exposure to sonic booms. Internoise2001, The Hague, Holland, 2001: 1095-98. (ISBN: 9080655422)

[10] Araujo A, Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA (2004). Vibroacoustic disease in a ten-year-old male. Internoise2004. Prague, Czech Republic, 2004; No. 634, 7 pages. (ISBN: 80-01-03055-5)

[11] Alves-Pereira M, Castelo Branco, NAA (2007). In-home wind turbine noise is conducive to vibroacoustic disease. Second International Meeting on Wind Turbine Noise, Lyon, France, Sep 20-21, Paper No. 3, 11 pages.

[12] Castelo Branco NAA, Costa e Curto T, Mendes Jorge L, Cavaco Faísca J, Amaral Dias L, Oliveira P, Martins dos Santos J, Alves-Pereira M (2010). Family with wind turbines in close proximity to home: follow-up of the case presented in 2007. 14th International Meeting on Low Frequency Noise, Vibration and Its Control. Aalborg, Denmark, 9-11 June, 2010, 31-40.

[13] Lian J, Wang X, Zhang W, Ma B, Liu D (2017). Multi-source generation mechanisms for low frequency noise induced by flood discharge and energy dissipation from a high dam with a ski-jump type spillway. International Journal of Environmental Research and Public Health, 14 (12): 1482.

[14] Rapley BI, Bakker HHC, Alves-Pereira M, Summers SR (2017). Case Report: Cross-sensitisation to infrasound and low frequency noise. ICBEN 2017, Zurich, Switzerland (Paper No. 3872).

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AN EVALUATION OF ENVIRONMENTAL, BIOLOGICAL, AND HEALTH DATA FROM THE ISLAND OF VIEQUES, PUERTO RICO

MARCH 19, 2013

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
Atlanta, Georgia 30333



AN EVALUATION OF ENVIRONMENTAL, BIOLOGICAL, AND HEALTH DATA FROM THE ISLAND OF VIEQUES, PUERTO RICO

Prepared By:

U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry (ATSDR)
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Eastern Branch

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This document is presented in English and Spanish for the convenience of the reader. We have made every effort to ensure the translation is accurate. Readers should be aware, however, that the English version is the official version of the text.

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Executive Summary

Scope of Review

ATSDR recognizes that Viequenses[1] are concerned about the health of everyone who lives on their island. ATSDR further recognizes that Viequenses are concerned about whether past military exercises[2] on Vieques might have contributed to health conditions some on the island have reported.

ATSDR wants to be responsive to those concerns. We have worked to ensure that this analysis of Viequense environmental data is thorough; that it considers all readily available investigations and research, especially research completed since release of our 2001–2003 public health assessments (PHA).[3] Simply stated, this report's nine chapters endeavor 1) to assess critically all of the available exposure and health information relevant to Viequense public health issues, 2) to draw conclusions—albeit often with some degree of uncertainty—and 3) to make recommendations for environmental and public health agencies as well as for scientific researchers that will assist in reducing that uncertainty.

The circumstances on Vieques typify many of the difficulties faced by the public and by public health officials when responding to concerns about the effects of hazardous substances. Although numerous questions arise regarding exposures and people's health, the means are limited to answer such questions definitively. When reaching conclusions about hazardous waste and public health, some degree of uncertainty will always remain. Thus in this evaluation, ATSDR identifies the available data as well as the data gaps.

This report's principal focus is to review and update environmental data on Vieques air, water, soil, seafood, and locally grown foods. In addition, this report adds human biomonitoring and health outcome data. It begins with a review and update of ATSDR's 2003 Fish PHA data on consumption of fish caught off the Vieques coast. Next are two chapters on human biomonitoring data and health outcome data. These are the chapters whose subject matter was not included in previous ATSDR reports on Vieques. At the end of each chapter, we provide conclusions, recommendations, and references specific to that chapter. Four chapters are dedicated to examination of exposure pathways from food, air, soil, and water. Chapter 9 summarizes all the conclusions and recommendations from each chapter.

The public health question in the chapters on food, air, soil, and water is whether residents were or are exposed to bombing-related contaminants and whether there were or are any public health consequences. The chapter on seafood consumption focused on mercury in seafood because Puerto

[1] Throughout this document, the Spanish noun form "Viequenses" refers to the residents of Vieques, and the adjectival form "Viequense" refers to Viequenses and Vieques.

[2] The term "military exercises" subsumes all forms of naval, Marine, and other military service operations—including aerial bomb and naval gunfire practice—on and in the vicinity of Vieques.

[3] A public health assessment (PHA) is an ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health.

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Rican scientists and Viequenses raised that as a concern. Mercury in seafood is not from bombing-related activities.

The public health question addressed in the biomonitoring and health outcome chapters is different. We could not use these data to assess whether bombing-related contaminants are present in Viequenses or are causing morbidity or mortality. The purpose of the biomonitoring chapter is to identify whether excessive exposure to metals is occurring in Vieques residents and whether there is a risk of harmful effects from metal body burdens. The purpose of the health outcome chapter is to assess the overall health status of the Viequenses population. Finally, the report includes four appendices. Appendix A summarizes the studies reviewed previously by ATSDR for this report. Appendix B contains summaries of ATSDR's previous documents evaluating Vieques-related environmental data, as well as summaries of two ATSDR-funded panel reviews: heart echocardiograms and elemental hair analysis. Appendix C contains the peer review comments received and ATSDR responses to those comments. Appendix D contains ATSDR's responses to comments following the public release of this report.

Background

Isla de Vieques, or Vieques Island, is a 55-square mile tract of land 7 miles off the Commonwealth of Puerto Rico's east coast. Approximately 10,000 persons live in the central 7,000 acres or about 20% of the island, mostly in the towns of Isabel Segunda and Esperanza.

In 1941, as the United States entered World War II, the U.S. Navy began to acquire Vieques property by condemnation of private lands. By 1950, the Navy owned the island's entire eastern and western portions. With its wide beaches, shallow approaches, and warm water temperatures, as well as its distance from commercial air and shipping lanes, the Navy believed that Vieques was not only ideal for naval and Marine warfare training, it was one of the few locations in the Western Hemisphere that met all the Navy's requirements.

The Navy's Atlantic Fleet Weapons Training Facility (AFWTF) established its so-called Inner Range on Vieques, comprising the Eastern Maneuver Area (EMA), and the Live Impact Area (LIA) (see Figure 1-2). Marines conducted live-fire exercises in the EMA. Aerial explosive-ordnance and naval gunfire practice were limited to the 900-acre LIA, on the island's easternmost end. Camp García,[4] the principal Marine Corps encampment on Vieques, was also within the EMA's southern section.

For decades—particularly after the mid-1970s—ships and aircraft fired, launched, and dropped live bullets, artillery rounds, rockets, missiles, and bombs into the LIA. In the years following World War II, Viequenses, other Puerto Ricans, scientists, and activists increasingly opposed the Navy's activities on Vieques, contending that those activities deprived many residents of their livelihood (primarily fishing), exposed them to injury and, because of chemicals in the explosives, to long-term illness.

Opposition increased after April 1999, when an F-18 pilot mistook an observation tower outside the LIA for a target. The exploding bomb killed the civilian observer inside the tower and prompted protestors

[4] Named for Puerto Rican native and Marine PFC Fernando García, a Korean War Medal of Honor recipient.

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to occupy the LIA. All live-fire exercises ceased. After protracted negotiations between the federal government and the Commonwealth of Puerto Rico, in early 2003 then-President George W. Bush ordered cessation of all military activities on Vieques. Former Navy properties on the eastern and western ends of the island, now mostly under U.S. Fish and Wildlife Service (FWS) jurisdiction, are national wildlife refuges. In August 2005, the U.S. Environmental Protection Agency (USEPA) added sites in the former AFWTF Inner Range to the National Priorities List, making the area subject to federally supervised remediation, which continues today.

Site investigations and environmental assessments began while the Navy still operated on Vieques. After the Navy's departure, a thorough, in-depth evaluation of the current and long-term human health risks posed by exploded and unexploded ordnance was initiated.[5] The Navy also looked at the risks military exercises generally might pose in Viequense air, water, biota, and in Viequenses themselves. The latest of these evaluations is ATSDR's review of environmental data that began in 2009. This report describes and discusses the results of our review. In addition, ATSDR examines some biomonitoring studies and health outcome data not available in our previous investigations.

Previous Site Investigations and Assessments

From 2001 to 2003, ATSDR released four Vieques-related PHAs. To investigate possible exposure to chemicals emanating from exploded and unexploded ordnance, each PHA assessed a specific exposure pathway: seafood (e.g., fish, shellfish, and land crabs), drinking water, air, and soil. After analyzing data and after extensive modeling, each PHA concluded that with one specific exception involving a single local well, contaminants in the evaluated pathways were not at levels expected to cause health effects. But these PHAs did not evaluate the risk of physical injury from unexploded ordnance or consider cumulative effects of exposure to multiple contaminants through multiple pathways. Unfortunately, current science does not adequately support a robust analysis of multiple chemical exposures and their interactions. Debate continues in the scientific community about how best to evaluate exposure to a chemical mixture both from a single pathway and from multiple, combined pathways. In addition, estimating combined doses from multiple pathways on Vieques is hampered by a lack of knowledge of the levels of chemicals residents are exposed to through various pathways (e.g., eating seafood, ingesting soil, drinking water, and breathing air).

Viequenses and other Puerto Ricans—together with some scientists, elected officials, and citizen groups—disagreed with ATSDR's findings. They believed the Navy's decades-long island presence left behind residual environmental hazards that directly and indirectly affected Viequenses' health. Scientists in Puerto Rico, for example, produced heart and hair studies that challenged ATSDR's findings (see Chapters 3 and 4). For this current site review, ATSDR not only revisited available data used in the previous ATSDR reports—and revisited Vieques itself—but also identified new environmental,

[5] Throughout this document, "exploded or unexploded ordnance" refers to naval gunfire projectiles, aerial bombs, and projectiles from other weapons fired, launched, or dropped primarily into the Vieques Live Impact Area (LIA).

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biomonitoring, and health outcome data.[6] In November of 2009, ATSDR hosted a meeting in Atlanta in which interested scientists discussed available scientific data concerning Vieques. The scientists suggested how ATSDR could look at the data in additional ways. For example, the group suggested that ATSDR reconsider exposure scenarios for fish consumption. They also expressed concern about the lack of information about potential exposure to contaminants through consumption of locally grown produce. Discussions such as these resulted in some new analyses for this report. A summary of the November 2009 meeting is available at http://www.atsdr.cdc.gov/sites/vieques/notes_vsc_toc.html.

This Report's Conclusions and Recommendations

As part of this evaluation, ATSDR reviewed many of its previous conclusions and recommendations and identified some new findings. Again, Chapter 9 contains all of this report's conclusions and recommendations according to the topics described in each chapter. Certain conclusions and recommendations are highlighted in this Executive Summary.

Conclusions and Recommendations from Environmental Data

Consumption of Fish from Reefs off the Vieques Coast

- ATSDR has identified mercury exposure from frequent consumption of marine seafood as a potential public health hazard. Mercury is present in most seafood; it is particularly high in some fish species and low in other species. Due to mercury, children born to women who eat fish daily from waters surrounding Vieques are at increased risk of language, attention, and memory deficits, and to a lesser extent visual/spatial and motor function deficits. Children who frequently eat fish from waters surrounding Vieques are also at risk of similar harmful effects. That said, we could find no relationship between mercury in fish and military operations on Vieques. A more plausible explanation for the mercury levels found in fish is that they resulted from the global reservoir of mercury circulating through the environment.
- Due to the many nutritional benefits, women and young children in particular should include in their diets fish or shellfish that are low in mercury. Fish and shellfish are a part of a healthy diet. They contain high-quality protein, omega-3 fatty acids, other essential nutrients, and are low in saturated fat. A well-balanced diet that includes a variety of fish and shellfish can contribute to heart health and children's proper growth and development.
- Statistical analysis showed that some fish and shellfish from certain reefs surrounding Vieques had higher levels of some metals and lower levels of other metals—iron, aluminum, copper, zinc, arsenic, barium, potassium and selenium were all slightly higher—compared with other reefs surrounding Vieques. These metals are materials found in bombs and in metal ships, suggesting possible localized contamination. But the levels were only slightly higher and the difference was statistically significant only for some reefs compared with other reefs

[6] In this regard, note that the Navy is still involved in remedial activities on Vieques and these activities should be conducted in a way that ensures public health is protected from contaminant exposure.

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surrounding Vieques. ATSDR recommends the following for consideration by environmental and public health agencies and scientists:

- People who frequently consume marine seafood should follow available fish advisories and fishing restrictions in Vieques. Maintain the fishing restrictions in the waters adjacent to the LIA.
- Conduct a survey of Vieques residents to determine the types, frequency, and quantity of fish consumed.
- Conduct additional risk assessments and statistical analyses using new information gathered from the previously recommended fish consumption survey.
- Should the proposed survey and statistical analysis not provide sufficient public health information, collect and analyze additional fish samples from Vieques.
- Collect sufficient fish samples to allow analysis by species and by location.

Biomonitoring

- The data from biomonitoring studies in Vieques showed elevated levels of some metals in residents' blood, urine, hair, or feces. While cigarette use, seafood consumption, or hair dyes might explain some elevated levels in these studies, the Puerto Rico Department of Health (PRDOH) manuscript reported they do not explain all. Because the source of these metals could not be identified, the biomonitoring results do not permit any conclusions about whether these elevated levels resulted from exposure to military exercise-related contaminants.
- Viequenses may be exposed to mercury in fish and cadmium in pigeon peas. These exposures may warrant additional environmental investigations, such as sampling locally grown produce for cadmium and gathering more information about fish consumption and possibly mercury in fish. The information could be used to decide whether to undertake human testing for mercury and cadmium in blood or urine. If other environmental exposures are identified, additional human biomonitoring investigations may be considered. More detailed information about ATSDR's recommendations concerning fish and locally grown produce can be found in Chapter 2, Section 2.3.2 and Chapter 5, Section 5.3.2, respectively.
- ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time. We found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation.

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- Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing. A qualified laboratory should do the testing and analysis.
- CDC/ATSDR can provide a list of qualified laboratories that can perform the tests. And if requested, CDC/ATSDR can provide information to healthcare providers about tests for metals in biologic samples.

Health Outcome Data

- Elevated morbidity and mortality in Vieques, coupled with problems accessing health care, paint a complex health picture for Viequenses.
- Data indicate elevations in chronic disease prevalence, cancer incidence, and cancer mortality among the Viequense population relative to the rest of Puerto Rico. The limitations associated with these analyses, particularly the methodological concerns discussed in this report, introduce considerable uncertainty and make interpretation difficult.

Local Produce and Livestock Pathway

ATSDR evaluated the available data and information on contaminants detected in locally raised garden produce and in livestock.

- The overall data are insufficient to quantify human exposures or draw health conclusions. Limited sampling data suggest cadmium toxicity may be a concern for excessive consumption of pigeon peas, but not for typical consumption rates of pigeon peas.
- A preliminary data evaluation completed for this report has concluded that the level of cadmium reported in a few samples of locally grown pigeon peas would not contribute excess dietary cadmium to preschool children who eat less than five of the largest (6 ounces) servings per week of locally grown pigeon peas. Adults who eat the largest serving sizes (12 ounces) should limit intake to 11 servings per week. Typical serving sizes for preschool children (1.5 ounces) do not contribute excess cadmium below 20 meals per week and adults who eat a typical serving (3

ATSDR Recommends

- Developing an educational program about mercury in fish that incorporates local habits and information about Viequenses' seafood consumption.
- Maintaining fishing restrictions near the former bombing range.
- Viequenses talk to their healthcare providers about the need for biologic testing.
- Additional sampling of locally grown foods to better evaluate this exposure pathway.
- Sampling surface soil in the island's residential areas to address uncertainties regarding residential soil contamination issues.
- Clean-up activities continue in the LIA and other former military exercise areas to prevent human exposures to harmful contaminants.

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ounces) may eat up to 44 meals per week without exceeding recommended cadmium intake levels.

- The significant uncertainty in the evaluation of cadmium in pigeon peas stresses the need for further sampling. Preliminary evaluation suggests a potential for uptake of metals from soil into food crops. These results warrant further investigation.

Air

- We reviewed the data on airborne contaminants from military exercises at the former Vieques Naval Training Facility. This review confirmed our previous findings and indicated that airborne contaminants from past military operations were very unlikely to have had health effects on Viequenses.

Soil

- Sufficient data are available to conclude that people who lived on the LIA during the 1999–2000 protests were not exposed to soil contaminants at levels high enough to cause adverse health effects.
- Recent data, and the presence of unexploded ordnance at the LIA, support the need for continued, restricted access to the LIA and to other potentially contaminated former military exercise areas. Environmental assessment and remediation activities should continue.
- In the island's residential areas, no soil data are adequate to characterize potential exposures fully. To address remaining uncertainties about residential soil contamination issues, ATSDR recommends surface-soil sampling in the island's residential areas.

Drinking Water

- With the possible exception of one private well found to contain harmful nitrate-nitrite levels, all drinking water supplies in Vieques are acceptable for their current uses. ATSDR recommends no one drink from the one private well until further testing confirms its water is safe.
- Ongoing monitoring of the current pipeline-source water is required to ensure the supply meets drinking water standards. Repeating previous sampling of storage tanks, residential taps, and wells still in use would address any remaining uncertainty.

Peer Review and Public Comment

Scientists from the Commonwealth of Puerto Rico and from the United States peer-reviewed the Vieques Summary Report before we released a public comment draft. Appendix C of this report contains peer comments and ATSDR's responses. Members of the public had an opportunity to review our public comment draft for a 90-day period between December 8, 2011 and March 11, 2012. ATSDR reviewed all public comments and has revised the text of this report as appropriate. We have prepared responses to each public comment received. The comments and responses are in Appendix D of this report.

Chapter 1 Introduction

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1.1 Island Overview



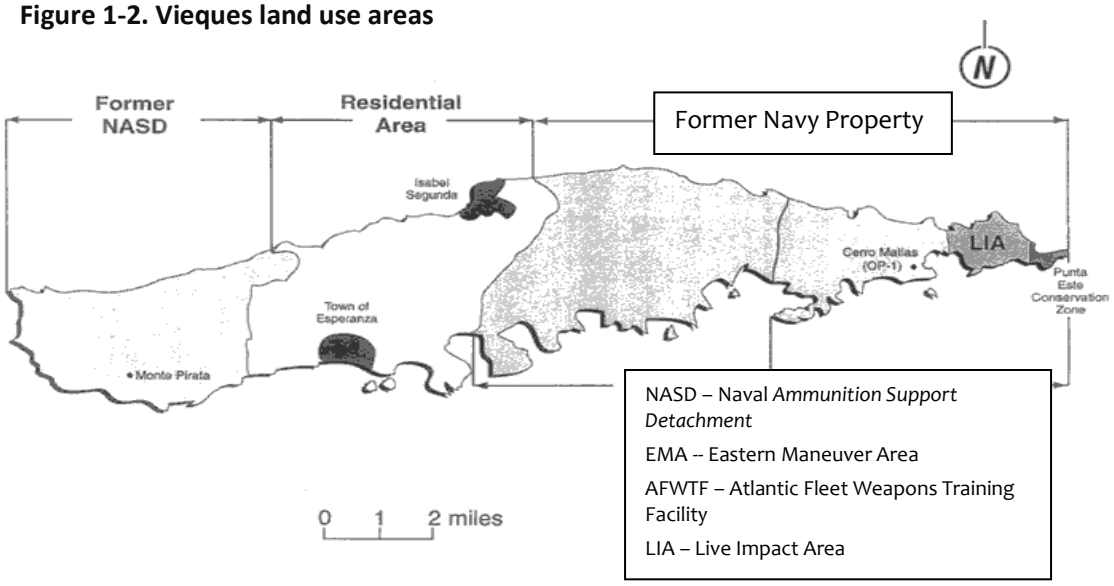
Figure 1-1. Puerto Rico and Vieques Island

Vieques is the Commonwealth of Puerto Rico's largest offshore island (see Figure 1-1). Twenty miles long and 4.5 miles wide at its widest point, Vieques comprises some 33,000 acres, or 51 square miles. Puerto Rico, the easternmost island in the Greater Antilles chain, is approximately 7 miles west of Vieques. The U.S. Virgin Islands, including St. Thomas, St. John, and St. Croix, are 20 miles or more northeast and southeast. Drinking water for the island is supplied by pipeline from the main island of Puerto Rico—Vieques groundwater is not used for drinking purposes. Figure 1-3 contains a topographic map showing groundwater flow on the island.

1.2 Vieques and the U.S. Navy

From 1941 until 2003, the U.S. Navy owned much of Vieques; residents were largely confined to areas slightly west of the of the island's center (see Figure 1-2). On the island's eastern half, the Navy's Atlantic Fleet Weapons Training Facility (AFWTF) administered the Eastern Maneuver Area (EMA) and, at the island's easternmost end, the Live Impact Area (LIA). Major training was generally restricted to two 90-day periods each year. Operating from Camp García in the EMA's southern section, U.S. Marines rehearsed amphibious assaults on Vieques beaches, maneuvered inland with armored and other vehicles, and generally worked with naval task forces to ensure combat-readiness before embarking on major deployments.

Figure 1-2. Vieques land use areas



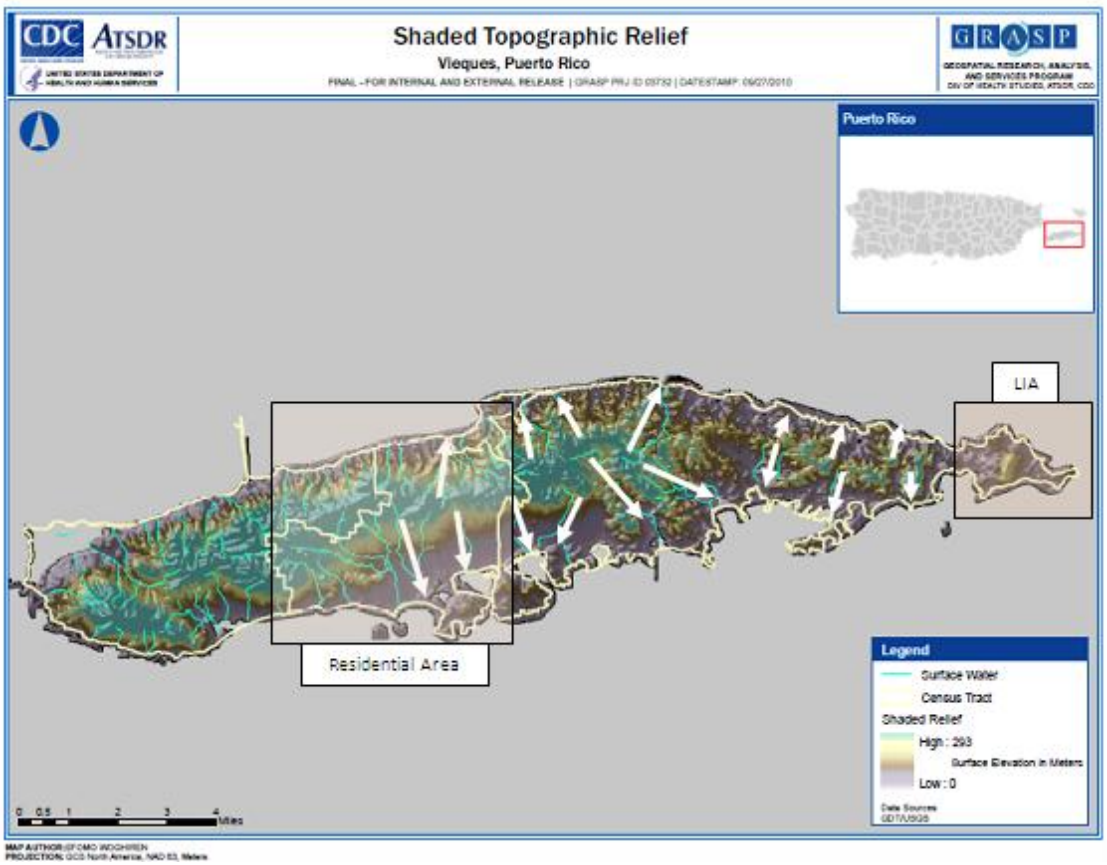
Over and off the Vieques east coast, the Navy used the LIA for naval gunfire training. Arrows in Figure 1-3 below show directions of surface water, stream, and shallow groundwater for air-to-ground, explosive-ordnance exercises (CH2MHILL and Baker 1999). After the Culebra Island range closed in the

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mid-1970s (see Figure 1-1), air-to ground and ship-to-shore training activities increased in the LIA. Major exercises were usually in the spring and fall, but smaller exercises occurred throughout the year (IT 2000). Unexploded ordnance (UXO), that is, bombs or explosive ordnance and other waste military munitions, were treated and detonated in the LIA. Today, when UXO are identified, they are transported to an authorized area and detonated with a remote control charge. UXO that cannot be transported safely to the open burning/open detonation area are detonated onsite in accordance with prescribed procedures.

Figure 1-3. Topographic map of Vieques showing directions of water flow.

Arrows show directions of surface water, stream, and shallow groundwater flow. Waterborne contaminants cannot flow from LIA to residential area.



The Navy is still involved in remedial activities on Vieques. These activities should be conducted in a way that ensures public health is protected from exposure to contaminants. In 2001, the Navy transferred ownership of approximately 7,500 acres of land on the west end of the island to the municipality of Vieques, the Puerto Rico Conservation Trust, and the U.S. Fish and Wildlife Service (FWS) but retained about 100 acres of the former Naval Ammunition Support Detachment (NASD) lands for radar and communication facilities (US Navy 2001). Some NASD areas were leased to local farmers for cattle grazing and other agricultural purposes (USEPA 2004). On May 1, 2003, after civilian protests and negotiations between the federal and Puerto Rican governments, all military operations ceased on and around the island. The Navy transferred its remaining 14,500-acre property on the eastern end of the

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island to the FWS (USEPA 2004). Lands on the eastern and western ends of the island are now a national wildlife refuge. The Navy has initiated site investigations and cleanup actions pursuant to the Resource Conservation and Recovery Act (RCRA) and the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), sometimes referred to as the Superfund Act.

1.3 Chemicals in Munitions and Detonation Byproducts

During the peer review process for this report, several peer reviewers pointed out that the report failed to discuss any link between historical, military activity-related contaminants that might have entered the Viequense environment and any resultant human exposure from those contaminants. Previous PHAs looked at this issue; their findings are summarized in the following statements.

- ATSDR's Water PHA acknowledged that very low levels of RDX, tetryl, ammonia, and nitrate plus nitrite might have been present in drinking water samples taken by the Navy in 1978, but also expressed doubts about the validity of these data. Additionally, water samples from the Esperanza aquifer contained metals, high levels of total dissolved solids (TDS), and high salt. Two of the metals found in the Sun Bay wells (iron and manganese) and the TDS in all wells were above their secondary maximum contaminant levels (SMCL) established by U.S.EPA as a national secondary drinking water regulation. SMCLs are established only as guidelines to assist public water systems in managing their drinking water for aesthetic considerations, such as taste, color, and odor. TDS—including iron, manganese, and sodium—are commonly found in groundwater. The presence of these metals was considered directly related to the high levels of TDS in the water and probably reflected the natural geology of the island, given that the igneous and volcanic rocks that make up Vieques bedrock are a common source of iron and manganese (ATSDR 2001).
- ATSDR's Soil PHA concluded that Navy activities appeared to influence LIA soils. The concentrations of heavy metals found at the LIA were 1.4 to 2.9 times higher than background concentrations collected from the west end of the island. Current soil data from the residential area of Vieques were not available. Therefore, ATSDR conducted a very conservative health evaluation that included concentrations of metals detected in areas where access was restricted (i.e., land previously owned by the Navy, including the LIA) (ATSDR 2003a).
- ATSDR's Air PHA stated the Navy's past military exercises at Vieques released contaminants to the air, including dusts, chemical byproducts of explosions, and metals. Using particulate ambient air monitoring data from the residential areas of the island, the PHA concluded that wind-blown dust from the LIA was not a health hazard on days without bombing exercises and that the Navy's past military training exercises with practice bombs did not pose a health hazard. The modeling analysis of the Air PHA predicted that chemicals emitted from live bombing exercises dispersed to extremely low levels over the 7.9 miles that separate the emissions source (the LIA) and the residential area of Vieques. For a majority of the contaminants released, the estimated concentrations in the residential areas are so low that even highly sensitive air sampling devices would likely not be able to measure them (ATSDR 2003b).

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- As a part of ATSDR's Fish and Shellfish PHA, fish and shellfish were analyzed for explosive compounds and heavy metals. Some evidence showed that explosive compounds might have contaminated the marine environment. HMX and a chemical similar to RDX[7] were detected in fiddler crabs collected on the LIA. Because the fiddler crabs had not been rinsed before sampling, whether the explosive residues were present in the fiddler crabs or were due to external sand and dirt contamination was unclear. One trunkfish from the fish market was found to contain trace amounts of a chemical similar to RDX.[7] Explosive compounds were not detected in any of the other 142 edible fish or shellfish samples. Several heavy metals were detected in the fish and shellfish (ATSDR 2003c).
- Statistical analysis showed that some fish and shellfish from certain reefs surrounding Vieques had higher levels of some metals and lower levels of other metals—iron, aluminum, copper, zinc, arsenic, barium, potassium and selenium were all slightly higher—compared with other reefs surrounding Vieques. These metals are materials found in bombs and in metal ships, suggesting possible localized contamination. But the levels were only slightly higher and the difference was statistically significant only for some reefs compared with other reefs surrounding Vieques.

ATSDR evaluated the known environmental data associated with military activities. Our evaluation showed that residents living in the central portion of the island did not have direct contact with LIA soils at levels that could harm their health. Nor were residents likely to be exposed to contaminants in air at levels that could harm their health. While explosive compounds were found in a few sampled marine animals, residents did not consume these animals. Therefore, the available data indicate that no exposure occurred at levels that would harm health.

The subsections that follow briefly describe 1) the organic and inorganic compounds and elements present in munitions and detonations, 2) what is known about these constituents in the Vieques environment, and 3) whether these constituents can be linked to human exposure on the island.

1.3.1 Organic Compounds in Munitions and Detonation Byproducts

In Vieques' eastern region, various branches of the U.S. military used many different ordnance types (e.g., firebombs, parachute flares, rockets, inert rockets, machine guns, practice bombs, and live explosives) (Young 1978). Organic compounds are typically found in the explosive charge of the military ordnance, and not in the casings. The explosive components of bombs varied over the years, but usually included some combination of

- 2,4,6-trinitrotoluene (TNT),
- Cyclotrimethylene trinitramine (RDX),
- Methyl-2,4,6-trinitrophenylnitramine (tetryl),
- Cyclotetramethylene tetranitramine (HMX), or

[7] The laboratory that conducted the analyses could not confirm a conclusive identification.

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- Ammonium picrate (explosive D).

When ordnance detonates, the organic explosives are rapidly consumed and release large amounts of energy. In the process of detonation, the organic explosives are largely—but not completely—destroyed. Thus, they form other compounds. The scientific understanding of detonation byproducts of explosions has advanced over the years. For example, some early Navy publications (Young 1978) listed various detonation byproducts anticipated for the types of organic explosives used at Vieques. These estimates included the following compounds and compositions: carbon dioxide (35%), nitrogen (27%), carbon monoxide (16%), water (8%), ethane (5%), carbon (6%), propane (2%), plus several minor by products (e.g., ammonia, hydrogen, hydrogen cyanide, methane, methanol, formaldehyde) formed in trace quantities (<1%). These estimates are based on theoretical calculations, not on field-testing of actual detonations.

Scientists have since developed more sophisticated and rigorous testing procedures for identifying and measuring detonation byproducts. "BangBox" studies (e.g., US Army 1992a, 1992b, 1992c) have been particularly useful for directly measuring organic chemicals released following detonations. The BangBox refers to a flexible structure in which ordnance is detonated. Because the BangBox is completely enclosed, byproducts formed during the detonation do not escape the structure. Air sampling equipment can measure them. Scientists have used the BangBox to estimate emission factors for various types of ordnance, many of which are similar or identical to those used at Vieques. The BangBox studies have identified more than 50 organic compounds expected to be emitted to the air in trace quantities as detonation byproducts. In addition to the byproducts listed in the previous paragraph, the BangBox studies found the following byproducts in greatest quantities (but all were less than 0.1% of the total emissions): sulfur dioxide, benzene, naphthalene, acetylene, di-butyl phthalate, and various aromatic compounds. The modeling analysis in the Air PHA considered emission rates for every organic byproduct identified in the BangBox studies for the types of ordnance most similar to those the Navy used at Vieques.

1.3.2 Inorganic Compounds in Munitions and Detonation Byproducts

Inorganic compounds in munitions, including metals, are found in the metal bomb casings and sometimes in the explosive charge. Both sources of inorganic compounds (primarily metals) are reviewed here.

ATSDR's Air PHA characterized the composition of ordnance dropped onto the LIA and emitted into the air because of the training exercises. The most abundant elements in bomb casings were iron (93%), aluminum (5%), copper (2%), manganese (2%), and zinc (0.5%). The percentages shown total more than 100%—not uncommon when expressing metal content in alloy mixtures (i.e., an upper bound concentration is often used when characterizing the composition of individual constituents). Numerous other metals were present in bomb casings at concentrations less than 0.02%, including boron, chromium, molybdenum, nickel, and titanium (ATSDR 2003b). The predominant metal in explosive charges was aluminum, which accounted for as much as 21% of the explosive charge in some live bombs (ATSDR 2003b).

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The emission factors for the BangBox studies identified the following five metals as having the highest emissions—in decreasing order: copper, zinc, aluminum, calcium, and lead. Eleven other metals were detected in these studies, but at lower levels. It should be noted that these studies did not measure concentrations of certain metals (e.g., iron, manganese) found in highest concentrations in the bomb casings (ATSDR 2003b).

Given the previous review, iron, aluminum, copper, manganese, zinc, and lead are the metals most likely to be elevated in LIA soils from military activity, though other metals were also found in bombs. It should be noted that all of these metals are also naturally present in the soils and rocks of Vieques (ATSDR 2003a, Learned 1973, USGS 1997 and 2001).

Because mercury was historically used in certain detonators, mercury has been mentioned as a contaminant of concern (García et. al. 2000). Bomb casing composition data originally provided by the Navy indicated that casings did not contain mercury, which is consistent with information on Material Safety Data Sheets available for steel. The Air PHA indicated that the total annual estimated mercury emissions from high explosive ordnance used at the LIA were very low (i.e., less than 0.5 kg per year). Following detonation, the mercury was presumably a trace constituent in the explosive charge and in the soil ejected into the air. This estimated mercury emission of less than 0.5 kg per year can be compared with the mercury National Emission Standard for Hazardous Air Pollutants (NESHAP) developed by the U.S. EPA. The mercury NESHAP air emission rate for a chlor-alkali plant is 844 kg per year. More information is available at: http://www.gpo.gov/fdsys/pkg/CFR-2011-title40-vol8/xml/CFR-2011-title40-vol8-part61.xml#seqnum61.52 . Therefore, the live ordnance dropped on the LIA does not appear to be a significant mercury source in the Vieques environment.

1.3.3 What Bomb-related Constituents Were Found in Vieques

Vieques has seen numerous environmental sampling efforts over the past few decades. This section reviews the evidence for ordnance-related constituents in the local environment. Information is first presented for explosive compounds, then for metals.

What follows is a summary of explosive compounds found in various media from Vieques. Because explosive compounds do not occur naturally, the presence of these contaminants in environmental media point to the past military activities as the most likely source.

- In 2000, surface soil samples collected from the LIA showed rare detections of HMX, RDX, 2-amino-DNT, and TNT (CH2MHIll 2000).
- In 2003, seawater samples from inside and near a 2000-lb bomb in Bahia del Sur showed explosive compounds, but explosives were not detected in samples near the sunken target vessel, the former USS Killen (Barton and Porter 2004).
- In 2003, sediment samples collected from within 2 meters of a 2000-lb bomb in Bahia Salina del Sur showed TNT. Farther away, the concentration declined to nondetectable. Sediment samples collected from the vicinity of the former USS Killen contained no detectable explosive residues (Barton and Porter 2004).

- In 2001, fish and shellfish were collected and their tissues analyzed for explosive compounds. No explosive residues were detected in fish tissues from any species or sample location, except for trunkfish. One trunkfish from the fish market was found to contain trace amounts of a chemical similar to RDX. Of the four shellfish species sampled, only fiddler crabs were shown to contain the explosive compound HMX. A chemical similar to RDX[8] was also detected in fiddler crabs, but the level was so low that an accurate determination could not be made. Because the fiddler crabs had not been rinsed prior to sampling, it was unclear whether the explosive residues were present in the crabs or were due to external sand and dirt contamination (ATSDR 2003c).
- In 2003, other marine organisms (one damselfish, one feather duster worm, and one sea urchin) collected near a 2000-lb bomb in Bahia Salina del Sur showed explosive residues. 1,3,5-trinitrobenzene was detected in a damselfish sample; TNT was found in a feather duster worm and in a sea urchin. Explosive residues were not detected in fish and lobster samples collected near the former USS Killen. One coral sample from the USS Killen area contained detectable TNT residues (Barton and Porter 2004).

The previous results show that small amounts of explosive compounds remain in the Vieques terrestrial and marine environments. While explosives have been detected in or on some organisms (e.g., feather duster worm, sea urchin, fiddler crabs), nearly every fish tissue sample to date has failed to contain detectable levels of explosives, with the only exception being a single damselfish tissue sample—a species not typically consumed by people—and possibly one trunkfish sample.

The remainder of this section evaluates the presence of munitions-related metals in the Vieques environment. Metals were clearly present in the bombs the Navy dropped on the island and into the nearby ocean. The metals that landed in the Vieques environment might change chemical form over time, but the metals themselves will not decay or decompose. Nevertheless, because many metals occur naturally in soil and sediment, distinguishing is often difficult between naturally occurring concentrations and concentrations that represent human-activity contamination. The text that follows considers whether metals in the LIA soils have been found at levels believed above background.

In 2006, the Navy's contractor (CH2MHILL) surveyed soil inorganics in east Vieques (CH2MHILL 2007). The survey's goal was to establish background levels within the naval training areas that could be used to determine whether other soil samples from suspected contaminated areas exceeded background levels. In cases where sampling results exceed background ranges, a logical inference is that the elevated measurements reflect contributions from past military activities or some other manufactured source. For each metal and element in the report, an upper tolerance limit (UTL) was developed for the various soil types in east Vieques. The UTL is a statistically derived value, with the exact derivation depending on the shape of the distribution of the sampling results (e.g., normal versus lognormal). The inference to be drawn from the UTLs is that measured soil concentrations below these values are indistinguishable from background concentrations. Not surprisingly, the UTLs were highest for the

[8] The laboratory that conducted the analyses could not confirm a conclusive identification.

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predominant elements in soil, including aluminum (35,000 ppm for all soil types), iron (38,100–43,200 ppm, depending on soil type) and magnesium (3,710–22,200 ppm, depending on soil type).

Other elements in soil had UTLs with the following ranges depending on soil type (CH2MHIll 2007):

- Arsenic 1.6–9.2 ppm
- Beryllium 0.27–0.95 ppm
- Cadmium 2.2–2.4 ppm
- Chromium 70–72 ppm
- Cobalt 16–26 ppm
- Copper 53–94 ppm
- Lead 5.4–16 ppm
- Mercury 0.057–0.31 ppm
- Nickel 22–41ppm
- Vanadium 56–144 ppm
- Zinc 32 ppm for all soil types

To identify metals possibly associated with military exercises, ATSDR compared soil samples collected in 2000 at the LIA (CH2MHILL 2000) with the above-listed UTLs. For the following metals, at least 30% of the measured concentrations exceeded background levels in LIA soils, as characterized by the UTLs. The maximum soil concentration for each metal follows:

- Arsenic 20 ppm
- Beryllium 0.48 ppm
- Chromium 120 ppm
- Cobalt 32 ppm
- Iron 59,000 ppm
- Lead 33 ppm
- Vanadium 220 ppm
- Zinc 180 ppm

Mercury was also considered in this data comparison. The average mercury levels in LIA soils from the 2000 dataset was 0.02 ppm, and the highest level was 0.086 ppm (CH2MHILL 2000). Only one of 29 soil samples had a mercury level that could be considered above naturally occurring levels when compared with the background UTLs (0.057–0.31 ppm; CH2MHILL 2007).

In May and October 2007, the National Oceanic and Atmospheric Administration (NOAA) randomly collected 78 sediment samples and 35 coral samples from near-shore waters and a number of inland lagoons (sediments only) on Vieques (Bauer and Kendall 2010). The samples were grouped according to

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adjacent land use. A series of statistical tests were carried out to understand the distribution of the chemicals in the sediments. The concentrations of cadmium collected from sediments on and near the LIA were significantly higher than in sediments collected from the residential area of the island. The concentrations of cadmium were also found significantly higher in coral tissues than in sediments (Bauer and Kendall 2010). In addition, sediment concentrations of arsenic, copper, and chromium from the inland lagoons at the LIA were, on average, higher than sediment concentrations of these metals found in the other inland lagoons sampled elsewhere on Vieques.

In conclusion, soil or sediment concentrations of arsenic, beryllium, cadmium, chromium, cobalt, copper, iron, lead, vanadium, and zinc appear above naturally occurring levels in some areas in and around the LIA. The presence of these metals above background concentrations could reflect the influence of military activity, given the lack of other contamination sources at the LIA.

1.3.4 Linking Contaminants in Vieques with Human Exposure Pathways

ATSDR's soil PHA evaluated how elevated metals in LIA soils could affect Viequenses. Those living in the residential portion of the island do not have direct contact with LIA soils 7 to 8 miles away. Thus, exposures from any elevated metals in LIA soils cannot affect their health. Specific environmental sampling showed that Viequenses who camped on portions of the LIA in 2000–2001 were not exposed to metals (or other contaminants) in soil at levels that could harm their health. Similarly, the Air PHA concluded that metals in air from military activities could reach residential areas of the island, but not at levels that could harm people's health.

Some migration of contaminants from the soils to the marine environment undoubtedly occurs at Vieques, just as weathering of soils naturally occurs in virtually any shoreline setting. Precipitation and wind can carry soils from the LIA—and chemicals found in these soils—either directly into marine waters or into inland lagoons. And those lagoons have the potential to overflow into the marine environment. Environmental decay processes can gradually transform explosives at the LIA into other substances over time and thus reduce the potential migration of these chemicals into marine waters. But these chemical decay processes do not decompose the metals at the LIA. From an environmental health perspective, although migration of contaminants clearly occurs, the rate and amount of chemical migration is more important.

Military activities on Vieques have directly contaminated the marine environment with explosive compounds, such as HMX and RDX. Some of the ordnance previously fired at the LIA landed in the ocean rather than hitting onshore targets. Dr. James Porter provided direct evidence of this in photographs viewed during a meeting at ATSDR in November 2009. Some ordnance found in Vieques waters apparently included unexploded material. While explosive organic compounds were found in some marine species, these species are not consumed by humans; therefore, the available sampling data indicate that elevated human exposures are not occurring. Explosive compounds were not found in the 104 edible fish sampled in 2001 except for a chemical similar to RDX in one trunkfish collected from the fish market.

Two observations suggest that the LIA is not currently the primary source of mercury in fish tissues. First, most mercury levels in LIA soils appear to be at naturally occurring levels, with no significant

spatial variation to suggest that mercury levels are higher at the LIA. Second, ATSDR's fish sampling study did not find unusually elevated tissue concentrations of mercury in Vieques reef fish. A more plausible explanation for the mercury levels found in fish is that they resulted from the global reservoir of mercury circulating through the environment and not from mercury in some bombs and other ordnance.

In conclusion, ATSDR evaluated the known environmental data associated with military activities. Our evaluation shows that residents living in the central portion of the island did not have direct contact with LIA soils at levels that could harm their health. Nor were residents likely to be exposed to contaminants in air at levels that could harm their health. While explosive compounds were found in a few sampled marine animals, residents did not consume these animals. Therefore, the available data indicate that no exposure occurred at levels that would harm health. ATSDR has concluded in this report that some residents who frequently ate reef fish were exposed to mercury at levels that could harm a developing fetus (see Chapter 2). But this mercury appears associated with global mercury circulating in the environment and not with the small amount of mercury in some military ordnance.

1.4 The Vieques Civilian Population

The entire, approximately 10,000-member civilian population of the island—referred to here as Viequenses—resides in the island's central 7,000 acres, mostly in the towns of Isabel Segunda and Esperanza. The residential areas are about 7 miles from the LIA. The civilian areas comprise residences and agricultural, commercial, and industrial activities. In the past, sugarcane was the principal crop; other crops have included coconuts, grains, sweet potatoes, avocados, bananas, and papayas. Construction of a General Electric plant in 1969 spurred manufacturing employment in the 1970s and released 1,1,1 trichloroethane and small amounts of copper to outdoor air (Bermudez 1998, ATSDR 2003b). Currently, however, the island is home to only minimal manufacturing activity. Commercial fishing fleets are home-ported at Isabel Segunda and Esperanza. Tourism has recently increased in economic importance. The 2000 Census indicates that 65% of residents of Vieques live in poverty compared with 48% for all of Puerto Rico[9]. The 1990 Census results were similar, with 73% of the residents of Vieques living in poverty compared with 59% in all of Puerto Rico. According to the 2000 Census, about 19% of the population consists of women of childbearing age (15 to 44 years), and children 6 years and younger account for 11% of the population.

1.5 ATSDR's Evaluation of Vieques

The agency's evaluation of public health concerns on Vieques is the latest in a series of Vieques investigations. In 1999, a Vieques resident petitioned ATSDR for a determination of whether the Navy's use of live and inert ordnance resulted in health risks. From 2001 to 2003, ATSDR released four public health assessments (PHAs). Each PHA assessed a specific pathway for possible exposure to chemicals: seafood (e.g., fish, shellfish, and land crabs), drinking water, air, and soil. Each of these evaluations is available at http://www.atsdr.cdc.gov/sites/vieques/publications.html. ATSDR followed the public

[9] Using 2000 Census data, the poverty thresholds in 2000 were $17,603 for a family of four and $13,738 for a family of three (http://www.census.gov/hhes/www/poverty/data/incpovhlth/2000/briefing.html).

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health assessment procedures described in its Public Health Assessment Guidance Manual.[10] Scientific experts peer-reviewed each PHA before its final release. After analyzing data and modeling, each PHA concluded that with one specific exception, no health hazard was associated with any pathways evaluated. The exception: water from one local well was contaminated with nitrates and not fit to drink.

But some scientists from the University of Puerto Rico, the University of Georgia, and Yale University, as well as some Viequenses, disagreed with ATSDR's findings. Most scientists and residents believed the Navy's decades-long island presence left residual environmental hazards that affected public health on Vieques.

ATSDR has since 2009 gathered more than 75 documents relating to environmental sampling, health outcomes, and biomonitoring. Most of these documents were available when ATSDR released the 2001–2003 public health assessments, but some were completed later.

In August 2009, ATSDR scientists and staff visited Vieques and met with community leaders, Puerto Rican scientists, and health officials. From those meetings emerged a commitment to involve local experts in ATSDR's review of Vieques environmental data. ATSDR invited Puerto Rican scientists and others who had studied Vieques to meet in Atlanta, Georgia, on November 5–6, 2009. Participants engaged in a thorough review of multiple studies, identified the strengths and limitations of many of them, and made recommendations for further work. The group suggested that ATSDR reconsider its fish consumption exposure scenarios. The group also expressed its concern about potential exposure to contaminants through consumption of locally grown produce. Discussions such as these resulted in ATSDR performing new analyses for this report. A summary of that meeting is available at http://www.atsdr.cdc.gov/sites/vieques/notes_vsc_toc.html.

1.6 The Report

ATSDR prepared this report after review of all relevant, available data and related information and after the November meeting with invited scientists. We have divided the report into sections on environmental data (i.e., air, soil, drinking water, and food consumption), human biomonitoring data, and health outcome data. Conclusions and recommendations are made in each chapter and are summarized again in Chapter 9.

The circumstances on Vieques typify many of the difficulties faced by the public and by officials concerned about the effects of hazardous substances. Numerous questions arise regarding exposures and people's health, and at times, relatively few measurements are available to answer those questions directly. Environmental data are often limited in spatial coverage, number, or analytical quality control documentation. Consequently, some degree of uncertainty always remains. That means a key part of any review is to consider, before drawing public health conclusions, the adequacy of the available data. Note, however, that public health conclusions often can be drawn from limited data as long as uncertainties are recognized. ATSDR's conclusions can be reevaluated if new, improved information becomes available. Thus in this evaluation, ATSDR identifies the available data as well as the data gaps.

[10] Available at: http://www.atsdr.cdc.gov/HAC/PHAManual/index.html.

In contrast to environmental data, human biomonitoring can demonstrate how much of a chemical has entered the human body. Blood and urine levels of metals and organic chemicals can be useful in the comparative sense, where potentially exposed groups can be compared with nonexposed or lesser-exposed groups. This comparison assesses whether the target group has unusual or higher than expected exposure. Limitations to biomonitoring include the appropriate timing and collection of samples that will affect data interpretation. Also, in most situations, biomonitoring provides information about current or recent exposures; only in certain situations where a chemical may persist in the body (e.g., lead in bone) does biomonitoring provide information about exposures long past.

The November, 2009 Atlanta meeting resulted in ATSDR performing new analyses, as detailed in the chapters of this report. Appendix A includes short summaries of each report analyzed as a part of this evaluation. After careful examination of previously reported data, new data, and new science, ATSDR has arrived at the conclusions and recommendations contained at the end of each environmental data chapter, the health-outcome data chapter, the biomonitoring data chapters, and in Chapter 9.

Because outside peer review and the public comment period are now complete and because the document is now final, ATSDR hopes the report will guide the future work of environmental and public health agencies and scientists on Vieques.

1.7 References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003a. Public health assessment, soil pathway evaluation, Isla de Vieques bombing range. Atlanta: US Department of Health and Human Services; February.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003b. Public Health Assessment, Air Pathway Evaluation, Isla De Vieques Bombing Range Vieques, Puerto Rico. Atlanta: US Department of Health Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry; 2003 Aug 26.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003c. Public Health Assessment, Fish and Shellfish Evaluation, Isla De Vieques Bombing Range Vieques, Puerto Rico. Atlanta: US Department of Health Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry; 2003 June 27.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2001. Public health assessment, drinking water supplies and groundwater pathway evaluation, Isla de Vieques bombing range. Atlanta: US Department of Health and Human Services; October.

Barton J, Porter J. 2004. Radiological, Chemical, and Environmental Health Assessment of the Marine Resources of the Isla de Vieques Bombing Range, Bahia Salina del Sur, Puerto Rico. Underwater Ordnance Recovery, Inc.; University of Georgia. March 8, 2004.

Bauer, L.J. and M.S. Kendall (eds.). 2010. An Ecological Characterization of the Marine Resources of Vieques, Puerto Rico Part II: Field Studies of Habitats, Nutrients, Contaminants, Fish, and Benthic Communities. NOAA Technical Memorandum NOS NCCOS 110. Silver Spring, MD.

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Bermudez W. 1998. Brief history of Vieques. Available from URL: http://www.vieques-island.com/hisindx.html.

CH2MHILL. 2000. Surface Soil Investigation Report, Live Impact Area, Vieques Island, Puerto Rico. October 2000.

CH2MHILL. 2007. Final East Vieques Background Soil Inorganics Investigation Report, Former Vieques Naval Training Range, Vieques, Puerto Rico. October 2007.

García N, Lopez AM, Soto M, Rosado S, Berrios B. 2000. Environmental impact of Navy activities in Vieques. San Juan, PR: Servicios Cientificos y Tecnicos, Inc.; July.

[IT] IT Corporation. 2000. Atlantic Division Naval Facilities Engineering Command, Air dispersion modeling and related analyses for Inner Range operations, Vieques, Puerto Rico. Knoxville, TN: February 2000.

Learned RE, Grove GR, Boissen R. 1973. A geochemical reconnaissance of the Island of Vieques, Puerto Rico. US Geological Survey and Puerto Rico Department of Natural Resources. Open-file report 73-155 (order number 1866).

US Army. 1992a. Development of methodology and techniques for identifying and quantifying products from open burning and open detonation thermal treatment methods-Bang Box Test Series, Volume 1, Test Summary. Rock Island, IL: AMMCOM 61299-6000; January.

US Army. 1992b, Development of methodology and techniques for identifying and quantifying products from open burning and open detonation thermal treatment methods-Bang Box Test Series, Volume 2, Test Development. Rock Island, IL: AMMCOM 61299-6000; January.

US Army. 1992c, Development of methodology and techniques for identifying and quantifying products from open burning and open detonation thermal treatment methods-Bang Box Test Series, Volume 3, Quality Control and Quality Assurance. Rock Island, IL: AMMCOM, 61299-6000; January.

US Navy. 2001 Installation Restoration Program: Community Fact Sheet. U.S. Naval Ammunition Support Detachment, Vieques Island, Puerto Rico. Issue Number 2. April, 2001.

[USEPA]US Environmental Protection Agency, Region 2. 2004. Vieques Island, Puerto Rico—Sectors. Available at: http://www.epa.gov/Region2/vieques/sectors.htm. [accessed 18 January 2006].

[USGS] U.S. Geological Survey. 1997. Collecting rocks. RM Barker. June 24, 1997. Available at http://pubs.usgs.gov/gip/collect1/collectgip.html, [accessed 2012 Aug 28].

[USGS] U.S. Geological Survey. 2001. Understanding our planet through chemistry. Compiled by JE Taggart. Edited by CA Bales and SK Runberg. Composition by ML Johnson. Graphics by RP Walker, ST Smith, and SM Smith.

Young, GA. 1978. Environmental dispersion of the products of explosions of conventional ordnance at Vieques Island, Naval Surface Weapons Center: August 28, 1978.

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Chapter 2 Consumption of Fish from Reefs off the Vieques Coast

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Chapter 2 Summary

At the request of scientists who visited ATSDR in 2009, ATSDR evaluated the risk from mercury in fish and shellfish by comparing the estimated mercury intakes with guidelines recommended by the National Academy of Sciences and by the U.S.EPA.

ATSDR has identified mercury exposure from frequent consumption of marine seafood as a potential public health hazard. After a review of estimated mercury intakes from seafood consumption, ATSDR concludes that children born to women who eat fish daily from waters surrounding Vieques are at increased risk of adverse health effects. Possible harmful effects identified from studies of non-Viequense children exposed in utero involve language, attention, and memory, and to a lesser extent visual/spatial and motor functions. In addition, even if children were not exposed in utero, some children who frequently eat fish from waters surrounding Vieques are also at risk of harmful effects. This conclusion about the risk of harmful effects to the fetus and to children is somewhat uncertain, primarily because a person's response to mercury is itself somewhat uncertain. Contributing to that uncertainty is how the body handles mercury, and the sex, genetics, health, and nutritional status of the person who eats the fish, or how mercury is handled in the body. Estimating mercury intake from eating reef fish is likewise uncertain. The intake could vary depending on the type, frequency, and quantity of fish eaten. Current information about Viequenses fish-eating habits and mercury levels in fish could reduce this uncertainty.

Statistical analysis showed that some fish and shellfish from certain reefs surrounding Vieques had higher levels of some metals and lower levels of other metals—iron, aluminum, copper, zinc, arsenic, barium, potassium and selenium were all slightly higher—compared with other reefs surrounding Vieques. These metals are materials found in bombs and in metal ships, suggesting possible localized contamination. But the levels were only slightly higher and the difference was statistically significant only for some reefs compared with other reefs surrounding Vieques.

ATSDR recommends the following for consideration by environmental and public health agencies and scientists:

Following available fish advisories and maintaining fishing restrictions near the LIA;

Conducting a survey to determine the type, frequency, and quantity of fish consumed;

Conducting additional risk assessments and statistical analyses using information gathered from the previously recommended fish consumption survey;

Collecting and analyzing additional fish samples from Vieques should the proposed survey and statistical analysis not provide sufficient public health information. Collect sufficient fish samples to allow analysis by species and by location; and

Developing an educational program about mercury in fish that incorporates local habits and information about Viequenses' seafood consumption.

2.1 Introduction to Viequense Fish Consumption

Many Viequenses remain concerned that the U.S. Navy's past military exercises might have contaminated fish and other marine life near the island. Locally harvested fish and other species are an important part of the Viequense diet. High contaminant levels in fish and other marine animals could affect the health of those who eat them. But heavy metals in fish and shellfish are not unusual. Depending on an area's geology and chemical composition, fish and shellfish will assimilate a variety of metals in varying concentrations. Thus in populations who eat substantial quantities of fish, evaluation of heavy metal exposure is quite reasonable.

During ATSDR's November, 2009 meeting in Atlanta, several scientists raised questions about mercury levels in fish. They reported finding residents with elevated mercury levels in hair. If these mercury levels are indicative of mercury body burdens generally, they might put a developing fetus at risk for neurological effects. The scientists were concerned that the conclusions in ATSDR's 2003 Fish PHA were inconsistent with the 2004 U.S.EPA/FDA national fish advisory for mercury. They were also concerned that ATSDR did not use the National Academy of Sciences' recommendations to U.S.EPA concerning mercury toxicity. ATSDR therefore is reevaluating its 2003 conclusions and recommendations about mercury in fish from reefs surrounding Vieques.

ATSDR's evaluation of mercury in the Vieques environment indicates that the mercury is most likely coming from the global reservoir of mercury in the environment and not from past military exercises. A more thorough discussion of this topic occurs in Chapter 1, Section 1.3. Nevertheless, because of continued public health concerns, ATSDR revisited its evaluation of mercury in fish.

2.1.1 A Brief Review of Mercury

Mercury is found in several chemical forms: elemental mercury, inorganic mercury, and methylmercury. Elemental mercury is the familiar silver material found in some thermometers. Mercury in soil is often inorganic mercury, while mercury in fish and shellfish is predominantly methylmercury, with small amounts of inorganic mercury. When elemental or inorganic mercury enters freshwater and saltwater environments, some of it is transformed into methylmercury, which accumulates in fish and seafood. It is the methylmercury form in fish that is harmful to the developing fetus and to young children. Tests for mercury in fish, however, often measure all forms of mercury. We refer to these tests as total mercury concentration or just mercury concentration. Identification of just the methylmercury or inorganic mercury concentrations in fish requires specific tests.

2.1.2 Summary of ATSDR's 2003 Fish PHA

In July 2001, ATSDR collected 104 fish and 42 shellfish samples. ATSDR analyzed the edible tissue for metals and explosive compounds. The 42 shellfish samples consisted of 20 conch, 7 lobster, 11 blue land crab, and 4 fiddler crab. Twenty-five blue land crabs comprised 11 composite samples, and 146 fiddler crabs comprised four composite samples. All samples were analyzed individually except the blue crab (combined into 11 composite samples) and the fiddler crabs (combined into four samples). As expected, the results showed various metals in fish and shellfish tissue. Therefore, in its 2003 Fish PHA, ATSDR evaluated whether fish and shellfish muscle tissues contained levels of heavy metals that would pose a health risk. In its 2003 Fish PHA, ATSDR used fish intake rates that focused on people who ate large

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amounts (i.e., 8 ounces for adults and 4 ounces for children) and who ate fish daily. The agency also used standard body weights of 70 kilograms (kg) (or 154 pounds) for adults and 16 kg (35 pounds) for children. ATSDR further assumed that all the mercury detected in fish and shellfish was methylmercury—numerous fish studies support this (ATSDR 1999; Greib et al. 1990; Bloom 1992). In the 2003 Fish PHA, the estimated doses were compared with ATSDR's chronic Minimal Risk Level (MRL) for methylmercury. In June 2003, ATSDR released its evaluation of these data as a public health assessment with these conclusions:

- A variety of fish and shellfish were safe to eat every day;
- Fish and shellfish were safe to eat from any of the locations sampled, including from around the LIA and the sunken navy target vessel USS Killen; and
- Snapper, the most commonly consumed species, was safe to eat every day.

These recommendations were based on the low level of mercury and other metals detected in fish collected from reefs, from the sunken target vessel, and from a commercial fish market on the island. Organic chemicals associated with past military activities were rarely detected in the seafood samples and then only at low levels.

2.1.3 U.S.EPA and FDA Joint National Fish Advisory

In March 2004, a year after the public health assessment, the U.S.EPA and the Food and Drug Administration (FDA) released a joint national fish advisory. It emphasized that fish and shellfish were an important part of a healthy diet. The advisory pointed out that fish and shellfish contained high-quality protein and other essential nutrients, were low in saturated fat, and provided omega-3 fatty acids, a heart healthy chemical. A well-balanced diet that included a variety of fish and shellfish could contribute to heart health and to children's proper growth and development. The advisory concluded that in particular, women and young children should include fish or shellfish in their diets (USEPA 2004; FDA 2004).

The joint FDA/USEPA advisory acknowledged that nearly all fish and shellfish contained traces of mercury. For most people, the risk of mercury-related health effects from eating fish and shellfish was not a concern. Yet some fish and shellfish may contain levels of mercury considered unhealthy. Women and young children are advised not to eat some fish and shellfish, especially in large quantities. The risks from mercury in fish and shellfish depend on the mercury levels in, and the amount of fish and shellfish eaten. The FDA and the U.S.EPA have advised women who might become pregnant, women already pregnant, nursing mothers, and young children to avoid some types of fish and to eat only fish and shellfish known to have lower mercury levels (USEPA 2004; FDA 2004).

The joint advisory said that by following these recommendations for selecting and eating fish or shellfish, women and young children would receive dietary benefits. At the same time, they would reduce their exposure to mercury's harmful effects. U.S.EPA and FDA made the following recommendations and statements:

- Do not eat shark, swordfish, king mackerel, or tilefish—they contain high levels of mercury.
- Eat up to 12 ounces (2 average meals) a week of a variety of fish and shellfish lower in mercury.

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- In the continental United States, five of the most commonly eaten fish low in mercury are shrimp (0.012 ppm),[11] canned light tuna (0.12 ppm), salmon (0.01 ppm), pollock (0.04 ppm), and catfish (0.05 ppm). Another commonly eaten fish in the continental United States, albacore ("white") tuna (0.36 ppm), has more mercury than does canned light tuna. U.S.EPA and FDA recommend limiting albacore tuna to only one 6 ounce-meal (one average meal) per week.
- U.S.EPA and FDA recommend checking local advisories about the safety of fish caught by family and friends in local lakes, rivers, and coastal areas. In the absence of a local advisory, eat only up to 6 ounces (one average meal) per week of fish caught in local waters but do not consume any other fish during the week.
- U.S.EPA and FDA advise following these same recommendations when feeding fish and shellfish to young children, but serve smaller portions (USEPA 2004; FDA 2004).

Note that the FDA currently compares the risk from mercury in fish with the benefits of eating fish (FDA 2009). According to its draft risk and benefit assessment, the FDA estimated the net effect of consumption of different amounts of fish. The results indicated that as measured by verbal development, consumption of fish species low in methylmercury was likely to result in a modest net benefit. When FDA modeled actual consumption for the range of methylmercury concentrations (low to high) in fish, the likelihood was small of an adverse effect in children. In addition, FDA concluded that fish consumption prevented a significant number of deaths from coronary heart disease and stroke each year in adults. FDA pointed out, however, that its risk and benefit assessment should not be construed as altering the agency's then-existing fish advisory (FDA 2009). More information about FDA's risk and benefit assessment is available at http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm088794.htm.

2.1.4 The Vieques Marine Environment

The following brief descriptions provide some insight into the ecological effects to the coral reefs around Vieques.

- In July 2001, the U.S.EPA's Environmental Response Team (USEPA/ERT) collected fish and shellfish from near-shore reefs around Vieques for ATSDR's public health assessment. The divers conducting the sampling reported that unexploded ordnance (UXO) was a common sight around the former USS Killen (a Navy target vessel intentionally sunk to the south of the LIA) and was occasionally seen in a seagrass bed to the north of the LIA. Despite the presence of UXO, the divers observed that all sample locations supported diverse, healthy populations of marine organisms and that the reefs visited were in good condition. The divers also noted that with very few exceptions, the fish and shellfish collected appeared healthy (USEPA 2001).
- In November 2001, a Navy contractor (Geo-Marine) characterized the biological organisms on and around the former USS Killen. The purpose was to assess the health of the marine species and to assess potential effects on the surrounding biota. The overall conclusion was that the

[11] Average mercury level as reported by FDA (FDA 2004).

sunken vessel and its contents had no negative effects on the coral reef ecosystem; rather, they acted as a productive artificial reef habitat (Geo-Marine 2002).

- In June 2003, researchers from the University of Georgia and Underwater Ordnance Recovery collected data on the environmental integrity and ecosystem health of Vieques coral reefs. They reported the presence of underwater UXO and numerous 55-gallon drums in Bahia Salina del Sur (the bay to the southwest of the former LIA) and documented damage to the coral reefs (Barton and Porter 2004).
- In May and October 2007, the National Oceanic and Atmospheric Administration (NOAA) conducted an ecological characterization of the marine resources of Vieques. They found little evidence of any differences in marine resources, nutrients, or contaminants among the different former land-use zones. Biota, nutrients, and contaminant levels around Vieques generally match those of other coral reef ecosystems (Puerto Rico and U.S. Virgin Islands) and appear shaped by regional-scale processes rather than local factors. The results of their study did not support the hypothesis that military activities negatively affected the marine environment around Vieques. Nor did their study support the opposite hypothesis: that the lack of development on two-thirds of the island positively influenced the marine environment (Bauer and Kendall 2010).
- In July 2008, the National Coral Reef Institute published the results of a study that investigated whether the coral reefs around Vieques were in better or worse shape than the reefs around St. Croix. Even though the researchers observed evidence of past military activity at Vieques (e.g., unexploded bombs, casings, and ammunition shells to the north and south of the LIA), they found no differences in living benthic cover and coral assemblage structure between the two islands. They concluded that the effects of Caribbean-wide natural disturbances (disease and storms) had a greater impact on the coral reefs than did the former military activities on Vieques (Riegl et al. 2008).

2.2 Assessment of Available Fish Data

Two sets of data are available on metal levels in fish. One is from the Metropolitan University in Puerto Rico, and the other is from ATSDR. Both sets of data originate from sampling events conducted from 1999–2000 or in 2001.

2.2.1 Fish Data from Metropolitan University

From December 1999 to April 2000, Dr. Doris Caro with the Metropolitan University (i.e., Universidad Metropolitana), School of Environmental Matters, collected 52 fish from two markets in Vieques (northern Vieques and Esperanza) and compared the results with 26 fish collected from fish markets from the Parguera area on Puerto Rico's mainland (Caro 2000). She also conducted a survey of 51 Vieques residents and fishers to determine the most frequently consumed fish. Among the 51 residents surveyed, Dr. Caro reported the following fish consumption frequencies:

- 20% never eat fish,
- 47% eat fish 1–2 times a week,
- 18% eat fish 3–4 times a week, and

- 16% eat fish five or more times a week[12] (Caro 2000).

Dr. Caro thus reported that 34% of Viequenses ate fish 3–4 or more times a week. The more extensive, random PRDOH survey in 2004 supported her findings. The PRDOH manuscript reported that 38% of Viequenses ate fish three or more times a week. Both of these surveys show that a significant portion of Viequenses eat fish regularly. ATSDR's assumption that a significant proportion of Viequenses eat fish daily therefore seems reasonable, although the precise percentage is not known.

Dr. Caro also provided information about the type of fish the 51 respondents ate. Table 2-1 shows these results from Dr. Caro's survey along with the number of responders who ate certain fish (Caro 2000). Dr. Caro concluded that the detected metal concentrations in Vieques fish did not show that metals in fish bioaccumulate, pointing to the absence of any clear relationship between fish weight, size, and metal content (Caro 2000). ATSDR's data, however, did show bioaccumulation in some species.

Mercury concentrations in fish collected by Dr. Caro are shown in Table 2-2 as ppm, although whether the reported concentrations are dry weight or wet weight is uncertain. We have attempted to contact the laboratory to ascertain how the data were reported, but we have not received a response. The described method produces raw results in dry weight. The mercury concentrations were extremely low (lower than ATSDR's results on wet or dry weight basis), but this latter fact could be explained by the very low weights of the fish. Uncertainty thus remains about whether the results are wet weight or dry weight. Regardless, Dr. Caro concluded that the results show that mercury concentrations are higher in fish from markets in Vieques compared with the control population from Parguera (Caro 2000).

These conclusions were reached using averages calculated in a manner inconsistent with U.S.EPA methods. Dr. Caro states that the results clearly justify a need for further sampling. We agree with this last statement.

Dr. Caro further states that her report should be viewed as a starting point for a more comprehensive survey. She recommended the following:

1. Choose sentinel species, including some close to sediments.
2. Obtain fish samples in other areas of the island, mainly in ordnance-free areas.
3. Sample fish after the beginning of military exercises and analyze it accordingly.[13]
4. Sample and analyze crustaceans (e.g., crabs, lobster) and other aquatic life, such as snails.
5. Assign additional funds for the enhancement of the proposed study.
6. Perform a risk-assessment study of the species analyzed and the results obtained.

[12] Percentages are rounded to whole numbers.

[13] Note that sampling fish after the beginning of military exercises is no longer possible—all such exercises have ceased.

Table 2-1. The type of fish that the 51 respondents ate as reported by Dr. Doris Caro in her survey of Viequenses.

Number of respondents	*Spanish Common Name*	*Scientific Name*	*English Common Name*
41	Colirrubia	*Ocyurus chrysurus*	Yellowtail snapper
32	Mero cabrilla	*Epinephelus guttatus*	Rock hind
29	Langosta	*Palinurus argus*	Spiny lobster
26	Peje puerco	*Balistes vetula*	Queen triggerfish
24	Sierra	*Scomberomorus maculatus*	Spanish mackerel
23	Capitán	*Lachnolaimus maximus*	Hogfish
21	Cotorro	*Sparisoma* sp.	Parrotfish
20	Chapin Gallina	*Lactophrys trigonus*	Trunkfish
13	Bonito	*Euthynnus alletteratus*	Little tunny
11	Negra	*Lutjanus buccanella*	Blackfin snapper
11	Dorado	*Coryphaena hippurus*	Dolphinfish (Mahi-mahi)
11	Chillo	*Lutjanus vivanus*	Silk snapper
11	Boquicolora'o	*Haemulon plumier*	Striped (white) grunt
10	Dentex (Corvina)	*Odontoscion dentex*	Reef croaker
8	Atun	*Thunnus*	Tuna
8	Tibarón	*Numerous genera (e.g.,* Etmopterus, Carcharhinus*)*	Shark
5	Sharpsnout*	*unknown*	Sharpsnout
4	Abanico†	*Makaira nigricans*	Blue marlin
4	Salmón	*Salmo* and *Oncorhynchus*	Salmon
3	Carrucho	*Strombus*	Conch
1	Robalo	*Centropomus*	Snook
1	Salmonete	*Mulloidichthys* or *Pseudupeneus*	Goatfish
1	Arrayao	*Lutjanus synagris*	Lane snapper
1	Jurel aleta amarilla	*Caranx hippos*	Crevalle Jack

*Because a genus name is not provided, whether "sharpsnout" refers to the sharpsnout stingray, sharpsnout seabream, or sharpsnout flounder is unclear.

†ATSDR is unsure of the Viequense Spanish common name for the blue marlin. Other possible names include *aguja, castero, prieta,* and *voladora*.

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Table 2-2. Average fish-mercury levels from fish markets in northern Vieques, southern Vieques (Esperanza), and Parguera, Puerto Rico (Caro 2000).

Fish	*Northern Vieques (n) ppm**	*Southern Vieques (Esperanza) (n) ppm*	*Parguera (n) ppm*
Arrayao (lane snapper)	0.048 (3)	0.018 (2)†	0.000 (1)
Boquicolora'o (striped grunt)	0.029 (6)	0.024 (6)‡	0.008 (4)
Colirrubia (yellow tail snapper)	0.019 (7)	0.022 (3)	0.004 (2)
Cotorro Azul (blue parrotfish)	0.002 (3)	NA	0.001 (2)
Cotorro Rojo (stoplight parrotfish)	0.000	NA	0.001 (3)
Cotorro Verde (redband parrotfish)	0.010 (1)	NA	0.007 (5)
Salmonete de Altura (goatfish)	0.038 (1)	NA	0.001 (2)
Pluma (dogfish)	NA	0.018 (3)	0.008 (2)
Mero Cabrilla (Red hind)	0.010 (4)	0.015 (1)	0.011 (3)

n = number of samples

NA = No sample available.

*Whether the concentrations represent wet weight or dry weight is unknown; no indication has been provided by the laboratory or Dr. Caro's report. The table here uses the values calculated by Caro et al. 2000 from Appendix 10. Her averages include values below the detection level of the instrument.

† An apparent typographical error appears in the original table in the main text of Dr. Caro's report. Using data from Appendix 10 in the report shows that the average is 0.018 ppm, not 0.048 ppm.

‡ An apparent typographical error appears in the original table in the main text of Dr. Caro's report. Using data from Appendix 10 in the report shows that the average is 0.024 ppm, not 0.029 ppm.

The Metropolitan University data have several strengths, but some limitations as well. To ensure that metal concentrations in fish were in the fish that people were consuming, investigators collected their fish samples from two local markets. Investigators interviewed 51 residents and fishers about their fish-eating habits and where those fish were bought or caught. Investigators learned their Vieques fish market samples included fish from the reefs and the open ocean. Metal concentrations in Vieques market fish did not therefore necessarily represent metal concentrations in reef fish near Vieques. Also, the participants were not randomly selected, and only a limited number of surveys (i.e., 51) were conducted. All this adds some uncertainty to identifying all the fish species that Viequenses eat and to the frequency of Viequense fish consumption. And as stated, with regard to the fish sampling results, the authors did not specify whether their results were wet or dry weight. This prevents a reliable estimation of human doses from eating fish. Despite these limitations, however, one peer reviewer of this report requested additional analysis of Caro's data.

Therefore, with qualification, after Dr. Caro's data are regrouped and reaveraged, Dr. Caro's data can be compared with ATSDR's data. Because Dr. Caro reported averages for fish according to species rather than fish family, some locations have just one or two fish to compare. Also, in calculating averages, Dr. Caro used values below the detection level.

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ATSDR used the raw data reported in Appendix 10 of Dr. Caro's report to group fish species into families from both Vieques markets. In this way, the results provide statistically significant numbers comparable with ATSDR's data:

- Arrayo was combined with colirrubia to represent the snapper family at both Vieques markets,
- Cotorro rojo was combined with cotorro azul and cotorro verde to represent the parrotfish family at both Vieques markets,
- Mero mantequilla were combined with mero cabrilla to represent the hind family at both Vieques markets, and
- Boquicolora were combined with pluma to represent the grunt family at both Vieques markets.

To calculate averages, all laboratory results below the analytical detection levels were then set at one-half the detection levels. Table 2-3 shows average mercury levels in various fish families using Dr. Caro's (regrouped) 2000 data and using ATSDR's 2001 data. Dr. Caro's data show average mercury levels by fish family that are below the average levels reported by ATSDR for both wet weight and dry weight. Note that if Dr. Caro's data are dry weight, the wet weight concentration will be lower.

Although for Dr. Caro's data the mercury concentration basis (wet vs. dry weight) is unknown, the fish families' relative rankings provide insight for exposure assessments. The fish families shown in Table 2-3 are in order of increasing average mercury concentration (according to ATSDR's order). Three of five families in Dr. Caro study have similar relative ranking to ATSDR's ranking. Goatfish might have a higher relative ranking in Dr. Caro study because of the small number (4) of Goatfish samples. The snapper Dr. Caro collected might have a higher relative ranking, but grunt and hind fish she collected were much smaller than ATSDR's. Some conclusions from these combined data follow:

1. Parrotfish have low mercury levels.
2. All of Dr. Caro's fish families have average mercury levels below U.S.EPA's screening level of 0.049 ppm (wet weight)[14]. This conclusion is true whether Dr. Caro's average is wet weight or dry weight.
3. ATSDR identified three families (goatfish, hind, and grunt) above the U.S.EPA screening level of 0.049 ppm (wet weight).

Because Dr. Caro's grunt and hind were much (~30%) smaller than ATSDR's grunt or hind and because we know that mercury accumulates in larger fish, it is likely that all (these) Vieques fish have very low levels—that is, until they grow larger. In addition, grunt and hind are two families that accumulate more mercury when they are larger.

[14] Using the Reference Dose (RfD) for methylmercury, U.S.EPA established a fish screening level of 0.049 ppm mercury for recreational and subsistence fishers. More information about U.S.EPA's screening levels is located at this Web address: http://water.epa.gov/scitech/swguidance/fishshellfish/techguidance/risk/volume2_index.cfm .

Table 2-3. Average fish-mercury levels: Vieques

Fish	*Caro et al. (ppm)**	*ATSDR (ppm-wet)*	*ATSDR (ppm-dry)*
Parrot	0.004	0.009	0.046
Snapper	0.026	0.048	0.218
Goatfish	0.038	0.080	0.344
Hind	0.016	0.116	0.551
Grunt	0.024	0.185	0.887
All fish (fin fish)	0.020	0.091	0.427

*Whether Dr. Caro's results are wet or dry weight is unknown

2.2.2 Fish and Seafood Data from ATSDR

ATSDR's 2001 sampling and analysis activities focused on whether muscle tissues from commonly consumed fish and shellfish contained levels of heavy metals and explosives compounds that would result in health effects. To assist in these activities, ATSDR sought advice and worked with the U.S.EPA's Environmental Response Team (EPA/ERT) to collect and analyze fish and shellfish from the coastal waters around Vieques and from land and beaches near the shore.

ATSDR collected fish and shellfish from five reefs surrounding Vieques and one local fish market. Two reefs were off the eastern portion of the island near the Live Impact Area, where military exercises formerly occurred and one of these locations included the area where the USS Killen is located. The three other reefs were off the central and western portions of the island. Land crabs were collected from two locations within the Live Impact Area and one location on the western portion of the island. Efforts were made to collect from each reef location and from the fish market five fish within a family (e.g., grunt and hind from the genus *Epinephelus* in the *Serranidae* family or snappers from the genera *Lutjanus* and *Ocyurus* within the family *Lutjanidae*). At most locations, ATSDR was able to collect five fish from each family (e.g., *Epinephelus, Sparisoma* and *Haemulon* families). But at several locations, five fish could not be collected for some families (see Table 2-4).

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Table 2-4. Fish and shellfish collected by ATSDR

Common Name and Taxonomic Family	***Genus and species***	***Species # and Family Total***	***Loc. #1 Reef North LIA***	***Loc. #2 Reef South LIA***	***Loc. #3 Reef South Esperanza***	***Loc. #4 Reef NW Isabel Segunda***	***Loc. #5 Fish Market***	***Loc. #6 West End***
Red hind	*Epinephelus guttatus*	19	5	1	0	5	5	3
Rock hind	*Epinephelus adscensionis*	8	0	4	4	0	0	0
Graysby	*Epinephelus cruentatus*	1	0	0	1	0	0	0
Coney	*Epinephelus fulvus*	2	0	0	0	0	0	2
Total # *Serranidae* family		Total = 30	Total = 5	Total = 5	Total = 5	Total = 5	Total = 5	Total = 5
Schoolmaster snapper	*Lutjanus apodus*	5	0	3	2	0	0	0
Grey snapper	*Lutjanus griseus*	3	0	2	1	0	0	0
Yellowtail snapper	*Ocyurus chrysurus*	11	0	0	1	2	5	3
Total # *Lutjanidae* family		Total = 19	Total = 0	Total = 5	Total = 4	Total = 2	Total = 5	Total = 3
Stoplight parrotfish	*Sparisoma viride*	19	4	1	5	4	0	5
Redband parrotfish	*Sparisoma aurofrenatum*	5	1	3	0	1	0	0
Redfin parrotfish	*Sparisoma rubripinne*	1	0	1	0	0	0	0
Total # *Scaridae* family		Total = 25	Total = 5	Total = 5	Total = 5	Total = 5	Total = 0	Total = 5
White Grunt	*Haemulon plumieri*	7	0	2	0	0	5	0
Spanish Grunt	*Haemulon macrostomum*	1	0	0	1	0	0	0
Bluestriped Grunt	*Haemulon sciurus*	12	5	3	4	0	0	0
French Grunt	*Haemulon flavolineatum*	4	0	0	0	0	0	4
Total # *Haemulidae* family		Total = 24	Total = 5	Total = 5	Total = 5	Total = 0	Total = 5	Total = 4

009510

Yellow Goatfish	*Mulloidichthys martinicus*	4	0	0	3	0	0	1
Spotted Goatfish	*Pseudupeneus maculatus*	1	1	0	0	0	0	0
Total # *Mullidae* family		Total = 5	Total = 1	Total = 0	Total = 3	Total = 0	Total = 0	Total = 1
Honeycomb Cowfish	*Lactophrys polygonia*	1	0	0	0	0	1	0
Queen Conch	*Strombus gigas*	20	5	5	5	0	0	5
Spiny Lobster	*Panulirus argus*	7	1	0	1	0	5	0
Blue Land Crab	*Cardisoma guanhumi*	11	5	5	0	0	0	1
Fiddler Crab	*Uca* sp.	4	2	2	0	0	0	0
Note: Blue land crabs and fiddler crabs are listed by the number of composite tissue samples analyzed, not by the number of individuals captured. The meat from several land crabs was composited until a sufficient quantity of meat was obtained. To ensure a sufficient sample was collected for analysis, the whole fiddler crabs were composited and were analyzed.								

Fish and shellfish tissues were analyzed for explosives compounds. No explosive residues were detected in fish tissues from any sample location. Of the four shellfish species, only fiddler crabs were shown to contain the explosives compound HMX. RDX was detected in fiddler crabs, but the level was so low that an accurate concentration could not be measured. We should note that the laboratory analysis identified trace levels of a chemical with the same characteristics (i.e., retention time) of RDX (hexahydro-1,3,5,-triazine) in two samples: the fiddler crabs and the trunkfish. These trace levels were well below the RDX detection level and estimated doses were well below the RDX health guideline. Furthermore, the natural toxins within the trunkfish have the same retention time on the gas chromatogram as RDX. Therefore, we cannot conclusively determine whether RDX was present in the trunkfish tissues. The 2003 Fish PHA concluded that because residents did not eat fiddler crabs, the fiddler crab HMX would not cause harmful effects in people. No explosive compounds were detected in conch, lobster, or land crab samples from any location. Fiddler crabs and land crabs were collected from locations 1 and 2 on the eastern end of the island, and land crabs were collected from location 6 on the western end of the island (see Figure 2-1).

009511

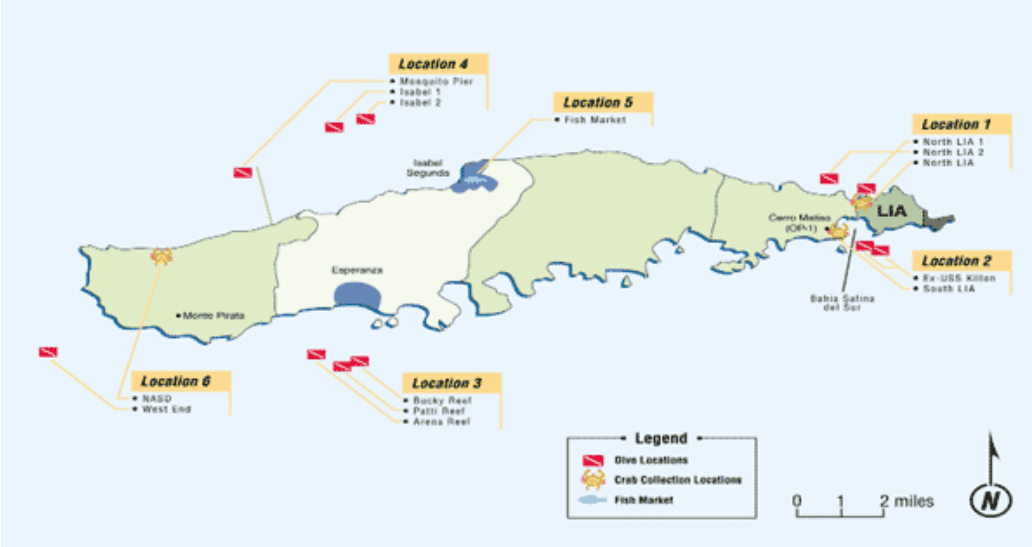


Figure 2-1. ATSDR fish and shellfish sample locations.

In addition to explosive compounds, ATSDR analyzed the same fish and shellfish tissues for heavy metals. ATSDR collected 104 fish and 42 shellfish samples and analyzed the edible tissue for metals and explosive compounds. The fish samples represent 17 different species and can be grouped into these fish families: 30 hinds (or groupers), 19 snappers, 25 parrotfish, 24 grunts, 5 goatfish, and 1 cowfish. The 42 shellfish samples consisted of 20 conch, 7 lobster, 11 composite, blue land crab, and 4 composite fiddler crab samples. Twenty-five blue land crabs comprised 11 composite samples and 146 fiddler crabs comprised four composite samples. All samples were analyzed individually except the blue crab (combined into 11 composite samples) and the fiddler crabs (combined into four samples). Here we refer to the individual and composite samples collectively as 42 shellfish samples.

2.2.3 Strength and Limitations of ATSDR's 2001 Fish and Seafood Data

ATSDR's 2001 sampling effort included several strengths:

- ATSDR used multiple information sources to identify the preferred types of fish and shellfish for collection. One important source was a Puerto Rican university report containing survey information about the frequency and types of fish eaten (Caro 2000). ATSDR substantiated the survey results with information from the petitioner and other residents, the Vieques Special Commission Report, and visits to the local fish markets on Vieques.
- ATSDR identified snapper (*Lutjanus* and *Ocyurus* species) as the most frequently eaten fish followed by hind (*Epinephelus* species) and grunt (*Haemulon* species).
- Fish and shellfish were collected from five marine locations and from one commercial fish market on the island. Land crabs were collected from three surface locations (see Figure 2-1).
- In designing the sample plan and in collecting the samples, ATSDR partnered with the U.S.EPA's Environmental Response Team, which has extensive experience in sampling and analyzing fish. The U.S.EPA recommended that ATSDR collect five fish per family group.

009512

- ATSDR collected 104 fish and 42 shellfish samples. Although most samples represented one organism, crab samples represented a composite of three or more organisms.
- ATSDR used standard-default fish consumption intakes. These included intake rates that focused on high-end fish consumers as well as standard body weights for adults and children. ATSDR also used the agency's chronic, oral MRL for mercury. This MRL underwent rigorous scientific and peer review when it was developed.
- ATSDR followed many of the criteria described in the U.S.EPA's Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories, Volume 1, Fish Sampling and Analysis, Third Edition (USEPA 2000). These guidelines describe among other techniques how to identify those fish that should be sampled, how to identify the fish tissue that should be analyzed, and how to composite fish tissue.

In hindsight, ATSDR's 2001 sampling effort also had limitations. The U.S.EPA and the State of California recommend these newer criteria when developing fish consumption advisories. Regarding the appropriate number of fish to sample, the State of California believes the sample design should include either 1) a minimum of three composite samples with each composite consisting of three fish, or, preferably, 2) nine individual fish samples for each species of concern from each water body. When a species has a large size range, fish samples, when feasible, should be collected from multiple legal or edible sizes. Following this sampling protocol allows estimation of the range and variation of contaminant concentrations at a particular site and derivation of a representative mean concentration for use in developing fish consumption advisories (State of California 2009). The U.S.EPA currently recommends a statistical analysis to determine the most appropriate number of fish in a composite sample and the most appropriate number of composite samples. At a minimum, U.S.EPA says the sample design should consist of three composite samples, with three fish per composite. If the contaminant concentration in a fish species is highly variable, more composite samples and more fish in each composite are needed to estimate the average concentration of the contaminant in that species. These recommendations were developed to determine whether average mercury levels in a fish species would exceed U.S.EPA's 0.049 ppm (wet weight) screening level for mercury in fish. When estimating human exposure to mercury, other sample designs might also provide reliable data.

2.2.4 Mercury Levels in Reef Fish from Vieques

Using ATSDR's 2001 fish sample results, the agency chose to analyze contaminant levels in individual fish—acceptable when a sufficient number of fish of each species or each family from each location are caught and analyzed. The maximum number of individual fish caught and analyzed for each species at each location was five. Table 2-4 summarizes the number of fish species and number of fish in each family analyzed at each location.

Although the U.S.EPA recommends against combining species when determining contaminant levels, the State of California says similar species can be combined (e.g., bass) because species within a family group are likely to have similar lifestyles. And because more than nine fish were collected within each family, sufficient samples were available to evaluate hind (*Epinephelus* species), snapper (*Lutjanu* and *Ocyurus* species), parrotfish (*Sparisoma* species), and grunt (*Haemulon* species)—if the data from all

009513

locations were combined. But grouping the data in this way introduced some uncertainty or bias. For example, certain species within a family had higher or lower contaminant levels than the average for that family. So having too many French grunt will create a high bias for all grunt. This was important for the fish ATSDR collected in Vieques—the mercury concentrations were highly dependent on species (and family). Grouping the data also limited evaluation of fish data by location. Table 2-4, above, identifies those locations with more samples of one species or another. This table helps identify where comparisons can be made by species and where comparisons are only possible by combining species within a family. For example, in Table 2-4, five red hind (first line of data) were collected from locations 1, 4, and 5—this suggests that red hind can be compared between locations 1, 4, and 5. Therefore, should we want to compare other locations, we would have to rely on other species of hind, which introduces added uncertainty due to the differences among the hind species.

ATSDR compensated for this uncertainty in part by a statistical analysis of the 2001 fish data. Table 2-5 provides the average concentrations for each species and family collected at the six locations.

Table 2-5 permits comparison of mercury levels in seafood by family, by location, and, in some cases, by individual species at a location. ATSDR's evaluation of the fish data resulted in new conclusions. A principal new conclusion was that the fish data represented enough fish to make numerous, statistically significant determinations. Statistical tests provided in Chapter 2, Section 2.5, Appendix 2A-1 indicate that mercury concentrations were highly dependent on family, on species within some families, and by size of fish within some species. But the fish-mercury levels near the LIA were not statistically different than in the other locations.

Other new conclusions in this report include

- Mercury was lower in shellfish and higher in finfish.
- Mercury in fish was highly dependent on the family and on fish species. Mercury was higher in two fish families (i.e., grunt and hind, 0.18 ppm and 0.12 ppm, respectively) compared with other families sampled (e.g., parrotfish and snapper, 0.01 ppm, and 0.05 ppm, respectively).
- Mercury in hind was associated with fish weight; larger hind typically had more mercury.
- The average mercury concentration in grunt and hind was higher than U.S.EPA's 0.049-ppm screening level. Mercury levels in parrotfish were statistically lower than U.S.EPA's screening level of 0.049 ppm and lower than the overall average of 0.1 ppm.
- Snapper averaged less than, but not statistically different from, the U.S.EPA 0.049 ppm screening level, and statistically less than 0.1 ppm.

ATSDR's evaluation would improve by having more information about the consumption frequency of

- Reef fish, ocean-going fish, and canned fish by sex and by age group,
- Various fish species by sex and by age group, and
- Portion size by sex and by age group.

009514

Table 2-5. Average total mercury in ATSDR-collected fish and shellfish

Common Name and Taxonomic Family	***Genus and species***	***Arithmetic Mean Mercury Levels (ppm wet weight)***						
		All Locations	***Loc #1 Reef North LIA***	***Loc #2 Reef South LIA***	***Loc #3 Reef South Esperanza***	***Loc #4 Reef Isabel Segunda***	***Loc #5 Reef Market***	***Loc #6 Reef West End***
Red Hind	*Epinephelus guttatus*	0.091	0.078	(0.038)	NS	0.10	0.12	0.057
Rock Hind	*Epinephelus adscensionis*	0.15	NS	0.13	0.17	NS	NS	NS
Graysby	*Epinephelus cruentatus*	(0.12)	NS	NS	(0.12)	NS	NS	NS
Coney	*Epinephelus fulvus*	0.21	NS	NS	NS	NS	NS	0.21
Total average *Serranidae* family		0.12	0.078	0.12	0.16	0.10	0.12	0.12
Schoolmaster snapper	*Lutjanus apodus*	0.045	NS	0.058	0.053	NS	NS	NS
Grey snapper	*Lutjanus griseus*	0.061	NS	0.032	(0.067)	NS	NS	NS
Yellowtail snapper	*Ocyurus chrysurus*	0.046	NS	NS	(0.048)	0.073	0.037	0.040
Total # *Lutjanidae* family		0.048	NS	0.055	0.045	0.073	0.037	0.040
White grunt	*Haemulon plumieri*	0.098	NS	0.068	NS	NS	0.11	NS
Spanish grunt	*Haemulon macrostomum*	(0.21)	NS	NS	(0.21)	NS	NS	NS
Bluestriped grunt	*Haemulon sciurus*	0.22	0.21	0.24	0.22	NS	NS	NS
French grunt	*Haemulon flavolineatum*	0.22	NS	NS	NS	NS	NS	0.22
Total # *Haemulidae* family		0.18	0.21	0.17	0.22	NS	0.11	0.22
Stoplight parrotfish	*Sparisoma viride*	ND*	ND*	(ND)*	ND*	ND*	NS	ND
Redband parrotfish	*Sparisoma aurofrenatum*	0.015**	(ND)*	0.020	NS	(ND)*	NS	NS
Redfin parrotfish	*Sparisoma rubripinne*	(0.016)	NS	(0.016)	NS	NS	NS	NS
Total # *Scaridae* family		0.023M	ND*	0.016	ND*	ND*	NS	ND
Yellow goatfish	*Mulloidichthys martinicus*	0.088	NS	NS	0.098	NS	NS	(0.059)
Spotted goatfish	*Pseudupeneus maculatus*	(0.045)	(0.045)	NS	NS	NS	NS	NS
Total # *Mullidae* family		(0.080)	(0.045)	NS	0.098	NS	NS	(0.059)

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Common Name and Taxonomic Family	***Genus and species***	***Arithmetic Mean Mercury Levels (ppm wet weight)***						
		All Locations	***Loc. #1 Reef North LIA***	***Loc #2 Reef South LIA***	***Loc #3 Reef South Esperanza***	***Loc #4 Reef Isabel Segunda***	***Loc #5 Reef Market***	***Loc #6 Reef West End***
Honeycomb Cowfish	*Lactophrys polygonia*	(0.0082)	NS	NS	NS	NS	(0.0082)	NS
Queen Conch	*Strombus gigas*	0.016**	0.018M	0.020M	0.033	NS	NS	ND*
Spiny Lobster	*Panulirus argus*	0.031	(0.031)	NS	(0.047)	NS	0.028	NS
Blue Land Crab	*Cardisoma guanhumi*	0.028M	0.028M	0.021M	NS	NS	NS	(ND)*
Fiddler Crab	*Uca* sp.	ND*	ND*	ND*	NS	NS	NS	NS

NS = The species was not sampled at the given location
ND = Not Detected (range of detection levels in marine live varied 0.0039-0.0085)
(#) = Parenthesis around a value connote a single measurement rather than an average
*= All sample results for this species at this location were ND
**=The value was calculated with at least two samples that were ND
M =The preceding value represents the maximum collected at the given locations as most samples were ND

The 2001 U.S.EPA analytical report on the fish and shellfish samples provided chemical results and statistical metal analysis (USEPA 2001). U.S.EPA's statistical analysis of the fish and shellfish data identified that some fish and shellfish species had slightly higher levels of iron, aluminum, copper, zinc, arsenic, barium, selenium, potassium, and iron in the samples collected near the submerged bombs off the LIA coast. But the levels were only slightly higher and the difference was only statistically significant when compared with a few reef locations.

U.S.EPA's report did not study further the specific sources for these slight increases (USEPA 2001). The U.S.EPA report did provide results for many common explosive organic chemicals, but explosive chemicals rapidly decay into other chemicals that may not be as easily detected.

Nevertheless, the chemical results indicated the presence of HMX and the possible presence of RDX in fiddler crabs. Both of these chemicals are known to break down rapidly in the marine environment. For most fish, uptake of these chemicals is low; it is lowest in fish with higher oil content, and most species rapidly excrete the chemicals (Ownby et al. 2005; Yoo et al. 2002; Helene et al. 2003; Lotufo et al. 2005; Lotufo and Lydy 2005; Houston and Lutufo 2005; Blackburn et al. 2004). When reviewing information for a chemical that had not been ruled out, we identified picric acid as a possible bomb component at some bombing ranges; but we could not verify it was used in Vieques. Some samples were collected from groundwater and soil, but picric acid was either not detected or the sample was rejected because of analytical problems. Picric acid is one explosive compound that does not decay rapidly in the marine environment. While picric acid is not typically used in bombs, it is an explosive compound used by the military. It does not quickly degrade in the sediment, but fish metabolize and excrete it rapidly (Yost et al. 2007; Nipper et al. 2001; Burton et al. 1983 and 1984; Cooper et al. 1984).

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Slightly, but still statistically significant higher levels of military exercise-related chemicals were found in some seafood in the southern LIA area compared with other areas, such as,

- HMX and trace levels of RDX explosives compounds were found in the LIA fiddler crabs;
- Aluminum and potassium were slightly higher in some LIA fish (grunts, parrotfish) compared with other reef locations:
  - Aluminum in south LIA grunt compared with west end and market,
  - Aluminum in north LIA parrotfish compared with west end,
  - Aluminum in south LIA parrotfish compared with all locations except Esperanza,
  - Potassium in south LIA gruntfish compared with west end,
  - Potassium in south LIA parrotfish compared with Isabel 2,
- Copper was slightly higher in some LIA species (grunts, conch) compared with other reef locations:
  - Copper in south LIA conch compared with North LIA, Esperanza, and west end,
  - Copper in north LIA conch compared with Esperanza and west end,
  - Copper in south LIA grunt compared with North LIA and Esperanza
- Iron was slightly higher in some LIA species (land crabs, snappers) compared with other reef locations:
  - Iron in snapper at south LIA compared with West End,
  - Iron in snapper at south LIA compared with North LIA,
  - Iron in crabs at south LIA compared with North LIA (USEPA 2001).

Mercury levels in LIA fish were not significantly different from mercury levels in fish from other Vieques reefs. Mercury has been mentioned as a contaminant of concern because mercury was historically used in some detonators (García et. al. 2000). The data originally provided by the Navy on the composition of the bomb casings indicated the casings did not contain mercury. This is consistent with information on the Material Safety Data Sheets available for steel. The Air PHA indicated that the estimated total annual emissions of mercury to the air were very low (i.e., 0.06 kg per year). This estimated mercury emission of 0.06 kg per year can be compared with the mercury National Emission Standard for Hazardous Air Pollutants (NESHAP) developed by the U.S. EPA. The mercury NESHAP air emission rate for a chlor-alkali plant is 844 kg per year. More information is available at: http://www.gpo.gov/fdsys/pkg/CFR-2011-title40-vol8/xml/CFR-2011-title40-vol8-part61.xml#seqnum61.52.

The source of mercury air emissions was trace amounts in the explosive charge of some bombs and naturally occurring mercury in the soil ejected into the air following detonation. Therefore, past military exercises do not appear to be a significant source of mercury in the Vieques environment.

Average mercury levels in LIA soils was 0.02 ppm, with the highest level detected at 0.086 ppm. NOAA determined that one of 29 soil samples had a mercury level that could be considered above naturally occurring levels (NOAA 2010). But two observations suggest that the LIA is not currently the primary

source of mercury in fish tissues. First, most mercury levels in LIA soils appear to be at naturally occurring levels with no significant spatial variation to suggest mercury levels are higher in the LIA. Second, ATSDR's fish sampling study did not appear to find unusually elevated tissue concentrations of mercury in Vieques reef fish. A more plausible explanation for the mercury levels found in fish is that they resulted from the global reservoir of mercury circulating through the environment.

2.2.5 Toxicological Evaluation of Mercury in Vieques Fish

The wet-weight/dry-weight nature of the Metropolitan University fish data is unknown. Those data, then, provide only speculative insight into contaminant levels. That is, the data can be only compared with fish from Parguera, the study's control population. For quantitative dose estimates, the data are unusable;[15] doses calculated using these data would be unreliable. A toxicological evaluation will therefore incorporate ATSDR's fish data, which have been converted from dry weight in ppm to wet weight in ppm. Compared with Dr. Caro's data (see Table 2-3), ATSDR's data will provide a higher mercury exposure estimate as the fish were larger and the mercury concentrations are higher. Also compared with Dr. Caro's data, the higher mercury concentrations in ATSDR's data could possibly result from ATSDR having collected larger fish, which tend to accumulate higher mercury levels.

Several scientists attending ATSDR's November, 2009 meeting on Vieques raised concerns about ATSDR not using U.S.EPA's Reference Dose (RfD) for methylmercury and not considering the National Academy of Sciences' (NAS) recommendations concerning methylmercury. The scientists noted that Puerto Ricans typically have lower body weights and that Viequenses have high fish consumption rates. They suggested, therefore, that ATSDR evaluate the risk for residents with lower body weights and with higher fish intake. ATSDR used the following parameters to recalculate the doses for women of childbearing age and for children:

1. A range of body weights from 4.5 to 100 kg (9.9 to 220 pounds):
    a. Children (1–2 years; 3–5 years; 6–8 years; 9–14 years; 15–18 years); and
    b. Adult women (18–54 years) (USEPA 1997).
2. A range of age-specific meal portion sizes, including the 50th, 95th, and 99th percentile.
    a. Adult women's portion sizes were 3 oz, 8 oz, and 13.9 oz.
    b. Children's portion size varied by age (see Table 2-6). For example, 3–5 year old girls have portion sizes of 2.5 oz, 6 oz, and 8.5 oz at the 50th, 95th, and 99th percentile, respectively (USEPA 1997).
3. Average mercury levels were recalculated using ½ the detection limit for the approximately 25% of fish that had nondetectable mercury levels (ATSDR 2005). Most of the nondetectable mercury

[15] Laboratory results for chemicals in fish are initially measured as dry weight because the moisture is removed from fish tissue before analyzing for the chemical. Once the moisture is removed, a fish sample might contain 1 ppm of a chemical as dry weight. To estimate exposure, the dry-weight concentration is usually converted to a wet-weight concentration; fish consumption is usually measured as ounces of fresh (wet) fish consumed. Thus to know whether the results are reported as wet weight or dry weight is important.

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levels were found in parrotfish. Average mercury level for all fish was 0.1066 ppm using the 95th upper confidence limit (UCL) of the mean with a maximum mercury level in a bluestriped grunt of 0.33 ppm.

4. Some scientists were concerned that the 2003 Fish PHA used national average weights to represent exposure dose. This report provides exposure estimates using a wide range of weights for various ages. Estimated doses also were calculated on a continuous weight profile from 9.9 to 220 pounds and increasing meal size up to the 99th percentile (See Table 2A-1 in Chapter 2, Section 2.6, Appendix 2A-2.)
5. The estimated doses were compared with U.S.EPA's RfD for methylmercury of 0.1 µg/kg/day. Chapter 2, Section 2.2.5 provides more information about methylmercury's toxic effects.

ATSDR also considered the National Academy of Sciences' recommendations to the U.S.EPA. The NAS recommended that U.S.EPA use the Faroe Islands study to develop the agency's Reference Dose. They also suggested the study and the effect levels that should be considered.

Table 2-6. 50th, 95th, and 99th fish portion size for women and girls of various ages.

	Meal size in ounces/portion		
Age group	50th	95th	99th
1–2 yr old girls	1.5	4.4	5.6
3–5 yr old girls	2	6	8.5
6–8 yr old girls	2.5	6	10.2
9–14 yr old girls	2.8	7.3	10.2
15–18 yr old girls	3	9.5	20
Adult women	3	8	13.9

Source: U.S.EPA's Exposure Factors Handbook (USEPA 1997).

Table 2A-1 (Chapter 2, Section 2.6, Appendix 2A-2) shows the estimated mercury doses for women and children who eat up to 20 ounces of fish daily and who weigh up to 220 pounds. Approximate age groups are provided for various body weights ranging from 10 to 220 pounds (4.5–100 kg). For example, for women who weigh 143 pounds (65 kg) and who eat 4 ounces of reef fish daily, the estimated dose is 0.19 µg/kg/day. Similarly, for women who weigh 121 pounds (55 kg) and who eat 4 ounces of reef fish daily, the estimated mercury does is 0.22 µg/kg/day. These estimated doses exceed U.S.EPA's RfD of 0.1 µg/kg/day. The 99th percentile portion size is used to estimate the maximum dose for each age group. Doses are not calculated beyond this point and appear as black in Table 2A-1. Table 2-6 shows the 50th, 95th, and 99th percentile fish portion size for various age groups. Precision is difficult regarding what constitutes average fish consumption for Viequenses. Using information from Dr. Caro's survey, a typical fish intake for an adult female might be three meals a week, with a portion size of 4 ounces and an average body weight of 55 kg (121 pounds). This person would have an estimated mercury dose of 0.094 µg/kg/day, which is below U.S.EPA's RfD of 0.1 µg/kg/day.

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Estimated doses less than U.S.EPA's RfD (0.1 µg/kg/day) are shown with a white background. Residents with these estimated doses are not at risk of harmful effects from mercury in fish. Estimated doses that exceed U.S.EPA's RfD for residents who eat fish daily are shown in three shades of blue (light, medium, and dark). Women with estimated doses indicated as light blue (light gray in black and white) have a small increased risk of harming a developing fetus if they are pregnant. The risk is greater for those women with estimated doses indicated in medium blue. These estimated doses approach the dose of 1.1 µg/kg/day identified by the National Academy of Science as a dose that results in a 5% increase in the incidence of abnormal scores on the Boston Naming Test (a picture-naming, vocabulary test). The NAS effect level is consistent with the range of 0.85 to 1.5 µg/kg/day identified by the U.S.EPA as the benchmark dose lower limit ($BMDL_{05}$.)

Similarly, most of the estimated doses in children exceed U.S.EPA's RfD as indicated by light, medium, and dark blue in Table 2A-1 (Chapter 2, Section 2.6, Appendix 2A-2). Young children with higher daily fish intake have estimated doses that exceed the effect level of 1.1 µg/kg/day. These doses are in dark blue. Whether children are as sensitive to the neurotoxic effects of mercury as is the fetus is uncertain. To be protective, U.S.EPA's and FDA's national fish advisory include a warning for children as well as women who are pregnant, who plan to become pregnant, and nursing mothers.

These conclusions about the risk of harmful effects are also somewhat uncertain; to know the exact dose that might cause harmful neurological effects in the fetus and in children is difficult. This uncertainty arises from limitations inherent in human studies and because a person's susceptibility may vary. Finally, estimating the mercury dose that someone might receive is similarly uncertain. If residents eat more snapper and parrotfish, which are lower in mercury, their estimated dose of mercury will be lower; they are likely to be at lower risk of harmful effects or at no risk at all. If residents eat more grunt and hind, which are higher in mercury, their estimated dose of mercury will be higher and they could be at greater risk of harmful effects.

The conclusions and recommendations from ATSDR's evaluation of fish and shellfish data appear at the end of this chapter (Section 2.3) and in Chapter 9 of this report.

2.2.6 Possible Mixtures Effects from Chemicals in Fish and Other Biota

In ATSDR's 2003 Fish PHA, the concentrations of individual metals in Vieques reef fish were not at levels of health concern. In its evaluation of these fish data for this report, ATSDR concluded that mercury in fish is a concern for children and for the developing fetus in women who frequently eat large amounts of fish. Unfortunately, current science does not adequately support a robust analysis of multiple chemical exposures and their interactions. Debate continues in the scientific community about the best methods by which to evaluate exposure to a chemical mixture both from a single pathway and from multiple, combined pathways. In addition, estimating combined doses from multiple pathways at Vieques is hampered by a lack of knowledge of the levels of chemicals residents are exposed to through various pathways (e.g., eating seafood, ingesting soil, drinking water, and breathing air). Because of this complexity and lack of knowledge, any additional risk from any possible, so-called mixtures effect assumed by residents eating seafood is likewise uncertain.

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2.3 Conclusions and Recommendations

2.3.1 Conclusions

During ATSDR's November, 2009 meeting, several scientists raised concerns about mercury levels in fish. In particular, they were concerned that the conclusions in ATSDR's 2003 Fish PHA were inconsistent with the 2004 U.S.EPA/FDA national advisory concerning mercury. Therefore, ATSDR reviewed its 2003 conclusions and recommendations about mercury in fish from reefs surrounding Vieques.

In its 2003 public health assessment regarding fish consumption, ATSDR used fish intake rates that focused on adults who ate 8 ounces of fish daily and who weighed 70 kg (or 154 pounds). Daily fish intakes rates for children were 4 ounces and children were assumed to weigh 16 kg (or 35 pounds). Estimated doses were compared with ATSDR's MRL of 0.3 µg/kg/day, which was derived from an analysis of the Seychelles Island and Faroe Islands studies. In ATSDR's evaluation as presented in this report, ATSDR used a broad range of daily fish intakes and body weights. Using information from U.S.EPA's Exposure Factor Handbook, ATSDR assumed that daily fish intakes for adult women were as high as 14 ounces—the 99th percentile—and that women weigh as little as 46 kg (or 100 pounds). For children, ATSDR assumed that daily fish intakes were as high as 6 ounces for 1- to 2-year old children, 8 ounces for 3- to 5-year old children, 10 ounces for 6- to 14-year old children, and 20 ounces for 15- to 18-year old children. The highest intakes for children represent the 99th percentile portion size for the stated ages. In addition, ATSDR used the NAS's recommendations and U.S.EPA's Reference Dose (RfD) concerning mercury as well as human toxicity studies from the Faroe Islands. From its evaluation of mercury in Vieques fish, ATSDR reached these new conclusions:

1. ATSDR has identified mercury exposure from frequent consumption of marine seafood as a potential public health hazard. Women with a varied fish diet who typically eat more than 2 oz of fish every day have estimated mercury doses that exceed U.S.EPA's chronic RfD. As portion size increases, the estimated doses approach the lowest level known to cause harmful effects to the developing fetus. ATSDR concludes that if these women are pregnant, their developing baby has a small increased risk of neurological effects later in life. The risk of harmful effects increases as portion size increases. Possible harmful effects identified from studies of non-Viequense children exposed *in utero* involve language, attention, and memory, and to a lesser extent visual/spatial and motor functions.

2. Women who eat grunt or hind more frequently than other reef fish and who typically eat more than 2 oz of fish every day have estimated mercury doses two times higher than women who eat a varied fish diet. As portion size increases, the estimated doses approach or exceed the lowest level known to cause harmful effects in the developing fetus. ATSDR concludes that if these women are pregnant, their developing baby has a small increased risk of neurological effects later in life. The risk of harmful effects increases as portion size increases. Possible harmful effects identified from studies of non-Viequense children exposed *in utero* involve language, attention, and memory, and to a lesser extent visual/spatial and motor functions.

3. Children with a varied fish diet who typically eat more than 0.5 oz of fish every day have estimated mercury doses that exceed U.S.EPA's chronic RfD. These children have a small risk of

neurological effects. But as portion size increases, the risk of harmful effects increases. Depending upon their age, children as young as 1 year who eat 3 to 4 ounces of fish every day have estimated doses that exceed doses known to cause neurological effects and have the greatest risk of harmful neurological effects. Possible harmful effects identified from studies of non-Viequense children exposed *in utero* involve language, attention, and memory, and to a lesser extent visual/spatial and motor functions.

4. Like women, children who eat grunt and hind more frequently than other reef fish have estimated doses two times higher than children who eat a varied fish diet. The estimated doses in these children exceed the doses associated with neurological effects.

5. Some uncertainty is associated with these findings because a person's mercury response is itself somewhat uncertain. The uncertainty could be due to sex, genetics, health and nutritional status, or how mercury is handled in the body. In the three human studies that focused on mercury exposure from eating fish and seafood, the identification of the lowest-effect levels was uncertain. Estimating the mercury dose from eating reef fish was likewise uncertain, given that the dose could vary depending on the type, frequency, and quantity of fish eaten.

6. While ATSDR supports the U.S.EPA's and the FDA's national fish advisory, portions of the advisory do not apply to the Viequenses who rely heavily on local seafood. For example, the advisory discusses fish that Viequenses do not eat, such as pollock, catfish, and tilefish. In addition, the advisory recommends that if a local advisory is not available, people should not eat more 6 ounces of local fish and should not consume any other fish during the week. We include links to the advisory for informational use and we recommend an educational program about mercury in locally consumed fish.

7. Residents need information so they can select local seafood lower in mercury over seafood higher in mercury. This will protect developing fetuses and young children from mercury in fish. For informational purposes only, the advisory and related information is available at: http://www.epa.gov/waterscience/fish/advice; http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm115662.htm, and Chapter 2, Section 2.7 of this report, Appendix 2A-3.

8. For the 104 fish samples collected from the marine areas near Vieques and from the commercial fish market, the average mercury level was 0.1 ppm and the range was nondetectable to 0.33 ppm. The mercury levels detected in Vieques reef fish are similar to levels reported by the FDA (FDA 2010). The mercury levels are in the low (0.02–0.2 ppm) to mid (0.2–0.6 ppm) mercury range identified by FDA in its recent risk and benefit assessment (FDA 2009).

9. ATSDR also conducted a statistical analysis of the 2001 fish data and concluded the following:

    a. Mercury detected in the seafood is higher in two fish families (grunt and hind) compared with other families sampled (e.g., parrotfish and snapper). The average mercury concentration in grunt and hind was higher than U.S.EPA's 0.049 ppm screening level. In testing some of the hypotheses, ATSDR used the overall average of 0.1 ppm to determine which families of reef fish were likely to exceed the overall average.

b. Mercury levels in parrotfish were statistically lower than U.S.EPA's 0.049-ppm screening level and lower than the 0.1-ppm overall average.

c. All snapper were lower than U.S.EPA's 0.049 ppm screening value. But an insufficient number of snapper were collected to determine whether the average snapper-mercury level was statistically different from the U.S.EPA 0.049 ppm screening level. A sufficient number of snapper were collected to determine that the average mercury level is statistically lower than the 0.1-ppm overall average.

10. Mercury is present in most seafood and is particularly high in some fish species and low in other species. While mercury was a component of the detonators of some bombs, only small amounts of mercury were introduced to the Vieques environment from this source. This conclusion is supported by the low mercury levels in LIA soils, which appear to be at naturally occurring levels. Mercury levels in fish in and around the LIA are most likely the result of the global reservoir of mercury circulating in the environment.

11. Statistical analysis showed that some fish and shellfish from certain reefs surrounding Vieques had higher levels of some metals and lower levels of other metals—iron, aluminum, copper, zinc, arsenic, barium, potassium and selenium were all slightly higher—compared to other reefs surrounding Vieques. These metals are materials found in bombs and in metal ships, suggesting possible localized contamination. But the levels were only slightly higher and the difference was statistically significant only for some reefs compared with other reefs surrounding Vieques.

2.3.2 Recommendations

ATSDR recommends environmental and public health agencies and scientists consider the following:

1. People who frequently consume marine seafood should follow available fish advisories and fishing restrictions in Vieques. Maintain the fishing restrictions in the waters adjacent to the LIA.
2. Conduct a survey of Vieques residents to determine the types, frequency, and quantity of fish consumed.
3. Conduct additional risk assessments and statistical analyses using new information gathered from the previously recommended fish consumption survey.
4. Collect and analyze additional fish samples from Vieques should the proposed survey and statistical analysis not provide sufficient public health information. Collect sufficient fish samples to allow analysis by species and by location.
5. Develop an educational program about mercury in fish that incorporates local habits and information about Viequenses' seafood consumption. Benefits accrue to the developing fetus with maternal intake of nutrients in seafood (FDA 2009) that can outweigh the concomitant intake of small amounts of mercury. The goal of this site-specific educational program should be to educate Viequenses about the benefits of eating seafood so they can choose fish lower in mercury and still maintain their healthy dietary customs of consuming local seafood.

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2.4 References

[ATSDR] Agency for Toxic Substances and Disease Registry. 1999 March. Toxicological profile for mercury (update). Atlanta: US Department of Health and Human Services, Public Health Service.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2005. Public Health Assessment Guidance Manual (Update). Atlanta: US Department of Health and Human Services, Public Health Service; January.

Barton J, Porter J. 2004. Radiological, Chemical, and Environmental Health Assessment of the Marine Resources of the Isla de Vieques Bombing Range, Bahia Salina del Sur, Puerto Rico. Underwater Ordnance Recovery, Inc.; University of Georgia. March 8, 2004.

Bauer, L.J. and M.S. Kendall (eds.). 2010. An Ecological Characterization of the Marine Resources of Vieques, Puerto Rico Part II: Field Studies of Habitats, Nutrients, Contaminants, Fish, and Benthic Communities. NOAA Technical Memorandum NOS NCCOS 110. Silver Spring, MD.

Blackburn W, Houston J, Lotufo GR. 2004. Dietary transfer of explosives in freshwater and marine systems: comparisons to DDT. Presented at: 2004 Society of Env Toxicology and Chemistry Conference. Bloom 1992. On the chemical form of mercury in edible fish and marine invertebrate tissue. Can J Fish Aquat Sci 49(5):1010–17.

Burton DT, Cooper KR, Goodfellow WL Bioconcentration, Elimination and Metabolism of Picric and Picramic Acid in Freshwater Fish and Estuarine Bivalves. Arch Environ Contam Toxicol 1983 Oct;41(3):308-18.

Burton DT, Goodfellow WL, Cooper KR Uptake, elimination, and metabolism of14C-picric acid and14C-picramic acid in the American oyster (Crassostrea virginica) Arch Environ Contam Toxicol 1984 V 13, No 6, November, 1984.

Caro DA, Nazario M, Díaz N. 2000. Toxicological Survey of Heavy Metals in Fish Populations, Vieques Island. Universidad Metropolitana, School of Environmental Matters. June 2000.

Cooper KR, Burton DT, Goodfellow WL, Rosenblatt DH. 1984. Bioconcentration and metabolism of picric acid and picramic acid in rainbow trout. J of Toxicology and Environmental Health 14:731-747.

Crump KS, Kjellstrom T, Shipp A, et al. 1998. Influence of prenatal exposure upon scholastic and psychological test performance: Benchmark analysis of a New Zealand Cohort. Risk Anal 18(6):701–13.

Davidson PW, Myers GJ, Cox C, et al. 1998. Effects of prenatal and postnatal methylmercury exposure from fish consumption on neurodevelopment: Outcomes at 66 months of age in the Seychelles child development study. JAMA 280(8):701–07.

Debes F, Budtz-Jorgensen E, Weihe P, White RF, Grandjean P. 2006. Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years. Neurotoxicol Tereatol 28:363–75.

[FDA] Food and Drug Administration. 2004. What you need to know about mercury in fish and shellfish: 2004 EPA and FDA advice for women who might become pregnant, women who are pregnant, nursing mothers, and young children. Available at:

http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm115662.htm [accessed 2012 Aug 27].

[FDA] Food and Drug Administration. 2010. Mercury concentrations in fish: FDA monitoring program. http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm191007.htm, and http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm115644.htm [accessed 2012 Aug 27].

[FDA] Food and Drug Administration. 2009. Report of quantitative risk and benefit assessment of consumption of commercial fish, focusing on fetal neurodevelopmental effects (measured by verbal development in children) and on coronary heart disease and stroke in the general population, January 15, 2009. Available at: http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm088794.htm. [accessed 2012 Aug 27].

Geo-Marine, Inc. 2002. Ex-USS Killen Site Investigation and Biological Characterization, Vieques Island, Naval Station Roosevelt Roads, Puerto Rico. June 2002.

García N, Lopez AM, Soto M, Rosado S, Berrios B. 2000. Environmental impact of Navy activities in Vieques. San Juan, PR: Servicios Cientificos y Tecnicos, Inc.; July.

Grandjean P, Weihe P, White RF, Debes F, et al. 1997. Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. Neurotoxicol Teratol 19:417–28.

Greib TM, Driscoll CT, Gloss SP, et al. 1990. Factors affecting mercury accumulation in fish in the upper Michigan peninsula. Environ Toxicol Chem 9:919–30.

Hélène DM, Augagneur S, Budzinski H, Mora P, Narbonne J, Garrigues P. 2003. Microcosm tributylin bioaccumulation and multibiomarker assessment in the blue mussel, Mytilus edulis. Environ Toxicology and Chemistry 22:2679–686.

Houston JG and Lutufo GR. 2005. Dietary exposure of fathead minnows to the explosives TNT and RDX and the pesticide DDT using contaminated invertebrates. Int J of Environ Res Public Health 2(2):286-292.

Lotufo GR and Lydy J. 2005. Comparative toxicokinetics of explosive compounds in sheepshead minnows. Env Contam and Toxicology 49:206-214.

Lotufo GR, Lydy J, Wild W. Toxicokinetics and toxicity of explosive compounds in fish. Presented at: Society of Env Toxicology and Chemistry Conference; 2005.

Myers GJ, Davidson PW, Cox C, Shamlaye CF, Palumbo D et al. 2003. Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study. The Lancet 361:1686–692.

Myers GJ, Thurston SW, Pearson AT, Davidson PW et al. 2009. Postnatal exposure to methylmercury from fish consumption: a review and new data from the Seychelles Child Development Study. Neurotoxicol 30;338–49.

Nipper M, Carr RS, Biedenbach JM, Hooten RL, Miller K, Saepoff S. 2001. Development of marine toxicity data for ordinance compounds. Environ Contam Toxicology 41(3):308-313.

[NOAA] National Oceanic and Atmospheric Administration 2010. An ecological characterization of the marine resources of Vieques, Puerto Rico. Available at: http://ccma.nos.noaa.gov/ecosystems/coralreef/vieques/, [accessed 2012 Aug 27].

[NRC] National Research Council. 2000. Toxicological Effects of Methymercury. Washington (DC): National Academy of Sciences, National Research Council. Pubs: National Academy Press.

Ownby DR, Belden JB, Lotufo GR, Lydy M. 2005. Accumulation of TNT in aquatic organisms: Part 1 – Bioconcentration and distribution in channel catfish (Ictalurus punctatus). Chemosophere 58(9):1153-1159.

Riegl B, Moyer RP, Walker BK, Kohler K, Gilliam D, and Dodge RE. 2008. A Tale of Germs, Storms, and Bombs: Geomorphology and Coral Assemblage Structure at Vieques (Puerto Rico) Compared to St. Croix (U.S. Virgin Islands). Journal of Coastal Research 24(4):1008–1021.Risher J. 2010. March 23rd e-mail from John Risher, ATSDR/DTEM to Dr. David Mellard, ATSDR/DHAC.

Rodríguez Sierra CJ, Jimenez B. 2002. Trace metals in striped majarra fish (*Diapterus plumier*) from Puerto Rico. Marine Pollution Bulletin 44;1039-1045.

State of California. 2009. Draft health advisory: safe eating guidelines for fish from Lake Sonoma (Sonoma County) and Lake Mendocino (Mendocino County) August 2006. Available at: www.oehha.ca.gov/fish/so_cal/pdf_zip/DSonomaMendo081706.pdf [2012 Aug 27].

[USEPA] US Environmental Protection Agency. 1997. Exposure factors handbook; August.

[USEPA] US Environmental Protection Agency. 2000. Guidance for assessing chemical contaminant data for use in fish advisories. Fish sampling and analysis. Third Edition, Volume 1. Available at: http://water.epa.gov/scitech/swguidance/fishshellfish/techguidance/risk/upload/2009_04_23_fish_advice_volume2_v2cover.pdf [accessed 2012 Aug 27].

[USEPA] U.S. Environmental Protection Agency/Environmental Response Team (EPA/ERT). 2001. Field Data Summary, Vieques Fish Assessment, Vieques, Puerto Rico. November 8, 2001.

[USEPA] US Environmental Protection Agency. 2004. Consumption advice, joint federal advisory for mercury in fish. Available at: http://www.epa.gov/waterscience/fish/advice/factsheet.html [accessed 2012 Aug 27].

[USEPA] US Environmental Protection Agency. 2012. Methylmercury, IRIS Database. US Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0073.htm [accessed 2012 Nov 14].

Yoo L, Houston J, Gibson A, Faulkner A, Stevens J. Distribution and target tissues of TNT in fathead minnow. Presented at: Society of Env Toxicology and Chemistry Conference; 2002 Nov 16-20.

Yost SL, Pennington JC, Brannon JM, Hayes CA. 2007. Environmental process descriptors for TNT, TNT-related compounds and picric acid in marine sediment slurries. Marine Pollution Bulletin 54:1262–66.

2.5 Appendix 2A-1: Statistical Analysis of ATSDR Fish Data

2.5.1 Addressing the Statistical Tests and Sample Numbers

Any investigation has strengths and weaknesses. The strengths of the 2001 sampling supported by ATSDR were

- It repeated the analysis of metals in market fish conducted by the Universidad Metropolitana, School of Environmental Matters (Caro et al. 2000).
- It provided previously unavailable, specific information about fish. Such information included measurements of explosive compounds in fish, measurements of contaminants in fish captured on specific reefs around the island (including reefs with visible signs of military presence), and measurements of contaminants in shellfish captured on and around the island.

The limitations were

- By collecting only larger specimens of the popular reef species, the average concentrations might have been biased high, which might have resulted in a mercury overestimate.
- Because larger fish were targeted and the reefs had only a few larger species, fish within the same family had to be combined to provide statistical significance. Consequently, these data could not establish differences in concentration between most species within a family. Fish within a family were grouped to provide sufficient number to represent each family at a sampling location. Although this was adequate to represent the concentrations of most metals in the families, more appropriate ways to address mercury were available. We determined that mercury concentrations were highly dependent on species within a family. For example, white grunt had lower mercury concentrations than the rest of the fish in the grunt family, and red hind had lower mercury concentrations than others in the hind family.

2.5.2 Sample Design

The original sample design did not focus only on mercury, but mercury was included. ATSDR and U.S.EPA proposed a sampling strategy to 1) evaluate many metals and explosive chemicals in fish, and 2) compare chemicals in fish caught in the various reefs with fish purchased at the market (USEPA 2001). From each location, the specimens collected were approximately the same size. The School of Environmental Matters collected 52 fish of varying sizes, with little variation in results. Each of these datasets provided unique and meaningful information about mercury and other metals.

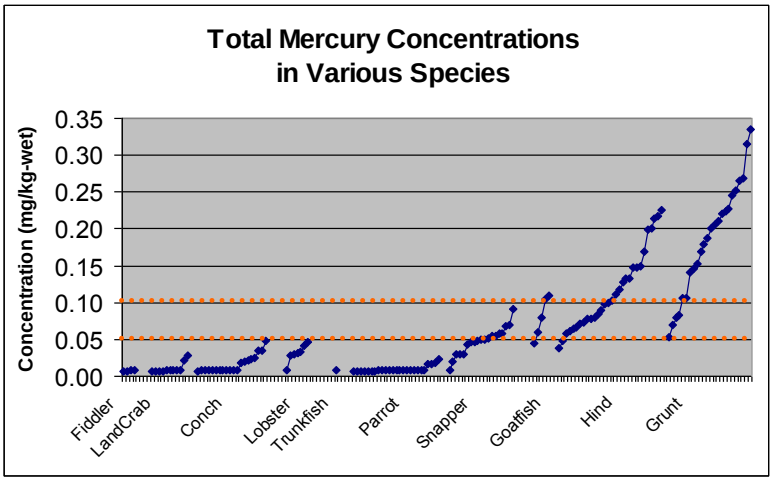
Statistical tests confirmed sufficient fish for numerous evaluations. U.S.EPA provided statistical tests to compare fish by location and by family for all metals (USEPA 2001), and ATSDR provided statistics to compare fish family with health-related concentrations for mercury. A summary of ATSDR's testing is provided below.

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2.5.3 Summary of Statistics for Health-Related Hypothesis Testing

Hypothesis tests were designed to determine whether mean mercury concentrations were above or below 0.1 ppm and above or below 0.049 ppm. Figure 2A-1 of total mercury in fish by family illustrates many of the results of the statistical tests.

Figure 2A-1. Total mercury in fish by family



Blue squares represent concentrations for individual fillet sample. The horizontal dotted line at 0.049 ppm (close to the 0.05 label) connotes the EPA screening value for subsistence populations and the horizontal dotted line at 0.1 ppm connotes the site-specific mean methylmercury level in Viequenses fish calculated using ATSDR's 2001 fish data.

A statistical analysis of the data supports the following conclusions:

1. Most of the goatfish, hind, and grunt had total mercury concentrations above the U.S. EPA screening level of 0.049 ppm;
    a. The mean goatfish concentration of 0.0797 ppm was statistically higher than 0.049 ppm;
    b. The mean hind concentration of 0.116 ppm was statistically higher than 0.049 ppm;
    c. The mean grunt concentration of 0.185 ppm was statistically higher than 0.049 ppm;
2. Many of the hind and grunt had total mercury concentrations above the average level of mercury measured in all Viequense fish analyzed for this report, 0.1 ppm.
    a. But the mean hind concentration of 0.116 ppm was not statistically different than 0.1 ppm;
    b. Follow-up review of the species shows that red hind are lower (with a mean concentration of 0.091 ppm—also not statistically different from 0.1 ppm)
    c. The mean grunt concentration of 0.185 ppm was statistically higher than 0.1 ppm;
    d. Follow-up review of the species shows that Bluestriped grunt and French grunt were higher with mean concentrations of 0.22 ppm (each) and that white grunt averaged 0.098 ppm (therefore, white grunt were not statistically different from 0.1 ppm)

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3. Many of the snapper and goatfish had total mercury levels in the range between the two levels (i.e., 0.049 ppm and 0.1 ppm):
    a. The snapper concentration of 0.0478 ppm was not statistically different from 0.049 ppm, but was statistically lower than 0.1 ppm;
    b. The mean goatfish concentration of 0.0797 ppm was statistically higher than 0.049, but not statistically different from 0.1 ppm;
4. The remaining fish families had total mercury levels below the 0.049 ppm level (many of those results were statistically significant):
    a. The mean lobster concentration of 0.0317 ppm was statistically lower than 0.049 ppm.
    b. The highest mercury concentration in 25 parrotfish was 0.0231 ppm, less than half of the 0.049 ppm screening level. Because mercury was not detected in most samples, the concentrations were not normally distributed; we therefore performed nonparametric tests using the median. The median of 0.00767 ppm has 95% confidence limits of 0.00747 and 0.00801 ppm, with a coverage of 95.7%, lower than 0.049 ppm.
    c. The highest mercury concentration in 11 composite land crab samples was 0.0287 ppm—less than the 0.049-ppm screening level. The concentrations were not normally distributed; mercury was not detected in many samples. We therefore identified that the median 0.00823 ppm (with its 95% confidence limits of 0.00725 and 0.0216 ppm and 96.1% coverage) was lower than 0.049 ppm.
    d. The highest mercury concentration in 20 conch samples was 0.0486 ppm, lower than 0.049 ppm. The conch concentrations were not normally distributed—mercury was not detected in many samples. We therefore found that the median 0.008277 ppm (with its 95% confidence limits of 0.00803 and 0.0179 ppm and 95.9% coverage) was well below 0.049 ppm.
    e. Four composite fiddler crab samples were normally distributed, with a mean concentration of 0.0077 ppm—statistically lower than 0.049 ppm.

We had only one trunkfish sample—insufficient for statistical analysis.

2.5.4 Testing Limitations

Testing of the thresholds had a high bias: larger fish were used, and total mercury was used to represent methylmercury. More samples in some locations would provide stronger results. But to collect enough samples for the test levels chosen (0.049 and 0.1 ppm) could be cost prohibitive, as some families had averages very close to those levels.

2.5.4.1 Summary of Statistics for Location Testing

Statistical analyses were conducted on the mercury (and other metals) levels in fish and invertebrate tissues to determine whether the locations were statistically different (USEPA 2001). Tissue from hind,

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grunts, parrotfish, snappers, conch, and land crabs were included in the analyses. Because of the limited numbers collected at most sampling locations, fiddler crabs, goatfish, and lobster were not tested.

The mercury statistical test found high influence of species and size. Even with these influences, the range of concentrations within families was relatively small. Thus any differences by location are hard to determine. Because the range of mercury concentrations was small and the influence of species and size was large, we cannot predict the number of samples needed to determine any differences between the levels found in the fish at each location. Further statistical tests are provided below.

2.5.5 Results of the Tests (Mercury focus)

A multivariate analysis of variance (MANOVA) was computed for mercury (and other metals). Individual analysis of variance (ANOVA) results indicated a statistical significance between several metals at several locations. For mercury, U.S. EPA found statistically lower levels of mercury near the LIA compared with some other locations. We later found, however, that the significance was due to differences between species within the groups.

Table C1 in Appendix C of U.S.EPA (2001) includes a complete summary of all comparisons between locations, parametric and nonparametric, for all families. The following is an abbreviated list of the summary of the mercury statistic tests provided by U.S.EPA in Appendix C of U.S.EPA (2001):

- Hind: A MANOVA was computed for mercury. Individual ANOVA results indicated a statistical significance between locations for other metals, but not for mercury. The statistical reports for hind are listed under “grouper” in U.S.EPA (2001) p C3.1.
- Grunt: A MANOVA was computed for mercury. Individual ANOVA results indicated a statistical significance between locations for some metals, but not for mercury.
- Snapper: A MANOVA was computed for mercury. Individual ANOVA results indicated a statistical significance between locations for some metals, but not for mercury.
- Parrotfish: Kruskal-Wallis’s nonparametric comparison of the locations indicated no significant difference ($p<0.05$) for mercury in comparisons between locations.
- Conch: Mercury at Esperanza was higher than the LIA North, LIA South, and the West End. Kruskal-Wallis’s nonparametric comparison of the locations resulted in significant probability values ($p<0.05$) from comparisons between locations for mercury. Tukey’s multiple comparisons on the ranks resulted in two distinct patterns for mercury: LIA-North (1), LIA-South (2), and the West End (6) were grouped together; Esperanza (3) was grouped separately.

  Land Crabs: Because more than 50% of mercury results were below the detection level, they were evaluated using nonparametric methods. Nonparametric testing indicated no significant difference between mercury levels at the locations.

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Table C1 from U.S.EPA's report (USEPA 2001) for locations 1 through 6.

Table C1. Results of Statistical Comparisons Between Sampling Locations, Target Species, and Analytes
Vieques Fish Assessment
Vieques, Puerto Rico
November 2001

Species: Locations Compared:	Probability Values					
	Conch 1, 2, 3, 6	Grouper 1, 2, 3, 4, 5, 6	Grunt 1, 2, 3, 5, 6	Land Crab 1, 2	Parrot 1, 2, 3, 4	Snapper 2, 3, 4, 5, 6
Aluminum	**0.0103** *	**0.0013** *	**<0.0001**	0.0508	**<0.0001**	**0.0284** *
Antimony	NE	NE	NE	NE	NE	NE
Arsenic	**0.0001**	**0.0053**	**0.0039**	NE	**0.0089** *	0.1415 *
Barium	**0.0069**	**0.0132** *	0.6473	0.1802	0.3259	0.5276
Beryllium	**0.0102** *	**0.0042** *	**0.0036** *	NE	**0.0045** *	**0.0103** *
Cadmium	**0.0435**	NE	NE	NE	0.8510 *	NE
Calcium	**0.0452**	**0.0050**	0.1826	**0.0498**	0.6766	0.7062
Chromium	0.6119	0.2764	0.0695	0.1676	0.0921	0.0919
Cobalt	0.1436 *	0.6493 *	0.1645 *	0.9168 *	0.7840 *	**0.0213** *
Copper	**0.0103**	0.1008	**0.0073**	0.6475	0.2887	0.1939
Iron	NU	0.1862 *	0.2557	**0.0070**	**0.0085** *	**0.0008**
Lead	**0.0086** *	0.0238 *	0.0792 *	0.0758 *	0.4978 *	0.3115 *
Magnesium	**0.0413**	0.5051	**0.0060**	0.0683	0.3419	0.2079 *
Manganese	0.6318	**0.0072** *	**0.0119** *	**0.0053**	0.9230	**0.0405** *
Mercury	**0.0195** *	0.3412	0.0728	0.1745 *	0.2011 *	0.1599
Nickel	NE	0.1960 *	NE	NE	NE	0.239 *
Potassium	0.8085	0.3901	**0.0064**	NE	**0.0256**	0.6473
Selenium	0.1064	**0.0002**	**0.0066**	**0.0162**	**0.0002**	0.2604
Silver	0.0558	NE	0.4209 *	**0.0160** *	0.3060 *	0.3214 *
Sodium	**0.0028**	0.7279	0.9993	0.2840	0.1410	0.2308
Thallium	NE	NE	NE	NE	NE	NE
Vanadium	0.6715 *	NE	NE	0.0864	0.8214 *	NE
Zinc	0.4960	**<0.0001**	0.3596	**0.0315**	**0.0063**	**<0.0001**

* - Non-parametric analyses conducted
NE - not evaluated - 100% of analytical results were below the method detection limit
NU - Analytical results were not useable

Statistical analyses conducted at an alpha- level of 0.05. Probability values <0.05 listed in bold type.

2.5.6 Testing Limitations

Because of peer-review comments, we performed additional tests. Because the grunt family averaged higher than 0.1 ppm, we tested to determine whether all grunt species were higher. Four French grunt were caught near the west end and nowhere else; all were over 0.1, with the lowest sample having 0.14 ppm and the 95% confidence interval being 0.13 ppm. These French grunt were much smaller than the other species of grunts. French grunt, even at full size, are small and tend to forage more near sand than near grasses at night when they leave the reef, resulting in different exposures from other grunt. Because French grunt had relatively high mercury levels for their size, they introduce a bias into the grunt family. This reduced our ability to compare all grunt at all reefs. Bluestriped grunt were caught at three reefs, North LIA, South LIA, and Esperanza, with similar averages (between 0.21-0.24 ppm). Furthermore, White grunt were statistically lower than Bluestriped grunt and French grunt. The market

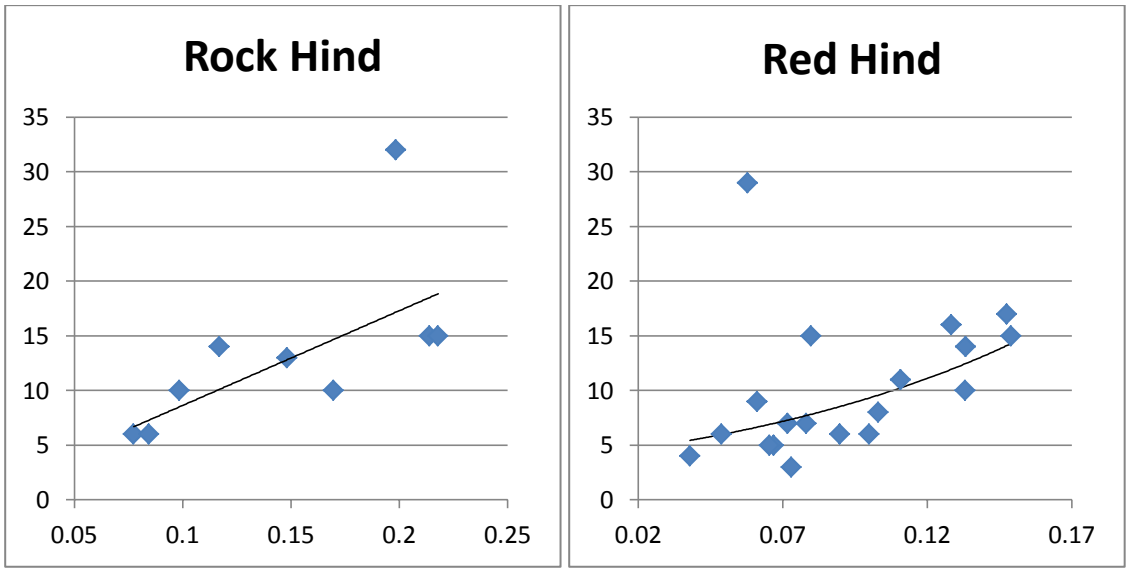
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only had white grunt for the investigation. We found no statistical difference between the Bluestriped grunt at the two LIA locations compared with those caught at Esperanza.

In Chapter 2, Section 2.2.2 we discussed the hind family as one case where it appeared that a location (location 1) had different (in this case lower) concentrations than other locations. We also mentioned additional statistical tests to determine other reasons for location 1 having lower levels. Looking into this family, we want to compare other species factors and to determine whether weight could have influenced the results.

We had sufficient samples within the hind family only to evaluate further the red hind and the rock hind. The red hind from the market, from Isabel Segunda, and from the North LIA averaged less than the rock hind from Esperanza and the South LIA (from Table 2-5). The three rock hind from the west end had the lowest average mercury levels for that species (0.057 ppm). Rock hind were found at two reefs, South LIA (averaging 0.13 ppm) and Esperanza (averaging 0.16 ppm). After accounting for species differences, we can determine whether weight influences the concentration by looking at concentrations within one species. Figure 2A-2 is a scatterplot of all the rock hind weights compared with their mercury concentrations.

Figures 2A-2a and 2A-2b. Weight (in oz) vs. mercury concentration (ppm)



The graphs suggest a relationship between fish size and mercury concentration. This was not evident in the fish Caro collected. Because ATSDR intentionally selected larger fish, the size variation is insufficient to make this comparison for all fish.

From the mercury tests, we concluded that only conch showed statistical differences by location. Those differences were that conch on the LIA-North, the LIA-South, and the West End had statistically less mercury than the conch collected in Esperanza. Even the Esperanza conch, however, were below screening levels.

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2.5.7 Mercury: Three Human Studies and U.S.EPA's Reference Dose

Several human studies have evaluated the neurological effects of methylmercury exposure in children. A long-term human study of children from the Faroe Islands, a small group of islands in the North Atlantic Ocean affiliated with Denmark, began in 1986 and focused on children born to women who lived on the islands. This population relies heavily on seafood and whales as a protein source. The investigators used various tests that monitor child development. They concluded that at birth, cord blood mercury levels in the mother were associated with harmful effects in children at age 7 years involving language, attention, and memory, and to a lesser extent visual/spatial and motor functions (Grandjean et al. 1997). Follow-up studies at age 14 years showed similar findings (Debes et al. 2006).

In 1978, New Zealand was the site of another human study. It focused on 61 children who were exposed *in utero* to high mercury levels that resulted from their mother's consumption of four or more fish meals a week. If the authors omitted one outlier, the data showed a decrease in children's intelligence quotient (IQ) at age 6 with increasing exposure to methylmercury as measured by their mother's hair mercury levels at birth (Crump 1998). The third study came from the Republic of Seychelles, where 85% of the population relied on local seafood for protein. Average ocean fish consumption in this population was 12 meals a week (Davidson 1998). The Seychelles study initially did not find harmful effects in children as they grew older. In one recent publication, the investigators reported that two of 21 endpoints (one positive and one negative) were associated with prenatal methylmercury exposure. The authors stated that these outcomes were probably due to chance. They concluded that their data did not support a neurodevelopment risk from prenatal methylmercury exposure from eating fish (Myers 2003). In another paper, the authors reported that they found several associations between postnatal methylmercury exposure and children's developmental endpoints. However, the investigators concluded that no consistent pattern of associations emerged to support a causal relationship (Myers 2009). In some cases in which several tests were used to evaluate a single domain, and only one of those tests was positive, the Seychelles investigators did not believe they could base a firm conclusion on only one of several tests for that domain. Rather, they believed that if a domain was adversely affected, more than one test should show it (Risher 2010).

More information about the harmful effects of methylmercury is available in ATSDR's Toxicological Profile for Mercury (ATSDR 1999).

U.S.EPA developed a methylmercury Reference Dose using a mathematical model that estimated a 5% adverse response in children for neurological effects.[16] Using the Faroe Islands study, U.S.EPA concluded that the methylmercury concentration in maternal cord blood that caused a 5% adverse response in children ranged from 46 to 79 ppb. This methylmercury concentration in maternal blood equated to a range of 0.8 to 1.5 µg methylmercury per kilogram per day (µg /kg/day) as a dietary intake. The doses were divided by an uncertainty factor of 10 to arrive at the Reference Dose of 0.1 µg/kg/day. The U.S.EPA's approach is consistent with the approach used by the National Academy of Sciences. The NAS recommended that U.S.EPA use the Faroe Islands Study and 58 ppb methylmercury in cord blood for

[16] More precisely, U.S.EPA estimated the lower 95th confidence limit of the concentration of methylmercury in maternal blood that gave a 5% response for neurological effects in offspring at 7 years of age.

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deriving its health guideline (NRC 2000). U.S.EPA's methods for deriving the agency's RfD are described in detail at http://www.epa.gov/iris/subst/0073.htm (USEPA 2012).

The National Academy of Sciences concluded that some risk is possible regarding anticipated harmful effects in children exposed *in utero* to 58 ppb methylmercury in cord blood. The cord blood concentration of 58 ppb methylmercury equates to 12 ppm methylmercury in maternal hair (NRC 2000).

2.6 Appendix 2A-2. Estimated Doses in Residents from Eating Vieques Fish

Table 2A-1. Estimated mercury dose for women and children of various body weights from eating Vieques reef fish. +

Approximate Age			Body Wt lbs	Body Wt kg	Estimated Mercury Dose in µg/kg/day													
18 yr and older	A		220	100	0.02	0.03	0.06	0.09	0.12	0.15	0.18	0.21	0.24	0.30	0.36	0.42		
18 yr and older			198	90	0.02	0.03	0.07	0.10	0.13	0.17	0.20	0.24	0.27	0.34	0.40	0.47		
18 yr and older	D		176	80	0.02	0.04	0.08	0.11	0.15	0.19	0.23	0.26	0.30	0.38	0.45	0.53		
18 yr and older			154	70	0.02	0.04	0.09	0.13	0.17	0.22	0.26	0.30	0.35	0.43	0.52	0.60		
18 yr and older	U		143	65	0.02	0.05	0.09	0.14	0.19	0.23	0.28	0.33	0.37	0.46	0.56	0.65	0.74	0.93
17 yr and older		C	132	60	0.03	0.05	0.10	0.15	0.20	0.25	0.30	0.35	0.40	0.50	0.60	0.71	0.81	1.01
16 yr and older	L		128	58.1	0.03	0.05	0.10	0.16	0.21	0.26	0.31	0.36	0.42	0.52	0.62	0.73	0.83	1.04
15 yr and older			121	55.1	0.03	0.05	0.11	0.16	0.22	0.27	0.33	0.38	0.44	0.55	0.66	0.77	0.88	1.10
14 yr and older	T		121	54.8	0.03	0.06	0.11	0.17	0.22	0.28	0.33	0.39	0.44	0.55				
13 yr and older		H	112	50.9	0.03	0.06	0.12	0.18	0.24	0.30	0.36	0.42	0.47	0.59				
12 yr and older	S		102	46.4	0.03	0.07	0.13	0.20	0.26	0.33	0.39	0.46	0.52	0.65				
11 yr		I	92	41.8	0.04	0.07	0.14	0.22	0.29	0.36	0.43	0.51	0.58	0.72				
10 yr			79	36.1	0.04	0.08	0.17	0.25	0.33	0.42	0.50	0.59	0.67	0.84				
9 yr			70	31.9	0.05	0.09	0.19	0.28	0.38	0.47	0.57	0.66	0.76	0.95				
8 yr		L	61	27.9	0.05	0.11	0.22	0.32	0.43	0.54	0.65	0.76	0.87	1.08				
7 yr			55	25	0.06	0.12	0.24	0.36	0.48	0.60	0.73	0.85	0.97	1.21				
6 yr			49	22.1	0.07	0.14	0.27	0.41	0.55	0.68	0.82	0.96	1.09	1.37				
5 yr		D	43	19.6	0.08	0.15	0.31	0.46	0.62	0.77	0.93	1.08	1.23					
4 yr			37	17	0.09	0.18	0.36	0.53	0.71	0.89	1.07	1.24	1.42					
3 yr			33	14.9	0.10	0.20	0.41	0.61	0.81	1.01	1.22	1.42	1.62					
2 yr		R	29	13	0.12	0.23	0.46	0.70	0.93	1.16	1.39							
1 yr			24	10.8	0.14	0.28	0.56	0.84	1.12	1.40	1.68							
< 1 yr		E	19.4	8.8	0.17	0.34	0.69	1.03	1.37	1.72	2.06							
< 1 yr		N	9.9	4.5	0.34	0.67	1.34											
				fish kg/day	0.014	0.028	0.057	0.085	0.113	0.142	0.170	0.198	0.227	0.284	0.340	0.397	0.454	0.567
				fish oz/day	0.5	1	2	3	4	5	6	7	8	10	12	14	16	20

+ Men with the same weight and fish intake have the same estimated dose as women. *These estimated doses are for teenagers 15 to 18 years who have a 99th percentile fish-meal portion of 20 ounces. The 99th percentile fish meal portion for adult women is 14 ounces.

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2.7 Appendix 2A-3. The Joint U.S.EPA and FDA National Fish Advisory

What You Need to Know About Mercury in Fish and Shellfish, March 2004:

2004 EPA and FDA Advice for Women Who Might Become Pregnant, Women Who Are Pregnant, Nursing Mothers, and Young Children

Fish and shellfish are an important part of a healthy diet. Fish and shellfish contain high-quality protein and other essential nutrients, are low in saturated fat, and contain omega-3 fatty acids. A well-balanced diet that includes a variety of fish and shellfish can contribute to heart health and children's proper growth and development. So, women and young children in particular should include fish or shellfish in their diets due to the many nutritional benefits.

However, nearly all fish and shellfish contain traces of mercury. For most people, the risk from mercury by eating fish and shellfish is not a health concern. Yet, some fish and shellfish contain higher levels of mercury that may harm an unborn baby or young child's developing nervous system. The risks from mercury in fish and shellfish depend on the amount of fish and shellfish eaten and the levels of mercury in the fish and shellfish. Therefore, the Food and Drug Administration (FDA) and the Environmental Protection Agency (EPA) are advising women who may become pregnant, pregnant women, nursing mothers, and young children to avoid some types of fish and eat fish and shellfish that are lower in mercury.

By following these three recommendations for selecting and eating fish or shellfish, women and young children will receive the benefits of eating fish and shellfish and be confident that they have reduced their exposure to the harmful effects of mercury.

1. Do not eat shark, swordfish, king mackerel, or tilefish because they contain high levels of mercury.
2. Eat up to 12 ounces (2 average meals) a week of a variety of fish and shellfish that are lower in mercury.
    a. Five of the most commonly eaten fish that are low in mercury are shrimp, canned light tuna, salmon, pollock, and catfish.
    b. Another commonly eaten fish, albacore ("white") tuna has more mercury than canned light tuna. So, when choosing your two meals of fish and shellfish, you may eat up to 6 ounces (one average meal) of albacore tuna per week.
3. Check local advisories about the safety of fish caught by family and friends in your local lakes, rivers, and coastal areas. If no advice is available, eat up to 6 ounces (one average meal) per week of fish you catch from local waters, but do not consume any other fish during that week.

Follow these same recommendations when feeding fish and shellfish to your young child, but serve smaller portions.

Chapter 3 Biomonitoring

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Chapter 3 Summary

The following chapter describes and evaluates the available biomonitoring studies of Vieques residents. These studies were conducted since 1999 by Puerto Rican scientists and physicians or by the Commonwealth's Department of Health. They consist of measurement of various metals in residents' blood, urine, hair, or feces. Data from these studies reported elevated levels of some metals in residents' blood, urine, or hair. While some of these elevated levels might be explained by cigarette use, seafood consumption, or hair dyes, they did not account for all the elevated levels. These studies were unable to investigate each person's environment to identify the metals' source for those residents who had excessive levels in hair, urine, or blood. The PRDOH study came closest to identifying possible sources but acknowledges an inability to identify the source or sources for all residents with excessive metals exposure.

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Viequenses may be exposed to mercury in fish and cadmium in pigeon peas. These exposures may warrant additional environmental investigations, such as sampling locally grown produce for cadmium and gathering more information about fish consumption and possibly mercury in fish. The information could be used to decide whether to undertake human testing for mercury and cadmium in blood or urine. If other environmental exposures are identified, additional human biomonitoring investigations may be considered. More detailed information about ATSDR's recommendations concerning fish and locally grown produce can be found in Chapter 2, Section 2.3.2 and Chapter 5, Section 5.3.2, respectively. ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time because we found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation. Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing. A qualified laboratory should do the testing and analysis. CDC/ATSDR can provide a list of qualified laboratories that can perform the tests. And if requested , CDC/ATSDR can provide information to healthcare providers about tests for metals in biologic samples.

3.1 Introduction to Biomonitoring

Biomonitoring is the measurement of metals or chemicals in a biological matrix such as blood, urine, or hair. It can sometimes be a useful method of evaluating environmental chemical exposure in people. Biomonitoring examples include measuring lead in blood, cadmium in urine, and mercury in hair. The concentration of environmental chemicals in a biological matrix may allow assessment of excessive exposure. In some cases, the levels of environmental chemicals could be predictive of the risk of harmful effects occurring in the population. Depending on the chemical, biomonitoring studies can provide insight into a population's exposure over the previous days, weeks or months, or even years. Scientists usually use biomonitoring to assess populations at risk for exposure (e.g., average exposures or the prevalence of unusually high exposures). Less often, biomonitoring can be useful in assessing the exposure of a single person.

But like all exposure assessments, biomonitoring has limitations and requirements. These fall into five categories:

- Many chemicals are not measureable with current technology.
- Measurements require defined precision and accuracy to be applicable.
- Proper timing and collection techniques are required. In fact, they are essential to interpretation, appropriate comparison, or identification of reference groups.

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- Although biomonitoring can assist in exposure assessment, the toxicological significance of biomonitoring results is sometimes not possible to ascertain.

Biomonitoring results from Vieques can be compared with national data, such as the Fourth National Report on Human Exposure to Environmental Chemicals (CDC 2009). But more appropriate comparisons should include comparison with biomonitoring data from residents of the main island of Puerto Rico. These limitations are described in more detail later in this chapter.

3.2 Assessment of Available Vieques Human Biomonitoring Studies Data

Because ATSDR's previous public health assessments did not review biomonitoring data from Vieques, this chapter and its appendices describe the biomonitoring data in detail. Because of confidentiality issues, ATSDR does not have access to individual results. Thus our evaluation relies on information reported by the investigators. The important fact is, however, that biomonitoring data available for Viequenses cannot be used to determine whether residents of Vieques were exposed to past, military exercise-related constituents.

Since 1999, either the Puerto Rico Department of Health or Puerto Rican scientists and doctors have conducted several human biomonitoring studies in Vieques. These studies have focused on measuring metals in hair, urine, blood, or stool samples. They are listed in Table 3-1 (Colón de Jorge undated; Ortiz Roque 2004; Ortiz Roque 2002; PRDOH 2006; Rodríguez Sierra 2009). Highlights are provided here. The studies are described in more detail in Appendix 3-A.

Dr. Carmen Colón de Jorge conducted one of the first studies. She analyzed hair samples from 30 residents with preexisting health conditions and analyzed seven stool samples for metals. At about the same time, Dr. Carmen Ortiz Roque's study analyzed hair samples from about 200 Viequenses for various metals. The doctors concluded their data showed Viequenses had heavy metal exposure and that the exposure had resulted in harmful health effects (Colón de Jorge; Ortiz Roque 2002, 2004).

Dr. Colón de Jorge reported numerous metals in hair. That report stated that based on the ranges provided by the service laboratory that made the measurements (i.e., Doctor's Data, Chicago, Illinois) a sizeable percentage of the 30 tested residents exceeded the reference range established by Doctor's Data. Dr. Colón de Jorge noted that in about 70% of the 30 tested residents, arsenic and antimony in hair were above the reference ranges selected by the reporting laboratory. Dr. Colón de Jorge's report stated that because antimony levels in bombs are high, the antimony exceedences proved residents were exposed to metals from munitions. The report, however, did not explain how residents could have been exposed to antimony or to other metals.

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Table 3-1. Vieques human biomonitoring studies

Source	*Title*
Puerto Rico Department of Health	Executive summary of the prevalence study of heavy metals in Vieques, 2006, draft unpublished manuscript
Dr. Carmen Ortiz Roque	Mercury contamination in reproductive age women in a Caribbean island: Vieques. J. Epidemiol Community Health 58:756-757, 2004
Dr. Carmen Ortiz Roque	Heavy metal exposure and disease in the proximity of a military base. Unpublished manuscript, 29 January 2002
Dr. Carmen Colón de Jorge	Innocence battered on Vieques, scientific investigation of toxic metals present in the biological terrain of Vieques children and adults and their effects on nutrient minerals utilizing hair and feces analysis (preliminary report). Unpublished manuscript, no date
Dr. Carlos Rodríguez Sierra	Webcast, October 30, 2009, New look at the opening of the case of Vieques, www.telecoqui.net

Most commercial laboratories that report results will develop their own reference range for what they consider normal or typical concentrations of a chemical in a biological matrix. But such ranges can vary with the populations tested. Inevitably, exposures will vary from population to population based on factors such as age, sex, race/ethnicity, and underlying health conditions. Laboratories will sometimes develop a concentration that they consider above the expected range or that they sometimes even consider toxic. Often for these ranges (and, if reported, for the toxic levels) the basis is the company's own cumulative experience from many years of laboratory testing. Such reference ranges or identified toxic levels are not regulated by the government nor are they approved by a medical or scientific organization.

Dr. Ortiz Roque's study identified several residents with hair-mercury levels above 12 ppm—the level the National Research Council identified as causing a 5% increase in abnormal results on a neurocognitive test (i.e., the Boston Naming test) for children who were exposed *in utero* (NRC 2000).

Dr. Ortiz Roque showed that higher mercury levels were associated with Viequenses' higher fish consumption (Ortiz Roque 2002, 2004); but, the patients in this investigation were not randomly selected. Thus, insofar as the prevalence of Viequenses' elevated mercury levels is concerned, any firm conclusions are difficult to draw. That said, Dr. Ortiz Roque's identification of three residents with hair mercury levels above 12 ppm is an important finding. A major limitation of hair analysis in a free-living population is the difficulty in discerning whether the metal was *internally* deposited from the blood stream or *externally* deposited from the air or the use of products on the hair. While hair measurements for mercury have been used in previous population studies, efforts to understand or control external deposition are usually investigated.

From 2006 to 2008, Dr. Carlos Rodríguez Sierra collected hair, nail, and urine samples from 52 Viequense adults. He divided the study subjects into high and low fish-consumption groups. This division is necessary because persons who eat fish regularly will have high levels of urinary arsenic. This arsenic is predominantly arsenobetaine—a nontoxic arsenic form. An October 30, 2009 Webcast sponsored by

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Casa Pueblo de Adjuntas carried a report that high levels of arsenic in hair, nails, and urine were not detected at the time of sampling. Hair and nail sample levels were below 1-µg/g (microgram per gram), which Dr. Rodríguez Sierra used as a reference value (Rodríguez Sierra C. 2009). The Webcast further reported that the sum of inorganic arsenic species and their metabolites in urine was less than 50 µg/g creatinine, which Dr. Rodríguez Sierra also used as a reference value. This value likely comes from occupational exposure studies; they might have used the value to assess excessive exposure to arsenic (ACGIH 2000; WHO 2001). The American Conference of Governmental Industrial Hygiene (ACGIH) currently recommends 35 µg/g creatinine as an action level of the sum of inorganic arsenic species and their metabolites for occupational testing (ATSDR 2007).

Dr. Rodríguez Sierra reported that the median and 95th percentile of total inorganic arsenic (As V, As III, DMA, MMA) levels in Viequenses' urine were slightly above the respective levels reported in the 2003–2004 Fourth National Report on Human Exposure to Environmental Chemicals.[17] Dietary sources of inorganic arsenic include chicken as well as rice, particularly when rice is grown in arsenic-contaminated soils (Lasky et al. 2004; Potera 2007; Williams et al. 2007). Whether these slightly higher levels of inorganic arsenic in urine are associated with harmful effects, however, is impossible to determine.

As a follow up to these studies, PRDOH conducted a comprehensive biomonitoring study in which it collected hair, urine, or blood samples from 500 randomly selected Viequenses. The results were shared with some of those who participated, but no public report was ever released (PRDOH 2006). In August 2009, ATSDR obtained a draft of the unpublished manuscript. ATSDR found that inappropriate units were used for some of the results, thus rendering some measurements uncertain.

Nevertheless, the PRDOH manuscript reported that in 20% of the participants, either aluminum, lead, or mercury in blood; uranium, cadmium, or nickel in urine; or nickel or arsenic in hair exceeded Quest Laboratories' reference range.[18] For some—but not all—of the elevated levels, the PRDOH manuscript identified as possible sources cigarettes, hair dyes, and seafood consumption. For other residents, the origin of high metal levels in urine, hair, and blood could not be identified. As mentioned previously, the elevated levels of metals (except mercury) in hair are difficult to interpret. Hair analysis leaves unanswered whether the metals were ingested and were bound to the hair from direct contact. Note also that the PRDOH manuscript stated that they did not test children 4 years and younger.

All that said, however, Dr. Colón de Jorge's and Dr. Ortiz Roque's biomonitoring studies did support the conclusions in the PRDOH manuscript.

[17] The basis for CDC's Fourth National Report on Human Exposure to Environmental Chemicals is an ongoing national survey that samples the U.S. population every 2 years. Each 2-year sample consists of about 2,400 persons. The *Fourth Report* includes findings from national samples for 1999–2000, 2001–2002, and 2003–2004. The data are analyzed separately by age, sex, and race/ethnicity groups. The Fourth Report is available at www.cdc.gov/exposurereport/.

[18] While the draft PRDOH manuscript states that Quest Laboratories in San Juan, PR conducted the clinical analyses, the correct name is most likely Quest Diagnostics.

3.3 Strengths and Limitations of Human Biomonitoring for Vieques

The use of biomonitoring results to assess public health issues requires consideration of biomonitoring's previously enumerated strengths and limitations. Biomonitoring data will allow an investigator to look at markers of exposure and markers of effect. When toxicological information is available, markers of effect allow an investigator to determine whether harmful consequences might result from the measured level of a chemical or its metabolite. Extensive toxicologic data support current research on lead in blood, cadmium in urine, and mercury in hair. Once the concentration is known, health scientists can draw conclusions about the presence or absence of risk. For other metals in blood or urine, toxicological data and research are limited, thus reducing the ability to draw firm conclusions. For example, no human toxicity data are available for urine levels of antimony, beryllium, cesium, molybdenum, platinum, and tungsten. Thus while biomonitoring might identify populations with excessive exposure, assessing the risk of that exposure's harmful effects might not be possible (CDC 2009).

But a particular biomonitoring strength is the availability of comparison data for a representative sample of the U.S. population. Those comparison data are in the National Reports on Human Exposure to Environmental Chemicals at http://www.cdc.gov/exposurereport/ (CDC 2009). National data are also available on mercury in hair (McDowell 2004).

An important consideration when using National Health and Nutrition Examination Survey (NHANES) biomonitoring data is that differences in levels between populations (e.g., regional, cultural, and ethnic) are likely due to differences in exposures, pharmacokinetics, and other factors. Thus U.S. population data can only be cautiously applied to the Commonwealth of Puerto Rico. If any future biomonitoring investigations are undertaken for the Vieques population, a comparison population from the Commonwealth of Puerto Rico or another nearby island-based population may be more appropriate.

To understand possible exposure sources, subjects with an elevated metal or chemical concentration should be subsequently investigated for attributable sources of exposure.

3.4 The Special Case of Hair Analysis for Metals and Other Elements

3.4.1 ATSDR's 2001 Hair Panel

During ATSDR's November, 2009 meeting with invited scientists, we were asked to reconsider the agency's position on testing metals in hair. In 2001, ATSDR sponsored a workshop and invited researchers in the medical, environmental, and toxicological fields to advise the agency of the utility of measuring metals and organic chemicals in hair. The workshop panelists agreed on the following summary statement related to the overall usefulness of hair analysis in evaluating environmental exposures:

> For most substances, insufficient data currently exist that would allow the prediction of a health effect from the concentration of the substance in hair. The presence of a substance in hair may indicate exposure (both internal and external), but does not necessarily indicate the source of exposure (ATSDR 2001).

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The panelists agreed that a relationship between contaminant concentrations in hair and any kind of measurable outcome—provided external contamination could be ruled out—have only been established for methylmercury and, to a limited extent, for arsenic (e.g., segmental analysis for forensic analysis). Unique forensic settings might be available for other substances (ATSDR 2001).

The panelists identified several factors that limit interpretation of even the most accurate, reliable, and reproducible laboratory results. These include

- A lack of reference (or background) ranges in which to frame the interpretation of results.
- Difficulties in distinguishing endogenous (internal) from exogenous (external) contamination in hair.
- Some panelists thought the then-current literature suggested the absence of any reliable washing method capable of separating external contamination from internal element deposition. One potential remedy was that where possible, identifying metabolites—or other unique markers of internal exposure—for substances of interest would be most helpful in distinguishing internal and external contamination.
- A lack of understanding of how and to what extent environmental contaminants are incorporated into the hair.
- A lack of correlation between levels in hair and blood and other target tissues, as well as the lack of epidemiologic data linking substance-specific hair levels with adverse health effects.
- Little pertinent information is available for the study of environmentally relevant organic compounds in hair. The panel recommended taking advantage of what is known about hair analysis for testing for drugs of abuse (ATSDR 2001).

3.4.2 Current Status of Hair Analysis Validity

ATSDR staff checked with several sources to determine the current validity of hair analysis. The American Medical Association (AMA) supports the conclusions of those panelists who participated in ATSDR's 2001 workshop (AMA 2009). The German Federal Environment Agency (GFEA 2005) has a similar recommendation. One exception is carefully tested hair specimens for mercury. The NHANES survey includes mercury in hair from a national population for one 2-year period (1999–2000). Therefore, comparison of hair mercury levels with a national group is possible. This might help in identifying those with excessive mercury exposure.

3.5 Conclusions and Recommendations

3.5.1 Conclusions

1. Since 1999, Vieques has hosted at least five human biomonitoring investigations. Puerto Rican scientists, physicians, or the Puerto Rico Department of Health (PRDOH) conducted all of them. The PRDOH has conducted the most extensive sampling effort, collecting biological specimens from 500 randomly selected Viequenses. The PRDOH manuscript reported that in 20% of the participants, either

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a. Aluminum, lead, or mercury in blood;

b. Uranium, cadmium, or nickel in urine; or

c. Nickel or arsenic in hair

exceeded the laboratory's reference range. The PRDOH manuscript identified cigarette use and hair dyes, as well as the consumption of seafood, as possible sources for some but not all the elevated levels. The PRDOH manuscript acknowledged that for some residents, it could not identify the source of high metal levels in urine, hair, and blood. The PRDOH manuscript did not report mercury levels in hair. Results from Dr. Ortiz Roque's investigations showed that some residents had elevated levels of mercury in hair, and that the most likely source was fish consumption; other possible sources, however, were not completely ruled out.

2. Data from these studies showed that in blood, urine, hair, or feces, some residents of Vieques had elevated levels of various metals. While some of these elevated levels might be explained by cigarette use, seafood consumption, or hair dyes, they did not account for all the elevated levels. In particular, biomonitoring results from Dr. Ortiz Roque showed that some Viequenses had elevated mercury in hair above 12 ppm, the level identified by the NAS to cause harm in 5% of fetuses exposed *in utero*. Dr. Ortiz Roque also showed that mercury in hair was associated with fish consumption. In contrast, the PRDOH study did not find excessive mercury levels in blood, although the study either did not measure for or did not report mercury level in hair. Thus some uncertainty exists about the prevalence of high mercury levels in Viequenses. Except for mercury, metal content in hair is difficult to interpret. Metals can bind directly to hair from the use of commercial hair products, making difficult any distinction between internal metal exposure via ingestion and inhalation versus external exposure resulting from contact with the metal in the environment (e.g., shampoo, dyes, dirt).

3. These studies were unable to investigate each person's environment to identify the source for those who had excessive metals in hair, urine, or blood. The PRDOH study came closest to identifying possible sources (e.g., cigarette and hair dye use, seafood consumption) but acknowledged an inability to identify the source or sources for all residents with excessive metals exposure. Either through the survey instrument or through an in-home visit, it may be possible to identify other sources that increase metal exposure, such as cooking utensils, metal residues in foods (e.g., tea and vegetables), consumption of drinks with metallic packaging, religious rites using mercury-containing capsules, skin creams, antacid formulations and antiperspirant formulations.

4. ATSDR remains cautious in making decisions about using hair as an indicator of exposure to environmental contaminants and as an indicator of risk of harmful effects. A major problem in interpreting metal concentrations in hair is whether the metal content resulted from internal exposure (e.g., from ingestion or inhalation) or from external exposure (e.g., the hair coming in contact with a metal-containing product). Currently, no washing method is capable of removing exogenous metal contaminants while leaving endogenous metals undisturbed. Chemicals such as methylmercury, which originate generally from dietary sources, suffer less from this

drawback, provided unusual sources of inorganic mercury do not complicate the picture, (e.g., mercury vapor in occupational or home settings).

5. These biomonitoring results do not permit any conclusions about exposure to the bombing-related contaminants.

3.5.2 Recommendations

1. Viequenses may be exposed to mercury in fish and cadmium in pigeon peas. These exposures may warrant additional environmental investigations, such as sampling locally grown produce for cadmium and gathering more information about fish consumption and possibly mercury in fish. The information could be used to decide whether to undertake human testing for mercury and cadmium in blood or urine. If other environmental exposures are identified, additional human biomonitoring investigations may be considered. More detailed information about ATSDR's recommendations concerning fish and locally grown produce can be found in Chapter 2, Section 2.3.2 and Chapter 5, Section 5.3.2, respectively.

2. ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time because we found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation.

3. Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare providers to discuss the need for and cost of testing. A qualified laboratory should do the testing and analysis.

4. CDC/ATSDR can provide a list of qualified laboratories that can perform the tests. If requested, CDC/ATSDR can provide information to healthcare providers about tests for metals in biologic samples.

3.6 References

[ACGIH] 2000. Threshold limit values for chemical substances and physical agents and biological exposure indices. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.

[AMA] American Medical Association. 2009. Health and ethics policies of the AMA House of Delegates. Available at: http://www.ama-assn.org/ad-com/polfind/Hlth-Ethics.pdf [accessed 2012 Aug 28].

[ATSDR] Agency for Toxic Substances and Disease Registry. 2001. Summary report, hair analysis panel discussion: exploring the state of the science. Atlanta: US Department of Health and Human Services, Public Health Service; 12-13 Jun.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007. Toxicological Profile for Arsenic [Update]. Atlanta: US Department of Health and Human Services, Public Health Service; August.

Caldwell KL, Jones RL, Verdon CP, Jarrett JM, Caudill SP, Osterloh JD. 2009. Levels of urinary total and speciated arsenic in the US population: National Health and Nutrition Examination Survey 2003-2004. J Exp Sci Environ Epi 19:59-68.

[CDC] Centers for Disease Control and Prevention. 2001. Blood and hair mercury levels in young children and women of childbearing age—United States, 1999. MMWR 50(8):140-3; 2March.

[CDC] Centers for Disease Control and Prevention. 2009. Fourth National Report on Human Exposure to Environmental Chemicals. Available at: http://www.cdc.gov/exposurereport [accessed 2012 Aug 28].

[CDC] Centers for Disease Control and Prevention. 2009. The Third National Report on Human Exposure to Environmental Chemicals. National Center for Environmental Health, Atlanta, GA. NCEH Pub. No. 05-0570. Available at: http://www.cdc.gov/exposurereport [accessed 2009 Nov 12].

[CDC] Centers for Disease Control and Prevention. 1997. Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials, Atlanta, GA.

[CDC] Centers for Disease Control and Prevention. 2002. Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention, Atlanta, GA

Colón de Jorge C, Santiago E, Brooks JF, Pérez FL, Rivera J, Valle R. (Undated). Innocence battered on Vieques: scientific investigation of toxic metals present in the biological terrain of Vieques children and adults and their effects on nutrient minerals utilizing hair and feces analysis (preliminary report). (Unpublished manuscript.)

[GFEA] German Federal Environment Agency (Umweltbundesamt). 2005. Hair analysis in environmental medicine. Gesundheitsschutz 48(2);246–50. Available at: http://www.umweltdaten.de/gesundheit-e/monitor/hair_analysis.pdf [accessed 2012 Aug 28].

Lasky T, Sun W, Kadry A, Hoffman M. 2004. Mean total arsenic concentrations in chicken 1989-2000 and estimated exposures for consumers of chicken. Envir Health Perspect 112(1):18-21.

Mahaffey KR, Clickner RP, Jeffries RA. 2009. Adult women's blood mercury concentrations vary regionally in the United States: association with patterns of fish consumption (NHANES 1999–2004). Environ Health Perspect 117(1):47–53.

McDowell MA, Dillon CF, Osterloh J, Bolger M, Pellizzari E, et al. 2004. Hair mercury levels in U.S. children and women of childbearing age: reference range data from NHANES 1999–2000. Environ Health Perspect 112(11):1165-71.

[NRC] National Research Council. 2000. Toxicological Effects of Methymercury. Washington (DC): National Academy of Sciences, National Research Council. Pubs: National Academy Press.

Ortiz Roque C, López-Rivera Y. 2004. Mercury contamination in reproductive age women in a Caribbean island: Vieques. J. Epidemiol Comm Health 58:756–57.

Ortiz Roque C. 2002. Heavy metal exposure and disease in the proximity of a military base. (Unpublished manuscript) 2002 Jan 29.

Potera C. 2007. U.S. rice serves up arsenic. Envir Health Perspect 115(6):A296.

[PRDOH] Commonwealth of Puerto Rico, Department of Health. 2006. Executive summary of the prevalence study of heavy metals in Vieques. San Juan: PR DOH; November.

Rodriquez Sierra C. 2009. New look at the opening of the case of Vieques. Webcast sponsored by Casa Pueblo de Adjuntas (www.casapueblo.org) on October 30, 2009 from this url: www.telecoqui.net.

[USEPA] US Environmental Protection Agency. 2012. Methylmercury, IRIS Database. US Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/iris/subst/0073.htm [accessed 2012 Nov 14].

Williams PN, Raab A, Feldman J, Meharg AA. 2007. Market basket survey shows elevated levels of As in South Central U.S. processed rice compared to California: Consequences for human dietary exposure. Envir Sci & Tech 41(7):2178-2183.

[WHO] World Health Organization. 2001. Arsenic and arsenic compounds. Environmental Health Criteria 224, Geneva.

3.7 Appendix 3-A. Biomonitoring

3.7.1 Vieques, Puerto Rico-Related Human Biomonitoring Study Summaries

Several biomonitoring investigations have been conducted on Vieques, with the first samples collected in 1999 (see Table 3A-1). These investigations are summarized in Table 3A-1 and described in detail in the text of this appendix (Colón de Jorge undated; Ortiz Roque 2004; Ortiz Roque 2002; PRDOH 2006; Rodriquez Sierra 2009).

Table 3A-1. Vieques human biomonitoring studies

Source	*Title*
Puerto Rico Department of Health	Executive summary of the prevalence study of heavy metals in Vieques, 2006, draft unpublished manuscript
Dr. Carmen Ortiz Roque	Mercury contamination in reproductive age women in a Caribbean island: Vieques. 2004. J. Epidemiol Comm Health 58:756–57
Dr. Carmen Ortiz Roque	Heavy metal exposure and disease in the proximity of a military base. Unpublished manuscript, 29 January 2002
Dr. Carmen Colón de Jorge	Innocence battered on Vieques, scientific investigation of toxic metals present in the biological terrain of Vieques children and adults and their effects on nutrient minerals utilizing hair and feces analysis (preliminary report). Unpublished manuscript, undated
Dr. Carlos Rodriquez Sierra	Webcast, October 30, 2009, New look at the opening of the case of Vieques, www.telecoqui.net

3.7.2 PRDOH Biomonitoring Study

The most extensive human biomonitoring study conducted to date is a two-phase study of 500 Vieques residents conducted by the Puerto Rico Department of Health starting in May 2004. This study was conducted after ATSDR's public health assessments were published and so represent new data to consider. For this reason, the study is described in detail.

In the first phase, the PRDOH manuscript reported interviewing 500 Vieques residents, collecting biological samples, and analyzing for the following:

- Arsenic, cadmium, and nickel in hair and urine,
- Aluminum, lead, and mercury in blood, and
- Uranium in urine.

As stated in the PRDOH manuscript, the second phase involved collecting and analyzing blood samples from Phase 1 participants with metal concentrations deemed toxic according to the reference values provided by the analytical laboratory. Phase II was from December 2005 to March 2006. The PRDOH manuscript stated that the information obtained from the study would be used to determine the magnitude and scope of the problem, generate hypotheses and identify areas that warrant intervention from a public health perspective. The PRDOH report includes recommendations for follow-up activities.

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3.7.2.1 PRDOH Study, Phase 1

Five hundred persons were randomly chosen from residents 5 years of age or older who lived in Vieques before 2000. The interview collected information about age, sex, residence history, smoking status, employment, water consumption and use, food preparation and consumption, medicines, hobbies, and chemicals/solvent use. In particular, information was collected about fish and shellfish consumption 3 days before the sample collection.

To reduce external contamination from the environment, hair samples were collected from the pubic region or, if that was not possible, a 1-inch hair sample from the scalp was collected near the root. Table 3A-2 provides a summary of metal concentrations in blood, urine, and hair from Phase 1. Note that comparing pubic hair results with scalp hair may not be appropriate.

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Table 3A-2. PRDOH Phase 1 results of heavy metal analysis of Vieques residents.

Note: The geometric means for some metals (e.g., uranium) in the study population appear to be the average of detectable concentrations. For other metals (e.g., mercury), nondetectable levels were replaced by zero while for other metals (e.g., aluminum), nondetectable levels were replaced by 1 µg/L. For cadmium, the report states that nondetect levels were replaced by a value but did not specify the value. The treatment of nondetects is very important when calculating means and comparing those means to either a reference or toxicity value or to a national average. Complicating the comparisons even further is that for some metals, the detection limit was high. When nondetectable levels are part of the database, the calculation of a geometric mean could be biased high or low depending upon what value was substituted for the nondetect. Therefore, caution is warranted when comparing these study population means to means from reference populations, who are likely to have handled nondetectable concentrations differently or had lower detection limits. For example, the NHANES means replace nondetectable concentrations with the detection limit divided by the square root of two. For some metals, the limitations of the detection limit are acknowledged in the PRDOH report.

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	% Detection	*Geometric Mean of PRDOH Sample (n=500)*[1]	*Geometric Mean General Population Selected by PRDOH*	*Geometric Mean 2003-2004 NHANES*[2]	*Level of Possible Toxicity (as reported by service laboratory)*	*Comments*
Aluminum in blood µg/L	93	17.6 (unreliable, see comment)	1-4[3]	NA	≥ 60[5]	Quest Laboratories reports a high detection limit (10 µg/L), which adds uncertainty to the reported geometric mean of 17.6 because 1 µg/L was used for non-detect values. Nevertheless, some persons appear to have elevated Al in blood; 109 persons ≥ 40 µg/L ; 10 persons ≥ 60 µg/L
Lead in blood µg/dL	99	3.53	1.66[4]	1.43	20 µg/dL[5]	Six persons ≥ 10 µg/dL; 2 persons ≥ 20 µg/dL Median age = 56 years; All males; 3 work with petroleum products No safe level identified; CDC reference value, 5 µg/dL
Mercury in blood µg/L	99	5.02 (unreliable, see comment)	1.02[6]	0.797	46-79[7]	The PRDOH report states that the detection limit (5 µg/L) was too high to estimate a reliable population mean; Maximum level detected was 13 µg/L. While the draft report states that zero was used for non-detects, it is uncertain if this is actually the case for the average of 5.02 µg/L as reported in the PRDOH manuscript.
Total arsenic in urine µg/L	95	33.6	38.2[4]	8.3	> 50 µg/L[5]	23% > 50 µg/L (117 of 500) 39 of 257 persons who did not eat fish had urine arsenic > 50 µg/L. Another possible source of urinary arsenic is rice.

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	% Detection	*Geometric Mean of PRDOH Sample (n=500)*[1]	*Geometric Mean General Population Selected by PRDOH*	*Geometric Mean 2003-2004 NHANES*[2]	*Level of Possible Toxicity (as reported by service laboratory)*	*Comments*
Arsenic in hair ppm	65	0.15	0.15[4]	NA	--	One person > 3 ppm
Uranium in urine µg/L	1	0.14 (6 individuals)	0.007[4]	0.008	--	Six women with detectable levels. Mean of these 6 women is 0.14 µg/L. Nondetectable concentrations were not included in calculating the mean.
Cadmium in hair ppm	82	0.14	<0.15[4]	NA	>3.4[5]	No participant exceeded 3.4 µg/L
Cadmium in urine µg/g	87	0.43	0.307[6,8]	0.211	>2[5]	Eleven persons >1.03 µg/g Two persons >lab's toxicity levels of 2 µg/g—for some persons, probably smoking-related
Nickel in urine	79	2.05	< 2[4]	NA	>100[5]	Three persons exceeded the lab's threshold reference range. No participant exceeded the lab's toxicity levels of 100 µg/g creatinine
Nickel in hair	97	0.64	0.39[4]	NA	--	

1 Nondetectable samples were handled in different ways for various averages. For aluminum, 35 missing values were replaced by 1, which is the minimum concentration expected in a population. For cadmium in urine, 64 values were replaced by a value, although the actual value was not stated in the report. For uranium, the average consists of just the six samples with detectable concentrations. A low percent detection indicates that many people had nondetectable levels, thus the mean of the detectable levels is uncertain

2 NHANES National Survey, 2003-2004. This 2-year period was chosen because Phase I of the PRDOH study was conducted in May 2004.

3 PRDOH 2006 report. Levels reported by Poison Line of Puerto Rico

4 PRDOH 2006 report, source not identified

5 Source = Quest Laboratories

6 Source = 1999-2002 NHANES

7 $bmdl_{05,}$ Source = U.S.EPA (IRIS, Methylmercury)

8 The value 0.307 µg/L could not be confirmed from NHANES

NA = not available

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Aluminum: The geometric mean blood aluminum was 17.6 µg/L (95% CI 17.02–18.34); Quest Laboratories reported, however, the detection limit for aluminum in blood was 10 µg/L. Because the PRDOH manuscript reported using 1 µg/L for 35 nondetectable levels, some uncertainty exists in the geometric mean of 17.6 µg/L. The PRDOH manuscript reported using a geometric mean for aluminum in blood in the United States of 1 to 4 µg/L, which was provided by the Poison Line of Puerto Rico. Twenty-two percent (109 persons) showed levels of 40 µg/L or greater—the laboratory's upper threshold level of the reference range for a population. Two percent (10 persons) had levels greater than 60 µg/L, the laboratory's toxicity threshold. Persons between the ages of 20 and 44 were five times more likely to have aluminum levels above 40 µg/L than those 65 and older (PRR=4.99, $p<0.05$). Medicines and occupation were not identified as possible sources.

Lead: The geometric mean blood lead level for all ages in the study population was 3.53 µg/dL compared with the geometric mean of 1.66 µg/dL from the 1999-2000 NHANES survey (as reported in the PRDOH manuscript) and 1.42 µg/dL from the 2003-2004 NHANES survey. In addition, 16% (81/500) of participants (all ages) had blood lead levels greater than 5 µg/dL compared with 5% from NHANES. Older participants (46–64 years and 65+) were more likely to have levels greater than 5 µg/dL than were the youngest age group tested (5 to 19 years). Participants who worked with metals and with petroleum-derived products, and those lacking a water purification system were more likely to have elevated blood lead levels. For example, the following occupations or hobbies showed a high prevalence risk ratio for elevated blood lead levels: car/truck maintenance, electrical repair, metalwork, solvent use, petroleum product use, or metal use. Smokers and participants who consumed seafood within 3 days of the test showed a higher probability of elevated blood lead levels. The PRDOH manuscript stated, however, that smoking did not account for all participants with elevated blood lead levels. This conclusion is supported by the observation that the geometric mean of blood lead levels for nonsmokers was twice the national average. ATSDR agrees with this conclusion.

Mercury: A comparison of average blood mercury levels in Vieques residents to other populations is not reliable—the detection limit for mercury in blood was too high (i.e., 5 µg/L). The NHANES survey finds the average blood mercury level for the U.S. population is 1 µg/L. But coastal populations have higher fish intake and a higher frequency of women with elevated blood mercury levels compared with the national average and inland areas. For example, 8% of women in coastal areas have blood mercury levels greater than 5.8 µg/L compared with 2% of women from inland areas (Mahaffey 2009). Interestingly, Mahaffey reported blood mercury levels by race/ethnicity as well: almost 16% of women whose ancestry is Asian, Native American, Pacific Islands, and the Caribbean Islands have blood mercury greater than 5.8 µg/L. The highest blood level detected in the PRDOH study was 13 µg/L.

To provide perspective, U.S.EPA estimated a benchmark-dose, lower confidence limit of a 5% ($BMDL_{05}$) adverse response rate in fetal development. U.S.EPA identified the $BMDL_{05}$ cord blood mercury level in women to be between 46–79 µg/L (USEPA 2012). U.S.EPA derived this range of blood mercury levels using human data from the Faroe Islands fish-eating study and the agency's benchmark dose methods (USEPA 2012). U.S.EPA's values are similar to those derived by the National Academy of Sciences' (NAS) Committee on the Toxicological Effects of Methymercury. Using a similar benchmark dose approach, the NAS identified the 5% adverse effect level (i.e., a benchmark dose, BMD) at 85 µg/L as a cord blood

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mercury level and estimated a 5% lower confidence limit of 58 µg/L ($BMDL_{05}$). NAS stated that corresponding values in hair result in a benchmark dose of 17 ppm and $BMDL_{05}$ of 12 ppm (NRC 2000).

Arsenic: The PRDOH manuscript stated that the geometric mean for arsenic in urine was below what was expected (see Table 3A-2, 33.6 vs. 38.2 µg/L). The report did not state the source of the reference levels. The geometric mean of the study population is higher than the geometric mean from the 2003-2004 NHANES survey (33.6 vs. 8.3 µg/L. Total urine arsenic levels were above 50 µg/L in 25% of the participants (i.e., 117/473). The median age of participants with levels greater than 50 µg/L was 50 years (range = 5 to 90 years). The PRDOH manuscript stated that risk factors associated with elevated urine arsenic were the age groups 45–64 and over 65, smokers, seafood eaters, seafood consumption within 3 days of the test, regular contact with animals, and those who worked with metals.

Still, the urine arsenic test did not distinguish between inorganic arsenic and organic arsenic. When people eat fish a few days before providing a urine sample, nonharmful organic arsenic arsenobetaine is often found in urine at high levels. Freshwater and saltwater fish contain the organic arsenicals, arsenobetaine, and to a much lesser extent, arsenocholine.

Whenever total arsenic levels exceed 50 µg/L and people eat fish, the high levels of total arsenic are probably the result of measuring arsenobetaine and arsenocholine and thus do not indicate toxicity (Caldwell et al. 2009). Arsenobetaine and arsenocholine are excreted very quickly through the kidneys without being metabolized and are relatively nontoxic to humans. Some shellfish contain small amounts of inorganic arsenic in addition to organic arsenic; consequently, the arsenic levels reported in the PRDOH study are likely to be mostly organic arsenic with some inorganic arsenic

The PRDOH report stated that 267 participants did not eat seafood and that 39 of these participants had urine total arsenic levels above 50 µg/L. This result points to another source of arsenic, or it points to dietary recall problems. Other dietary sources of arsenic in the human diet include chicken as well as rice, particularly when rice is grown in arsenic-contaminated soils (Lasky et al. 2004; Potera 2007; Williams et al. 2007).

Uranium: Six of the 500 participants had detectable levels of uranium in urine. The geometric mean uranium level in urine in these six participants was 0.14 µg/L, which is greater than the 2003-2004 NHANES geometric mean of 0.008 µg/L and the 95th percentile of 0.031 µg/L. All six participants are women, and five were not employed when interviewed. The median age was 57. No uranium levels exceeded Quest Laboratories' threshold level or toxic level. The source of uranium is unknown.

Cadmium: The geometric-mean hair-cadmium level in the study population (n=429) was 0.14 ppm. The PRDOH report states that this value is similar to its reference value of less than 0.15 ppm. But the report does not identify the source of the reference value of less than 0.15 ppm. In any event, no participant exceeded Quest Laboratories' hair toxicity threshold of 3.4 ppm.

The PRDOH report stated that the geometric mean cadmium level in urine in the study population was higher than the 1999-2000 NHANES geometric mean (0.43 vs. 0.31 µg/g). Because of interference from molybdenum oxide, CDC has since corrected the 1999-2000 NHANES value. The corrected geometric mean from the 1999-2000 NHANES is 0.181 µg/g. The geometric mean from the 2003-2004 NHANES is 0.21 µg/g. Eleven participants had cadmium urine levels above 1.03 µg/g, the 95th percentile from the

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1999-2000 NHANES (uncorrected for molybdenum oxide). Two participants showed urine cadmium levels above the laboratory's toxic level of 2 µg/g creatinine. OSHA's worker action level is 3 µg/g creatinine. The median age for participants with urine cadmium levels above 1.03 µg/g creatinine was 55. Smokers showed urine cadmium levels five times higher than did nonsmokers.

Nickel: Participants' geometric mean nickel levels in urine (2.05 µg/g creatinine) was similar to the reference value (less than 2 µg/g creatinine). The report does not identify the source of the reference value. Three participants (2 women and 1 man) had urine nickel levels that exceeded Quest Laboratories' threshold of 6.2 µg/g creatinine in women and 10.2 µg/g creatinine in men. No urine nickel levels exceeded the Quest Laboratories' 100 µg/g- creatinine toxicity threshold.

The geometric mean for hair-nickel levels in participants (0.64 ppm) was much greater than the reference level (0.39 ppm) reported in the PRDOH manuscript. The PRDOH manuscript did not identify the source of this reference level. Sixty-three participants had hair nickel levels exceeding the laboratory's 2-ppm threshold level. Of these, 80% were women. In addition, participants in age groups 5–19 and 20–44 showed higher levels than did persons over 65 years. Risk factors associated with higher nickel hair levels included dyed hair, colorants and pigments use, and solvent use. Most of the elevated nickel levels in hair were probably from external contact via hair treatment.

3.7.2.2 PRDOH Discussion of Phase 1

At the total population level, the average concentration of some metals was above expected norms as established by Quest Laboratories (i.e., the laboratory's reference value) but the average concentrations for the population were not above toxic levels as also stated by Quest Laboratories. The PRDOH manuscript stated that this suggests the need for further investigation concerning the various risk factors, with particular emphasis on how smoking contributes to metal body burden. In addition, a better understanding is needed for how fish consumption affects metal body burdens, especially for arsenic and mercury. The PRDOH manuscript stated that eating fish within 3 days of a test or smoking could partly explain some but not all of the elevated metal body burdens in participants. The PRDOH manuscript stated that smoking was associated with lead in blood, and arsenic and cadmium in urine. In addition, seafood consumption was associated with mercury in blood, and arsenic in hair and urine.

The PRDOH manuscript pointed out that the percentage of people with high metal levels who were neither exposed to tobacco smoke nor exposed from eating seafood was still greater than expected. For example, the PRDOH manuscript stated that 10% of nonsmokers had urine cadmium levels above 5 µg cadmium per gram creatinine where only 5% was expected. The PRDOH report did not clarify whether these nonsmokers lived with smokers. Using information from the survey, the PRDOH manuscript identified factors or sources that were associated with elevated metals. These factors or sources include age, sex, hair dyes, and years living on Vieques, and soil contact, as well as working with petroleum products, solvents, metals, or animals. Table 9 from the PRDOH manuscript provides information about the estimated number of residents with elevated levels of metals in residents' blood, urine, or hair.

The PRDOH manuscript stated the following:

- Being an adult between the ages of 20 to 65 was associated with higher aluminum levels in blood.

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- Being an adult over age 61 and working with animals was associated with higher arsenic in urine.
- Working with petroleum products was associated with higher blood lead levels.
- Contacting the earth's crust and working with solvents was associated with higher cadmium in urine.
- Being a female; dying hair; using colorants, pigments, and solvents; and years living in Vieques was associated with higher nickel in hair.

What follows is Table 9 from the PRDOH manuscript.

Table 9 from Heavy Metal Prevalence Study, Vieques, 2004

Table 9. Summary of heavy metal findings; estimate of persons affected in the population and their possible exposure factors, Heavy Metal Prevalence Study, Vieques, 2004.

METAL	Prevalence n (%)	Estimate of Persons with Above-Normal Levels in the Community Universe=9,106	Exposure
Aluminum in Blood	23(4.6)	419	Age 20-65
Lead in Blood	6 (1.2)	109	Petroleum products Smoking
Mercury in Blood	7 (1.4)	127	Consumption of fish
Arsenic in Urine	117 (23.4)	2.130	Older than age 61 Smoking Consumption of fish Working with animals Use of metals
Cadmium in Urine	22 (4.4)	401	Earth's crust Work with solvents Smokes > 10 cigarettes per day
Nickel in Urine	3 (0.60)	55	-
Uranium in Urine	6 (1.2)	109	-
Arsenic in Hair	1 (0.23)	21	Consumption of fish
Cadmium in hair	0	0	-
Nickel in hair	63 (12.7)	1,154	Female Hair dying Use of colorants and pigments Solvents Years living in Vieques

The PRDOH manuscript stated that further research was needed to understand how metal body burden in Vieques residents compared with Puerto Rico. In addition, research was needed concerning the possible long-term effects of high body burdens of metals in some Viequenses.

At the individual level, the PRDOH manuscript stated that according to Quest Laboratories, 15 persons showed toxic levels of metals:

- 10 had toxic levels of aluminum in the blood,

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- Two had toxic levels of blood lead,
- Two had toxic levels of cadmium in the urine, and
- One had toxic levels of mercury in blood.

Quest recommended follow-up tests and medical examinations for these persons and investigation of their environments for possible metals sources.

It is not clear why the PRDOH manuscript did not include the 117 persons who had arsenic urine levels that exceeded 50 µg/L (see Table 3A-2).

3.7.3 PRDOH Study, Phase 2

In Phase 2, from December 2005 to March 2006, the PRDOH study undertook follow-up activities on 15 adults whose metal body burdens exceeded toxicity levels as identified by Quest Laboratories. Ten of 15 agreed to participate. Blood samples were collected and measured for aluminum (6 persons), arsenic (3 persons), lead (2 persons), and mercury (1 person)[19]. Arsenic blood tests included inorganic arsenic. Why the PRDOH study did not test urinary cadmium in the two persons with elevated urinary cadmium in Phase 1 is not clear. Why the PRDOH study did not measure speciated urinary arsenic in the 117 persons with elevated urinary arsenic in Phase 1 is similarly unclear. LabCorp conducted the tests and provided the reference levels.

Of the six persons in Phase 1 with elevated aluminum in blood who agreed to retesting, none were elevated in Phase 2. Similarly, the one person with elevated blood mercury in Phase 1 was not elevated in Phase 2. Of the two participants from Phase 1 with elevated blood lead levels, one was elevated in Phase 2. The PRDOH manuscript stated that a follow-up investigation of the home environment for this person would take place. Table 3A-3 shows the results of Phase 2, although because of confidentiality issues, the PRDOH manuscript did not report the specific values.

In addition to the 10 persons who participated in Phase 2 because they had high metal levels, the PRDOH manuscript included four additional residents who were not part of Phase I. These four residents volunteered for blood tests because they were interested in being tested. The PRDOH manuscript provides their results in the report but states that none showed levels considered potentially toxic.

[19] Note that the number of tests is 12—some persons were apparently tested for more than one metal.

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Table 3A-3. PRDOH biomonitoring study Phase 2 biomonitoring results for 10 persons

Metal (Blood Samples)	*Reference Threshold Level*	*NHANES (4th Report)*		*Number of Persons Above Toxicity Level, Phase 1*	*Number of Persons Above Toxicity Level Phase 2*
		Geometric Mean	**95th Percentile**		
Aluminum	0 – 9 µg/L	Not measured	Not measured	6	0
Total Arsenic	< 50+ µg/L	Not measured	Not measured	3	2
Inorganic Arsenic	0 – 19 µg/L	Not measured	Not measured	Not available	0/3
Lead	0 – 19 µg/dL	1.2 µg/dL	3.7 µg/dL	2	1 (Class III)
Mercury	0 – 14.9 µg/L	0.77 µg/L	4.6 µg/L	1	0

+ Table 1 (Phase II) in the PRDOH report is the source of the Reference Threshold Levels reported in Table 3A-3. The value of 50 µg/L appears to be a mistake as this level is often used for total urinary arsenic.

3.7.4 PRDOH Findings

The PRDOH manuscript reported that the main findings of this study were

- In over 90% of the population, detectable levels were found of at least one heavy metal.
- In more than 20% of the study participants, the levels of aluminum in blood, arsenic in urine, or nickel in hair were over the laboratory reference threshold.
- Geometric means for uranium in urine, mercury in blood, lead in blood, aluminum in blood, nickel in hair, and cadmium in urine were significantly higher than the geometric means from the 1999 NHANES survey. But the geometric means for mercury and aluminum in blood reported in the PRDOH manuscript were unreliable because of the high detection limit reported by the laboratory.
- None of the identified geometric means were over the toxicity threshold identified by the reporting laboratory.
- Cigarette use, hair dye use, and seafood consumption were identified as risk factors for levels above the laboratory threshold for arsenic, cadmium, and nickel.
- Fifteen persons (3%) were identified with levels above the toxicity threshold for aluminum in blood (10 persons), lead in blood (2 persons), mercury in blood (1 person), and cadmium in urine (2 persons).
- In the follow-up study on 10 persons with metal body burdens that exceeded toxicity levels, only one person with levels above the toxicity threshold was identified.
- Fewer than 4% of the participants showed elevated metal levels associated with possible development of signs and symptoms related to acute exposure. Among the metals studied, levels of aluminum in blood, nickel in hair, and arsenic in urine were reported with the greatest frequency as over the laboratory detection reference threshold. But the test for arsenic

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measured total arsenic, which includes nontoxic forms of arsenic from eating seafood (i.e., arsenobetaine and arsenocholine).

Multiple factors might account for these observations of elevated metals in blood, hair, and urine. For example, aluminum is abundant in soil worldwide. Average aluminum levels in LIA soils is 16,200 ppm compared with average aluminum in U.S. soils of 57,000 ppm. The absence of reference populations from Puerto Rico or information about aluminum levels in soils in Viequense residential areas limits interpretation of the aluminum results. The PRDOH manuscript also evaluated possible risk factors associated with elevated metal levels below the laboratory's toxicity level—significant risk factors were in fact found. The PRDOH manuscript, however, points out that the study did not consider possible risk factors such as exposures from explosives, armaments, use of metal cooking utensils, metal residues that might be in foods such as tea and vegetables, consumption of drinks with metallic packaging, religious rites using mercury-containing capsules, skin creams, antacid formulations, and antiperspirants.

In Phase 1, the PRDOH study measured total arsenic in urine, which consists of inorganic and organic arsenic. Because high levels of nontoxic organic arsenic are likely from seafood consumption, in Phase 2 the PRDOH study measured total and inorganic arsenic for the three persons who had elevated total arsenic in Phase 1. No evidence of inorganic arsenic was found. The PRDOH manuscript pointed out that hair arsenic levels in the study participants were similar to the national average.

Nickel levels in urine in the study participants were similar to the national average. Sixty-three (63) persons had nickel levels in hair above the laboratory reference threshold. Hair dye, used by 27 of these 63 persons (43%), and the use of colorants and solvents were identified as risk factors and thus possible sources of the nickel in hair. The PRDOH manuscript mentioned hair analysis's inability to provide information about internal (i.e., ingestion) exposure versus external contact with metals (except for mercury). In addition, nickel measurements in hair do not predict that exposure will produce adverse health effects.

The PRDOH manuscript also said the geometric means for aluminum (blood), mercury (blood), lead (blood), uranium (urine), cadmium (urine), and nickel (hair) were higher than the national average (using NHANES and other reference sources); average levels, however, were not above toxicity levels cited by Quest Laboratories. The PRDOH manuscript identified three factors associated with increased arsenic, cadmium, and nickel in hair, urine, or blood: cigarettes , hair dye , and fish consumption. At the time of the study, seventy (15%) participants self-reported as smokers. Elevated lead levels were 4.75% more likely in smokers, who were also nearly two times as likely to have elevated levels of arsenic and nickel. The PRDOH manuscript added that the percentage of people with elevated levels of these metals who were not exposed to cigarette use or consumption of seafood was still higher than expected.

The study design attempted to use a stratified random sample of Viequenses who would represent the island's population. But the PRDOH manuscript acknowledged the sample had a higher percentage of women, older persons, and the unemployed compared with the island's population. The PRDOH manuscript said this might have been due to interviews conducted during the day. Nevertheless, the PRDOH study may have underrepresented fishers and island residents who worked during the day. The PRDOH manuscript agreed that the difference between the study population characteristics and the island characteristics limited generalizations of the data to all island residents.

The PRDOH manuscript also acknowledged that some of the tests failed to distinguish between the different forms of a metal. As mentioned previously, urine arsenic tests in Phase 1 failed to distinguish inorganic from organic arsenic. This also was the case for mercury. For uranium, laboratory tests failed to distinguish between natural uranium and depleted uranium.

3.7.5 PRDOH Actions Taken After Phase 1 and Phase 2

After completion of Phase 1, the following actions were taken:

- Dr. Luis Santiago gave a presentation of preliminary results to a group of about 15 Vieques community leaders.
- Participants' results were provided to Dr. Mackenzie at the Vieques clinic. Of the 500 participants, 182 collected their results.
- Dr. Braulio Jimenez, a toxicologist with the University of Puerto Rico, gave three talks in Vieques about the signs and symptoms associated with exposure to heavy metals.
- A Phase 2 sampling was conducted to follow up on residents with toxic levels of metals.
- A working group was formed to analyze the studies conducted by the health department. This group came up with the suggestions for continuity and follow-up activities listed here.

3.7.6 PRDOH's Suggested Follow-up Activities

The PRDOH manuscript suggested follow up with

- People identified with toxic levels of heavy metals,
- People with possible acute exposure to heavy metals,
- People with possible chronic exposure to heavy metals, and
- High-risk groups.

The PRDOH study used the CDC-provided recommendations to decide follow-up activities for the one remaining person with elevated blood lead levels after Phase 2 testing (CDC 1997; CDC 2002). The PRDOH study offered additional urine arsenic tests for those people who had elevated total urine arsenic in Phase 1. The follow-up tests measured inorganic arsenic. Inorganic arsenic levels in urine were within the laboratory reference levels (PRDOH 2006).

3.7.6.1 Follow up for people with possible acute exposure to heavy metals

The PRDOH manuscript stated that a protocol will be developed for the management of persons with suspected acute poisoning of heavy metals using CDC guidelines as a basis (CDC 1997; CDC 2002). It should be noted that CDC guidelines apply only to lead. The protocol will contain 1) training of local health care providers, 2) ways to promote additional testing using the Commonwealth's laboratory, 3) use of the poison center to provide recommendations for managing acute exposure victims, and 4) referral of suspected cases to the Medical Center of Puerto Rico.

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3.7.6.2 Follow up for people with possible chronic exposure to heavy metals

The PRDOH manuscript suggested establishing a voluntary health registry for Vieques residents. A health registry would 1) allow for systematic documentation of factors related to potential risks, 2) identify people with early signs and symptoms that may be associated with illness, and 3) facilitate early care and health service access needs. The registry would gather demographic information, risk history, family history, past illness history, present status, and history of signs and symptoms associated with the chronic heavy metal exposure. A health registry could also provide comprehensive medical evaluations that include physical examinations with, among other tests, neurological examination and laboratory tests, including tests for kidney function, liver function, and CBC.

3.7.6.3 Follow up with a monitoring program for children under 6 years of age

Blood lead levels are unknown for the 1,000 Vieques children 6 years of age and younger—a high-risk group more vulnerable to lead exposure than are adults. This initial evaluation proposed in the PRDOH manuscript will include all children 6 years of age and younger and will subsequently incorporate in future years an evaluation of children 1 and 2 years of age as part of their primary care. In 2006, when the report was written, the PRDOH manuscript stated that an epidemiologist would be appointed to work towards establishing this program, and this program will follow CDC guidelines for case management (PRDOH 2006).

With funding from USEPA, PRDOH and CDC are currently conducting a prevalence study of blood lead levels in 1 to 5 year old children from mainland Puerto Rico.

3.7.7 Investigations by Dr. Carmen Ortiz Roque

Dr. Carmen Ortiz Roque is a physician who has treated patients and conducted investigations on Vieques. In 2000, she began collecting hair samples from residents and had the samples analyzed for various metals. She has reported some of her results in unpublished manuscripts. In 2004 she published one peer-reviewed journal article.

3.7.7.1 Summary of Dr. Ortiz Roque's 2004 Journal Article

In 2004, Dr. Ortiz Roque published the mercury data from her 2000/2001 hair study of Vieques residents (Ortiz Roque 2004). The study investigated hair mercury levels in women 16 to 49 years of age who lived on the island of Vieques and compared those levels with women from San Juan and Ceiba, Puerto Rico. Women with chemically treated hair within 3 months of the test were excluded. One of two licensed laboratories analyzed a 1.5-cm proximal hair sample.

Dr. Ortiz Roque reported statistics on 41 Viequense women together with information about seafood consumption and compared these data with mainland Puerto Rico and with results from the 1999 NHANES survey in the United States. Table 3A-4 summarizes her data. Dr. Ortiz Roque used margin of exposure (MOE) to define unsafe exposure levels. The MOE approach used by Dr. Ortiz Roque came from a 2001 MMWR article and the National Research Council. The formula for MOE is 5% effect level for mercury in hair / 90th percentile value for mercury in hair in the study population.

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Table 3A-4. Mercury hair results in women published by Dr. Ortiz Roque in 2004.

	#	Age	*Mercury in ppm*			*Margin of Exposure*	*Seafood Consumption*			*% women > RfD*
			Median	*Mean*	*90th Percentile*		*Total*	*Local*	*Local fish*	
Vieques	41	31.8	0.66	4.4	8.96	1.3	4.9	2.9	1.9	26.8
PR	45	29.9	0.38	0.4	1	12	2.8	1.1	0.7	6.6
USA (1999 NHANES)	702	NA	0.2		1.4	8.6	NA	NA	NA	7

The National Research Council (NRC) recommended deriving the 5% effect level from the 95% lower confidence limit of a benchmark dose ($BMDL_{05}$) that resulted in abnormal scores on cognitive function tests in children exposed to methylmercury *in utero*. The NRC recommended that the U.S.EPA derive their Reference Dose using a $BMDL_{05}$ of 58 ppb methylmercury in cord blood, which was identified as the 5% effect level using statistical models from the Faroe Islands study. The 58-ppb cord blood translates to 12 ppm in hair (NRC 2000). Therefore, the MOE = 12 ppm / 90th percentile mercury in hair. A MOE less than 1 means that for 10% of the study population the mercury level in hair is above the 5% effect level of 12 ppm, while an MOE greater than 1 means that for 90% of the study population the mercury level in hair is below the 5% effect level of 12 ppm (Ortiz Roque 2004; CDC 2001). It is important to note that Dr. Ortiz Roque found three women with hair mercury levels greater than 12 ppm, the level identified by the NAS as an effect level for the developing fetus.

Results showed that the 90th percentile mercury hair concentration in Vieques women was 8.96 ppm compared with 1 ppm in women from Puerto Rico and 1.4 ppm in women from the United States. With a MOE of 1.3, Dr. Ortiz Roque concluded that because hair mercury levels were close to the 5% effect level of 12 ppm, some Viequense women of reproductive age were exposed to mercury concentrations unsafe to their developing fetus. While ATSDR agrees with this conclusion, it should be noted that the percentage of Viequenses women with hair mercury levels above 12 ppm is uncertain because of the small and biased sample size in Dr. Ortiz Roque's investigation.

Dr. Ortiz Roque also has unpublished data showing other metal concentrations in hair of Vieques residents. These data are summarized in Chapter 2.

3.7.8 Summary of Dr. Ortiz Roque 2002 Unpublished Manuscript

3.7.8.1 Study Design

Dr. Carmen Ortiz Roque collected hair samples from 203 Viequenses from January 2000 to July 2001 along with an extensive residential, occupational, nutritional, and health survey. Hair samples were analyzed for mercury, aluminum, cadmium, lead, and arsenic. The group consisted of 110 females and 93 males and ranged in age from 0.8 to 81 years. Participants were distributed among 18 neighborhoods on the island and were later informed of their individual hair results. Scalp hair samples were obtained from most participants. However, when scalp hair was limited or not available or hair was treated chemically, pubic or chest hair was collected (Ortiz 2002). Note that comparing results from pubic hair or

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chest hair with scalp hair may not be appropriate. Hair analysis were conducted by either the Mayo Medical Laboratory (Rochester, MN) or the King James Medical Laboratory (Cleveland, OH).

3.7.8.2 Hair Study Results

Dr. Ortiz Roque reported the following results concerning metals in hair (Table 3A-5):

Table 3A-5. Results of metal analysis of hair samples collected from Viequenses from January 2000 to July 2001.

Metal (sample number)	***% elevated***	***Average in ppm***			
		Overall (95% CI)	***Females***	***Males***	***Children <10 years old***
Mercury N= 205	33% > 1 ppm	2.07 (0–8.9)	2.87	1.08	1.89
Aluminum N=145	56% > 17 ppm	25.74 (10.52–25.75)	26.45	24.18	34.94
Cadmium N=205	26% > 0.47 ppm	0.65 0–5.0)	0.75	0.55	Not available
Lead N=205	2.9% > 25 ppm	8.07 (0–19.0)	4.28	12.47	Not available
Arsenic N=205	0 > 1 ppm	0.18 (0–3.0)	0.141	0.233	Not available

Dr. Ortiz Roque stated that elevated levels of mercury (33%), aluminum (56%), cadmium (26%), and lead (2.9%) were found in hair samples from residents in Vieques. Hair arsenic levels were not elevated (Ortiz 2002).

Dr. Ortiz Roque provided additional information about hair mercury in women and children, shown in Table 3A-6. She stated that 22% of women and 60% of children sampled in Vieques had mercury levels above the 90th percentile in the United States as identified from the 1999 NHANES national survey (Ortiz 2002). Dr. Ortiz Roque further analyzed a subset of 22 Viequense matched pairs of mothers and their children 5 years of age or less. Dr. Ortiz Roque reported a significant correlation between mercury hair levels in mothers and their children (Pearson's correlation 0.93, p = 0.0001). Dr. Ortiz Roque published these matched pairs in 2003 (see Table 3A-4).

Dr. Ortiz Roque stated that the average aluminum hair concentration for Viequenses was 25.74 ppm. She reported that this concentration exceeded the upper limit of a standard human population, although the manuscript does not report the source of the upper limit. Dr. Ortiz Roque pointed out that aluminum levels in children's hair was higher than in adults (Ortiz 2002). As stated previously, it is not possible to determine whether elevated levels of aluminum, cadmium, and lead in hair are from internal

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exposure via ingestion or inhalation or external exposure from direct contact of the hair with a metal-containing commercial product. While Dr. Ortiz Roque's data show that these metals are elevated in Viequenses at the 75th and 90th percentile, her population was not chosen randomly from the Vieques population. This nonrandom participant selection might account for some confounding factor elevating the rates. And for other metals, the nonrandom sample might account for the lower than expected metal concentrations.

Table 3A-6. Viequense women and children mercury hair analysis compared with United States

Population	***Number***	***% Population Sampled 2000***	***75th percentile ppm hair***	***90th Percentile ppm hair***	***% above 90th percentile for U.S.***
USA Women 16–49 years*	702	702/146,250,000 0.00048%	0.5	1.4	10%
Vieques women	45	45/4,594 0.97%	1.14	7.54	22%
USA Children* 1–5 years	338	338/19,175,798 0.00256%		0.4	10%
Vieques Children 1–5 years	38	38/771 4.9%	1.31	2.67	60%

* Reference ranges were derived from the 1999 NHANES survey

3.7.8.3 Dr. Ortiz Roque's Conclusions

Dr. Ortiz Roque maintained her data showed that Viequenses had internal exposure to heavy metals, supported by the high percentage of women and children with hair mercury levels above the 90th percentile compared with the continental United States. While none of the women in the 1999 NHANES survey had mercury hair levels above 12 ppm, 3 of 45 Viequense women had hair levels above 12 ppm in Dr. Ortiz Roque's survey (i.e., 15.41, 25.26, and 101.3 ppm) (Ortiz 2002).

In her 2002 draft manuscript, Dr. Ortiz Roque stated further that the positive correlation between mother and offspring hair showed that mercury exposure was occurring *in utero;* Viequenses' exposure to mercury was too high to be considered safe; and mercury was used in explosive ordnance detonators (Ortiz 2002). The correlation between mother and offspring hair with seafood consumption is expected, has been shown in numerous human studies, and points to seafood as a major source of mercury in humans. The correlation of hair mercury levels in mother and children is not conclusive proof that these children were exposed *in utero*. A study of mothers and newborns would be needed to make that statement conclusively. Assuming that mothers maintained similar fish-eating habits before and during their pregnancy, the positive hair mercury correlation between mother and offspring suggests that *in utero* exposure to mercury occurred and could occur in future pregnancies. While Dr. Ortiz Roque's

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patients were not selected randomly, identifying several women with very high hair mercury levels is an important finding.

3.7.9 Investigations by Dr. Carmen Colón de Jorge

Dr. Carmen Colón de Jorge also conducted human biomonitoring of metals in hair and in addition analyzed several stool samples for metals. Dr. Colón de Jorge collected hair and stool samples from Vieques residents in 1999 and 2000 and wrote a manuscript describing her results and findings (Colón de Jorge, undated).

Dr. Carmen Colón de Jorge began her manuscript by describing the case history of one of her patients who lived on Vieques. According to the reporting laboratory, a 1991 hair analysis in a sick patient showed toxic levels[20] of lead and aluminum. The patient was treated using naturopathic remedies, and the patient's health improved. The patient was tested again in 1993 and again in 1996 because the patient was again sick. The hair tests showed high levels of lead, aluminum, and antimony. Arsenic and mercury were also present in hair. In 1999, toxic levels of aluminum, antimony, arsenic, and mercury were much higher as well, now showing toxic levels of cadmium and bismuth. The patient also had low levels of zinc and selenium and increased boron. Dr. Colón de Jorge stated the low levels of zinc and selenium confirmed endogenous poisoning with heavy metals. Representing the Scientific Committee of the Association of Licensed Naturopaths of Puerto Rico, Dr. Colón de Jorge collected hair and stool samples from Vieques residents in 1999 and 2000. She stated that the nutritional imbalance in residents as shown by hair analysis was evidenced by deficiencies in calcium, phosphorus, and magnesium and excessive boron in hair. She further stated that the decline in selenium, resulting from heavy metal contamination, was a contributor to cancer development.

3.7.9.1 Dr. Colón de Jorge's Conclusions

Dr. Colón de Jorge stated that 45–50% of the people tested in Vieques were poisoned with mercury. Using results from stool samples, she found that 3 out of 6 children tested in Vieques had metal concentrations in stools[21] above the reference range provided by the clinical laboratory (i.e., Doctor's Data) that conducted the tests. She also reported that 5 out of 6 children tested had antimony and arsenic in stool samples that exceeded the laboratory's reference range. She pointed out that children could not be exposed to metals in paint, cigarette paper, tobacco, old pots, make-up, and hair dye as adults might be.[22] Uranium levels in children's stool samples were below the reference range established by the laboratory.

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[20] Dr. Colón de Jorge's manuscript does not provide a definition for toxic level. Apparently Dr. Colón de Jorge used the term "toxic level" because it is used in the laboratory report provided by Doctor's Data for potentially toxic elements. Although Doctor's Data, Inc. also provides no "toxic level" definition, it appears to be any level above the 95th percentile and is based on the laboratory's observation of over 1 million hair samples.

[21] Determination of metals in stools samples is not a standardized or recommended means of determining exposure.

[22] Note, however, that children can be exposed to lead in paint when they contact painted surfaces such as walls, doors, windows, stairs, and fences.

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Dr. Colón de Jorge reported that 30 hair samples were collected from Vieques residents with various health conditions:

- 50% of the people tested were contaminated with antimony compared with 29% from a control population,
- 50% of the people tested were contaminated with arsenic compared with 29% from a control population,
- 50% of the people tested had a selenium imbalance.
- Dr. Colón de Jorge pointed out that antimony levels were high because antimony was used in explosive ordnance on Vieques, thus making the case that military exercises were the reason antimony levels were high. No supporting evidence, however, was provided to show how residents were exposed.
- Dr. Colón de Jorge also summarized the results of seven control patients with the following statements:
    - None of the seven patients showed toxic levels.
    - Two cases had antimony above the reference range established by the laboratory compared with 29% of those investigated outside of Vieques. The selenium was normal for exogenous contamination.
    - Two cases had arsenic above the reference range compared with 29% of the cases outside Vieques. The selenium is normal for exogenous contamination.
    - The seven control patients exhibited no endogenous contamination with heavy metals—the selenium was normal and within the reference range. According to Dr. Colón de Jorge, heavy metal contamination in a patient will often result in lower levels of selenium.

In summary, Dr. Colón de Jorge collected hair samples from seven random persons in Rio Piedras, a community on the main island of Puerto Rico. These persons' health status remains unknown. None of the results show toxic levels of heavy metals, while 21 of 30 persons (i.e., 63%) from Vieques with known health conditions—and thus not randomly selected—showed toxic levels of various metals. As stated previously, current science is simply incapable of determining whether elevated levels of these metals in hair is from internal exposure via ingestion or inhalation, from external exposure via direct contact of the hair with a metal-containing commercial product, or due to accumulation secondary to an underlying disease. The exception is that current science can determine whether mercury in hair is from internal or external exposure.

Dr. Colón de Jorge provided a report section that described the relationship between metal toxicities, which can cause mineral deficiencies and excesses, and the risk of various diseases and health conditions. This relationship, or risk pattern, is described for 1) cardiac conditions, 2) cardiovascular, 3) emotional disturbances, 4) violence 5) poor absorption, and 6) cancer.

3.7.9.2 Sample Results and Interpretation

Dr. Colón de Jorge summarized elemental levels in hair (Table 3A-7) and in stool samples (Table 3A-8). Dr. Colón de Jorge's report contained several appendices with additional information, mostly focused on how to evaluate and interpret elemental results in hair. One of the appendices contained a report by Doctor's Data entitled Comprehensive Interpretations for Hair Elements from Al to Zn.

Table 3A-7. Summary of hair analysis of 30 samples as reported in Dr. Colón de Jorge's unpublished manuscript (Colón de Jorge undated).

Metal	***Range of levels in Dr. Colón de Jorge's patients in µg/g***	**Reference Range in Dr. Colón de Jorge's manuscript***	***Percent above reference***	***Percent below reference***
Aluminum	4.4 – 68	<7	90	
Antimony	0.016 – 2.7	<0.05	69	
Arsenic	0.028 – 2.8	<0.06	66	
Barium	0.23 – 4.7	0.026 – 3; 0.16 – 1.6	14 21	3
Beryllium	<0.001	<0.02	0	
Bismuth	0.011 – 2.4	<0.06; <0.1	48 41	
Boron	0.42 – 19	0.3 - 2 0.4 - 3	41 48	0
Calcium	145-2240	300 - 1200 200 - 750	17 24	24
Cadmium	0.024 – 0.98	<0.1 0.15	69 52	3
Zinc	64-630	140 - 220 130 - 200	21 24	28 14
Zirconium	0.012 - 0.76	0.02 - 0.42 0.02 - 0.44	7 7	3 0
Cobalt	0.008 – 0.16	0.013 - 0.05 0.013 - 0.035	14 28	21
Copper	8.9 – 110	12 - 35	31	21

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Metal	***Range of levels in Dr. Colón de Jorge's patients in µg/g***	**Reference Range in Dr. Colón de Jorge's manuscript***	***Percent above reference***	***Percent below reference***
		10 - 28	35	3
Chromium	0.2 – 0.69	0.2-0.4	10	0
Tin	0.08 – 1.3	<0.3	66	
Strontium	0.29 – 9	0.5 - 7.6 0.3 - 3.5	7 28	17 3
Germanium	0.022 – 0.082 (12 N/A)	0.045 - 0.0.65 (sic)	18	77
Iron	6.4 – 70	5.4 - 14 5.4 - 13	52 62	0
Lithium	0.005 – 0.056	0.007 - 0.023	7	66
Magnesium	23 – 560	35 - 120 25 - 75	31 52	21 3
Manganese	0.21 – 4.8	0.15 - 0.65	31	0
Mercury	0.06 – 3.1	<1.1	38	
Molybdenum	0.02-0.12		48 31	10 3
Nickel	0.05 - 2.9	<0.4	28	
Silver	0.01 - 1.7	<0.14; <0.12	21 35	
Platinum	<0.003	<0.005	0	
Lead	0.34 – 26	<0.1;<2	55 35	
Rubidium	0.018 – 0.38	0.007 – 0.096;	28 24	0 0

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Metal	***Range of levels in Dr. Colón de Jorge's patients in µg/g***	**Reference Range in Dr. Colón de Jorge's manuscript***	***Percent above reference***	***Percent below reference***
		0.11 – 0.12		
Selenium	0.52 – 2.5	0.95 – 1.7	3	
Thallium	<0.001 – 0.005	<0.01	0	
Thorium	<0.001	<0.005	0	
Uranium	<0.001 – 0.021	(<0.06)	0	
Vanadium	0.013 – 1.2	(0.018 – 0.065)	38	3

* Reference values provided by Doctor's Data, Inc., St. Charles, IL. For several elements, the laboratory reported two reference ranges on different samples.

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Table 3A-8. Summary of the results of feces analysis as reported in Dr. Colón de Jorge unpublished manuscript (Colón de Jorge undated).

Metal	*Range in Dr. Colón de Jorge's patients in µg/g (Reference range)*	*Reference Range**	*Percent above reference*	*Percent below reference*
Aluminum	43-750	130	57	42
Antimony	0.06 – 0.127	NA	83	16
Arsenic	0.098 – 0.866	NA	83	16
Beryllium	0.01 – 0.012 (2 < dl) *	NA	50	50
Bismuth	0.014 – 0.55	NA	50	50
Cadmium	0.14 – 1	0.47	14	86
Copper	17 – 68	50	14	86
Mercury	0.028 – 0.1 (0.02 with no amalgam) (0.26 with amalgam)	0.02 with no amalgam 0.26 with amalgam	100 0	0 100
Nickel	3.4 – 7.5 (4.4)	4.4	86	14
Platinum	2.92 (6< dl) [a]	NA	16	84
Lead	0.23 – 0.59	0.75	30	100
Thallium	0.005 – 0.015	NA	0	100
Tungsten	0.014 –0 0.475	NA	50	50
Uranium	0.025 – 0.061	NA	0	100

*Reference values provided by Doctor's Data, Inc., St. Charles, IL.

dl = detection limit

NA = not available

3.7.10 Investigations by Dr. Carlos Rodríguez Sierra

On October 30, 2009, Dr. Carlos Rodríguez Sierra gave a presentation at Casa Pueblo de Ajuntas that was broadcast via the Internet. Dr. Rodríguez Sierra discussed arsenic in fish and biomonitoring data concerning urine arsenic levels in Viequenses. Among the 162 fish from 8 species that he collected, he said only arsenic exceeded the international criterion of 2 µg arsenic per gram fish wet weight.

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In July 2006, he also collected biological samples from 52 adults, whom he divided into two groups: a high fish-consumption group (n=30) and a low fish-consumption group (n=22). He collected hair, nail, and urine samples. Urine samples were measured for total arsenic and for various arsenic species:

- Trivalent arsenic (As III)
- Pentavalent arsenic (As V)
- Monomethylarsensonic acid (MMA),
- Dimethylarsenonic acid (DMA), and
- Arsenobetaine.

The inorganic arsenic species are AsIII and AsV and their organic metabolites (MMA and DMA). The following data were presented:

	Minimum	***Median***	***75th***	***95th***	***max***[23]
Total As	5.33	22.54	33.7	161.69	1414
Arsenobetaine µg/g creatinine	0	7.64	22.26	141.63	1373

Total inorganic arsenic was compared with the 2003-2004 NHANES results of the U.S. population:

	N	***Median***	***95th***
Vieques	52	10.3	35.3
NHANES	2557	6.0	18.9

Dr. Rodríguez Sierra announced these conclusions from his analysis of arsenic in residents of Vieques:

1. High levels of arsenic in hair, nails, and urine were not detected at the time of sampling
   a. Hair and nail sample levels were below the reference values of 1 µg/g,
   b. The sum of the inorganic arsenic species and its metabolites was less than 50 µg/g creatinine, which was used as a reference value.
2. Levels of inorganic arsenic in urine samples from Vieques are slightly above the median and 95th percentile levels reported in 2003–2004 NHANES.

Dr. Rodríguez Sierra has informed ATSDR that he plans to publish these data; more complete information should then become available.

[23] ATSDR is unsure of the maximum level of total arsenic and arsenobetaine reported during Dr. Rodríguez Sierra's presentation.

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3.8 Appendix 3-B. Biomonitoring

The NHANES Fourth National Report on Human Exposure to Environmental Chemicals (The 4th Report) provides biomonitoring data for the U.S. population for the years 1999–2004. The 4th Report also provides geometric mean, 50th, 75th, 90th, 95th percentile as well as statistics by the following age groups, sex, race, and ethnicity:

- 6–11
- 12–19
- 20 years of age and older
- Mexican-American
- Non-Hispanic blacks
- Non-Hispanic whites

The 4th Report contains toxicological information about metals when available. The report is now available at: http://www.cdc.gov/exposurereport. The 4th Report also contains summary statistics from previous NHANES surveys that cover 1999–2000 and 2001–2002. Table 3B-1 provides a brief summary of statistics (CDC 2009).

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Table 3B-1. Fourth National Report on Human Exposure to Environmental Chemicals: urine and blood levels toxicity

Metal	*Media*	*Year*	*Geometric Mean*	*95th Percentile*	*Age*
Antimony	Urine	99-00	0.132 µg/L	0.42 µg/L	6 and older
		01-02	0.134 µg/L	0.34 µg/L	
		03-04*	--	0.28	
		99-00	0.124 µg/g	0.382 µg/g	
		01-02	0.126 µg/g	0.364 µg/g	
		03-04*	--	0.277 µg/g	
Total Arsenic	Urine	03-04	8.3 µg/L	65.4 µg/L	6 and older
		03-04	8.24 µg/g	50.4 µg/g	
Arsenobetaine	Urine	03-04	1.55 µg/g	35 µg/g	6 and older
		03-04	1.54 µg/g	29.4 µg/g	
Barium	Urine	See report	See report	See report	
Beryllium	Urine	See report	See report	See report	
Cadmium	Urine	99-00	0.193 µg/L	1.2 µg/L	6 and older
		01-02	0.210 µg/L	1.2 µg/L	
		03-04	0.211 µg/L	1.15 µg/L	
		99-00	0.181 µg/g	0.993 µg/g	6 and older
		01-02	0.199 µg/g	0.917 µg/g	
		03-04	0.21 µg/g	0.94 µg/g	
	Blood	99-00	0.412 µg/L	1.3 µg/L	1 and older
		01-02*	--	1.3 µg/L	
		03-04	0.304 µg/L	1.6 µg/L	
Cesium	Urine	See report	See report	See report	
Cobalt	Urine	See report	See report	See report	
Lead	Blood	99-00	1.66 µg/dL	4.9 µg/dL	1 and older
		01-02	1.45 µg/dL	4.4 µg/dL	
		03-04	1.43 µg/dL	4.2 µg/dL	
	Urine	99-00	0.766 µg/L	2.9 µg/L	6 and older
		01-02	0.667 µg/L	2.9 µg/L	
		03-04	0.636 µg/L	2.29 µg/L	

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Metal	*Media*	*Year*	*Geometric Mean*	*95th Percentile*	*Age*
		99-00	0.721 µg/g	2.37 µg/g	6 and older
		01-02	0.639 µg/g	2.03 µg/g	
		03-04	0.632 µg/g	1.97 µg/g	
Total Mercury**	Blood	99-00	0.343 µg/L	2.3 µg/L	1-5 year old
		01-02	0.318 µg/L	1.9 µg/L	1-5 year old
		03-04**	0.326 µg/L	1.8 µg/L	1-5 year old
		99-00	1.02 µg/L	7.1 µg/L females	16-49 years
		01-02	0.833 µg/L	4.6 µg/L females	16-49 years
		03-04	Not available	Not available	Not available For women 16-49
		03-04	0.979 µg/L	4.9	1 and older
	Urine	99-00	0.719 µg/L	5.0 µg/L	16-49 years
		01-02	0.606 µg/L	3.99 µg/L	16-49 years
		03-04	Not available	Not available	Not available for women 16-49
		03-04	0.447 µg/L	0.319 µg/L	1 and older
		99-00	0.71 µg/g	3.27 µg/g females	16-49 years
		01-02	0.62 µg/g	3.0 µg/g females	16-49 years
		03-04	Not available	Not available	Not available For women
		03-04	0.443 µg/g	2.35 µg/g	1 and older
Molybdenum	Urine	See report	See report	See report	
Platinum	Urine		<LOD	<LOD	
Thallium	Urine	99-00	0.176 µg/L	0.45 µg/L	6 and older
		01-02	0.165 µg/L	0.44 µg/L	
		03-04	0.155 µg/L	0.44 µg/L	
		99-00	0.166 µg/g	0.366 µg/g	6 and older
		01-02	0.156 µg/g	0.348 µg/g	

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Metal	*Media*	*Year*	*Geometric Mean*	*95th Percentile*	*Age*
		03-04	0.154 µg/g	0.350 µg/g	
Tungsten	Urine	See report	See report	See report	
Uranium	Urine	99-00	0.008 µg/L	0.046 µg/L	6 and older
		01-02	0.009 µg/L	0.046 µg/L	
		03-04	0.008 µg/L	0.039 µg/L	
		99-00	0.007 µg/g	0.034 µg/g	6 and older
		01-02	0.008 µg/g	0.04 µg/g	
		03-04	0.008 µg/g	0.029 µg/g	

* A large percentage of participants had < LOD, thus preventing the calculation of an overall mean

** Additional information about mercury levels in blood for various age groups is now available from a 2003–2006 survey. These data are summarized in Caldwell KL et al, "Total blood mercury concentrations in the U.S. population: 1999–2006." Int J Hyg Environ Health 2009 (Nov) 212;6:588–98.

Note: Total blood mercury was measured in children aged 1–5 years and in women aged 16–49 years in 1999–2002. Total blood mercury and inorganic blood mercury were measured in all participants aged 1 year and older in 2003–2004. Urinary mercury was measured in women aged 16–49 years in 1999–2002. For the 2003–2004 survey, urinary mercury was measured in a random one-third subsample of participants aged 6 years and older.

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Chapter 4 Health Outcome Data

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Chapter 4 Summary

Assessing accurately the health status of Viequenses requires quantifying morbidity and mortality as well as identifying possible factors that may lead to adverse health outcomes. Quantifying morbidity and mortality and assessing possible contributing factors can help to identify specific opportunities for intervention in public health programs or delivery of health services. An understanding of public health data collection and reporting systems, public health programs, and the health care delivery system on the island of Vieques is required to determine future activities for improving the health status of Viequenses.

In this chapter we review public health data collection and reporting systems used in quantifying morbidity and mortality, review several reports published in the last 10 years that assess morbidity and mortality in Vieques, and provide a brief review of available health services on Vieques. All of these studies are descriptive in nature and provide some insight into the Vieques health picture. The studies do not contain analyses that relate potential environmental or other factors to the identified health conditions. Because of the small population, all studies suffer to some degree from a lack of statistical power and methodological limitations that make interpretation difficult. Despite these limitations, the studies are valuable for describing the health status of Viequenses. The studies indicate elevations in the prevalence of chronic disease, cancer incidence, and cancer mortality in Vieques relative to the rest of Puerto Rico.

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4.1 Public Health Data Systems and Programs

The primary data systems supporting the estimation of incidence and prevalence of health outcomes in Puerto Rico are the Central Cancer Registry (RCCPR), Behavioral Risk Factor Surveillance System (BRFSS), Birth Defects Surveillance System (BDSS), and the Puerto Rico Vital Records Office. CDC helps to fund each of these data collection and reporting systems. Their strengths and weaknesses affect data quality and the resulting ability to develop appropriate public health actions based on this information.

The data reported by the RCCPR have several strengths. Cancer registries typically represent the best population-based dataset when compared with data collection systems and associated datasets for other chronic diseases. Most cancer cases are captured by registries due to legal reporting requirements, redundancies in the reporting system (i.e., reporting by labs, clinics, hospitals, and specific oncologists), and the clinical course of cancer that usually requires substantial follow up and repeat visits. The analysis of cancer registry data should then represent the most accurate assessment of cancer incidence in Vieques. Through a cooperative agreement in place since 1998, CDC has continuously funded the cancer registry. And the registry's data accuracy has steadily improved.

That said, however, several weaknesses in the registry data should be noted. First, funding has been inconsistent throughout the life of the registry, but recently it has become more stable. The RCCPR was funded in part by the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program from 1973–1989. The contract between the RCCPR and SEER was not renewed, and until 1997 RCCPR operated on local funding. In 1997, the RCCPR applied for and received funding from the Centers for Disease Control and Prevention (CDC) through a cooperative agreement via the National Program of Cancer Registries. The RCCPR provided data to the CDC beginning with cancer cases diagnosed in calendar year 1998. Completeness and timeliness have also been problematic due to a shortage of trained personnel to perform cancer abstracting and delays in reporting by facilities because of lack of funds and lack of reporting requirements by some facilities. The RCCPR has made significant progress toward improving completeness. In 2003, a CDC review concluded that 95.3% of all cancer cases diagnosed or treated in hospital facilities in Puerto Rico were appropriately reported to the RCCPR; a result comparable to the US median (95%) (ORC MacroSM 2000).

BRFSS is the primary surveillance system for estimating the prevalence of chronic diseases and health behaviors in the United States and in Puerto Rico. BRFSS is the primary data source for prevalence estimates of asthma, diabetes, hypertension, and heart disease. One limitation of this system is the ability to provide accurate prevalence estimates in small populations. Vieques has a population of less than 10,000 residents. Providing accurate prevalence estimates is difficult in populations this small. This is particularly due to the complex survey design and the overarching goals of producing state and national prevalence estimates. The Selected Metropolitan/Micropolitan Area Risk Trends (SMART) project was initiated to develop prevalence estimates at the local level. The SMART project methods develop prevalence estimates using BRFSS data for areas with 500 or more respondents. These methods may represent a viable option for using BRFSS to develop prevalence estimates for Vieques.

CDC funds the Puerto Rico BDSS, which tracks thirteen congenital conditions diagnosed at birth. Current program goals include expanding the capacity of the surveillance system to identify birth defects cases and developing legislation that mandates the reporting of birth defects in Puerto Rico to the BDSS. The

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BDSS is one of 14 CDC-funded birth defects surveillance systems. The population-based system uses active case ascertainment. Abstractors in each public health region visit hospitals and other facilities to review medical records to identify cases for each specific type of birth defect. Although this approach is considered the gold standard, it is resource-intensive. A limitation of birth defects surveillance in general is that some defects cannot be diagnosed at birth and thus go undetected.

Another morbidity and mortality data source is the Puerto Rico Vital Records Office. The Vital Records office collects data on births and deaths for the entire population. Advantages of birth and death data include low cost, availability, and, because of the statutory reporting requirements, near-complete coverage. Mortality data limitations include multiple causes of death, completeness of records, and changes in diagnostic practices from improvements in medical technology (Rothman and Greenland 1998). Birth certificate data limitations include changes to birth certificates over time, incomplete birth certificates, and self-reported information.

4.2 Assessment of Available Public Health Data

Several studies have evaluated morbidity and mortality in the Viequense population. All are descriptive in nature and provide some insight into the health of that population. But these studies do not contain analyses that relate potential environmental or other factors to the identified health condition. In addition, because of the small study population, all the studies suffer from a lack of statistical power. Despite their limitations, these studies are valuable for describing the health status of Viequenses and providing insight that will inform next steps. An evaluation of studies published within the past decade are summarized and discussed in this chapter. Conclusions and recommendations for further action are outlined below and in Chapter 9. These recommendations are viewed as a starting point for dialogue with the scientific community and Viequenses to develop an action plan that will help improve Viequenses' health.

4.2.1 Cancer

The RCCPR released reports in 2006 and 2009 detailing cancer incidence and mortality in Puerto Rico. The 2006 report evaluated cancer incidence and mortality from 1990–2001. In contrast, the 2009 report evaluated cancer incidence and mortality from 1990–2004. Both studies assessed whether, using similar methods, cancer incidence and mortality rates were higher in Vieques than in the main island of Puerto Rico. The standardized incidence and mortality ratios with 95% confidence intervals were calculated. The authors defined statistical significance as a 95% confidence interval that did not include 1. Similarly, the authors defined marginal statistical significance as a 90% confidence interval that did not include 1.

The analyses have several strengths. Most cancer cases are captured by registries. This is because of legal reporting requirements, redundancies in the reporting system (i.e., reporting by labs, clinics, hospitals, and specific oncologists), and the clinical course of most cancers that requires substantial follow up and repeat visits. The analysis of cancer registry data should then represent the most accurate assessment of cancer incidence on Vieques.

Although the analyses have several strengths, several limitations were also noted. As mentioned briefly in the PRDOH reports and confirmed by Dr. Figueroa (personal communication, July, 2009), some follow

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up to identify cases in Vieques occurred that was not uniformly applied across the rest of Puerto Rico. One example is related to the documentation of age for cases in the registry.

For both assessments, all cases indicating residence on the Island of Vieques were age-confirmed. This practice was not mirrored on the main island of Puerto Rico. If a case did not have a known age, it was not included in any rate calculations. In addition, cases were actively sought in Vieques and not in the rest of Puerto Rico. This potentially introduces bias; it omits cases from the reference population (i.e., Puerto Rico) and increases the proportion of cases captured by the registry in Vieques relative to Puerto Rico, thus potentially, artificially inflating standardized incidence ratios. Other differential methods for seeking out and reviewing cases in Vieques not applied on the main island would similarly bias the results (RCCPR 2006, 2009).

4.2.2 Noncancer

We reviewed two studies that evaluated noncancer data. The primary noncancer morbidity study was released in March 2000 by Yadiris Lopez and Crisarlin Carrosquillo (Lopez and Carrosquillo 2002). We also reviewed noncancer mortality in “Heavy metal exposure and disease in the proximity of a military base” by Dr. Carmen Ortiz Roque (Ortiz Roque 2002).

The Lopez and Carrosquillo survey was conducted because of growing concerns regarding environmental contamination from the past military activities and their potential adverse human health effects on Viequenses. The survey was a cross-sectional design with a sample size of 1,043. The field work for the study was conducted between April and November of 2000. Lopez and Carrosquillo attempted to include all residents in each neighborhood and allowed the head of a household, typically the matriarch, to provide proxy responses for all household members. The survey collected data on demographics, employment history, and dietary habits, along with self-reported disease prevalence.

The overall age distribution of the sample selected, based on 1990 and 2000 U.S. census data, appears older than the Vieques population. Lopez and Carrosquillo reported that 36% of the sample was ages 60 and older. Data from the 1990 U.S. Census indicated that 16% of the Vieques population was 60 years and older. Similarly, the 2000 Census indicated that 19.5% of the Vieques population was 60 years and older. Participants were selected from Lujan, Puerto Rico Reconstruction Administration, Santa Maria, and Esperanza. The 132 residents of Lujan reported an age range of 5–25, whereas the 306 residents of the Puerto Rico Reconstruction Administration (PRRA) reported an age range of 59–70. The age distributions of Santa Maria and Esperanza were not reported separately. These neighborhoods were collectively described as "representative” of the Vieques population but appear to be older.

The survey participants identified 28 medical conditions. These conditions are listed in Table 4-1. Of the 28 medical conditions identified by participants, the authors presented comparisons using risk ratios for six health conditions: cancer, heart disease, hypertension, diabetes, asthma, and arthritis. The prevalence of health conditions was compared with the expected number of cases, calculated by multiplying the population of Vieques by the percentage of cases that occurred in the 1994 Puerto Rico Chronic Morbidity Study (PRCMS). In addition to medical conditions, the authors described the frequency of cigarette use, alcohol consumption, eating habits, and abortions. The authors stated that cancer was not connected to cigarette smoking or alcohol consumption—the frequency of each factor in

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the survey was similar to the totality of Puerto Rico. The frequency of cigarette use and alcohol consumption among participants with the most prevalent health conditions was not presented (Lopez and Carrosquillo 2002).

Table 4-1. Viequenses-reported health conditions

Health Conditions	*Number of Cases*	*Percentage*
High Blood Pressure	189	18.1
Asthma	112	10.7
Diabetes	102	9.8
Arthritis	89	8.5
Heart Disease	77	7.4
Skin Conditions	61	5.8
Sinusitis	56	5.4
High Cholesterol	54	5.2
Muscular Spasms	52	5.0
Allergies	49	4.7
Other	36	3.5
Circulation	36	3.5
Stress	32	3.1
Migraine	24	2.3
Cancer	19	1.8
Anemia	16	1.5
Kidneys	14	1.3
Uric Acid	13	1.2
Epilepsy	11	1.1
Tumor	10	1.0
Prostate	10	1.0
Fatigue	10	1.0
Nodules	5	0.5
Pneumonia	4	0.4
Lead in blood	4	0.4

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Health Conditions	*Number of Cases*	*Percentage*
Osteoporosis	3	0.3
Lupus	1	0.1
Spinal Column Deviation	1	0.1

Dr. Ortiz Roque briefly presented noncancer mortality and infant mortality in a report. In January 2002, Dr. Ortiz Roque, together with the College of Physicians and Surgeons in San Juan, PR, released a report entitled "Heavy metal exposure and disease in the proximity of a military base." This effort was initiated because of community concern that past military activities on the island may have adversely affected Viequenses' health. Ortiz Roque reported mortality statistics from the Puerto Rico Department of Health for 1991–1998. Age standardized mortality data between Vieques and mainland Puerto Rico were compared with total deaths and cause-specific deaths. Additionally, infant mortality rates in Vieques were also compared with those of Puerto Rico for 1975–1995 (Ortiz Roque 2002).

The mortality data presented did not enumerate all analyses performed—only those that were elevated. Finally, infant mortality is only briefly mentioned, and to ascertain specific times is difficult, especially those for specific causes.

Symptom-disease prevalence surveys of a population, such as the Lopez and Corrosquillo survey, are useful hypothesis-generating tools. This is especially true when little preexisting knowledge is available regarding the population's potential exposures and associated health effects. Another strength of the Lopez and Carrosquillo survey was its large sample size (n=1,043) relative to a population of approximately 10,000. Mortality data may also provide meaningful insight into the health status of Viequenses. Analyses of mortality patterns in a population can be useful for hypothesis generation; however, using these data to quantify potential relationships in exposure-disease relationships is difficult. Many potential confounding variables cannot be assessed (e.g., access to care, lifestyle factors, and dietary habits). Typically, mortality data are population-based, with near universal coverage, and are less prone to bias.

Despite such strengths, however, symptom-disease prevalence surveys and the mortality data reviewed have several limitations. Their ability is restricted to test inferences regarding associations between exposure and health status. They assess exposure and disease simultaneously; thus to determine whether the exposure preceded the disease is not possible. Because these surveys rarely contain quantitative exposure data—either through direct contaminant measurements in a participant's immediate environment or through biomonitoring—using this approach to understand the relationship between exposure and health status is inherently difficult. The Lopez and Carrosquillo symptom prevalence survey, for example, did not include environmental exposure information. In addition, cross sectional surveys of symptom-disease prevalence are often severely compromised by reporting bias from participants' real or perceived problems. For the Lopez and Corrosquillo survey, all data were self-reported or reported via proxy, with no attempt to validate the information using medical records. In such cases, the analysis would benefit from a medical records review to validate self-reported data.

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A third limitation of this survey was a lack of statistical power. The sample was large relative to the population, but still may have lacked the power necessary to identify differences in the observed frequencies and, for many health conditions, in the expected frequencies. Confidence intervals, with risk ratio estimates to identify differences, were not presented.

4.3 Findings

- Access to oncology and other specialty services (e.g., neurology, cardiology) requires travel to the main island of Puerto Rico.
- Analyses of data from the RCCPR (cancer incidence) and the Vital Statistics office (cancer mortality) identified some statistically significant elevations. Statistically significant results (α=0.05) from these analyses are presented in Tables 4-2 and 4-3.
- When compared with all of Puerto Rico, the Lopez and Carrosquillo survey indicated an increased occurrence in Vieques of hypertension, asthma, diabetes, heart disease, and some cancers. A key survey limitation is the lack of supporting medical record inspection to support self-reported claims and to reduce the possibility of information bias.
- An increase in infant mortality was also reported and increases in noncancer mortality for hypertension, cirrhosis, and diabetes.

Table 4-2. Summary of Statistically Significant Standardized Incidence Ratios for Cancer

Outcome	*Period*	*Group*	*SIR, 95% CI*
All cancers	1990 – 1995	Men and Women together	1.19 (1.02, 1.39)
Lung and bronchus	1990 – 1995	Men and Women together	2.25 (1.35, 3.52)
Lung and bronchus	1990 – 1995	Men	2.24 (1.19, 3.83)
Prostate	1990 – 1995	Men	1.47 (1.03, 2.03)
All cancers	1995 – 1999	Men and Women together	1.26 (1.08, 1.47)
All cancers	1995 – 1999	Men	1.31 (1.07, 1.60)
Prostate	1995 – 1999	Men	1.53 (1.09, 2.09)

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Table 4-3. Summary of Statistically Significant Standardized Mortality Ratios for Cancer

Outcome	*Period*	*Group*	*SMR, 95% CI*
All cancers	1990 – 1995	Men and Women together	1.26 (1.01, 1.57)
All cancers	1990 – 1995	Women	1.43 (1.01, 1.96)
Breast	1990 – 1995	Women	0.26 (0.03, 0.92)
Colorectal	1990 – 1994	Women	2.75 (1.11, 5.67)
Oral Cavity and Pharnyx	1990 – 1995	Men	3.96 (1.08, 10.0)
All cancers	1995 – 1999	Men and Women together	1.35 (1.08, 1.67)
All cancers	2000 – 2004	Men	1.40 (1.07, 1.80)
Prostate	2000 – 2004	Men	2.62 (1.66, 3.93)

Vieques has a population of approximately 10,000. Typically, around 30 new incident cases and 15 deaths for all cancers combined occur annually. Therefore, statistical power to detect any meaningful differences in cancer incidence on the island is low when evaluating specific anatomical sites, short time frames, or both. Assessing rates over a longer time span would be useful rather than the traditional 5 years, as in standard cancer surveillance publications. As differential ascertainment for the Vieques residents probably biased the presented, standardized incidence ratios, uniform case identification methods should be applied across all study and referent populations.

4.4 Conclusions and Recommendations

4.4.1 Conclusions

1. The documents ATSDR reviewed paint a complex health picture for Viequenses. The findings of these reports indicate elevations in chronic disease prevalence, cancer incidence, and cancer mortality among the population between Vieques and the rest of Puerto Rico. In addition, increased mortality in Vieques particularly from cancer may indicate lack of access to appropriate medical care. The limitations associated with these analyses, particularly the methodological concerns discussed in this report, introduce considerable uncertainty and make interpretation difficult. Some of the methodological concerns previously noted include more exhaustive cancer case finding in Vieques than the rest of Puerto Rico, differential follow-up, chronic disease prevalence estimates based on a symptom disease prevalence survey without medical record confirmation of health outcomes, and limited noncancer mortality data. Differential follow up and exhaustive case finding may result in biased estimates of cancer incidence and mortality. Uncertainty is also evident in the wide confidence intervals reported indicating imprecise estimates of cancer incidence and mortality ratios in Vieques relative to the rest of Puerto Rico (RCCPR 2009). This is not unusual when calculating estimates for small populations.

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2. These findings can nonetheless serve as a guide for future investigations of Viequenses' health status.

4.4.2 Recommendations

Assess the feasibility of applying the SMART BRFSS methods for generating stable Vieques specific prevalence estimates on asthma, diabetes, hypertension, and other chronic diseases.

4.5 References

[RCCPR] Commonwealth of Puerto Rico Department of Health. 2006. Cancer incidence and mortality in Vieques 1990–2001.

[RCCPR] Commonwealth of Puerto Rico Department of Health. 2009. Cancer incidence and mortality in Vieques 1990–2004.

Lopez Y, and Corrosquillo C. 2002. Exploratory study of health and other conditions in Vieques.

ORC MacroSM. National Program of Cancer Registries, Technical Assistance and Audit Puerto Rico Central Cancer Registry 2000, Case Completeness and Data Quality Audit.

Ortíz Roque C. 2002. Heavy metal exposure and disease in the proximity of a military base. San Juan, Puerto Rico: College of Physicians and Surgeons

Rothman K and Greenland S. 1998. Modern epidemiology. Philadelphia PA: Lippincott-Raven.

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Chapter 5 Local Produce and Livestock Pathway

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Chapter 5 Summary

This chapter evaluates available data and information on contaminants detected in local garden produce and livestock; Chapter 7 considers the effects of direct contact with soil contaminants.

The overall data are insufficient to quantify adequately human exposures or draw any valid health conclusions about whether consuming locally grown produce and livestock would result in harmful health effects.

Limited sampling data are available for pigeon peas, a staple food among Viequenses. A preliminary data evaluation completed for this report has concluded that the level of cadmium reported in samples of locally grown pigeon peas would not contribute excess dietary cadmium to preschool children who eat no more than 5 of the largest (6 ounces) servings per week of locally grown pigeon peas. Adults who eat the largest serving sizes (12 ounces) should limit intake to 11 servings per week. Typical serving sizes for preschool children (1.5 ounces) do not contribute excess cadmium below 20 meals per week and adults who eat a typical serving (3 ounces) may eat up to 44 meals per week without exceeding recommended cadmium intake levels. Exceeding recommended levels of cadmium in the diet would not typically result in immediate health consequences. Excessive cadmium intake over decades, however, could contribute to harmful levels of cadmium accumulation in the kidneys, possibly resulting in kidney disease.

To be protective, these exposure estimates assumed that people consistently ate the largest portions typically consumed at a meal, and that the pigeon peas contained the highest level of cadmium measured. These assumptions, however, likely overestimate the extent of exposure for the majority of the population.

Whether the limited sampling results are representative of cadmium concentrations in other locally grown pigeon peas is unclear; but the significant uncertainty in the evaluation stresses the need to conduct further sampling. Preliminary evaluation results and the totality of the available data suggest a potential for uptake of metals from soil into food crops—thus further investigation is

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warranted. To evaluate better this exposure pathway, ATSDR has recommended a collaborative effort to conduct additional sampling of locally grown foods.

5.1 Introduction

Many Viequenses remain concerned that past military training activities in the eastern portion of their island left elevated levels of heavy metals in the surface soil. Plants could take up these metals. And when people eat those plants, they could be exposed. Viequenses also question whether animals could accumulate heavy metals if they forage in areas where past military activities occurred, and whether this could result in exposure for people who consume meat and milk products from these animals. Viequenses depend on many local animal and vegetable products as food sources and for economic support. Thus, for Viequenses the safety of locally produced foods has both health and economic implications.

5.2 Assessment of Available Data

Since 2000, a number of independent studies have explored levels of heavy metals in Vieques vegetation and livestock. This section provides a brief summary of the results and discusses their strengths and limitations, together with ATSDR's evaluation and response to the findings.

The studies discussed in this section were prepared by scientists associated with the University of Puerto Rico and Casa Pueblo, a local community advocacy organization. Several of these studies were presented and discussed by the authors during the November 2009 meeting in Atlanta, GA.

5.2.1 Studies from 2000–2002

- In 2000, researchers from Casa Pueblo and the University of Puerto Rico sampled the prevailing vegetation in the Live Impact Area (LIA) (specifically Carrucho Beach, Monte David, and Gato and Icacos Lagoons) for heavy metals (Massol Deyá and Díaz 2000). Reference populations of the same species were also collected in Bosque Seco de Guánica and RUM Alzamora Ranch. The plants collected included *Urochloa maxima* (root and stem), *Sporobolus virginicus* (root and stem), *Syringodium filiform* (stem), *Ipomoea violaceae* (fruit), *Faidherbia albida* (stem), *Calotropis procera* (leaves), among other species. The authors reported that the concentrations of heavy metals such as lead, cobalt, and manganese in Vieques vegetation were significantly higher than concentrations detected in the reference locations.
- In 2001, the same researchers randomly collected agricultural and common vegetation from three sites within the residential section of Vieques: an agricultural area in Monte Carmelo, another area of Monte Carmelo that bordered the former Camp García, and an agricultural farm in Barrio Monte Santo, Gobeo sector (Massol Deyá and Díaz 2001). In this paper, discussion was limited to the two farms sampled in the civilian area of Vieques and focused on plants grown for an agricultural economy. Sampling included leaves and stems of pumpkin, pepper, pigeon peas, pineapple, and yucca. The study reported that the concentration of cadmium, cobalt, copper, lead, and manganese exceeded levels reported in typical vegetation in temperate zones (from Smith and Huyck 1999). The authors stated that roots, stems and leaf samples of pumpkin, pepper, pigeon pea, pineapple and yucca had elevated lead and cadmium concentrations while guamá and mango trees did not show any elevated metals concentrations. On August 18, 2001,

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the researchers from Casa Pueblo collected hair samples from goats that grazed in Mount Santos and Santa María on Vieques (Massol Deyá 2002). As a reference, samples were collected from goats that pastured in the main island of Puerto Rico. The publication reported that hair from goats grazing in Vieques contained higher levels of lead than did goats on the main island. No mercury was detected in the goat hair (detection limit was 0.03 µg/g).

Because of these results and other studies by the same researchers reporting elevated levels of heavy metals in plants and livestock, on August 7, 2001, the Puerto Rico Department of Agriculture (PRDA) placed an embargo on Vieques produce and livestock. The Puerto Rico Department of Agriculture, in cooperation with the Farmers Association of Puerto Rico, sampled grass, fruit-bearing trees, and bovine livestock from Monte Carmelo, Martineau, Monte Santo, Esperanza, Lujan, Gubeo, and western Vieques for cadmium, cobalt, copper, lead, manganese, and nickel. The Department of Agriculture and the Farmer's Association concluded that the agricultural products from Vieques were suitable for consumption and did not contain toxic levels of these metals (PRDA 2001). When the Department of Agriculture and the Farmer's Association could not duplicate the Massol Deyá and Díaz' studies, the embargo was lifted (El Nuevo Día 2001).

5.2.1.1 ATSDR Evaluation

During preparation of the public health assessment evaluating soil exposure (ATSDR 2003), ATSDR cited two of the studies (Massol Deyá and Díaz 2000, 2001). These studies reported elevated metals in local vegetation and agricultural areas. This issue was originally addressed as a community concern in the Soil Pathway Evaluation Public Health Assessment (ATSDR 2003).

ATSDR requested that an agronomist with the U.S. Department of Agriculture independently review the 2000 and 2001 Massol Deyá and Díaz studies. The agronomist determined (USDA 2002) that although the studies provided evidence that heavy metals were accumulating in plants, the study's limitations prevented estimation of a human exposure dose:

1. Humans do not eat many of the species sampled in this study. When edible species were sampled, the edible portions were not. Human exposures from locally grown foods are best estimated from the edible portions of the food source. In general, the edible portions of plants are less likely to accumulate metals from soil because of normal plant processes (e.g., physiological barriers that prevent contaminants from getting to the tops of plants) (ATSDR 2001).
2. The study lacked the use of standard reference materials to demonstrate that the results were accurate. It also lacked background corrections for lead, cobalt, nickel, and cadmium to eliminate the effect of light scattering by any nonelement materials in the samples, which can result in a concentration overestimate.
3. The metals analysis did not indicate whether the metals were in a bioavailable form (i.e., in a chemical form readily absorbed into plant tissues from the surrounding environment). This is important because vegetation samples include both contaminants taken up by the plant and soil particles from the surrounding environment. Even with a thorough washing, small soil particles will adhere to the plant materials and can actually carry more contaminants than those taken up

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by the plant from the soil (ATSDR 2001). The researchers did not determine how much of the chemical was adhered externally as soil and how much was in the plant tissue.

Because of these limiting factors, ATSDR could neither quantify human exposures appropriately nor draw any valid health conclusions about whether consuming Vieques plants would result in harmful health effects. ATSDR has recommended additional sampling of locally grown produce that would improve the evaluation of this exposure pathway. Details of ATSDR's recommendations are at the end of this chapter and in Chapter 9 of this report.

ATSDR was unable to evaluate human exposure to goat meat and milk products from the sampling data presented in Massol Deyá (2002). Information was missing on similarities and differences between the goats in Vieques and the reference population (e.g., goat species, age, whether differences were statistically significant) and how they might affect interpretation of the hair sampling results. The report was unclear regarding how the hair samples were prepared for analysis. Inadequate processing of the hair for chemical analysis can lead to misleading results and to difficulty in interpreting whether the data represent contamination internal to the hair shaft or incomplete removal of any external contamination. Additionally, limitations in interpreting the health significance of hair sampling data precluded ATSDR from using the goat hair data reported in Massol Deyá (2002). Three key limitations of hair analysis generally prevented further characterization of exposures:

1. Hair analysis results cannot pinpoint the sources of detected chemical contaminants. For instance, hair analysis results typically cannot distinguish substances deposited onto hair (e.g., dusts) from substances that might have distributed into hair following an environmental exposure, such as ingestion of contaminated drinking water or food. In other words, hair analysis generally cannot differentiate internal from external exposure.
2. Currently, the scientific community does not know the range of contamination levels typically found in hair. Without reliable data in the peer-reviewed literature on baseline or background hair contamination levels in the general population, and without hair samples collected from comparison populations, health agencies cannot determine whether hair analysis results from a given site are unusually high or low. In Massol Deyá (2002) a comparison population of goats was sampled, and the results supported the author's conclusion that the goats sampled on Vieques had higher concentrations of heavy metals in hair relative to the comparison population.
3. A critical input into public health assessments is an understanding of the dose—whether measured or estimated. Because we currently know so little about rates at which substances in human and animal bodies distribute into hair, calculation was not possible of internal doses from hair sampling results. Hair analysis, then, would provide no added insight into estimating exposures to humans, but it does imply a need to sample directly meat and milk from these animals to determine whether the meat and milk are safe for consumption.

For most substances, insufficient data are currently available to support any prediction of adverse health effects from concentrations in hair. Detection of a substance in a hair sample generally will not tell how, when, or where exposure to that substance occurred. Data from plant and animal foods that people

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actually consume are best suited for determinations about the possible health implications for people who eat those foods. Accordingly, as part of its evaluation, ATSDR recommends additional sampling of local produce and livestock.

5.2.2 Studies from 2003

In 2003, researchers from University of Puerto Rico published a study discussing the same data the authors published in April 10, 2000—plant samples collected in February and March 2000 and analyzed for heavy metals (Díaz and Massol Deyá 2003). This paper compared concentrations found on Vieques with concentrations detected on the main island of Puerto Rico. The 2000 sampling included plant stems and leaves but did not include the edible portions of the plants sampled. This peer-reviewed publication provided more information on sampling, preparation, and analytical techniques than did the April 2000 report. In addition to the 2000 sampling data, this paper included new samples of *C. cajan* (pigeon peas) leaves and fruit taken August 18, 2001. It also discussed the ratio of contamination detected in the fruit to contamination in the leaves. The study compared concentrations detected in pigeon pea fruit and leaf samples collected August 18, 2001. For some metals, higher concentrations were found in the fruit (zinc, nickel, cadmium and cobalt). For other metals, higher concentrations were found in the leaves (copper and lead).

5.2.2.1 ATSDR Evaluation

In May 2009 ATSDR obtained a copy of the 2003 Díaz and Massol Deyá study. As part of its evaluation of Vieques, ATSDR reviewed the pigeon pea data in this study. The data reported in Díaz and Massol Deyá have significant limitations that restrict ATSDR's ability to interpret the study findings' significance. These limitations include no mention of comparison samples and no indication of statistical significance in the data reporting pigeon-pea metal concentrations. The sampling results from only one study are not representative of the extent of cadmium in local produce and do not represent the potential for cadmium exposure to the general population from consuming locally grown pigeon peas.

A preliminary evaluation concluded that the level of cadmium reported in a few samples of locally grown pigeon peas would not contribute excess dietary cadmium to preschool children who eat no more than 5 of the largest (6 ounces) servings per week of locally grown pigeon peas. Adults who eat the largest serving sizes (12 ounces) should limit intake to 11 servings per week.

To be protective, these exposure estimates assumed that people consistently ate the largest portions typically consumed at a meal, and that the pigeon peas contained the highest level of cadmium measured. These assumptions likely overestimate the extent of exposure for the majority of the population. Typical serving sizes for preschool children (1.5 ounces) do not contribute excess cadmium below 20 meals per week. Adults who eat a typical serving (3 ounces) may eat up to 44 meals per week without exceeding recommended cadmium intake levels. Still, excessive cadmium intake over decades could contribute to harmful levels of cadmium accumulation in the kidneys, possibly resulting in kidney disease. Table 5-1 provides details on the methodology used in this evaluation.

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Table 5-1. Exposure dose for pigeon pea fruit (Díaz and Massol Deyá 2003 data)

	Percentile		*C mg/kg*	*IR kg/day*	*Portion size in Oz*	*Meals per week*	*Meals per Year*	*EF days/yr*	*ED Yrs*	*BW kg*	*AT days*	*FA unit less*	*DOSE (mg/kg/day)*	Target dose (mg/kg/day)
Chemical	IR													
cadmium	95th	adult	3.17	0.10	12	5	260	260	64	70	23360	0.05	0.00016	0.00036
	95th	child	3.17	0.05	6	5	260	260	6	16	2190	0.05	0.00035	
Number of weekly meals to reach tolerable daily intake level														TDI
	95th	child <6 yr	3.17	0.05	6	5	260						0.00036	0.00036
	50th	child <6 yr	3.17	0.01	1.5	20	1040						0.00036	
	95th	adult	3.17	0.10	12	11	572						0.00036	
	50th	adult	3.17	0.02	3	44	2288						0.00036	

Where

C Concentration (mg/kg)

IR Ingestion rate in percentile (e.g. 95th means that 95% of the population has an intake level below that amount): see below

EF Exposure frequency or events: 260 days/year = 5 days/week for 52 weeks/year; 156 days/year = 3 days/week for 52 weeks/year

ED Exposure duration over which exposure occurs: 64 years (adult); 6 years (child)

AT Averaging time: period over which exposures are averaged (expressed in days) for noncancer AT=ED*365 days/year

FA Food absorption factor: Assumes dietary cadmium absorption of 5%. (ATSDR Toxicological Profile for Cadmium, Sep 2008)

BW Body weight in kilograms

TDI Tolerable daily intake, 0.00036 mg/kg-bw/day, European Food Safety Authority (EFSA) 2011

dry weight
rate

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95th adult 98.7 g/day See EFH Chapter 9, Section 9.2.5 for conversion between as consumed and dry weight intake rates

50th adult 24.8 g/day used 71.8 percent water content for "beans –dry–black-eyed peas (cowpeas)–cooked," see Table 9-27

95th child 1.6 g/kg/day

50th child 0.4 g/kg/day

USEPA. 1997. Exposure Factors Handbook. August 1997. Available from: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20563

Tolerable daily intake

TWI= tolerable weekly intake

2.5 µg/kg BW EFSA 2009

0.00036 mg/kg/day Converted to daily intake

5.2.3 Studies from 2004–2005

- In 2004, researchers from the University of Puerto Rico's Agricultural Extension Service in Vieques and the Land Authority of the Commonwealth of Puerto Rico collected a total of 72 samples of smooth cayenne pineapple fruit, leaf tissues, and soil. They collected these samples from two pineapple plantations (one located in Barrio Luján in the civilian zone in Vieques and a control located in Barceloneta on the mainland of Puerto Rico). The samples were to determine the total concentration of arsenic, cadmium, chromium, cobalt, and lead in the fruit. The author concluded that no contamination was found in the pineapple fruit or leaf samples from either plantation. In all fruit samples arsenic, cadmium, cobalt, and lead either were below levels of detection or were not detected. No contamination was observed in the pineapples from Vieques when compared with those from the Barceloneta plantation (Lopez Morales 2005). Higher levels of metals were, however, found in the soil at the Barceloneta plantation, which the author attributed to the soil's natural composition. The study concluded that the results were within accepted regulatory levels.
- In 2005, Massol Deyá et al. published a paper discussing the same data that the authors published in April 10, 2000, and again in 2003—plant samples collected in February and March 2000 and analyzed for heavy metals. The authors noted that "distinctive profiles are observed within the studied species thus reflecting differences in their physiological properties." In addition to the 2000 sampling data, this paper included new samples of *Syringodium filiforme* (Manatee grass) taken from the Atlantic Fleet Weapons Training Facilities (AFWTF) in 2004 and from Guánica State Forest in 2003 and 2004. Concentrations of heavy metals were higher at AFWTF. The authors proposed the hypothesis of bioaccumulation through the food chain: manatee grass to crustaceans, fish eat the crustaceans and then are eaten by people. A similar food-chain scenario was hypothesized for migratory birds (Massol-Deyá et al. 2005).

5.2.3.1 ATSDR Evaluation

The data in Massol-Deyá et al. (2005) might well provide ecologically important information, but they are not directly relevant to evaluating human health—manatee grass is not typically a Viequense food source. ATSDR has recommended additional produce sampling.

The López Morales (2005) study was unique in that its purpose was specifically to sample the edible portion of a commonly consumed agricultural product grown in Vieques, *smooth cayenne* pineapples. As part of the evaluation of Vieques, ATSDR evaluated the level of chromium—the only metal detected—in the pineapple fruit samples. Using a standard intake rate for fruit consumption,[24] ATDSR calculated the estimated exposure dose at below levels of concern for adverse health effects.

López Morales (2005) cited two additional studies that found "no contamination in the civilian zones of Vieques": 1) College of Agriculture & Environmental Science: Soil, Plant and Water Laboratory (September 6, 2001), and 2) A & L Southern Agricultural Laboratories, Inc. (August 31, 2001). Both

[24] About 7 ounces of fruit a day (mean total fruit intake; USEPA 1997)

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studies analyzed for heavy metals in guinea grass, sugar apple, wild tamarind, mango, green banana fruit, pigeon peas, genip fruit, grapefruit, and buffet grass. These studies sampled species Viequenses regularly eat. ATSDR is attempting to obtain copies of these studies, but to date has not been successful.

5.2.4 Studies from 2008

In January 2008, leaves from cilantro, spinach, beans, peppers, and papaya plants were collected from a Viequense organic farm and analyzed for cadmium, chromium, copper and lead. The researchers believed the organic farm's soil would be representative of naturally occurring soil plus any local, airborne contamination. The researchers found that concentrations of lead and cadmium in the Viequense vegetation were significantly higher than those detected in the Mayagűez reference plants (Díaz de Osborne et al. 2008). Mayagűez is a town on the western end of Puerto Rico, approximately 120 miles from Vieques

5.2.4.1 ATSDR Evaluation

In reviewing the Díaz de Osborn et al. (2008) report, ATSDR was initially concerned that the data appeared to indicate potential harm to humans consuming this produce. Due, however, to the report's lack of any data quality assurance information, ATSDR questioned whether those data accurately represented typical heavy metal concentrations in locally grown produce. ATSDR consulted with the U.S. Department of Agriculture (USDA) about the study (R. Chaney, U.S. Department of Agriculture, Agricultural Research Station, Beltsville, MD, personal communication, 2008 Oct 28–30). USDA noted that the reported levels of copper would be phytotoxic to the plants. USDA commented that the reported measurements were consistent with plants grown in soils that had been changed to increase metal uptake in the plants (Sterrett et al. 1996). As a result, how ATSDR should interpret these data is unclear. ATSDR has recommended additional sampling of locally grown produce to resolve uncertainties in the available data. Still, until additional data are available and this exposure pathway is more thoroughly evaluated, the USDA suggests following several simple methods for reducing metal uptake into garden produce from soil. More information is available at USDA's National Agriculture Library Web site: http://www.nal.usda.gov/. Specialized gardening information is available online at: http://riley.nal.usda.gov/nal_display/index.php?info_center=8&tax_level=2&tax_subject=7&topic_id=1063.

The conclusions and recommendations from ATSDR's evaluation of local produce and livestock data follow in section 5.3 of this chapter and in Chapter 9 of this report.

5.3 Conclusions and Recommendations

5.3.1 Conclusions

1. The overall data are insufficient to quantify adequately human exposures or draw any valid health conclusions about whether consuming locally grown produce and livestock would result in harmful health effects.

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2. Limited sampling data are available for pigeon peas, a staple food among Viequenses. Interpretation of these data is uncertain because of the lack of adequate QA/QC information for the analytical findings.

    a. A preliminary data evaluation completed for this report has concluded that the level of cadmium reported in a few samples of locally grown pigeon peas would not contribute excess dietary cadmium to preschool children who eat less than 5 of the largest (6 ounces) servings per week of locally grown pigeon peas. Adults who eat the largest serving sizes (12 ounces) should limit intake to 11 servings per week.

    b. Typical serving sizes for preschool children (1.5 ounces) do not contribute excess cadmium below 20 meals per week and adults who eat a typical serving (3 ounces) may eat up to 44 meals per week without exceeding recommended cadmium intake levels.

    c. Exceeding recommended levels of cadmium in the diet would not typically result in immediate health harm. Excessive intake of cadmium over decades could contribute to harmful levels of cadmium accumulation in the kidneys, possibly resulting in kidney disease. Typically, only a very small fraction (5%) of cadmium in food is absorbed in the body, especially in individuals with healthy diets containing adequate amounts of essential minerals such as zinc, iron and calcium (Reeves and Chaney 2008).

3. Whether the limited sampling results are representative of cadmium concentrations in other locally grown pigeon peas is unclear; but the significant uncertainty in the evaluation stresses the need to conduct further sampling.

4. Preliminary evaluation results and the totality of the available data suggest a potential for uptake of metals from soil into food crops—thus further investigation is warranted. To evaluate better this exposure pathway, ATSDR has recommended a collaborative effort to conduct additional sampling of locally grown foods.

5.3.2 Recommendations

ATSDR supports additional sampling and collaborative data collection to evaluate more thoroughly this human exposure pathway. The following recommendations are intended to promote collection of high quality data of maximum utility, and to minimize data uncertainties:

1. Sampling data should represent edible portions of a cross-section of local produce from local farms, home gardens, and local markets. Sample produce that tends to accumulate heavy metals more easily (e.g., leafy vegetables such as lettuce and spinach) rather than leaves of grain or fruit crops.

2. Survey residents to identify the prevalence and type of locally grown and commonly consumed garden produce. Sampling plan design should include produce types identified from this survey.

3. Collect representative surface soil samples at the same location and time that garden and farm produce samples are taken. Soil samples should be of appropriate depth to represent the root zone.

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4. Collect samples of meat and milk products from Vieques forage-consuming livestock.
5. To avoid false high or low values, sample collection and analysis should conform to high standards of data quality, including detailed quality assurance/quality control information, standard reference materials for analysis, and background correction. Design the sampling plan to collect a sufficient number of samples to ensure high statistical confidence.
6. Collect samples from an appropriate background or control location for comparison with food samples produced on Vieques.
7. To facilitate exposure assessment, report data as consumed (wet weight).
8. Plan and conduct sampling protocols in collaboration with local scientists. One way to avoid misunderstandings about the data is for local scientists and an independent expert jointly collect and split several samples and provide analytical reports with QA/QC supporting information. A QA/QC standard could be split at the time the samples are collected.
9. Request technical assistance from the U.S. Food and Drug Administration (FDA) or the U.S. Department of Agriculture (USDA) in sample collection and analytical protocols. The USDA Agriculture Research Service has offered to provide, if requested, technical assistance in sampling plan design, collection, and analysis with proper QA/QC protocols.

5.4 References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2001. Summary report for the ATSDR expert panel meeting on tribal exposures to environmental contaminants in plants. Atlanta: Division of Health Assessment and Consultation; 23 March.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003. Public Health Assessment, Isla de Vieques Bombing Range, Vieques, Puerto Rico. Atlanta: US Department of Health and Human Services, Public Health Service; 7 February.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2008. Toxicological profile for cadmium. Atlanta: US Department of Health and Human Services, Public Health Service; September.

Díaz de Osborne E, Acevedo-Acevedo P, Massol Deyá A. 2008. Evaluation of heavy metals in agricultural products of a farm with organic practices in the Bo. Lujan Sector Destination of Vieques (Puerto Rico). Casa Pueblo de Adjuntas, PR: University of Puerto Rico Biology Department.

Díaz E and Massol Deyá A. 2003. Trace element composition in forage samples from a military target range, three agricultural areas, and one natural area in Puerto Rico. Caribbean Journal of Science 39;2:215–20.

El Nuevo Día. 2001. They declare suitable the agricultural products in Vieques; 13 December.

[EFSA] European Food Safety Authority. 2011. Scientific Opinion, Statement on the tolerable weekly intake for cadmium, EFSA Panel on Contaminants in the Food Chain (CONTAM). EFSA Journal 2011, 9(2): 1975. Available online at: http://www.efsa.europa.eu/en/efsajournal/pub/1975.htm [accessed 2012 Nov 15]

López Morales JL. 2005. Determination of arsenic (As), cadmium (Cd), chromium (Cr), cobalt (Co) and lead (Pb) in "smooth cayenne" pineapple fruit, leaves tissue and soil using inductively coupled plasma-optical emission spectrometry (ICP-OES). Mayagüez, PR: University of Puerto Rico. Available at: http://www.conucopr.org/ViewRecord.do;jsessionid=9352FA03CF29511445F3F457499BD7F1?id=11161028 [accessed 2012 Aug 28].

Massol Deyá A and Díaz E. 2000. Heavy metals in the impact area prevailing vegetation, Vieques, Puerto Rico. Casa Pueblo de Adjuntas, PR: University of Puerto Rico, Recinto Universitario Mayagüez, Biology Department; 10 April.

Massol Deyá A and Díaz E. 2001. Toxic metals in the vegetation of the civilian zone of Vieques, Puerto Rico. Casa Pueblo de Adjuntas, PR: University of Puerto Rico, Recinto Universitario Mayagüez, Biology Department; 10 January.

Massol Deyá A. 2002. Herbivorous: additional evidence of heavy metal mobilization through the food web. *In:* Ciencia y Ecología: Vieques en Crisis Ambiental, Third edition. Casa Pueblo de Adjuntas, PR.

Massol-Deyá A, Pérez D, Pérez E, Berrios M, Díaz E. 2005. Trace elements analysis in forage samples from a U.S. Navy bombing range (Vieques, Puerto Rico). Int J Environ Res Public Health 2(2):263–66.

[PRDA] Puerto Rico Department of Agriculture. 2001. Letter from Luis Colón to the Undersecretary of Agriculture concerning heavy metal tests on Vieques bovine livestock; 17 October.

Reeves PG, Chaney RL. 2008. Bioavailability as an issue in risk assessment and management of food cadmium: A review. Science Total Environ 398:13–19.

Smith KS, Huyck HLO. 1999. An overview of the abundance, relative mobility,

bioavailability, and human toxicity of metals. Reviews in Economic Geology 6A:29-70.Sterrett SB, et al. 1996. Influence of fertilizer and sludge compost on yield and heavy metal accumulation by lettuce grown in urban soils. Environ Geochem Health 18:135-42.

[USDA] US Department of Agriculture. 2002. March 5th letter from Rufus Chaney (USDA) to Jeff Kellam (ATSDR) concerning expert review of two vegetation sampling studies by Massol-Deya and Díaz.

[USEPA] US Environmental Protection Agency. 1997. Exposure factors handbook (Final Report). EPA/600/P-95/002F a-c, 1997; Aug. Available online at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20563 [accessed 2012 Aug 28]

Chapter 6 Air Pathway

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Chapter 6 Summary

This chapter reviews how potential airborne exposures to contaminants from military operations at the former Vieques Naval Training Facility might have affected Viequense residential areas. Although our review draws primarily from sources used in the 2003 ATSDR Air PHA, we also summarize air monitoring data and air dispersion studies developed or collected after publication of the 2003 Air PHA. These later data assessed effects of ongoing or proposed open detonation or open burning and included site-specific meteorological data collected after 2003.

We further evaluate the modeling process used in the 2003 Air PHA, the assumptions and data used in that modeling process, and compare the results with monitored data collected during recent open burning events. While this review finds the Air PHA modeling process consistent with established practices, it does identify two minor errors in estimating detonation source concentrations. These errors, however, do not affect the results or conclusions of the Air PHA.

Collectively, the available sampled and modeled data are adequate for the determination of any potential public health hazard arising from exposure to airborne contamination. By overestimating numerous aspects of the contaminant emissions and by assessing exposures for worst-case conditions, the 2003 ATSDR air dispersion model adequately addressed uncertainties inherent in the air dispersion modeling process.

The model results indicate that in the residential areas of Vieques, airborne contaminants from historic military exercises on the Vieques Naval Training Range would have been essentially nondetectable and unlikely to have resulted in harmful effects. These results are supported by post 2003 air monitoring studies conducted in support of the ongoing site remediation.

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6.1 Introduction to Air Pathway

In 1980, not long after ATSDR's inception, Viequenses began to ask the agency whether the island's air was safe to breathe. The residents were and are concerned about contaminants released to the air during the Navy's military training exercises. These exercises included the Navy's past use of explosive ordnance (early 1970s to April 1999), as well as exercises using inert ordnance (May 2000 to May 2003).[25] Among other concerns, Viequenses questioned whether toxic dusts from the explosive-ordnance range could have blown into their neighborhoods.

During the time the Navy still conducted explosive-ordnance exercises on Vieques, three air-sampling studies occurred (1972, 1978, and 1979). Direct references for these studies are not available, but other documents reported and summarized the results (TAMS 1979; Cruz Pérez 2000). Appendix C of the Air PHA (ATSDR 2003) includes a review of the studies' data, the availability of that data, and each dataset's limitations. The Puerto Rico Environmental Quality Board conducted two of these studies, and the Navy conducted the other. None of the studies' measurements detected air pollution at levels of health concern. Those conclusions are limited by the small number of air sampling locations and the small number of measured analytes. And because those historic sampling studies did not employ data quality control procedures that are accepted today, the studies could not conclusively determine by current standards whether airborne contaminants were present in Viequense residential areas.

Thus ATSDR could not base health conclusions on these studies alone. Modeling, however, could provide another dataset that might clarify earlier findings.

When ATSDR first began evaluating the air pathway, two dispersion-modeling studies were available that produced distinctly different conclusions about potential exposures to particulate matter on Vieques. One was prepared by Navy contractors (IT 2000) and a second by Dr. Cruz-Pérez, a local professional engineer (Cruz Pérez 2000). As part of the 2003 PHA process, ATSDR hired three independent air modeling experts to review the studies. Appendix D of the Air PHA includes a summary and review of each of the modeling studies. After receiving feedback from the external reviewers, ATSDR decided to conduct an additional, independent modeling study using recommendations from the peer reviewers. Note that given the limited extent of air sampling that occurred at Vieques while the Navy used explosive ordnance, modeling was necessary. Appendix D in the Air PHA contains model documentation, including defining assumptions and results.

6.2 Assessment of Available Data

6.2.1 Assessment of Air Exposures in the Public Health Assessment

To estimate effects of explosive ordnance on air quality, ATSDR used a modeling analysis that evaluated 86 different contaminants known to be released to the air when ordnance explodes (49 organic compounds, 29 metals, 5 explosive compounds, plus chaff, depleted uranium, and particulate matter such as PM10). The model results found that as dust and smoke traveled from the explosive-ordnance

[25] The Air Public Health Assessment includes a more complete summary of the Former Vieques Naval Training Facility operational history.

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range toward areas where people lived, chemicals released to the air by explosions dispersed to extremely low levels. For most contaminants, the predicted air quality effects at residential areas were so low that even if highly sensitive air sampling devices were in place they likely could not measure such low levels of the contaminants.

For example, particulate-matter contaminants emitted during explosive ordnance exercises were predicted to account for less than 1 percent of the particulate-matter[26] concentrations measured in Viequense residential areas.

This comparison suggests that LIA emissions had a small effect on residential air quality. Using this modeling analysis and the previously referenced air sampling data, ATSDR concluded that of the 86 contaminants evaluated that might have been part of emissions from the former Vieques Naval Training Range, by the time they reached the residential areas of Vieques none exceeded any health comparison values. In other words, even the total effect of the chemicals combined did not result in residential air pollution.

From May 2000 through May 2003, the Navy conducted military training exercises with inert (nonexplosive) ordnance. During 2000–2001, the Puerto Rico Environmental Quality Board conducted ambient air monitoring at two stations in Viequense residential areas. On days when such inert ordnance fell on the explosive-ordnance range, the Puerto Rico Environmental Quality Board collected more than 50 particulate-matter samples in the residential areas of Vieques. These data and supporting information are reported in the U.S.EPA Air Quality System (AQS)—formerly the Aerometric Information Retrieval System (AIRS)—and summarized in the Air PHA (Appendix C). In every sample, the levels of particulate matter were much lower than health-effects levels. In fact, no clear relationship was ever established between the quantity of inert ordnance dropped in the LIA and the levels of air pollution measured in the island's residential areas. These observations on estimated contaminant air concentrations led ATSDR to conclude that on days when inert ordnance fell into the LIA, levels of air pollution did not pose a health hazard to the island's residents.

In addition to the modeling study and the analysis of historic air monitoring data, ATSDR conducted on-site air monitoring during the June 2001 Navy air-to-ground, inert ordnance exercises. The results of this sampling effort are documented (ERG 2001), and the Air PHA describes their limitations. Due to station location/siting problems (as discussed in the Air PHA), the sampling data do not meet applicable quality control standards. The measured contaminant concentrations are nonetheless consistent with the public health conclusions.

The following Air PHA conclusions are based largely on ATSDR's modeling effort and on data from air monitoring stations at Isabel Segunda and Esperanza established and maintained by the Puerto Rico Environmental Quality Board:

[26] The term "particulate matter" refers to solid particles and liquid droplets in the air. PM10 and PM2.5 refer to particulate matter having aerodynamic diameters less than or equal to 10 and 2.5 microns, respectively. Also note that while current emphasis on the health effects of particulate matter focus on PM2.5, past health-based standards used PM10. The 2003 Air PHA used PM10 to make direct comparisons with earlier measurements and model results.

- The Navy's ordnance exercises at the LIA did not pose a health hazard in the residential areas of Vieques.
- Wind-blown dust from the explosive-ordnance range did not and does not pose a health hazard to residents.

The Air PHA also contains a thorough review of the then-available modeling studies, the ATSDR modeling study, and other reported air monitoring datasets.

6.2.2 Review of the Air Modeling Process Used in the Air PHA

Eastern Research Group (ERG) and Trinity Consultants conducted the ATSDR air modeling study to evaluate potential air exposures from historic operations at the former Vieques Naval Training Range. The Air PHA and a follow-up report (Wilhemi et al. 2006) describe the specific procedures, assumptions, and results of the modeling effort. Trinity Consultants conducted the "Calpuff" dispersion modeling and submitted the results to ERG (Trinity Consultants 2002).

Direct measurement of airborne emissions produced by military ordnance explosions is not possible.[27] Quantitative modeling, however, is a useful approach for assessing potential emissions produced by explosive ordnance events. To evaluate potential exposures to airborne contamination, the Air PHA used the following modeling procedure:

1. Make health-protective estimates of airborne emission concentrations from individual ordnance items using the Combined Obscuration Model for Battlefield Induced Contaminants (Army Research Laboratory 2000);
2. Use a U.S.EPA-accepted air dispersion model (Calpuff) to estimate event-specific, health-protective, 24-hour and annual contaminant concentrations at points of maximum off-site exposure;
3. Calculate maximum daily and annual contaminant concentrations at off-site exposure points using documented ordnance usage data to scale up the event-specific, dispersed-contaminant concentrations; and
4. Evaluate the potential public health effects of exposure to those contaminants by comparing the estimated maximum 24-hour and annual contaminant concentrations with appropriate health comparison values.

This review finds that the overall rationale for that approach is sound, and each of the assumptions underlying the selection of model parameters is well documented in the Air PHA and in the literature (text and Appendix D; Wilhemi et al. 2006). The modeling procedure was developed from comments and recommendations by external, independent reviewers who assessed the existing studies.

A number of factors or model assumptions within the 2003 study collectively led to an intentional overestimation of potential exposures, thus helping to ensure the conclusions are health-protective:

[27] The blasts would destroy the monitoring equipment; indirect downwind sampling is possible, however.

- Overestimate particulate emission rates from explosions by assuming all particulates are composed of the PM10 fraction (larger particles not susceptible to significant air dispersion are included as PM10).
- Overestimate emission rates of explosives (e.g., TNT, RDX, HMX, aluminum powder) by assuming 10% of explosives are not consumed in the explosion and are available for dispersion. By contrast, available data show that less than 1% of explosives remain after detonation.
- Because of variations in explosive composition of different ordnance types, maximum composition percentages of TNT, RDX, and aluminum powder were assumed to comprise collectively more than 125 percent of total explosive use.
- The emission estimates also assumed that all ordnance dropped, fired, or launched into the LIA exploded, and that all emissions emanated from a single-source location. Thus bombs exploding in multiple locations would spread the emissions over a relatively large area, leading to greater dispersion and lower concentration at any single downwind site.
- Potential exposure evaluations are all based on worst-case meteorological conditions for both 24-hour and annual exposures for a person living directly on the site boundary (i.e., the location of highest off-site concentration estimates). Residential areas are at least 3 miles farther west which, for off-site residents, would result in commensurately lower exposure doses.

Three EXCEL spreadsheets developed by ERG (METALS.xls, ORGANICS.xls, and EXPLOSIVES.xls) contain the modeling study's specific results. These spreadsheets were transmitted to ATSDR via e-mail (John Wilhelmi, ERG, to Mark Evans, ATSDR, personal communication, 2009 Dec 3). A review of the spreadsheets has revealed two minor errors or discrepancies.

First, metals concentrations within the particulate plume are calculated by adding metals in the explosive compounds, metals in the ordnance casing, and metals in the soil ejected into the air by the explosion. Concentrations of explosives (e.g., TNT, RDX, and HMX) present in Live Impact Area soils are not similarly accounted for in assessing plume concentrations of those materials. Still, because the concentrations of explosives compounds of LIA soils are so low, the relative contribution from soil ejecta is insignificant.

As an example, the concentration of TNT in LIA soil averages 2.9 parts per million (ppm). Maximum annual emissions of TNT as soil ejecta (based on the TNT soil concentration of 2.9 ppm) would be approximately 1.6 pounds per year (or 0.00027 pounds per hour; based on 5,840 hours per year operations). In comparison, the ATSDR air dispersion model assumes that airborne emissions from undetonated TNT in bombs (assuming 10% of TNT does not ignite) is 10.76 pounds *per hour*. If only 5% of TNT remains after the explosion, this hourly TNT emission rate becomes 5.38 pounds *per hour*. Considering that available studies show less than 1% of TNT does not ignite, the amount of TNT in the air is overestimated and, relative to the overestimate from TNT ignition, the contribution from TNT in soil ejecta is very small.

The second discrepancy detected in the ERG spreadsheets concerns calculation of maximum 24-hour emission rates of metals in air particulates. This calculation neglected to include a conversion factor for

adjusting emission rates in pounds per hour to emissions in grams per second. Considering that an emission rate of 1 pound per hour is about 8 times greater than an emission rate of 1 gram per second, the use of an input value 8 times too large results in an eightfold overestimate of the maximum 24-hour emission rate. Collectively, however, the discrepancies in the air dispersion modeling process do not significantly affect the results or conclusions expressed in the 2003 Air PHA.

The Air PHA's Appendix E contains 39 comments on the modeling process and the PHA conclusions, and ATSDR's responses to those comments. We reviewed those comments and responses to ensure that each response adequately addressed each concern. This review finds that the responses were appropriate and provided adequate support for the Air PHA assessment procedure and the resultant public health conclusions.

We note, however, one specific concern and the response thereto (Air PHA, comment/response #8, Appendix E). The stated concern was "No data is (sic) available related to PM2.5 . . ." The response indicates that in fact PM2.5 was not explicitly modeled. But it is addressed by making the most health protective assumption possible (i.e., that the estimated PM10 concentrations consisted entirely of PM2.5 particulates). Thus the annual average and maximum 24-hour concentration increases caused by explosive ordnance events (0.04 µg/m^3 for annual average; 10.2 µg/m^3 for 24 hour average) would still be (from the 2003 Air PHA):

> " . . . considerably lower than EPA's current (PM2.5) health-based standards (15 µg/m3 for annual average, and 65 µg/m^3 for 24 hour average concentrations). Therefore, the modeling data indicate that air emissions of particulate matter, whether coarse or fine, from the military training exercises were not at levels of health concern for the residential areas of Vieques."

6.2.3 Post-PHA Air Monitoring and Air Modeling Studies

After the Air PHA's release, the Navy conducted additional air monitoring and modeling related to the "Time Critical Removal Actions" for the blow-in-place (BIP) munitions detonations. That is, for the removal of dropped, launched, or fired unexploded ordnance and for a planned effort to burn vegetation from the live-ordnance training areas. These efforts were designed to allow safe access to historic live fire areas, and were documented in a series of reports:

1. Air Dispersion Modeling of the TCRA/BIP Activities on the Former Vieques Naval Training Range, Draft (US Navy Contract Task Order 0047, February 2007; prepared by CH2MHill, Herndon, VA).
2. Air Dispersion Modeling for TCRA-Prescribed Vegetation Burns on the Former Vieques Naval Training Range, Draft Final (US Navy Contract Task Order 0047, June 2008; prepared by CH2MHill, Herndon, VA).
3. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: August 2005 through December 2006, Draft (US Navy Contract Task Order 0047, February 2007, prepared by CH2MHill, Herndon, VA).

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4. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: January 1, 2007 through March 31, 2007, Draft (US Navy Contract Task Order 0047, June 2007, prepared by CH2MHill, Herndon, VA).
5. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: April 1, 2007 through June 30, 2007, Draft (US Navy Contract Task Order 0047, January 2008, prepared by CH2MHill, Herndon, VA).
6. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: January 1, 2008 through June 30, 2008, Draft (US Navy Contract Task Order 0047, February 2009, prepared by CH2MHill, Herndon, VA).

The air monitoring data summaries describe real-time, on-site (downwind) sampling events conducted before, during, and after 51 in-place ordnance detonation events at the LIA. The information in these reports shows that the monitoring analyses were conducted using accepted methods, and that the results were within acceptable limits for sampling and analytical quality assurance.

Analytes measured were PM10, metals, and explosives at hourly or 8-hour intervals. Measurements were summarized as 8-hour or 24-hour averages. No explosives or metals in any of the air samples have been reported in concentrations above regulatory or applicable health based standards. One detonation event might have ignited surrounding vegetation; it resulted in a 24-hour, PM 10 value of 153 µg/m3—just above the NAAQS standard of 150 µg/m3. This measurement, however, was taken at an on-site location immediately downwind of the detonation site. None of the 51 detonation events produced elevated PM 10 measurements at stations closer to areas of potential off-site exposure. The exposure levels for metals are more than 10 times below the health guideline. Thus interaction effects between chemicals are not likely, and additive or synergistic effects between chemicals are not likely to enhance the mixture's toxicity. In sum, harmful effects from the mixture of chemicals in air are not likely (ATSDR 2004).

An air dispersion model conducted in support of the BIP detonations (CH2MHill 2007a) supports the measured monitoring results. This modeling study used a different air dispersion model (Open Burn/Open Detonation Dispersion Model; Bjorkland et al. 1998) and site-specific meteorological data unavailable when the Air PHA was conducted. The results of this air dispersion model also support the monitoring results and the conclusions of the ATSDR modeling study. Estimated airborne contaminant concentrations from BIP events at areas of potential off-site exposure "are below both regulatory standards and reasonable analytical detection limits for all compounds."(CH2MHill 2007a).

The air dispersion modeling of the BIP detonations (CH2MHill 2007a) used meteorological data collected from the Camp García airfield on Vieques. These site-specific windspeed and direction data for the year 2005 were not available when the ATSDR PHA was conducted. Instead, the ATSDR air dispersion model used then-available meteorological data from the Roosevelt Roads Naval Station and the San Juan Airport station.

Figure 6-1 shows wind rose diagrams for Camp García, the San Juan LM Airport, and the Roosevelt Roads stations (from CH2MHill 2007a). The Camp García site is in a shallow topographic bowl on the south central portion of Vieques. The 2005 wind rose reflects this location with the predominant wind

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directions blowing from the northeast, while the San Juan LM Airport and Roosevelt Roads predominant wind directions are similar, but more southeasterly and easterly. Average annual wind speeds for the Roosevelt Roads and San Juan LM Airport stations are about two to four times greater than for the Camp García station (2.88 and 4.10 vs. 1.46 m/sec, respectively). No long-term meteorological data are available for the LIA. The residential areas of Vieques are west of the LIA; the use of wind data that maximizes the easterly component of dispersion and uses a higher annual average wind speed provides a health-protective estimate of contaminant transport. That is, application of the Camp García wind data would tend to disperse more contaminants a shorter distance and more toward the ocean (to the south), with lesser amounts to residential areas (to the east).

In addition to the recent evaluation of BIP detonations, the Navy has proposed prescribed burning of LIA vegetation to access safely and to remove remaining unexploded ordnance. In support of this proposal, the Navy has conducted additional air modeling to assess the potential effects of such prescribed burning (CH2MHill 2008a). Although the Air PHA did not address vegetation burning, it did address past operations of open burning/open detonation (OBOD) of excess or recovered ordnance. As the mass of ordnance involved in past OBOD events was small relative to the mass involved in then-ongoing Naval operations, the 2003 Air PHA found that air emissions from past OBOD events did not present a public health hazard to Viequenses.

The 2003 Air PHA included an assessment of the public health significance of exposure to dust from African storms based on measured or estimated PM10 concentrations and found that PM10 exposures attributed to such dust storms were not a public health hazard. At the time of publication of the Air PHA, there were no data available on the chemical-specific makeup of dust from these storms. A recent study by Gioda et.al. (2007) presented PM10 and metals concentrations from air samples for several Puerto Rican locations, including Vieques, which makes such chemical-specific data available. This study was able to correlate seasonal, measured air particulate concentrations at Vieques and other Puerto Rican locations with African dust storms. Most of the metals analyzed in these samples (cadmium, cobalt, copper, iron, nickel, lead, and vanadium) were well below their applicable health comparison values and do not represent a public health hazard.

The annual average arsenic concentration of 0.0003 $\mu g/m^3$ is just above the comparison value of 0.0002 $\mu g/m^3$, which is based on health protective exposure assumptions, a lifetime excess cancer risk of 0.000001, and the U.S.EPA unit inhalation cancer risk of 4.3e-3 (per $\mu g/m^3$). Using the Gioda et.al. (2007) data, the estimated lifetime excess cancer risk for inhalation exposure to arsenic on Vieques is

$$0.0003\ \mu g/m^3 \times 4.3e\text{-}3 \text{ per } \mu g/m^3 = 0.0000013$$

The measured annual average arsenic air particulate concentration and resulting estimated lifetime excess cancer risk at Vieques is midway between other Puerto Rican values and not significantly different from the Fajardo, PR background location identified by Gioda et.al. (2007). Dust from African storms provides a measurable contribution to background particulate loadings at Vieques and other locations only during summer months and thus represents only a seasonal contribution to the predominantly local air particulate loads. Inhalation of specific metals from African dust storms, while contributing to seasonal particulate loads at Vieques and other Puerto Rican locations do not present an explicit public health hazard.

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Table 6-1. Average annual concentrations of PM10 and metals: Vieques and Fajardo, PR

Vieques	PM10	As	Cd	Co	Cu	Fe	Ni	Pb	V
	23.6	0.00029	0.00002	0.0002	0.001	0.00033	0.0007	0.0009	0.0025
Fajardo	25.5	0.00023	0.00002	0.00014	0.002	0.00027	0.0008	0.0007	0.0019
CV	50	0.00020	0.01	0.1	150	2200	0.09	0.15	0.2

All data from (Gioda et al. 2007)

All data in $\mu g/m^{3*}$

6.3 Findings

Every environmental dataset, whether based on direct sampled analyses or a modeling approach, includes simplifying assumptions and inherent uncertainties. Analytically measured data assumes the sampling device adequately captures the target substance, that the sampler is at the correct time and place relative to contaminant releases and exposures, and that the chemical/substance analyses produce accurate concentration estimates. Quality control and assurance procedures ensure that field sampling and laboratory measurement processes produce reliable results. That said, however, field-sampling data remain only an estimate of real-world conditions.

Quantitatively modeled datasets, such as the air dispersion model used in the Air PHA, resolve some direct sampling limitations—the modeled datasets allow estimation of results for any time or location. But air dispersion models necessarily include other assumptions, such as relevant contaminant emission rates, appropriate meteorological data, and specific aspects of the site terrain (e.g., topography). All of these site-specific conditions or model parameters influence air movement patterns. As with field sampling methods, air dispersion models also include quality assurance procedures to improve result quality. The Calpuff model used in the ATSDR Air PHA has undergone appropriate regulatory review. The U.S.EPA Support Center for Regulatory Atmospheric Modeling has approved the model for such uses as those employed by ATSDR. See: http://www.epa.gov/scram001.

Model assumptions and input parameters used in the ATSDR study are specifically documented in the 2003 Air PHA Appendix D and further described by Wilhelmi et al. (2006). By overestimating numerous aspects of the contaminant emissions and by assessing exposures for worst-case conditions, the uncertainties inherent in this air dispersion model have been adequately addressed. And despite the overestimation of potential exposures, the model results indicate that in Viequense residential areas, airborne contaminants from historic uses of the Vieques Naval Training Range would have been essentially nondetectable. The PREQB's historic and recent air sampling data and the Navy's post-PHA air sampling and air dispersion modeling support these predicted results. Collectively then, the available data are adequate for the conclusions in the Air PHA.

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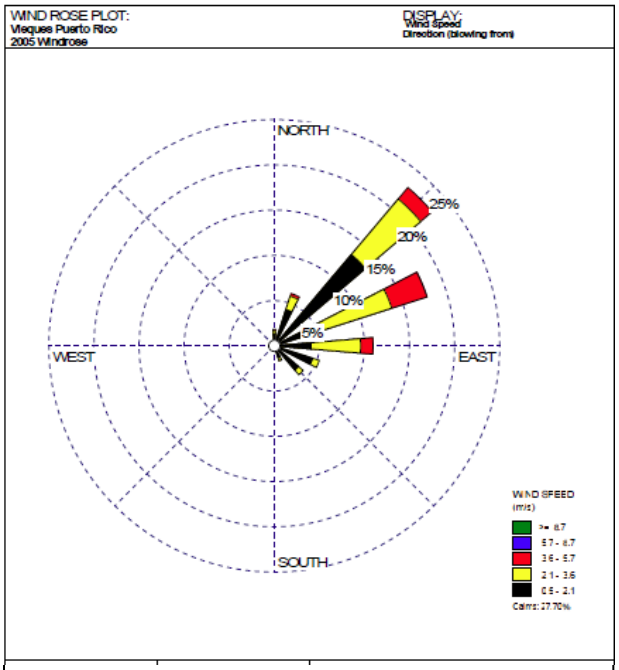


Figure 6-1a. 2005 Wind rose from Camp García, Isla Vieques.

Annual average wind speed is 1.46 meters per second. From CH2MHill 2007a.

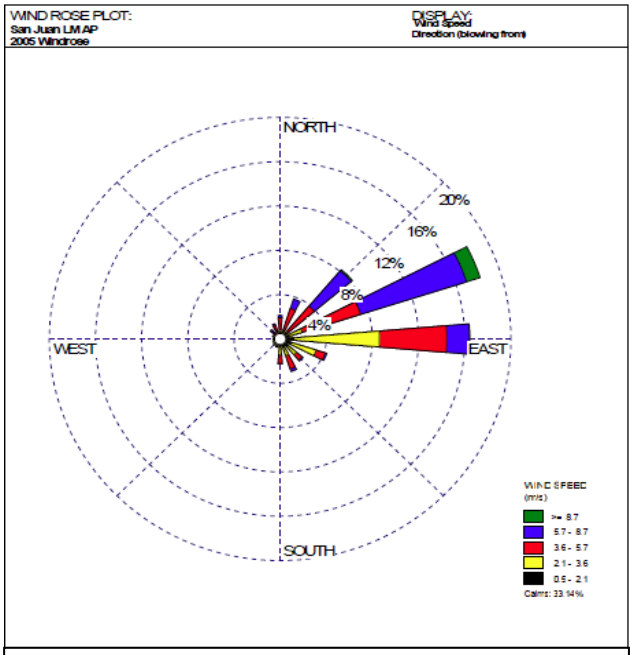


Figure 6-1b. 2005 Wind rose from San Juan LM Airport.

Annual average wind speed is 2.88 meters per second. From CH2MHill 2007a.

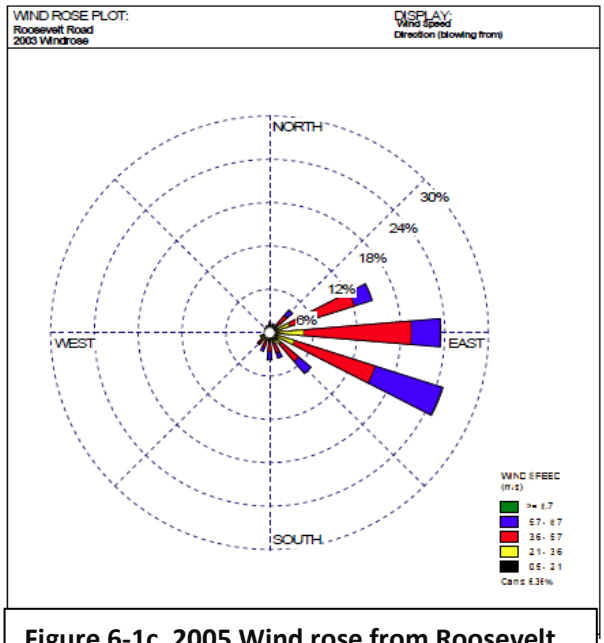


Figure 6-1c. 2005 Wind rose from Roosevelt Roads NAS.

Annual average wind speed is 4.1 meters per second. From CH2MHill 2007a.

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6.4 Conclusions and Recommendations

6.4.1 Conclusions

1. This review of potential airborne exposures within the residential areas of Vieques to contaminants from military operations at the former Vieques Naval Training Facility is based on the following sources of information that were used in the 2003 ATSDR PHA:
   a. U.S. Navy records of the types and amounts of ordnance used at the training range,
   b. A source term model used to estimate the amounts of material produced by ordnance detonations,
   c. An air dispersion model to estimate contaminant transport from the detonation area to the residential areas,
   d. Long term meteorological data from the San Juan PR airport and the Roosevelt Roads Naval Station, and
   e. Ambient air monitoring in the residential areas conducted by the PREQB for the 2001-2003 time period.
2. Also summarized are air monitoring data and air dispersion studies developed or collected after publication of the 2003 Air PHA for the purpose of evaluating ongoing or proposed open detonation or open burning.
3. This review of potential airborne exposures evaluates the modeling process used in the 2003 Air PHA, the assumptions and data used in the modeling process, and compares the results of that modeling process with monitored data collected during recent open burning events.
4. While this review finds the Air PHA modeling process consistent with established modeling practices, it does identify two minor errors in estimating detonation source concentrations. One results in an overestimate of metals concentrations. The other results in a minor underestimate of explosion byproducts in entrained soil that is overcompensated by the overall estimate of explosive compound concentrations. These errors do not affect the results or conclusions of the Air PHA.
5. Collectively, the available sampled and modeled data are adequate for the determination of the potential public health hazard from exposure to airborne contamination. The uncertainties inherent in the 2003 ATSDR air dispersion model have been adequately addressed by overestimating numerous aspects of the contaminant emissions and by assessing exposures for worst-case conditions. Despite the overestimation of potential exposures, the model results indicate that airborne contaminants from historic uses of the Vieques Naval Training Range would have been essentially nondetectable in the residential areas of Vieques and unlikely to have resulted in harmful effects. These predicted results are supported by the PREQB's historic and recent air sampling data and by the Navy's post-PHA air sampling and air dispersion modeling. Collectively, the available data provide an adequate basis for the public health conclusions in the Air PHA.

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6. To conduct its health protective evaluation of potential exposures to airborne contaminants from military exercises at the former Vieques Naval Training Facility, the ATSDR Public Health Assessment used appropriate modeling procedures and available monitoring data. The additional air dispersion modeling and monitoring data conducted after completion of the Air PHA further support ATSDR's public health conclusions. Because past military exercises would not likely have produced measurable concentrations of airborne contaminants in the residential areas of Vieques, no public health basis supports additional air studies or ambient air monitoring efforts related to past military exercises on Vieques.
7. Although the residents of Vieques could hear and possibly feel the explosions from military exercises on the former Vieques Naval Training Range, 8 miles is still a considerable distance. The physics of sound and seismic wave migration are very different from the physics of atmospheric dispersion of airborne particulates and gases.

6.4.2 Recommendations

No further recommendations at this time.

6.5 References

Army Research Laboratory, 2000. COMBIC, Combined Obscuration Model for Battlefield Induced Contaminants: Volume 1-Technical Documentation and Users Guide. Authors: Alan Wetmore; Scarlett D. Ayres; Army Research Laboratory, Adelphi MD. 2000 Aug. Available at: http://www.stormingmedia.us/44/4492/A449283.html.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003. Public Health Assessment, Air Pathway Evaluation, Isla De Vieques Bombing Range Vieques, Puerto Rico. Atlanta: US Department of Health Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry; 2003 Aug 26.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2004. Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures. Atlanta: US Department of Health Human Services, Public Health Service. May. Available at: http://www.atsdr.cdc.gov/interactionprofiles/ipga.html . Accessed April 1, 2010.

Bjorkland et al. 1998. Open Burn / Open Detonation Dispersion Model (OBODM) User's Guide, Volume 1. User's Instructions, DPG Document No. DPG-TR-96-008a. Prepared for the U.S. Army Dugway Proving Ground, West Desert Test Center. Meteorology & Obscurants Division. 2003 February. Model revision 2003 May 15.

CH2MHill. 2007a. Air Dispersion Modeling of the TCRA/BIP Activities on the Former Vieques Naval Training Range (Draft). Herndon, VA: US Navy Contract Task Order 0047, prepared by CH2MHill; 2007 Feb.

CH2MHill. 2007b. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: August 2005 through December 2006 (Draft). Herndon, VA: US Navy Contract Task Order 0047, prepared by CH2MHill; 2007 Feb.

CH2MHill. 2007c. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: January 1, 2007 through March 31, 2007 (Draft). Herndon, VA: US Navy Contract Task Order 0047, prepared by CH2MHill; 2007 Jun.

CH2MHill, 2008a. Air Dispersion Modeling for TCRA-Prescribed Vegetation Burns on the Former Vieques Naval Training Range (Draft Final). Herndon, VA: US Navy Contract Task Order 0047, prepared by CH2MHill; 2008 Jun.

CH2MHill. 2008b. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: April 1, 2007 through June 30, 2007 (Draft). Herndon, VA: US Navy Contract Task Order 0047, prepared by CH2MHill; 2008 Jan.

CH2MHill. 2009. BIP Air Monitoring Data Summary Report for the Former Vieques Naval Training Range: January 1, 2008 through June 30, 2008 (Draft). US Navy Contract Task Order 0047, prepared by CH2MHill, Herndon, VA; Feb 2009.

Cruz Pérez 2000. Contaminacion Producida por Explosivos y Residuos de Explosivos en Vieques, Puerto Rico. Ing Rafael Cruz Pérez. Dimension magazine, 2:11-18. June 2000.

[ERG] Eastern Research Group. 2001. Vieques Air Monitoring Program. Prepared for the Agency for Toxic Substances and Disease Registry, Division of Health Assessment and Consultation. Lexington, MA: Contract No. 200-2009-10039, Task Order No. 9; 2001 Jan 22.

Gioda et al. 2007. Particulate matter (PM_{10} and $PM_{2.5}$) from Different Areas of Puerto Rico. Fresenius Environmental Bulletin v. 16 No.8; 2007.

[IT] IT Corporation. 2000. Atlantic Division Naval Facilities Engineering Command, Air dispersion modeling and related analyses for Inner Range operations, Vieques, Puerto Rico. Knoxville, TN: February 2000.

[TAM] TAMS Consultants. 1979. Draft Environmental Impact Statement: Continued Use of the Atlantic Fleet Weapons Training Facility Inner Range (Vieques). December 1979.

Trinity Consultants, 2002. Calpuff Air Dispersion Modeling Report-Final, Vieques, Puerto Rico. Submitted to Eastern Research Group, Lexington MA. Parsippany, NJ: Project 023101.0024; Jun 2002.

Wilhelmi JL, Tsakaloyannis MK, Heath CH, Weber WM, Kellam JA, Campbell GH. 2006. Public health assessment of air emissions from military training exercises on the island of Vieques, Puerto Rico. San Diego, CA: Presented at the Air and Waste Management Association's 96th Annual Conference and Exhibition

Chapter 7 Soil Pathway

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Chapter 7 Summary

The following chapter evaluates the available data and information on Vieques soil contaminants and whether accidentally swallowing soil could make people sick. Indirect effects of soil contaminants through the terrestrial food chain are considered separately in Chapter 5, "Local Produce and Livestock Pathway." ATSDR evaluated potential direct soil exposures in two situations: people who stayed on the LIA property during 1999–2000 protests and people who live in the island's residential areas.

Sufficient data are available to conclude that people who lived on the LIA during protests were not exposed to soil contaminants at levels high enough to cause adverse health effects. In the island's residential areas, no soil data are adequate to characterize potential exposures fully. Nevertheless, consideration of soil data from other island locations and consideration of how contaminants might be transported through the air to residential areas suggests that in the island's residential areas, exposure to military activity-related soil contaminants is not high enough to result in adverse health effects. Since ATSDR's 2003 Soil PHA, more soil data have become available through investigations of known, localized areas of contamination on military lands. These were not areas accessed or occupied by the protesters, so they do not affect the conclusions drawn for those exposures. They are also not from the residential portion of the island; therefore, they do not improve the residential evaluation.

The recent data, along with the known presence of unexploded ordnance at the LIA, support the need for continuing to restrict access to the LIA and to other potentially contaminated military

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areas and the need to continue environmental assessment and remediation activities. To address remaining uncertainties about residential soil contamination issues, ATSDR recommends surface soil sampling in the island's residential areas.

7.1 Introduction to Soil Pathway

Several studies have examined soil contaminant levels in the Live Impact Area and elsewhere on Vieques. ATSDR evaluated the available data to determine potential effects for two situations: people who lived in areas of the LIA during 1999–2000, and people who lived (in the past and currently) on residential portions of the island. The chapter includes the following subjects:

- Ways in which people may be exposed to soil contaminants and ATSDR's focus in this chapter on direct exposures;
- Documentation of available soil data, including a discussion of the strengths and limitations of the data for assessing exposures in the LIA or the residential portion of the island and a fresh analysis of the exposures of interest focusing on uncertainties in the available data; and
- Discussion of how the above analysis might be affected by recently collected soil data in areas of concern or areas not affected by aerial bombing exercises, and a discussion of other considerations raised by interested parties related to soil exposures.

7.1.1 Soil Exposure Pathways

Environmental contaminants directly and indirectly affect the health of the planet and the people on it. ATSDR focuses on identifying exposures that might directly affect human health. For soil, we look at whether the small amount people might accidentally swallow or touch could contain enough of a particular chemical to cause health effects. Contaminated soil might also affect plants growing in it, which people might eat. And people might eat the animals that eat the plants growing in that contaminated soil. ATSDR considers such *exposure pathways* by looking at contaminant levels in the plants or animals that people actually use or eat. Characteristics of the contaminants, the soil, and the plants all affect the potential for plant uptake of contamination and may be important for food, medicinal, or ceremonial use of the plants or the animals feeding on them. For further information on exposure from plants or animals possibly affected by soil contaminants, please see Chapter 5 of this report, "Local Produce and Livestock Pathway."

ATSDR identified several ways soil contaminants might directly affect Viequenses. We considered past (i.e., both during military exercises and after), present (i.e., the past few years, after cessation of military exercises), and future potential soil exposure pathways. Table 7.1 below lists the identified direct-soil exposure pathways.

For evaluating soil exposures from accidentally swallowing or touching soil, surface soil data are preferred. Generally, in day-to-day activities people are assumed to contact only the top few inches of soil. Household dust is also considered a soil pathway constituent, given that soil may contribute contaminants to dust when it is brought indoors on shoes or hands. Windblown soil may contribute contaminants to surface soil, but its health effects are generally considered part of the air pathway. This section lists considerations on the air pathway's contribution to soil exposures, presumed to be the

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mechanism by which residential soil would have been initially contaminated by aerial bombing exercises several miles away in the LIA.

Table 7-1. Direct soil exposure pathways to people on Vieques

Time Frame: Past	***Point of Exposure***	***Exposed People***	***Status***
Accidentally swallowing or touching airborne dust blowing from past military activities	Residential Area of Island	Residents of Vieques	No historical data; no way to confirm model predictions
Accidentally swallowing or touching surface soil or household dust contaminated by past military activities	Residential Area of Island	Residents of Vieques	No historical data; no way to confirm assumptions, except perhaps for inorganics
Accidentally swallowing or touching surface soil while residing on Live Impact Area (during 1999 and 2000)	Live Impact Area of Navy Property	Protesters Occupying Beach Camps	Surface soil results available for the period and location of interest. Evaluated in Previous Assessment
Accidentally swallowing or touching surface soil or subsurface soil while conducting construction or excavation activities	Current and former Navy Properties	Navy Personnel or Contractors	Not evaluated at this time; focus on residents
Accidentally swallowing windblown dust from Live Impact Area	Residential Area of Island	Residents of Vieques	Current Live Impact Area soil data show contaminants too low to harm health; therefore same conclusion for windblown dust
Accidentally swallowing or touching previously contaminated surface soil or household dust	Residential Area of Island	Residents of Vieques	No data available; could collect to confirm conclusions based on other locations
Accidentally swallowing or touching surface soil in restricted access navy activity areas	Current and former Navy Properties	Navy Personnel or Contractors; Trespassers	Current data available at specific locations; few contaminants above safe levels used for screening; difficult to generalize
Accidentally swallowing or touching surface soil or subsurface soil while conducting remedial activities	Navy Property	Navy Personnel or Contractors	Not evaluated at this time; focus on residents
More frequent exposure of civilians to surface soil in navy activity areas or former navy land	Current and former Navy Properties	Residents of Vieques	Specific areas need sampling / assessment before opening to public

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7.1.2 Assessment of Available Data

Several investigations have analyzed Vieques soil samples:

- In 1972, the U.S. Geological Survey collected and described metals concentrations of samples of the soil's "C" horizon—in general, the parent rock beneath the surface soil and subsoil layers (Learned et al. 1973). This sampling was a reconnaissance geochemical survey for metals only and included 420 samples across the entire island. In a later report the data were re-reported (Marsh 1992).
- In 1978, soil samples collected from four locations within the Live Impact Area (LIA) and two locations in the Eastern Maneuver Area (EMA) were analyzed for explosives and explosion combustion products (Hoffsommer and Glover 1978; Lai 1978). The exact sampling locations and depths were not given in the reports describing this sampling.
- In 1998, to describe existing conditions before a proposed airport expansion project, five soil samples (4–12 inches below ground surface) from an area of the Naval Ammunition Support Detachment (NASD) buffer zone were collected and analyzed for a range of potential contaminants (PMC 1998).
- In 1999, about 33 surface soil samples (0–6 inches below ground surface) were collected along the western border of the EMA and analyzed for explosive compounds (CH2MHill and Baker 1999).
- In 1999 and 2000, soil and sediment samples from 55 locations were collected from the LIA and other locations and analyzed for metals and other inorganic compounds (García et al. 2000). The report describing this sampling event states that samples were collected from direct bomb-impact locations, stormwater runoff areas, adjoining conservation zones, and civilian areas, but exact locations of sampling and exact sampling depth and procedures were not given. Only the highest and second highest contaminant concentrations detected for 25 sample locations within the LIA were reported.
- In 2000, 37 surface soil samples (0–6 inches below ground surface) were collected within the LIA and adjoining conservation zones, including specific areas in the LIA occupied by protesters in 1999 and 2000 (CH2MHill 2000). These samples were analyzed for metals and explosives. Samples from the LIA were targeted to bomb craters or collection areas for stormwater runoff deemed likely to contain the highest level of explosive contaminants. Samples from protester camps were collected from locations thought to be representative of the general area.
- In 2001, to describe background conditions at the NASD, 24 surface soil samples (0–6 inches below ground surface) were collected and analyzed for inorganic compounds (CH2MHill 2002).
- Since 2003, several investigations of sites in the EMA, the Surface Impact Area (SIA), and the NASD have included surface soil sampling of between 4 and 50 samples per site (CH2MHill 2007a-c; 2008a-e). These investigations focused on known, localized areas of concern affected by Navy activities other than aerial explosive-ordnance exercises.

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- In 2007, 40 surface soil samples (0–6 inches below ground surface) were collected from areas of East Vieques not obviously affected by Navy activities. The samples were analyzed for inorganic compounds to describe background conditions on eastern Vieques (CH2MHill 2007d).

7.1.3 Strengths and Limitations of Available Data for Assessing Soil Pathway

Some of the above-listed data are not appropriate for assessment of residents' exposure to contaminants in Vieques soil. Thus to assess potential soil exposures, ATSDR focused on two pathways: exposures to protesters who occupied parts of the LIA in 1999 and 2000, and exposures to bomb-related contaminants of people in the island's residential area. The following section describes the strengths and limitations of these applicable, available data.

7.1.3.1 Protesters Living in the Live Impact Area during 1999–2000

Although limited in number, the CH2MHill 2000 data are otherwise ideal for assessing exposures to protesters during 1999–2000: they match the locations and time people may have been exposed. Less specific information is known about the locations, analysis, and results reported in García et al. (2000), but the samples were taken at the same general area and at the appropriate time. In both sets of data, samples from the LIA were selected from areas thought to be the most contaminated (i.e., bomb craters and stormwater runoff collection areas). The samples then were intended to describe conservatively the contaminant levels within the LIA. But the small number of samples compared with the large land area of the LIA leaves a great deal of uncertainty as to whether actual "worst case" samples were collected. This is a common limitation of environmental sampling. A further limitation of both datasets is that they only apply to periods near the protester occupation. Finally, evaluating these datasets for health hazards of chemical constituents gives little information about other serious hazards of LIA occupation, such as physical hazards posed by unexploded ordnance.

7.1.3.2 Exposures in the Residential Area of the Island

Because of current Navy property access restrictions, most exposures today would occur only in the island's residential areas. But the only data from the residential areas are the 1972 geological survey data, which just describe the soil's "C" horizon[28] and just analyze for metals content. These data were distributed across the residential and other areas of the island. They may not provide accurate or complete information about the surface soil people accidentally swallow or touch in their day-to-day activities. Although other data contain more complete contaminant information for soils at the surface, they were collected from uninhabited parts of the island. One limitation of these data in describing exposures is that they may not accurately represent residential exposures. Nevertheless, despite the limited nature of the available data on residential soil contaminant levels, ATSDR accessed a combination of information related to soil contaminant levels elsewhere on the island. ATSDR used this information 1) to model predictions of how contaminants could be transported through air to residential areas, and 2) to reach scientifically based conclusions about the likelihood of military exercises contaminating residential soil at levels that could cause health effects. More information on the

[28] In general, the parent rock beneath the surface soil and subsoil layers.

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uncertainties posed by using limited data to evaluate soil exposures is provided below in 7.2, the Analysis section.

7.2 Analysis

Among other Vieques health concerns ATSDR received were those related to contaminant exposure from military exercises involving explosive-ordnance in the LIA. Because of current Navy property access restrictions, most exposures would take place today only in the island's residential areas. During 1999 and 2000, however, some protesters did occupy areas of the LIA. The following sections discuss the data available for assessing direct soil exposures of protesters living in the LIA during that period, and of Viequenses generally at other times in the past or present. In addition, surface soil sampling data obtained since ATSDR's previous evaluation in 2003 are discussed as they relate to assessing potential soil pathway exposures.

7.2.1 Exposures of Protesters in the Live Impact Area During 1999–2000

In 2000, surface soil samples were collected from the specific LIA locations where protesters lived in 1999 and 2000, as well as other LIA locations (CH2MHill 2000). Samples included low-lying areas that stormwater runoff would likely affect. Around the same time, an independent organization also collected soil samples at the LIA and in areas where people lived during 1999–2000 (García et al. 2000). For this sampling, the exact location, depth of sampling, and full results were not reported. Because the García et al. report stated that the intent of the study was to characterize exposure, in its previous evaluation ATSDR considered the results as surface soil samples. This resulted in conservative exposure estimates because García et al. only reported the highest and next-to-highest values detected. ATSDR's inclusion of these reported values in estimating average exposures biased the estimate to the high side.

Although limited in number, the surface soil samples collected during 2000 were nearly ideal for assessing exposures to people occupying the LIA. Both in space and time the samples were representative of the soil people may have contacted while they were there. The CH2MHill 2000 data report included documentation of high standards of data quality such as quality assurance/quality control and chain of custody forms.

In its previous assessment, ATSDR estimated doses using the highest concentration measured in the locations where protesters lived; the doses were all below then-current health effects levels (ATSDR 2003). Some critics of this analysis suggested that people in the camps might have been exposed to contaminants in other parts of the LIA. In response we examined whether our conclusion would change if we used the average inorganic contaminant concentrations measured throughout the LIA as an exposure concentration (a person spending time in both the camp and in other parts of the LIA would have an average exposure over all the surface soils encountered). Although the average exposure concentrations were biased high because the averages included only the maximum and second highest soil measurement detected from the García et al. study (2000), the concentrations were only slightly higher than the maximum camp value used in ATSDR's previous assessment. Following the reasoning of the previous public health assessment, exposure to these concentrations in surface soil would not be expected to result in adverse health effects (ATSDR 2003). ATSDR also looked at surface soil explosives data from other locations of the LIA collected in 2000 (CH2MHill 2000). Although those samples showed

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rare detections of HMX, RDX, 2-amino-DNT, and TNT, even the highest detected values were lower than screening values.[29] The average soil concentration for every chemical measured is below the respective screening value, indicating that potential mixture effects are unlikely.

Using the available data, we found no indication that protesters living on the LIA in 1999–2000 were exposed to harmful levels of surface soil contaminants. A strength of this conclusion is that the camp data were collected specifically to assess this exposure and were collected to represent the camps where protesters lived. In addition, the LIA samples were collected to assess the most contaminated areas on the LIA, so the conclusions would be based on contaminant levels that were biased high and would be more protective. A limitation is that, as with any evaluation of this type, conclusions must be based on a small number of samples. If further sampling were to show the earlier sampling did not result in a conservative or representative description of the area of exposure, the previous conclusions might not be valid.

Another limitation of using these data to assess exposure is that they apply only to the specific scenario described. That is, is the data are restricted to protesters living in camps on the LIA in 1999–2000. In the years long before 1999-2000, contaminant levels (especially explosives) could have been much higher and could have had the potential for short- or long-term health effects (we have no way of obtaining these data today). Also, despite the fact that around the year 2000 the surface soil contaminant levels in the LIA were not high enough to expect adverse health effects, that finding alone does not mean people could safely occupy the LIA. Physical hazards remained, such as unexploded ordnance that could cause immediate injury or death to those who inadvertently disturbed it. Because these hazards remain, limiting access to the LIA and to other contaminated military areas is essential for protecting public health—at least until those areas undergo a complete environmental assessment and cleanup.

7.2.2 Exposures in the Island's Residential Areas

Lack of surface soil data is a major limitation for assessing soil exposure in the island's residential areas. The only soil data available from this section of the island were the 1972 U.S. Geological Survey data (Learned et al. 1973). Although the sample points were well distributed across residential and other areas of the island, the data described only sampling of the soil's "C" horizon (in general, the parent rock beneath the surface soil and subsoil layers). The report did state that at many points the surface soil and subsoil layers were absent (i.e., the parent rock was exposed at the surface). But the specific samples to which this applied were not stated, and the sampling depth and conditions were not available on a sample-by-sample basis. Therefore, whether the "C" horizon data truly represent the surface soil people accidentally swallow or touch in their day-to-day activities is unknown. Moreover, whether the parent rock is exposed or not, analyzing the "C" horizon provides little if any information about surface deposition of airborne contaminants (i.e., the mechanism by which contaminants from explosive-ordnance exercises are believed to reach soil).

Although the residential soil data were limited, ATSDR could use the limited data and other information to reach scientifically based conclusions about the potential for exposures to bombing-related

[29] Levels at which no adverse health effects would be expected from even continual exposure.

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contaminants in residential soil to result in adverse health effects. In its previous health assessment, because the residential data were limited, ATSDR used the USGS data and several other data sources from different areas of the island to evaluate the potential for residential exposure to soil to harm health (CH2MHill and Baker 1999; CH2MHill 2000; García et al. 2000; Hoffsommer and Glover 1978; Lai 1978; Marsh 1992; PMC 1998). The evaluation included data from

- The LIA,
- The Eastern Maneuver Area (EMA),
- The EMA border,
- The residential area, and
- The Naval Ammunition Support Detachment (NASD).

ATSDR first compared a particular contaminant's highest level found in any sample with screening levels below which no health effects would be expected. Contaminants above the screening level were evaluated further by estimating exposure doses based on an average soil concentration. None of the contaminants in soil were at levels expected to result in harmful effects (ATSDR 2003).

The soil pathway public health evaluation conforms to ATSDR procedures for reaching public health decisions when only limited environmental data are available. In addition, the air modeling results, discussed in the previous chapter, support the hypothesis that exploded or unexploded ordnance did not contaminate residential soils to harmful levels. Models indicated that airborne contaminants would disperse to undetectable levels away from the Live Impact Area.

Although the analysis of all the information indicated that health effects from exposure to contaminants in residential soil are unlikely, a point that needs reiteration is that the previous assessment's assumptions contain uncertainty. When using discrete environmental samples to describe a large area, uncertainty is always present. This is particularly true when data collected at one location form the basis for conclusions about another, unsampled location. Additional data uncertainties include

- Averaging soil results that might include subsurface samples is not necessarily representative of the concentrations present in the surface soil that people in their normal activities accidentally swallow or touch.
- Each investigation might have a different focus and might analyze for different sets of contaminants. Also, while inorganic contaminant levels are expected to remain relatively stable over time, other contaminant levels (e.g., those of volatile compounds, certain explosives) in surface soil might, through volatilization or chemical or biological degradation, be significantly reduced over time. Therefore, even if surface soil sampling were available, the results might not represent completely all potential past exposures.
- The majority of the more recent surface soil data were collected in the LIA. Because the LIA was the focus of a citizen's petition then under consideration, ATSDR's previous assessment concentrated on describing potential residential soil effects using LIA data. Because contaminants from the LIA would be dispersed by the time they reached the residential area,

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using LIA data was assumed to represent "worst case" exposures possible from LIA activities. But Vieques was the site of past military activities other than those involving explosive ordnance (e.g., transportation, storage, general training). Conceivably these activities—or even nonmilitary activities—could result in different contaminant profiles in the residential portion of the island compared with the LIA.

Such data limitations aside, ATSDR still maintains that any low-level, current residential area soil exposures to explosive ordnance-related contaminants in the LIA are unlikely to cause health effects. First, recent surface soil sampling in the LIA did not show contaminant levels of health concern. Second, air modeling indicates that any windborne contaminants from the LIA would be dispersed to undetectable levels away from the LIA. Nevertheless, given the uncertainties described above, ATSDR has updated its previous conclusion (ATSDR 2003) to acknowledge these limitations and indicate that the historical data are insufficient for us to determine whether past exposures to explosives or other compounds in surface soil could have been high enough for an elevated past risk of adverse health effects.

7.2.3 Recent Surface Soil Sampling Data on Areas of Concern

Since ATSDR's first assessment, several site investigations in the EMA, the Surface Impact Area (SIA), and the NASD have included surface soil sampling (CH2MHill 2007a-c; 2008a-e). The investigations focused on known, localized areas of concern affected by Navy activities other than explosive ordnance. This means the surface soil samples were probably not representative of residential area surface soils. ATSDR performed a preliminary evaluation of the surface soil results from these investigations, focusing on maximum detections of inorganic compounds above background and detections of organic compounds. Although several sites had contaminants higher than screening values,[30] current access restrictions due to ongoing remedial activity mean exposures to the public are unlikely. In any event, we saw no evidence that contamination associated with areas of concern was present outside the localized area. Examining the contaminants present may nonetheless provide clues to contaminants previously unrecognized but of possible concern in the residential area. For example, certain recent investigations have shown isolated detections of pesticides, semivolatile compounds, explosives, and dioxin compounds. We repeat that we have found no evidence these areas of contamination have spread beyond their isolated, local areas. Agencies responsible for characterizing the nature and extent of contamination in the future should ensure that all potential sources and contaminants are considered.

7.2.4 Recent Background Study Data

Recent soil background studies at the NASD and on East Vieques (the EMA) characterized the soils in areas unaffected by Navy operations—that is, not close to known, localized areas of concern and not in the runoff path from known military operations areas (CH2MHill 2001; CH2MHill 2007d). As described in the previous chapter, evaluation of the air pathway indicates that airborne contaminants would hardly be detectable after dispersing away from the bomb impact area. Examining results from background locations (not directly affected by military exercises) could be a way to confirm that soils are not

[30] Levels at which no adverse health effects would be expected from even continual exposure.

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currently affected by past airborne fallout of contaminants from military operations. The NASD background study, however, only included inorganics, and the East Vieques background study only included inorganics and explosives. While inorganic contaminant levels are usually relatively stable over time, levels of other contaminants (e.g., volatile compounds, certain explosives) in surface soil might, through volatilization or chemical or biological degradation, substantially reduce over time. For this reason, the background soil data were not useful for conclusions about past exposures to explosives or present and past exposures for nonanalyzed potential contaminants.

In background samples, very few contaminants were present above screening values.[31] Only total chromium slightly exceeded a screening value for hexavalent chromium. Chromium is a naturally occurring element as well as a potential contaminant. In soils it is found in several forms, including the most toxic hexavalent form (ATSDR 2000). Although the relative proportions of all the different forms of chromium were not analyzed, much of the total chromium measured probably consisted of less toxic chromium forms. In the EMA, the explosive 2-amino-DNT was detected in one sample, but it was well below the applicable screening value. The residential area of the island was much farther from the past military activities than were the background samples collected in these investigations. Dispersal would result in contaminant concentrations even lower in the residential areas than in the background sampling areas. These background data, then, further support the hypothesis that contaminants in surface soil in Vieques residential areas are not at levels known to cause health effects today. Yet detection of explosive residues in the background samples also suggested that all areas of the island, including the residential area, might have been affected by explosive compounds from past bombing activities. Although residual levels are low today, it is impossible to say what past levels were.

7.2.5 Additional Considerations

Some Puerto Rican scientists suggested to ATSDR that evaluating surface soil samples collected from 0–6 inches below land surface would underestimate potential exposure to contaminants present at high concentrations only at the very surface. To explore this concern, we considered alternate "worst case" concentration assumptions, such as assuming the contaminant was present only in the top inch rather than the entire 6 inches.

For the available data, however, we determined this was an unrealistic assumption. Most surface soil data came from the LIA, where years of widespread bomb and projectile impacts and explosions would have stirred up huge amounts of soil; any resulting contaminants were likely distributed throughout more than just the top 6 inches. Likewise, many inorganic compounds are naturally present throughout soil and would never be completely absent, regardless of depth.

Given these considerations, we decided an analysis of the available data assuming that all contaminants were present in the top inch of soil would be misleading. In future surface soil sampling events, ATSDR recommends that agencies or researchers explore this issue further by analyzing the top inch of soil separately from the typical 0–6 inches or 0–3 inches collected.

[31] Levels at which no adverse health effects would be expected from even continual exposure.

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The conclusions and recommendations from ATSDR's evaluation of the soil pathway are below and in Chapter 9 of this report.

7.3 Conclusions and Recommendations

7.3.1 Conclusions

1. Those who occupied the LIA from 1999–2000 were not at increased risk of adverse health effects from exposure to surface soil contaminants. Supporting data are limited, but they are of good quality and represent the location and the period of interest. In the years before the late 1990s tests, contaminant levels in the LIA (especially explosives) might have been higher, but we have no historical data with which to evaluate this assumption.
2. This does not mean anyone can now safely visit the LIA. Remaining unexploded ordnance could cause immediate injury or death to anyone who might inadvertently disturb it. Recently collected data on specific areas of concern within military lands—not the same areas accessed and occupied by protesters—demonstrate the remaining potential for localized contamination, which, if people frequented those areas, could be of health concern.
3. Limited available data from other locations and air pathway considerations suggest that the military exercises in the LIA did not result in current contamination of residential soils with inorganic or explosive compounds at levels considered harmful. ATSDR arrives at this conclusion using a scientific evaluation of the available data. But again, data from other areas are limited in number, data for all potential contaminants of concern are not available, and no adequate surface soil data are available from the residential area itself. ATSDR nonetheless understands that community members remain concerned about residential soil exposures.
4. Modeling described in the air pathway discussion has suggested that airborne transport of contaminants during past military exercises would not have been substantial enough to have affected soils in the island's residential area. Sufficient historical data are not available to confirm this, nor will such data ever become available. Consequently, we cannot determine whether past exposures to explosives or other compounds in surface soil could have been heavy enough to increase a past risk of adverse health effects.

7.3.2 Recommendations

1. Continue to restrict access to the LIA and other potentially contaminated military areas and continue environmental assessment and remediation activities to clear the way for public access.
2. To help address community concerns regarding residential soil, work with local residents to design sampling to identify residential surface soil quality issues, whether or not those surface soil issues relate to past military activities:
    a. Focus initial sampling on areas identified as potential high-exposure areas, such as areas free of vegetation or child-use areas.

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b. To identify any possible differences in the top layer, initial sampling should analyze the top inch of soil separately from the rest of the 0–3 or 0–6 inch surface samples.

c. Perform a full range of analyses to identify all possible contaminants.

d. Plan and conduct sample collection and analysis to ensure the results meet high data quality standards.

3. Although this sampling might provide inferences about past exposures for compounds stable over time, such as metals or other inorganic compounds, it will provide no definitive information on past surface composition and no information on past levels of compounds that react or degrade over time.

4. ATSDR also recommends collection of representative soil samples as part of its evaluation of the terrestrial food chain pathway. See Chapter 5 of this report for details.

7.4 References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003. Public health assessment, soil pathway evaluation, Isla de Vieques bombing range. Atlanta: US Department of Health and Human Services; February.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2000. Toxicological profile for chromium. Atlanta: US Department of Health and Human Services; September.

CH2MHIll and Baker Environmental, Inc. 1999. Results of the hydrogeologic investigation: Vieques Island, Puerto Rico; November.

CH2MHIll 2000. Live impact area soil sampling report: US Naval facility, Vieques Island, Puerto Rico. Tampa FL: CH2MHIll; October.

CH2MHILL, 2001, Soil, groundwater, surface water, and sediment background investigation report (draft), Former U.S. Naval Ammunition Support Detachment, Vieques Island, Puerto Rico. Tampa, Florida. June 2001.

CH2MHIll 2002. Final soil, groundwater, surface water, and sediment background investigation report, US Naval Ammunition Support Detachment, Vieques Island, Puerto Rico. Tampa, FL: CH2MHill; October.

CH2MHIll. 2007a. Remedial investigation report for solid waste management unit (SWMU) 6, CH2MHill; February.

CH2MHIll 2007b. Remedial investigation report for area of concern (AOC) J, CH2MHill; May.

CH2MHIll. 2007c. Remedial investigation report for area of concern (AOC) H, CH2MHill; July.

CH2MHIll. 2007d. East Vieques background soil inorganics investigation report, former Vieques Naval Training Range. Tampa, FL: October.

CH2MHIll. 2008a. Remedial investigation report for solid waste management unit (SWMU) 7. Tampa, FL: March.

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CH2MHIll. 2008b. Remedial investigation work plan addendum for solid waste management unit. Tampa, FL: (SWMU) 4; April.

CH2MHIll. 2008c. Final PA/SI report, 12 consent order sites and 8 PI/PAOC sites. Tampa, FL.

CH2MHIll. 2008d. Remedial investigation report for area of concern (AOC) I. Tampa, FL: June.

CH2MHill. 2008e. Remedial investigation report for area of concern (AOC) E. Tampa, FL: July.

García N, Lopez AM, Soto M, Rosado S, Berrios B. 2000. Environmental impact of Navy activities in Vieques. San Juan, PR: Servicios Cientificos y Tecnicos, Inc.; July.

Hoffsommer JC and Glover DJ. 1978. Vieques litigation support: explosives analysis of water and soil samples taken on Vieques Island, Puerto Rico, May 11 through May 16, 1978. Silver Spring, MD: Naval Surface Weapons Center, Explosives Chemistry Branch.

Lai MG. 1978. Explosion products content of water and soil samples taken on Vieques Island, Puerto Rico, May 11 through May 16, 1978. Silver Spring, MD: Naval Surface Weapons Center; July.

Learned RE, Grove GR, Boissen R. 1973. A geochemical reconnaissance of the Island of Vieques, Puerto Rico. US Geological Survey and Puerto Rico Department of Natural Resources. Open-file report 73-155 (order number 1866).

Marsh SP. 1992. Analytical results for stream sediment and soil samples from the Commonwealth of Puerto Rico, Isla de Culebra, and Isla de Vieques. Open-file report 92-353A. Denver, CO: US Geological Survey.

[PMC] Program Management Company. 1998. Environmental baseline survey: 400-acre site, Vieques municipal airport expansion, Vieques Island, Puerto Rico, 00765. Annapolis, MD: PMC; November.

Chapter 8 Drinking Water Pathway

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Chapter 8 Summary

The following chapter evaluates the available data and information on contaminants in past and present drinking water sources on Vieques and whether consuming drinking water could make people sick. ATSDR considered three past or present drinking water sources: the current pipeline supply, public and private supply wells used in the past and still occasionally used when pipeline water is interrupted, and rainwater collection systems that may have been used in the past and may still be used.

ATSDR determined that the limited available sampling data on the pipeline water supply system and the public and private wells indicate almost all these drinking water supplies are acceptable for their current uses. One private well was found to contain nitrate-nitrite levels that could be harmful; ATSDR recommended no one drink from this well. Ongoing monitoring of the current pipeline source water is required to ensure the supply meets drinking water standards, and repeating previous sampling of storage tanks, residential taps, and wells still in use would address remaining uncertainty associated with conclusions about the pipeline water and public and private wells. A lack of adequate historical data prevents ATSDR from making any conclusion about distant past (1970s and 1980s) public supply well exposures. Also, no data are available to evaluate past or present rainwater collection systems exposures. Since ATSDR's 2001 drinking water public health assessment, more groundwater data have been collected. These data are not from current drinking water sources, so they do not provide additional exposure information. One study, however, provided results supporting ATSDR's previous conclusion that contaminants in groundwater at the LIA could not reach the public water supply wells. The other studies focused on locally contaminated areas of concern not used for drinking water; they support the continuing need for characterization and eventual cleanup of source areas resulting from past military activities on Vieques.

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In addition to required testing of the pipeline source water, ATSDR recommends repeated sampling of the current public water supply (storage tanks and residential taps) and public and private wells still available for use to confirm the safety of the drinking water for consumption. Also, ATSDR recommends further evaluation of the use of rainwater collection systems and sampling if such systems are used for drinking water.

8.1 Introduction

Many Viequenses remain concerned that military activities on and near the island might have contaminated Vieques drinking water, either currently or in the past. Vieques has or has had only three past or present drinking water sources:

1. Since 1978, the Puerto Rico Aqueduct and Sewer Authority (PRASA) has provided drinking water through a pipeline from the main island. In some years, these operations were conducted by private companies contracted by PRASA. After transport from Puerto Rico, water is stored in tanks on Vieques. Most Viequenses use this water source.
2. Before 1978, public supply wells in the Esperanza well field (presumably operated by PRASA) and several private groundwater wells were sources of drinking water. Some of these wells are still used whenever water pipeline service is interrupted.
3. Reportedly, individual households use rainwater collection systems as sporadic, supplemental drinking water sources. How prevalent the systems are, or were in the past, is unclear.

ATSDR evaluated available data to describe the safety of these potential drinking water sources on Vieques. This chapter describes ATSDR's data analysis.

8.2 Assessment of Available Data

8.2.1 Pipeline Water (Current Supply)

In summer of 1999, the U.S.EPA and PRDOH collected and analyzed water samples from storage tanks for water piped from the main island and from residential water taps on Vieques (PRDOH 1999; USEPA 1999). To address quality control issues, in January 2000, U.S.EPA resampled some of the locations for explosives (USEPA 2000). In its previous assessment, ATSDR examined these data and found no contaminants above health or regulatory standards or obviously related to bombing activities, indicating that pipeline water was acceptable for consumption (ATSDR 2001). ATSDR recently obtained water quality reports of the source water dated from 2004–2009. These reports show that in 2004–2005, the action level for lead (15 μg/L) was occasionally exceeded. This was attributed to corrosion in the piping system. In 2006–2007, the number of exceedances went up sharply, with 28 in 2007 alone. For that year, the 90th percentile of lead concentrations measured (the level below which 90% of the lead concentrations measured fall) was 144 μg/L, greatly exceeding the action level. This level is a significant concern, especially to pregnant women and young children. The cause of the elevations in lead are unknown to ATSDR. But the water quality reports stated that immediate investigations were undertaken to identify the source of the lead and to address the problem. In 2008 and 2009, the number of

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exceedances was low (2 in 2008 and 4 in 2009); the 90th percentile of lead concentrations measured was well below the action level in both years.

To summarize, testing of the supplied water did not indicate any contaminants were related to military exercises on Vieques. Still, continued safety of the pipeline water supply relies on continued monitoring of the water supply according to federal regulations and, if necessary, on prompt action to address violations.

8.2.2 Public and Private Well Water

A 1978 sampling event reported low levels of explosives or explosives byproducts in drinking water samples taken from two public supply wells in Esperanza and Isabel Segunda (Hoffsommer and Glover 1978; Lai 1978). In its previous assessment, ATSDR questioned these reported explosive detections because of the small number of samples collected and because the sample descriptions left uncertainty as to the samples' identity and locations (ATSDR 2001). Even assuming the detections were present at that level in drinking water, the levels of explosives were too low to result in adverse health effects (ATSDR 2001). In 1995, PRDOH tested the public and private wells, as did U.S.EPA and Navy contractors in 1999 (PRDOH 1995a and 1995b; USEPA 1999; Baker 1999). ATSDR evaluated these results and found that most well water was safe to drink. One private well located on the residential portion of the island west of the EMA and LIA was found to contain high levels of nitrate-nitrite, which could pose a health hazard and were likely a result of agricultural activity or septic systems in the area. Residents were advised that this well's water was not safe for consumption. In addition to sampling data, ATSDR evaluated the island's geology and topography and concluded that activities at the Live Impact Area (LIA) were unlikely to affect groundwater in private and in former public supply wells—groundwater did not move in the direction of the wells (ATSDR 2001). Although wells used previously as public water supplies would have been subject to drinking water regulations, ATSDR did not review historical reports on the water quality from the 1970s and 1980s or before. On the basis of our experience reviewing historical records, even if the records had been maintained and could be found, too many questions would arise as to quality control measures used, adequacy of the analytical methods used, and analytes measured to make the historical records useful for assessing past exposures.

8.2.3 Rainwater Collection Systems

No information was available on rainwater collection systems. This included their prevalence, whether they had ever been used for drinking water, or whether they had ever been tested. In its previous Drinking Water PHA, ATSDR discussed but did not evaluate this potential exposure pathway. ATSDR had planned to learn about that pathway through its evaluation of the air pathway's potential to transport contaminants to the residential portion of the island and potentially contaminate rainwater collection systems. As discussed in a previous chapter, this air analysis suggested the air pathway was unlikely to transport contaminants to the residential portion of the island at levels that might result in adverse health effects (ATSDR 2003). ATSDR did recommend that Puerto Rican authorities identify representative systems and test them to ensure those systems deliver safe water. To our knowledge, such sampling has not occurred (ATSDR 2001). ATSDR also recommended sampling sediment from rainwater collection systems as an indication of potential past water quality.

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8.2.4 Strengths and Limitations of Available Drinking Water Pathway Data

8.2.4.1 Pipeline Water

The available data were collected and analyzed according to standard methods and procedures. But only one round of tank sampling occurred. Only one residential tap sample representative of the water supplied to residents was collected. It is possible that the available data may not fully represent the quality of this water source over time. In fact, recent water quality reports indicate that lead levels in the supplied water have exceeded drinking water standards, sometimes significantly, in some years. Although these elevated levels are not related to bombing activities, they highlight the need to monitor continually the public water supply to ensure its quality. According to the most recent water quality reports, lead levels in source water have only rarely exceeded standards since 2007. The levels of lead do not appear to be a current concern.

8.2.4.2 Public and Private Well Water

The available, recent data were collected and analyzed according to standard methods and procedures. But, similar to the pipeline water, only one or two rounds of sampling occurred, limiting the confidence that the recent results will fully represent the wells' status over time. Regarding the historical sampling results from the 1970s, these data are insufficient for public health conclusions regarding past conditions. Only two samples were collected, and the sampling and the analysis lacked documented quality assurance. We assumed that any available water quality reports or testing from that period would be similarly limited.

8.2.4.3 Rainwater Collection Systems

No information was available on the prevalence of rainwater collection systems—including whether they were ever used for drinking water or whether they were ever tested for contaminants.

8.3 Analysis

Scientists, Viequenses, and other Puerto Ricans questioned whether the island's drinking water was safe. In response, ATSDR first evaluated the available data. The drinking water pathway's public health evaluation conformed to ATSDR procedures for using limited available environmental data to reach public health conclusions. The focus was on exposures taking place at that time. Uncertainties did arise from the limited available data, which we discuss below. We also discuss new information that may add to the previous assessment of drinking water exposures in light of groundwater sampling data obtained since ATSDR's 2001 evaluation.

8.3.1.1 Pipeline Water

In 1999–2000, only one round of sampling appears to have been done for the tanks and for the residential tap water representative of the water supplied to residents. While conditions in the tanks and the tap water are not expected to change over time, greater confidence in the safety of the storage and distribution of the municipal supply could be gained by repeating the sampling and increasing the number of samples collected at the point of use. Although Puerto Rico water authorities regularly test

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the source water, recent (2004–2009) water quality reports have indicated intermittent problems with elevated levels of lead in the water. That water is from Puerto Rico and would not be affected by past military activities on Vieques. But regardless of the source, lead in drinking water is a concern, especially for pregnant women and growing children. Ongoing monitoring and prompt action to address drinking water violations are essential to ensure the pipeline water's continued safety.

8.3.1.2 Public and Private Well Water

ATSDR identified two general uncertainties associated with the evaluation of the former public water supply wells and the private wells:

- Number of Data Points: The conclusions about the recent safety of the drinking water wells were based on one or two sampling events for each well (June–August 1995 and September 1999). Because well conditions usually fluctuate over time, we would have greater confidence in the safety of water from these wells were multiple time points available, encompassing at least both the rainy and dry seasons. We recognize that due to shutdown, further sampling of some of the wells (e.g., Sun Bay) is impossible, and that if people are unable to drink the water, no exposure can occur. Also, that contaminants would fluctuate between extremely low levels and levels of health concern is unlikely. Further testing would nonetheless engender greater confidence in the conclusion.
- Lack of Historical Data: The arguments given in the previous assessment support the conclusion that groundwater movement is impossible from the Live Impact Area to the aquifers used for drinking water. ATSDR's previous assessment focused only on explosive ordnance in the Live Impact Area—that was the focus of the petition then under consideration. But other military activities took place on Vieques, such as transportation, storage, and general training. These activities could have had spills or other releases that resulted in contaminants reaching the former public water supply. In addition, nonmilitary contaminant sources, such as spills, sanitary landfills, septic systems, or agricultural run-off, could also have affected the former public water supply. That explosives are or were recently present in the aquifer supplying the former municipal wells is unlikely, given that in the late 1990s nearby wells showed no detections. Yet explosives were possibly detected in the 1978 samples. But this detection was uncertain: only two samples were collected, and any mechanism by which the explosives could have entered the groundwater is unknown.

Still, to be completely rigorous, we cannot dismiss entirely the possibility that explosive residues might have been present in the past in the drinking water supply. If such possible explosives detections were representative of the typical level in the public drinking water, that level was too low to result in health effects. But too few historical data points are available to confirm this conclusion. When the public supply wells were in operation, the owner would have been subject to drinking water regulations and should have collected and maintained water quality testing results. As described previously, however, ATSDR did not attempt to gather historical information on the water quality of these wells. Assuming data and testing reports were kept from the 1970s-1980s and could be located, numerous questions would remain, such as the accuracy and precision of the analytical methods used, the quality control

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measures employed, whether all contaminants of potential concern were analyzed, and whether the sampling in general was adequate to describe past water quality from the wells. Thus with regard to the past condition of any well-supplied public water, sufficient data will never become available to establish its safety fully.

8.3.1.3 Rainwater Collection Systems

In its previous drinking water assessment, ATSDR did not evaluate this pathway—it remains, however, a potential pathway of exposure. ATSDR's later air modeling suggested that air transport was, historically and currently, not likely a major contamination source for rainwater collection systems. Still, to confirm that the water is safe, sampling of representative systems would be necessary. In its previous assessment, ATSDR recommended that Puerto Rico authorities identify example systems and test them to ensure they delivered safe water. To our knowledge, this sampling has not yet occurred. If sampling were conducted today (i.e., years after active bombing ceased), it would not answer the question of whether contaminants might have entered such systems in the past. ATSDR also recommended sampling sediment from rainwater collection systems as an indication of potential past water quality. This sampling would be limited to insoluble contaminants or to contaminants that had precipitated out of solution, had settled out, and had remained unchanged over time. But as stated previously for well water systems, for rainwater collection systems complete information about every past potential contaminant and its past level will never become available.

8.3.1.4 Recent Data

Since ATSDR's 2001 assessment, several investigations have incorporated Vieques groundwater sampling.

- In 2004, additional sampling was conducted for the wells along the western border of the Eastern Maneuver Area (EMA) (CH2MHill 2005). Groundwater analysis detected no explosives, semi-volatile organic compounds, herbicides, pesticides, PCBs, or dioxins. One inorganic (barium) and two volatile organic compounds (bromodichloromethane and chloroform) were detected above screening values.[32] Because a groundwater flow study done in conjunction with the investigation showed that groundwater did not flow from the EMA toward the western border, such exceedances were thought more likely to result from background or from recharge of municipally treated water than from past military activities. ATSDR determined that all the substances found in the investigation were at levels not expected to cause adverse health effects if people drank the water. Although limited in scope, this study provided additional evidence supporting the former assessment's conclusion: groundwater, even if contaminated by military operations, could not physically move westward toward the aquifers formerly used as a public water supply. The results of the study do not exclude the possibility for other groundwater contamination caused by military or nonmilitary activities hydrogeologically connected to the groundwater that supplies public and private wells.

[32] Levels at which no adverse health effects would be expected from even continual exposure.

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- Other investigations conducted since ATSDR's initial assessment have focused on characterizing the extent of contamination at localized groundwater areas of concern identified through the remedial process (CH2MHill 2007a–c;2008a–e). Several contaminants were found at levels that would make the associated groundwater unsuitable for consumption. These studies do not address potential drinking water sources. But they do support the need for characterization and eventual cleanup of source areas resulting from past military activities on Vieques.

The conclusions and recommendations from ATSDR's evaluation of the drinking water pathway appear below and are in Chapter 9 of this report.

8.4 Conclusions and Recommendations

8.4.1 Conclusions

1. The available sampling data of Vieques storage tanks and representative drinking water taps indicate that at the time of sampling, the public drinking water supplied via pipeline from Puerto Rico was acceptable to drink. This conclusion, however, is uncertain due to the limited number of samples and lack of additional rounds of confirmatory sampling. Public water supplies are tested regularly and have to meet water quality criteria. We have reviewed recent water quality reports indicating that lead levels have been a problem in the drinking water supply, although those problems appear to have been addressed now. Ongoing monitoring of the current pipeline source water and prompt action to address problems is required to ensure the supply meets drinking water standards.
2. Limited, late 1990s sampling data from public and private wells indicated that most of the wells are acceptable for occasional consumption, such as when the pipeline source is interrupted.[33] Flow patterns preclude groundwater from the east (where most military operations occurred) affecting the public aquifers. And with the exception of one well, all groundwater contaminants were below ATSDR health screening values. But again, confidence in the conclusions is tempered by the limited number of sampling rounds, which might not have captured seasonal fluctuations in contaminant levels. Also, a lack of adequate historical data prevents ATSDR from making any conclusion about past public supply well exposures (i.e., the 1970s and 1980s).
3. Although additional sampling might change the result, ATSDR confirms its previous conclusion that nitrate-nitrite levels in one private well would pose a health hazard, especially to children. Regardless of the source of this contamination, water from this well should not be consumed unless proven safe by further, comprehensive testing.
4. No data are available to evaluate rainwater collection systems for past or present exposure potential to contaminants. Rainwater collection systems remain a potential exposure pathway.
5. Past military activities did affect groundwater, but the affected groundwater is not currently used for drinking water.

[33] Although the "Sun Bay" public wells have been abandoned, to our knowledge the "B" public wells and the private wells can still supply drinking water.

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8.4.2 Recommendations

1. Continue required monitoring of the public water supply source water and take prompt action to address any exceedances of drinking water standards. Repeat the 1999–2000 storage tank and residential tap sampling, including a wider selection of representative taps. This could confirm the conclusion that the present public water supply is acceptable to drink.
2. Conduct additional sampling of the wells still available for use during pipeline service interruptions. This too could confirm the safety of drinking water from these wells.
3. Determine whether people drink water from rainwater collection systems, and if so, test the collected water to evaluate its safety. Additionally, sample the sediment from these systems; the sample results might provide limited information about the water's past quality.
4. Do not use groundwater beneath the LIA and other former military operations for drinking water; continue environmental assessment and remediation activities to identify and clean up impacted groundwater.

8.5 References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2001. Public health assessment, drinking water supplies and groundwater pathway evaluation, Isla de Vieques bombing range. Atlanta: US Department of Health and Human Services; October.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003. Public health assessment, air pathway evaluation, Isla de Vieques bombing range. Atlanta: US Department of Health and Human Services; August.

[Baker] Baker Environmental, Inc. 1999. Vieques groundwater sampling information. Coraopolis, PA: 15 November.

CH2MHIll. 2005. Draft final groundwater baseline investigation at US Navy's Eastern Maneuver Area, Former Atlantic Fleet Weapons Training Facility. Tampa, FL: May.

CH2MHIll. 2007a. Remedial investigation report for solid waste management unit (SWMU) 6, CH2MHill; February 2007.

CH2MHIll. 2007b. Remedial investigation report for area of concern (AOC) J, CH2MHill; May 2007.

CH2MHIll. 2007c. Remedial investigation report for area of concern (AOC) H, CH2MHill; July 2007.

CH2MHIll. 2008a. Remedial investigation report for solid waste management unit (SWMU) 7. CH2MHill; March 2008.

CH2MHIll. 2008b. Remedial investigation work plan addendum for solid waste management unit (SWMU) 4, CH2MHill; April 2008.

CH2MHIll. 2008c. Final PA/SI report, 12 consent order sites and 8 PI/PAOC sites. CH2MHill; June 2008.

CH2MHill. 2008d. Remedial investigation report for area of concern (AOC) I, CH2MHill; June 2008.

CH2MHIll. 2008e. Remedial investigation report for area of concern (AOC) E, CH2MHill; July 2008.

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Hoffsommer JC and Glover DJ. 1978. Vieques litigation support: explosives analysis of water and soil samples taken on Vieques Island, Puerto Rico, May 11 through May 16, 1978. Silver Spring, MD: Naval Surface Weapons Center, Explosives Chemistry Branch.

Lai MG. 1978. Explosion products content of water and soil samples taken on Vieques Island, Puerto Rico, May 11 through May 16, 1978. Silver Spring, MD: Naval Surface Weapons Center; July.

[PRDOH] Puerto Rico Department of Health. 1995a. Data Sheets concerning contamination at three Sun Bay wells on Isla de Vieques. San Juan, Puerto Rico; May.

[PRDOH] Puerto Rico Department of Health. 1995b. Data Sheets concerning contamination at four B wells on Isla de Vieques. San Juan, Puerto Rico; May.

[PRDOH] Puerto Rico Department of Health. 1999. Investigation of the potable water in Vieques (in Spanish). San Juan, Puerto Rico: August.

[USEPA] US Environmental Protection Agency. 2000. Sampling of the Rio Blanco filter plant and Vieques public water supply tanks, Island of Vieques, January 18–19, 2000.

[USEPA] US Environmental Protection Agency. 1999. Vieques, Puerto Rico potable water storage tank and well sampling report, Vieques, PR, September 27–28, 1999

Chapter 9 Conclusions and Recommendations

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9.1 Introduction

As part of ATSDR's evaluation of Vieques, we have evaluated multiple documents and reports. We visited Vieques, where we discussed public health concerns. We discussed those concerns with local officials, with scientists, and with the Viequenses themselves. We held a meeting with other scientists in which we discussed the most current Vieques research and studies. Our conclusions and recommendations for the Vieques site and community regarding the environmental media, biomonitoring, and health outcomes follow.

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9.2 Consumption of Fish from Reefs off the Vieques Coast

9.2.1 Conclusions

During ATSDR's November, 2009 meeting, several scientists raised concerns about mercury levels in fish. In particular, they were concerned that the conclusions in ATSDR's 2003 Fish PHA were inconsistent with the 2004 U.S.EPA/FDA national advisory concerning mercury. Therefore, ATSDR reviewed its 2003 conclusions and recommendations about mercury in fish from reefs surrounding Vieques.

In its 2003 public health assessment regarding fish consumption, ATSDR used fish intake rates that focused on adults who ate 8 ounces of fish daily and who weighed 70 kg (or 154 pounds). Daily fish intakes rates for children were 4 ounces and children were assumed to weigh 16 kg (or 35 pounds). Estimated doses were compared with ATSDR's MRL of 0.3 µg/kg/day, which was derived from an analysis of the Seychelles Island and Faroese studies. In ATSDR's evaluation as presented in this report, ATSDR used a broad range of daily fish intakes and body weights. Using information from U.S.EPA's Exposure Factor Handbook, ATSDR assumed that daily fish intakes for adult women were as high as 14 ounces—the 99th percentile—and that women weigh as little as 46 kg (or 100 pounds). For children, ATSDR assumed that daily fish intakes were as high as 6 ounces for 1- to 2-year old children, 8 ounces for 3- to 5-year old children, 10 ounces for 6- to 14-year old children, and 20 ounces for 15- to 18-year old children. The highest intakes for children represent the 99th percentile portion size for the stated ages. In addition, ATSDR used the NAS's recommendations and U.S.EPA's Reference Dose (RfD) concerning mercury as well as human toxicity studies from the Faroe Islands. From its evaluation of mercury in Vieques fish, ATSDR reached these new conclusions:

1. ATSDR has identified mercury exposure from frequent consumption of marine seafood as a potential public health hazard. Women with a varied fish diet who typically eat more than 2 oz of fish every day have estimated mercury doses that exceed U.S.EPA's chronic RfD. As portion size increases, the estimated doses approach the lowest level known to cause harmful effects to the developing fetus. ATSDR concludes that if these women are pregnant, their developing baby has a small increased risk of neurological effects later in life. The risk of harmful effects increases as portion size increases. Possible harmful effects identified from studies of non-Viequense children exposed *in utero* involve language, attention, and memory, and to a lesser extent visual/spatial and motor functions.

2. Women who eat grunt or hind more frequently than other reef fish and who typically eat more than 2 oz of fish every day have estimated mercury doses two times higher than women who eat a varied fish diet. As portion size increases, the estimated doses for women who eat larger portions daily (e.g., 10 to 14 oz) approaches or exceeds the lowest level known to cause harmful effects in the developing fetus. ATSDR concludes that if these women are pregnant, their developing baby has a small increased risk of neurological effects later in life. The risk of harmful effects increases as portion size increases. Possible harmful effects identified from studies of non-Viequense children exposed *in utero* involve language, attention, and memory, and to a lesser extent visual/spatial and motor functions.

3. Children with a varied fish diet who typically eat more than 0.5 oz of fish every day have estimated mercury doses that exceed U.S.EPA's chronic RfD. These children have a small risk of neurological effects. But as portion size increases, the risk of harmful effects increases. Depending upon their age, children as young as 1 year who eat 3 to 4 ounces of fish every day have estimated doses that exceed doses known to cause neurological effects and have the greatest risk of harmful neurological effects. Possible harmful effects identified from studies of non-Viequense children exposed *in utero* involve language, attention, and memory, and to a lesser extent visual/spatial and motor functions.
4. Like women, children who eat grunt and hind more frequently than other reef fish have estimated doses two times higher than children who eat a varied fish diet. The estimated doses in these children exceed the doses associated with neurological effects.
5. Some uncertainty is associated with these findings because a person's mercury response is itself somewhat uncertain. The uncertainty could be due to sex, genetics, health and nutritional status, or how mercury is handled in the body. In the three human studies that focused on mercury exposure from eating fish and seafood, the identification of the lowest-effect levels was uncertain. Estimating the mercury dose from eating reef fish was likewise uncertain, given that the dose could vary depending on the type, frequency, and quantity of fish eaten.
6. While ATSDR supports the U.S.EPA's and the FDA's national fish advisory, portions of the advisory do not apply to the Viequenses who rely heavily on local seafood. For example, the advisory discusses fish that Viequenses do not eat, such as pollock, catfish, and tilefish. In addition, the advisory recommends that if a local advisory is not available, people should not eat more 6 ounces of local fish and should not consume any other fish during the week. We include links to the advisory for informational use and have recommended an educational program about mercury in locally consumed fish.
7. Residents need information so they can select local seafood lower in mercury over seafood higher in mercury. This will protect developing fetuses and young children from mercury in fish. For informational purposes only, the advisory and related information is available at: http://www.epa.gov/waterscience/fish/advice; http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm115662.htm, and Chapter 2, Section 2.7 of this report, Appendix 2A-3.
8. For the 104 fish samples collected from the marine areas near Vieques and from the commercial fish market, the average mercury level was 0.1 ppm and the range was nondetectable to 0.33 ppm. The mercury levels detected in Vieques reef fish are similar to levels reported by the FDA (FDA 2010). The mercury levels are in the low (0.02–0.2 ppm) to mid (0.2–0.6 ppm) mercury range identified by FDA in its recent risk and benefit assessment (FDA 2009).

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9. ATSDR also conducted a statistical analysis of the 2001 fish data and concluded the following:
    a. Mercury detected in the seafood is higher in two fish families (grunt and hind) compared with other families sampled (e.g., parrotfish and snapper). The average mercury concentration in grunt and hind was higher than U.S.EPA's 0.049 ppm screening level. In testing some of the hypotheses, ATSDR used the overall average of 0.1 ppm to determine which families of reef fish were likely to exceed the overall average.
    b. Mercury levels in parrotfish were statistically lower than U.S.EPA's 0.049-ppm screening level and lower than the 0.1-ppm overall average.
    c. All snapper were lower than U.S.EPA's 0.049 ppm screening value. But an insufficient number of snapper were collected to determine whether the average snapper-mercury level was statistically different from the U.S.EPA 0.049 ppm screening level. A sufficient number of snapper were collected to determine that the average mercury level is statistically lower than the 0.1-ppm overall average.
10. Mercury is present in most seafood and is particularly high in some fish species and low in other species. While mercury was a component of the detonators of some bombs, only small amounts of mercury were introduced to the Vieques environment from this source. This conclusion is supported by the low mercury levels in LIA soils, which appear to be at naturally occurring levels. Mercury levels in fish in and around the LIA are most likely the result of the global reservoir of mercury circulating in the environment.
11. Statistical analysis showed that some fish and shellfish from certain reefs surrounding Vieques had higher levels of some metals and lower levels of other metals—iron, aluminum, copper, zinc, arsenic, barium, potassium and selenium were all slightly higher—compared to other reefs surrounding Vieques. These metals are materials found in bombs and in metal ships, suggesting possible localized contamination. But the levels were only slightly higher and the difference was statistically significant only for some reefs compared with other reefs surrounding Vieques.

9.2.2 Recommendations

ATSDR recommends the following for consideration by environmental and public health agencies and scientists:

1. People who frequently consume marine seafood should follow available fish advisories and fishing restrictions in Vieques. Maintain the fishing restrictions in the waters adjacent to the LIA.
2. Conduct a survey of Vieques residents to determine the types, frequency, and quantity of fish consumed.
3. Conduct additional risk assessments and statistical analyses using new information gathered from the previously recommended fish consumption survey.
4. Collect and analyze additional fish samples from Vieques should the proposed survey and statistical analysis not provide sufficient public health information. Collect sufficient fish samples to allow analysis by species and by location.

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5. Develop an educational program about mercury in fish that incorporates local habits and information about Viequenses' seafood consumption. Benefits accrue to the developing fetus with maternal intake of nutrients in seafood (FDA 2009) that can outweigh the concomitant intake of small amounts of mercury. The goal of this site-specific educational program should be to educate Viequenses about the benefits of eating seafood so they can choose fish lower in mercury and still maintain their healthy dietary customs of consuming local seafood.

9.3 Biomonitoring

9.3.1 Conclusions

1. Since 1999, Vieques has hosted at least five human biomonitoring investigations. Puerto Rican scientists, physicians, or the Puerto Rico Department of Health (PRDOH) conducted all of them. The PRDOH has conducted the most extensive sampling effort, collecting biological specimens from 500 randomly selected Viequenses. The PRDOH manuscript reported that in 20% of the participants, either

   a. Aluminum, lead, or mercury in blood;

   b. Uranium, cadmium, or nickel in urine; or

   c. Nickel or arsenic in hair

   exceeded the laboratory's reference range. The PRDOH manuscript identified cigarette use and hair dyes, as well as the consumption of seafood, as possible sources for some but not all the elevated levels. The PRDOH manuscript acknowledged that for some residents, it could not identify the source of high metal levels in urine, hair, and blood. The PRDOH manuscript did not report mercury levels in hair. Results from Dr. Ortiz Roque's investigations showed that some residents had elevated levels of mercury in hair, and that the most likely source was fish consumption; other possible sources, however, were not completely ruled out.

2. Data from these studies showed that in blood, urine, hair, or feces, some residents of Vieques had elevated levels of various metals. While some of these elevated levels might be explained by cigarette use, seafood consumption, or hair dyes, they do not account for all the elevated levels. In particular, biomonitoring results from Dr. Ortiz Roque showed that some Viequenses had elevated mercury in hair above 12 ppm, the level identified by the NAS to cause harm in 5% of fetuses exposed *in utero*. Dr. Ortiz Roque also showed that mercury in hair was associated with fish consumption. In contrast, the PRDOH study did not find excessive mercury levels in blood, although the study either did not measure for or did not report mercury levels in hair. Thus the prevalence of high mercury levels in Viequenses remains somewhat uncertain. Except for mercury, metal content in hair is difficult to interpret. Metals can bind directly to hair from the use of commercial hair products, making difficult any distinction between internal metal exposure via ingestion and inhalation and external exposure resulting from contact with the metal in the environment (e.g., shampoo, dyes, dirt).

3. These studies were unable to investigate each person's environment to identify the source for those who had excessive metals in hair, urine, or blood. The PRDOH study came closest to

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identifying possible sources (e.g., cigarette and hair dye use, seafood consumption) but acknowledged an inability to identify the source or sources for all residents with excessive metals exposure. Either through the survey instrument or through an in-home visit, it may be possible to identify other sources that increase metal exposure, such as cooking utensils, metal residues in foods (e.g., tea and vegetables), consumption of drinks with metallic packaging, religious rites using mercury-containing capsules, skin creams, antacid formulations and antiperspirant formulations.

4. ATSDR remains cautious in making decisions about using hair as an indicator of exposure to environmental contaminants and as an indicator of risk of harmful effects. A major problem in interpreting metal concentrations in hair is whether the metal content resulted from internal exposure (e.g., from ingestion or inhalation) or from external exposure (e.g., the hair coming in contact with a metal-containing product). Currently, no washing method is capable of removing exogenous metal contaminants while leaving endogenous metals undisturbed. Chemicals such as methylmercury, which originate generally from dietary sources, suffer less from this drawback, provided unusual sources of inorganic mercury do not complicate the picture, (e.g., mercury vapor in occupational or home settings).
5. These biomonitoring results do not permit any conclusions about exposure to the bombing-related contaminants.

9.3.2 Recommendations

1. Viequenses may be exposed to mercury in fish and cadmium in pigeon peas. These exposures may warrant additional environmental investigations, such as sampling locally grown produce for cadmium and gathering more information about fish consumption and possibly mercury in fish. The information could be used to decide whether to undertake human testing for mercury and cadmium in blood or urine. If other environmental exposures are identified, additional human biomonitoring investigations may be considered. More detailed information about ATSDR's recommendations concerning fish and locally grown produce can be found in Chapter 2, Section 2.3.2 and Chapter 5, Section 5.3.2, respectively.
2. ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time because we found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation.

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3. Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing. A qualified laboratory should do the testing and analysis.

4. CDC/ATSDR can provide a list of qualified laboratories that can perform the tests. And if requested, CDC/ATSDR can provide information to healthcare providers about tests for metals in biologic samples.

9.4 Health Outcome Data

9.4.1 Conclusions

1. The documents ATSDR reviewed paint a complex health picture for Viequenses. The findings of these studies indicate elevations in chronic disease prevalence, cancer incidence, and cancer mortality among the population between Vieques and the rest of Puerto Rico. In addition, increased mortality in Vieques particularly from cancer may indicate lack of access to appropriate medical care. The limitations associated with these analyses, particularly the methodological concerns discussed in this report, introduce considerable uncertainty and make interpretation difficult. Some of the methodological concerns previously noted include more exhaustive cancer case finding in Vieques than in the rest of Puerto Rico; differential follow-up, chronic disease prevalence estimates based on a symptom disease prevalence survey without medical record confirmation of health outcomes; and limited noncancer mortality data. Differential follow up and exhaustive case finding may result in biased estimates of cancer incidence and mortality. Uncertainty is also evident in the wide confidence intervals reported indicating imprecise estimates of cancer incidence and mortality ratios in Vieques relative to the rest of Puerto Rico (RCCPR 2009). This is not unusual when calculating estimates for small populations.

2 These findings can nonetheless serve as a guide for future investigations of Viequenses' health status.

9.4.2 Recommendations

Assess the feasibility of applying the SMART BRFSS methods for generating stable Vieques specific prevalence estimates on asthma, diabetes, hypertension, and other chronic diseases.

9.5 Local Produce and Livestock Pathway

9.5.1 Conclusions

1. The overall data are insufficient to quantify adequately human exposures or draw any valid health conclusions about whether consuming locally grown produce and livestock would result in harmful health effects.

2. Limited sampling data are available for pigeon peas, a staple food among Viequenses. Interpretation of these data is uncertain because of the lack of adequate QA/QC information for the analytical findings.

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a. A preliminary data evaluation completed for this report has concluded that the level of cadmium reported in a few samples of locally grown pigeon peas would not contribute excess dietary cadmium to preschool children who eat less than 5 of the largest (6 ounces) servings per week of locally grown pigeon peas. Adults who eat the largest serving sizes (12 ounces) should limit intake to 11 servings per week.

b. Preschool children who eat a typical serving size (1.5 ounces) may eat up to 20 meals per week and adults who eat a typical serving (3 ounces) may eat up to 44 meals per week without exceeding recommended cadmium intake levels.

c. Exceeding recommended levels of cadmium in the diet would not typically result in immediate health harm. Excessive intake of cadmium over decades could contribute to harmful levels of cadmium accumulation in the kidneys, possibly resulting in kidney disease. Typically, only a very small fraction (5%) of cadmium in food is absorbed in the body, especially in individuals with healthy diets containing adequate amounts of essential minerals such as zinc, iron and calcium (Reeves and Chaney, 2008).

3. Whether the limited sampling results are representative of cadmium concentrations in other locally grown pigeon peas is unclear; but the significant uncertainty in the evaluation stresses the need to conduct further sampling.

4. Preliminary evaluation results and the totality of the available data suggest a potential for uptake of metals from soil into food crops—thus further investigation is warranted. To evaluate better this exposure pathway, ATSDR has recommended a collaborative effort to conduct additional sampling of locally grown foods.

9.5.2 Recommendations

ATSDR supports additional sampling and collaborative data collection to evaluate more thoroughly this human exposure pathway. The following recommendations are intended to promote collection of high quality data of maximum utility, and to minimize data uncertainties:

1. Sampling data should represent edible portions of a cross-section of local produce from local farms, home gardens, and local markets. Sample produce that tends to accumulate heavy metals more easily (e.g., leafy vegetables such as lettuce and spinach) rather than leaves of grain or fruit crops.

2. Survey residents to identify the prevalence and type of locally grown and commonly consumed garden produce. Sampling plan design should include produce types identified from this survey.

3. Collect representative surface soil samples at the same location and time that garden and farm produce samples are taken. Soil samples should be of appropriate depth to represent the root zone.

4. Collect samples of meat and milk products from Vieques forage-consuming livestock.

5. To avoid false high or low values, sample collection and analysis should conform to high standards of data quality, including detailed quality assurance/quality control information,

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standard reference materials for analysis, and background correction. Design the sampling plan to collect a sufficient number of samples to ensure high statistical confidence.

6. Collect samples from an appropriate background or control location for comparison with food samples produced on Vieques.
7. To facilitate exposure assessment, report data as consumed (wet weight).
8. Plan and conduct sampling protocols in collaboration with local scientists. One way to avoid misunderstandings about the data is for local scientists and an independent expert jointly collect and split several samples and provide analytical reports with QA/QC supporting information. A QA/QC standard could be split at the time the samples are collected.
9. Request technical assistance from the U.S. Food and Drug Administration (FDA) or the U.S. Department of Agriculture (USDA) in sample collection and analytical protocols. The USDA Agriculture Research Service has offered to provide, if requested, technical assistance in sampling plan design, collection, and analysis with proper QA/QC protocols.

9.6 Air Pathway

9.6.1 Conclusions

1. This review of potential airborne exposures within the residential areas of Vieques to contaminants from military operations at the former Vieques Naval Training Facility is based on the following sources of information that were used in the 2003 ATSDR PHA:
   a. U.S. Navy records of the types and amounts of ordnance used at the training range,
   b. A source term model used to estimate the amounts of material produced by ordnance detonations,
   c. An air dispersion model to estimate contaminant transport from the detonation area to the residential areas,
   d. Long term meteorological data from the San Juan PR airport and the Roosevelt Roads Naval Station, and
   e. Ambient air monitoring in the residential areas conducted by the PREQB for the 2001-2003 time period.

   Also summarized are air monitoring data and air dispersion studies developed or collected after publication of the 2003 Air PHA for the purpose of evaluating ongoing or proposed open detonation or open burning.
2. This review of potential airborne exposures evaluates the modeling process used in the 2003 Air PHA, the assumptions and data used in the modeling process, and compares the results of that modeling process with monitored data collected during recent open burning events.
3. While this review finds the Air PHA modeling process consistent with established modeling practices, it does identify two minor errors in estimating detonation source concentrations. One

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results in an overestimate of metals concentrations. The other results in a minor underestimate of explosion byproducts in entrained soil that is overcompensated by the overall estimate of explosive compound concentrations. These errors do not affect the results or conclusions of the Air PHA.

4. Collectively, the available sampled and modeled data are adequate for the determination of the potential public health hazard from exposure to airborne contamination. The uncertainties inherent in the 2003 ATSDR air dispersion model have been adequately addressed by overestimating numerous aspects of the contaminant emissions and by assessing exposures for worst-case conditions. Despite the overestimation of potential exposures, the model results indicate that airborne contaminants from historic uses of the Vieques Naval Training Range would have been essentially nondetectable in the residential areas of Vieques and unlikely to have resulted in harmful effects. These predicted results are supported by the PREQB's historic and recent air sampling data and by the Navy's post-PHA air sampling and air dispersion modeling. Collectively, the available data provide an adequate basis for the public health conclusions in the Air PHA.
5. To conduct its health protective evaluation of potential exposures to airborne contaminants from military exercises at the former Vieques Naval Training Facility, the ATSDR Public Health Assessment used appropriate modeling procedures and available monitoring data. The additional air dispersion modeling and monitoring data conducted after completion of the Air PHA further support ATSDR's public health conclusions. Because past military exercises would not likely have produced measurable concentrations of airborne contaminants in the residential areas of Vieques, no public health basis supports additional air studies or ambient air monitoring efforts related to past military exercises on Vieques.
6. Although the residents of Vieques could hear and possibly feel the explosions from military exercises on the former Vieques Naval Training Range, 8 miles is still a considerable distance. The physics of sound and seismic wave migration are very different from the physics of atmospheric dispersion of airborne particulates and gases.

9.6.2 Recommendations:

No further recommendations at this time.

9.7 Soil Pathway

9.7.1 Conclusions

1. Those who occupied the LIA from 1999–2000 were not at increased risk of adverse health effects from exposure to surface soil contaminants. Supporting data are limited, but they are of good quality and represent the location and the period of interest. In the years before the late 1990s tests, contaminant levels in the LIA (especially explosives) might have been higher, but we have no historical data with which to evaluate this assumption.
2. This does not mean anyone can now safely visit the LIA. Remaining unexploded ordnance could cause immediate injury or death to anyone who might inadvertently disturb it. Recently

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collected data on specific areas of concern within military lands—not the same areas accessed and occupied by protesters—demonstrate the remaining potential for localized contamination, which, if people frequented those areas, could be of health concern.

3. Limited available data from other locations and air pathway considerations suggest that the military exercises in the LIA did not result in current contamination of residential soils with inorganic or explosive compounds at levels considered harmful. ATSDR arrives at this conclusion using a scientific evaluation of the available data. But again, data from other areas are limited in number, data for all potential contaminants of concern are not available, and no adequate surface soil data are available from the residential area itself. ATSDR nonetheless understands that community members remain concerned about residential soil exposures.
4. Modeling described in the air pathway discussion has suggested that airborne transport of contaminants during past military exercises would not have been substantial enough to have affected soils in the island's residential area. Sufficient historical data are not available to confirm this, nor will such data ever become available. Consequently, we cannot determine whether past exposures to explosives or other compounds in surface soil could have been heavy enough to increase a past risk of adverse health effects.

9.7.2 Recommendations

1. Continue to restrict the LIA and other potentially contaminated military areas and continue environmental assessment and remediation activities to clear the way for public access.
2. To help address community concerns regarding residential soil, work with local residents to design sampling to identify residential surface soil quality issues, whether or not those surface soil issues relate to past military activities:

   a. Focus initial sampling on areas identified as potential high-exposure areas, such as bare areas or child-use areas.

   b. To identify any possible differences in the top layer, initial sampling should analyze the top inch of soil separately from the rest of the 0–3 or 0–6 inch surface samples.

   c. Perform a full range of analyses to identify all possible contaminants.

   d. Plan and conduct sample collection and analysis to ensure the results meet high data quality standards.
3. Although this sampling might provide inferences about past exposures for compounds stable over time, such as metals or other inorganic compounds, it will provide no definitive information on past surface composition and no information on past levels of compounds that react or degrade over time.
4. ATSDR also recommends collection of representative soil samples as part of its evaluation of the terrestrial food chain pathway. See Chapter 5 of this report for details.

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9.8 Drinking Water Pathway

9.8.1 Conclusions

1. The available sampling data of Vieques storage tanks and representative drinking water taps indicate that at the time of sampling, the public drinking water supplied via pipeline from Puerto Rico was acceptable to drink. This conclusion, however, is uncertain due to the limited number of samples and lack of additional rounds of confirmatory sampling. Public water supplies are tested regularly and have to meet water quality criteria. We have reviewed recent water quality reports indicating that lead levels have been a problem in the drinking water supply, although those problems appear to have been addressed now. Ongoing monitoring of the current pipeline source water and prompt action to address problems is required to ensure the supply meets drinking water standards.
2. Limited, late 1990s sampling data from public and private wells indicated that most of the wells are acceptable for occasional consumption, such as when the pipeline source is interrupted.[34] Flow patterns preclude groundwater from the east (where most military operations occurred) affecting the public aquifer. And with the exception of one well, all groundwater contaminants were below ATSDR health screening values. But again, confidence in the conclusions is tempered by the limited number of sampling rounds, which might not have captured seasonal fluctuations in contaminant levels. Also, a lack of adequate historical data prevents ATSDR from making any conclusion about past public supply well exposures (i.e., the 1970s and 1980s).
3. Although additional sampling might change the result, ATSDR confirms its previous conclusion that nitrate-nitrite levels in one private well would pose a health hazard, especially to children. Regardless of the source of this contamination, water from this well should not be consumed unless proven safe by further, comprehensive testing.
4. No data are available to evaluate rainwater collection systems for past or present exposure potential to contaminants. Rainwater collection systems remain a potential exposure pathway.
5. Past military activities did affect groundwater under some specific military areas, but the affected groundwater is not currently used for drinking water.

9.8.2 Recommendations

1. Continue required monitoring of the public water supply source water and take prompt action to address any exceedances of drinking water standards. Repeat the 1999-2000 storage tank and residential tap sampling, including a wider selection of representative taps. This could confirm the conclusion that the present public water supply is acceptable to drink.
2. Conduct additional sampling of the wells still available for use during pipeline service interruptions. This too could confirm the safety of drinking water from these wells.

[34] Although the "Sun Bay" public wells have been abandoned, to our knowledge the "B" public wells and the private wells can still supply drinking water.

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3. Determine whether people drink water from rainwater collection systems, and if so, test the collected water to evaluate its safety. Additionally, sample the sediment from these systems; the sample results might provide limited information about the water's past quality.
4. Do not use groundwater beneath the LIA and other former military operations for drinking water; continue environmental assessment and remediation activities to identify and clean up impacted groundwater.

9.9 Final Summary

ATSDR conducted a thorough review of environmental, biological, and health outcome data from the Island of Vieques. We carefully considered the data and information from Puerto Rican and other scientists who spent considerable effort and time, sometimes at their own expense, to research environmental health issues in Vieques. We are grateful for their efforts. Nevertheless, as with any site that has had such a long history of environmental investigations, those data have strengths and weaknesses that become important in public health decision-making. At times, the data can raise as many questions as they can provide answers.

One such situation is mixtures. ATSDR recognizes the possibility that this report cannot address accurately the effects of mixtures and cumulative exposures on the health of Viequenses. Sometimes this is the result of a lack of data; other times it is a limitation of the science. ATSDR has published guidance for mixtures assessments (ATSDR 2004) and has some of the world's experts on these subjects among our staff. But we recognize the continuing debate among scientists on these subjects and that no scientific consensus, or "best practice," has yet emerged on how to assess these effects accurately.

Further, we are aware of the literature that examines the physical and psychological health effects stress can have on a community such as Vieques, given its six decades of military activities and lingering concern for the effects of those activities on the population's health. Studies have shown that chronic stress is a risk factor in cardiac disease and contributes to the onset of autoimmune diseases. Chronic stress also is an important factor in premature aging.

Finally, as we stated at the outset of this report, ATSDR recognizes that Viequenses are concerned about the health of everyone who lives on their island. We hope the conclusions and recommendations in this report will raise public health awareness, increase the public's well-being, and protect the health of all Viequenses.

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Appendix A Study Summaries

Dramatic Increase in Radiation in Vieques A-3

Radioactivity in Vieques [trans.] A-5

Environmental Impact of Navy Activities in Vieques A-8

Heavy Metal Studies in Sediments from Gato and Anones Lagoons [trans.] A-11

Heavy Metals in the Impact Area Prevailing Vegetation, Vieques, Puerto Rico [trans.] A-13

Toxic Metals in the Vegetation of the Civilian Zone of Vieques, Puerto Rico [trans.] A-17

Trace Element Composition in Forage Samples from a Military Target Range, Three Agricultural Areas, and One Natural Area in Puerto Rico A-21

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Category:	Environmental Data – Radiation
Date of Publication:	November 9, 2001
Title:	**Dramatic Increase in Radiation in Vieques**
Author(s):	Frankie Jimenez
Affiliation:	Committee for the Rescue and Development of Vieques (CRDV)
Publication Status:	Press Release
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Air Pathway Evaluation)

Study Findings

In 2001, after two rounds of bombing in Vieques, radiation sampling resulted in these findings:

Table A-1. Radiation sampling in Vieques, 2001

Date	***Sampling Location***	***Radiation Level***
June 2001	Civilian areas Yayi Beach (near bombing range)	No increase 60% increase
September-October 2001	Esperanza Beach (civilian area, south coast) Main town (Isabel II, north coast)	248% increase 225% increase

The authors attributed the radiation increase to soil dispersion contaminated by past use of depleted uranium (DU) penetrators at the Live Impact Area (LIA). The press release did not indicate how levels of radiation were measured, what types of radiation were measured, or the actual amounts of radiation detected.

The press release did, however, condemn future bombing exercises planned for November–December 2001.

How ATSDR Addressed the Study Findings

In 2002, ATSDR contacted the Committee for the Rescue and Development of Vieques (CRDV) to learn more about this sampling effort, but did not receive a response. The allegation that radiation levels increased on Vieques during military training exercises could not be confirmed. No public health

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decisions could be made based on a reported 248% increase in radiation levels without data. The more important indicator of exposure is the actual level of radiation, not the relative increase. No confirmation of statements made in the press release could be obtained. Nonetheless, in Section V.D of the PHA's Air Pathway Evaluation, ATSDR used established background levels to investigate the public health implications of the reported radiation increase.

To estimate the effect of a reported 248% dose increase, ATSDR used data collected by the Nuclear Regulatory Commission (NRC) to establish background radiation levels. In June 2000, the NRC recorded radiation dose rate measurements at 29 locations in Vieques residential areas using a Ludlum Model 19 microR meter. These observations were collected at a distance of 1 meter above ground surface. The 29-measurement average exposure rate was 4 microroentgens per hour (µR/hour)—approximately equal to 4 microrem per hour (µrem/hour). ATSDR assumed this dose rate represented external radiation background levels in the Vieques residential areas. If the CRDV data were based on similar dose rate observations, a 248% increase in radiation would imply that radiation levels increased from 4 µrem/hour to 14 µrem/hour, or a net increase above background of 10 µrem/hour. Assuming this increase above background occurred 24 hours per day for 90 days per year (i.e., the maximum amount of time the Navy could conduct military training exercises on Vieques), the overall increase in radiation dose for the year would be 22 mrem—a level well below ATSDR's chronic minimal risk level (MRL) for ionizing radiation. The MRL is defined as an increase in ionizing radiation dose of 100 mrem above background per year. Thus given these parameters, ATSDR does not consider a 248% increase indicates radiation exposures at levels of concern presuming an exposure rate of 4 µrem/hour.

In fact, ATSDR noted that the levels of radiation measured in the NRC study appear to be well within background levels observed throughout the United States. And a non-CRDV press release (Fellowship of Reconciliation 2001) announced that the highest level of radiation measured during the recent survey on Vieques was 18 µR/hour—approximately equal to 18 µrem/hour. These levels are comparable to survey readings collected elsewhere in the United States; they are considerably lower than background measurements from many areas at elevations of several thousand feet such as Denver, Colorado. Due to its higher elevation, Denver would be expected to have higher-than-average radiation levels.

As part of the agency's analysis of data, ATSDR again requested more information on the study. In response to ATSDR's request, the report **"**Radioactivity in Vieques" was received on August 5, 2009.

Reference

Fellowship of Reconciliation. 2001. Vieques issue brief: environmental impacts of navy training; November 2001.

Category:	Environmental Data – Radiation
Date of Publication:	circa 2000
Title:	**Radioactividad en Vieques** Radioactivity in Vieques [trans.]
Author(s):	Frankie Jimenez
Affiliation:	Committee for the Rescue and Development of Vieques (CRDV)
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

From October 7, 1999 to February 3, 2000, radiation sampling was conducted on the Live Impact Area (LIA). The investigator conducted the sampling in response to the U.S. Navy's accidental use of depleted uranium projectiles on February 19, 1999. The investigator took gamma radiation measurements from around the targets (e.g., tanks and aircraft), from the roads leading to the targets, and from areas where former targets were buried. Yayí Beach was measured as the background location.

According to the investigator's report, samples were collected with an instrument "manufactured by the Nuclear Research Corporation." The report provided no further sampling device information nor did the report list actual amounts of radiation detected at other locations. It provided instead the percent increase above readings at Yayí Beach. Lastly, the report did not specify the averaging time for individual measurements. Thus whether the findings were based on instantaneous peak radiation levels or sustained averages is unclear.

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Table A-2. Vieques LIA radiation sampling, 1999–2000

Sampling Location	*Radiation Level*
Yayí Beach (background location)	7 µR/hr
Mayor of Cataño's camp	30% increase
Several aircraft behind the "Pro Vieques" camp	50% increase in front of the planes, "normal" away from the planes
Tank just before the mountain	200% increase
Tank at the top of the mountain	100% increase
Roads leading to the targets	100% increase, "normal" immediately beyond the roads
Tanks near the Monte David camp	50% increase
Slope of the mountain near the Monte David camp	220% increase
Tanks near Salina Sur Beach	100% increase

µR/hr = microroentgens per hour

At the beginning of the article, the investigator noted that radiation levels in the LIA were "over 500% higher than normal." But 220% was the highest percent increase listed. Because certain areas indicated "above-normal" radioactivity, the investigator concluded that radioactive material was present on and around the targets as well as under the surface (e.g., roads and burial grounds). After completing some sampling, on January 19, 2000, the investigator filed a complaint with the Nuclear Regulatory Commission (NRC). That complaint might have triggered the NRC study reviewed in the PHA's Air Pathway Evaluation.

How ATSDR Addressed the Study Findings

ATSDR received this report on August 5, 2009. Because this report—like a contemporary press release entitled *Dramatic Increase in Radiation in Vieques*—lacked an actual radiation level reading, ATSDR investigated the public health implications of the reported increase in radiation using the same approach as presented in Section V.D of the PHA's Air Pathway Evaluation. A 220% increase in radiation would imply that radiation levels increased from the background of 7 µr/hour (approximately equal to 7 microrem per hour [µrem/hour]) to 22 µrem/hour, or a net increase above background of 15 µrem/hour. Assuming this increase above background occurred 24 hours per day, 365 days per year, the overall increase in radiation dose for the year was 131 mrem. ATSDR's chronic minimal risk level (MRL) for ionizing radiation is defined as an increase in ionizing radiation dose of 100 mrem above background per year. Even given that this estimated dose exceeded ATSDR's MRL, the calculation did not take into account individual occupancy. That is, for the MRL to be exceeded, a person would have to find the elevated radiation-level location and remain there 24 hours per day for an entire year. A more realistic—yet still unlikely—scenario is for a person to remain at the location 4 hours per day over the course of a year. This would result in an increased radiological dose of 20 mrem for the year.

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Thus with respect to the actual method used to detect the radiation, the information supplied is of limited use: the background range was not specified. Typically, gamma radiation measurements will vary by 3 to 5 µR/h. As the author stated, the measured background was 7 µR/h, which ATSDR believes could vary up to 11 or 12 µR/h. Such a meter variation could also account for the large percentages observed. For example, if the average were 7 µR/h, an increase in 3 µR/h would represent a 50% increase.

Further, although depleted uranium (DU) is radioactive, its gamma radiation emission is low. It would not add to the ambient background radiation detectable by the type of radiation meter used in this study. In fact, the United Nations estimated that DU radiation is so weak it would be shielded by the soil in which it was buried.[35] Thus ATSDR believes the reported DU radiation was in error.

[35] World Health Organization (2001). Report of the World Health Organization. Depleted uranium mission to Kosovo. Available online at http://www.who.int/ionizing_radiation/pub_meet/en/Report_WHO_depleted_uranium_Eng.pdf [accessed 2011 June 13].

Category:	Environmental Data – Soil, Sediment, and Water
Date of Publication:	July 11, 2000
Title:	**Environmental Impact of Navy Activities in Vieques**
Author(s):	N García Martínez, AM López, M Soto, T García, S Rosado, B Berríos
Affiliation:	Servicios Científicos y Técnicos
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Soil Pathway Evaluation)

Study Findings

From May 1999 to April 2000, personnel from Servicios Científicos y Técnicos, Inc. collected and analyzed soil, sediment, and surface water samples from 55 Vieques locations. Their purpose was to determine levels of metals and other inorganic compounds in the Vieques ecosystem. Forty-four samples were collected from the LIA: specifically, areas of direct impact, target areas, and nearby areas. Five were collected from the Punta Este Conservation Zone, and six were taken from the residential area. Eleven water samples were collected from lagoons in the LIA, from Carrucho Beach, and from the Punta Este Conservation Zone. The sample analysis included highest and second highest concentrations:

Table A-3. Water samples from LIA lagoons

Chemical	*Highest Concentration (ppm)*	*Type of Sample*	*2nd Highest Concentration (ppm)*	*Type of Sample*
Aluminum	24,500	soil	23,600	soil
Ammonia	50.1	sediment	46.8	soil
Arsenic	20.2	soil	16.4	soil
Barium	1,170	soil	1,100	soil
Cadmium	31.3	soil	27.4	soil
Chromium	40.2	sediment	31.9	soil
Cobalt	26.4	soil	21.4	soil
Copper	501	soil	313	soil
Iron	67,900	soil	51,400	sediment

Chemical	*Highest Concentration (ppm)*	*Type of Sample*	*2nd Highest Concentration (ppm)*	*Type of Sample*
Lead	138	sediment	75.4	soil
Manganese	1,111	soil	811	soil
Mercury	4.21	sediment	0.329	sediment
Nickel	68.7	soil	32.9	sediment
Nitrates plus Nitrites	50.8	sediment	30.2	soil
Phosphorous	40.4	soil	16	sediment
Selenium	1.48	sediment	1.34	sediment
Silver	0.625	soil	0.526	sediment
Sulfate	8,590	NA	8,380	NA
Tin	38.7	surface water	NA	NA
Vanadium	178	soil	164	soil
Zinc	872	soil	325	soil

NA = not available

ppm = parts per million

The authors reported that higher levels of metals and other substances were in the sediment, soil, and water samples collected from the LIA as opposed to samples collected from the eastside territory and from the civilian zone. LIA zinc and copper concentrations were higher than those USGS reported in 1973. The authors recommended additional environmental sampling in the civilian area and western portion of Vieques, as well as biomonitoring samples from Vieques residents.

How ATSDR Addressed the Study Findings

In a statistical comparison with other LIA data, these data show nine of the 20 chemicals were significantly different (higher, $p < 0.05$): ammonia, barium, cadmium, copper, lead, mercury, nickel, nitrate/nitrite, and zinc. This does not imply any data discrepancy; it simply means that using only the highest and second highest detections rather than the complete dataset would skew any results. Therefore, these data were not used in the statistical analyses to evaluate general soil characteristics. They were, however, used to form the following conclusions during the public health evaluations in the PHA's Soil Pathway Evaluation (see Section IV in the Soil Pathway Evaluation):

- During the public health assessment process, all available soil sampling data were considered, including the data presented in the Servicios Científicos y Técnicos report.
- To evaluate human exposures, ATSDR compiled roughly 600 soil/sediment samples (for a soil sampling summary, see pages 13–15 of the PHA).
- Vieques residents are not currently exposed to harmful levels of chemicals in the soil.

- Although in the past, Navy training activities elevated the levels of some metals in the soil on the former LIA, those levels remain too low to be of health concern.
- Protestors who lived on the LIA for a year (1999–2000) endured the longest exposure to LIA soil contaminants. ATSDR analyzed the soil data collected by Servicios Científicos y Técnicos—together with Navy data—from areas where the protestors lived. ATSDR determined that the protestors were not exposed to harmful levels of chemicals in the soil.

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Category:	Environmental Data – Sediment
Date of Publication:	2002
Title:	**Metales pesados en sedimentos de las Lagunas Gato y Anones.** Heavy Metal Studies in Sediments from Gato and Anones Lagoons [trans.]
Author(s):	Arturo Massol-Deyá and Elba Díaz
Affiliation:	Casa Pueblo de Adjuntas; University of Puerto Rico, Mayaguez Campus
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

Gato and Anones lagoons are on the western side of the Live Impact Area (LIA), near the Surface Impact Area (SIA). On February 12, 2000, to characterize heavy metal contamination, a sediment sample was collected from each lagoon. The samples were taken only from the lagoon dry areas.

The samples were 50 and 30 centimeters deep and were analyzed in 10- and 5- centimeter intervals (Gato and Anones, respectively). The authors reported that with respect to depth, the metals were almost homogeneously distributed. No significant differences appeared in the concentrations of heavy metals at different depths, nor between the lagoons. The authors noted that the "results display a high degree of mixture in the sediments down to at least 50 cm (20 inches) caused primarily by the intensity of bomb impacts in the area."

The authors reported sampling results in a bar chart. An approximate interpretation of their data is presented below.

Table A-4. Sediment samples from Gato and Anones Lagoons

	mg/kg						
	Copper	***Cobalt***	***Cadmium***	***Lead***	***Nickel***	***Chromium***	***Manganese***
Gato	40–60	60–100	60–100	100–130	60–125	300–500	900–1750
Anones	40–85	75–110	75–110	90–140	75–100	200–250	600–1000

No health guideline was cited.

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How ATSDR Addressed the Study Findings

ATSDR obtained this study in July 2009, and reviewed the data as part of its evaluation of Vieques.

These data were collected from the former LIA—a restricted area. Thus no one is now continuously exposed to these chemicals. The longest potential exposure occurred from April 1999 to May 2000, when protestors occupied the LIA. As reported by Massol-Deyá and Díaz (2002), the maximum concentrations of cobalt, cadmium, lead, nickel, chromium, and manganese were higher than the concentrations evaluated in the 2003 Soil Pathway Evaluation. And when matched with health-based comparison values, the cadmium and chromium concentrations were higher as well. As noted, however, access remains restricted to the former LIA (including Gato and Anones lagoons).

Category:	Environmental Data – Plants
Date of Publication:	April 10, 2000
Title:	**Metales pesados en la vegetación dominante del área de impacto de Vieques, Puerto Rico.** Heavy Metals in the Impact Area Prevailing Vegetation, Vieques, Puerto Rico [trans.]
Author(s):	Arturo Massol-Deyá and Elba Díaz
Affiliation:	Casa Pueblo de Adjuntas and University of Puerto Rico, Mayagüez
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Soil Pathway Evaluation)

Study Findings

In February and March 2000, researchers from Casa Pueblo and the University of Puerto Rico's Recinto Universitario de Mayagűez (RUM), sampled vegetation for heavy metals in the Live Impact Area (LIA)—specifically, Carrucho Beach, Monte David, and Gato and Icacos Lagoons. Reference populations of the same species were also collected in Bosque Seco de Guánica and RUM Alzamora Ranch. Researchers sampled the following plants:

Urochloa maxima (root and stem),

Sporobolus virginicus (root and stem),

Syringodium filiform (stem),

Ipomoea violaceae (fruit),

Faidherbia albida (stem), and

Calotropis procera (leaves).

The average concentrations are presented in Table A-5.

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Table A-5. LIA vegetation sample results

Average concentrations (µg/g)									
	Lead	***Cobalt***	***Nickel***	***Manganese***	***Chromium***	***Cadmium***	***Copper***	***Magnesium***	***Zinc***
Playa Carrucho									
S. filiforme	33.32	29.60	28.66	58.23	2.78	2.78	30.48	9929.14	106.24
Sargazo	22.69	130.46	28.23	169.07	7.36	2.83	28.27	17914.70	187.54
Monte David									
U. maxima (root)	12.85	63.66	14.19	115.44	21.04	2.89	17.10	2550.16	65.78
U. maxima (stem)	10.25	48.65	5.08	135.02	5.99	1.57	4.03	1667.61	42.98
C. procera (leaves)	30.05	68.40	18.08	287.94	12.68	3.11	4.63	11401.98	112.47
A. farnesiana (stem)	8.13	10.15	5.33	18.27	2.86	ND	4.04	2095.67	15.14
Laguna Icacos									
Aquatic plant	2.69	36.31	29.31	1740.42	42.14	4.63	12.93	-	68.97
Laguna Gato									
I. violaceae (fruit)	32.61	25.75	7.64	40.31	1.17	ND	9.39	1867.95	50.86
S. virginicus (root)	17.17	68.61	78.26	882.35	182.62	1.18	10.36	5135.36	73.20
S. virginicus (stem)	30.45	34.81	ND	670.57	5.32	ND	ND	1553.96	24.40
S. pyramidatus (root)	18.77	22.91	7.14	156.23	15.77	2.25	9.02	4450.16	45.06
S. pyramidatus (stem)	12.19	2.73	1.25	31.75	3.03	ND	ND	2931.47	15.60
Bosque Seco de Guanica and Alzamora Ranch (reference populations)									
C. procera (leaves)	1.29	2.59	5.17	17.85	9.31	1.03	5.69	-	19.91
S. filiforme (stem)	5.57	4.19	14.64	251.44	27.93	2.79	15.39	-	59.31
S. virginicus (stem)	0.60	3.11	5.36	7.54	7.35	0.79	2.76	-	14.83
U. maxima (root)	ND	6.89	19.95	78.61	23.75	0.80	14.52	-	75.77

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U. maxima (stem)	ND	4.69	10.50	35.19	12.24	0.60	12.93	-	59.53

ND = not detected

The authors reported that in Vieques vegetation, concentrations of heavy metals such as lead, cobalt, and manganese were significantly higher than concentrations detected in the reference locations. The authors concluded that with the levels detected, if people were to eat these plants they would be exposed to critically hazardous doses. The authors recommended remediation to reduce the transport of chemicals from the LIA to the residential areas.

How ATSDR and the Puerto Rico Department of Agriculture Addressed the Study Findings

This and other studies by the same researchers reported metals in plants and livestock. Consequently, on August 7, 2001, the Puerto Rico Department of Agriculture placed an embargo on Vieques produce and livestock. The Puerto Rico Department of Agriculture, in cooperation with the Farmers Association of Puerto Rico, then sampled grass, fruit-bearing trees, and bovine livestock for cadmium, cobalt, copper, lead, manganese, and nickel. Samples were taken from Monte Carmelo, Martineau, Monte Santo, Esperanza, Lujan, Gubeo, and western Vieques. Sample analysis resulted in the conclusion that Vieques agricultural products were in fact suitable for consumption and did not contain toxic levels of the named contaminants (Department of Agriculture 2001). Because government and Farmer's Association researchers could not duplicate Dr. Massol's results, the embargo was lifted (El Nuevo Día 2001).

As part of the previous public health assessment, ATSDR requested that an agronomist with the U.S. Department of Agriculture (USDA) independently review the Casa Pueblo and RUM studies. The agronomist determined that although the studies provided evidence of heavy metals accumulation in plants, the studies' limitations prevented an estimation of human exposure dose. The agronomist further concluded that

> Many of the species sampled in this study were not eaten, and when edible species were sampled, the edible portions were not sampled. Human exposures from locally grown foods are best estimated from the edible portions of the food source. In general, the edible portions of plants are less likely to accumulate metals from soil because of normal plant processes (i. e., physiological barriers that prevent contaminants from reaching the tops of plants) (ATSDR 2001).

Additionally, the studies lacked standard reference materials to demonstrate that the results were accurate and lacked background corrections for lead, cobalt, nickel, and cadmium to eliminate the effect of light scattering by non-element materials in the samples.
The metals analysis also did not indicate whether the metals were bioavailable. This is important because vegetation samples include both contaminants taken up by the plant and soil particles from the

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growing media. Thus even with a thorough washing, small soil particles will adhere to the plant materials and can actually carry more contaminants than those taken up by the plant from the soil (ATSDR 2001). When the external soil contamination is ingested, the chemicals bound to soil are not usually in a form the body can absorb. While methods are available to determine how much of a chemical concentration is adhered as soil and how much is in the plant tissue, the researchers here did not use them.

Thus ATSDR could neither quantify human exposures nor draw any health conclusions about whether consuming plants grown in Vieques would result in harmful health effects. As part of its evaluation, ATSDR recommended additional sampling of subsistence produce.

References

ATSDR. 2001. Summary Report for the ATSDR Expert Panel Meeting on Tribal Exposures to Environmental Contaminants in Plants. Atlanta: Division of Health Assessment and Consultation; 2001 March 23.

Department of Agriculture. 2001. Letter from Luis Colón to the Undersecretary of Agriculture concerning heavy metal tests on Vieques bovine livestock. 2001 October 17.

El Nuevo Día. 2001. They declare suitable the agricultural products in Vieques. 2001 December 13.

Category:	Environmental Data - Plants
Date of Publication:	January 10, 2001
Title:	**Metales tóxicos en la vegetación de la zona** civil de Vieques, Puerto Rico. Toxic Metals in the Vegetation of the Civilian Zone of Vieques, Puerto Rico [trans.]
Author(s):	Arturo Massol-Deyá and Elba Díaz
Affiliation:	Casa Pueblo de Adjuntas and University of Puerto Rico, Mayagüez
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Soil Pathway Evaluation)

Study Findings

Researchers from Casa Pueblo and University of Puerto Rico's Recinto Universitario de Mayagűez (RUM) randomly collected agricultural and common vegetation from three sites within 1) a Vieques residential section, 2) an agricultural area in Monte Carmelo as well as a section of Monte Carmelo that borders Camp García, and 3) an agricultural farm in Barrio Monte Santo, Gobeo sector. The ensuing paper, however, limited its discussion to the two farms sampled in the civilian area of Vieques and focused its conclusions and recommendations on plants grown for an agricultural economy.

The study reported that all metals (cadmium, cobalt, copper, lead, and manganese) were found at toxic levels. Lead and cadmium had the highest absorption rates. Sampling included leaves and stems of pumpkin, pepper, pigeon peas, pineapple, and yucca. Only guamá and mangoes showed acceptable metals levels.

The study reported the following average concentrations found in plant stems and leaves. It did not report concentrations in the edible portions of the plants sampled (See Table A-6).

The authors concluded that the highest concentrations of metals were found in plants that had shallow roots—where higher contamination levels would be expected to be found in the soil— and plants requiring higher watering rates (more access to soil contamination).

Study recommendations included

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- Permanent cessation of contamination-causing activities.
- A moratorium on consumption of plants grown in Vieques and compensation for farmers.
- Preventative actions for children who are more sensitive to toxins.

How ATSDR and the Puerto Rico Department of Agriculture Addressed the Study Findings

Because this and other studies by the same researchers reported metals in plants (Massol 2000) and livestock (Massol 2001), the Puerto Rico Department of Agriculture on August 7, 2001, placed an embargo on Vieques produce and livestock. The Puerto Rico Department of Agriculture, in cooperation with the Farmers Association of Puerto Rico, sampled grass, fruit-bearing trees, and bovine livestock from Monte Carmelo, Martineau, Monte Santo, Esperanza, Lujan, Gubeo, and western Vieques for cadmium, cobalt, copper, lead, manganese, and nickel. The Department of Agriculture and the Farmer's Association concluded that the agricultural products from Vieques were suitable for consumption and did not contain toxic levels of the named contaminants (Department of Agriculture 2001). When the Department of Agriculture and the Farmer's Association could not duplicate Dr. Massol's results, the embargo was lifted (El Nuevo Día 2001).

As part of the 2001 public health assessment, ATSDR requested independent review of the study by an agronomist with the U.S. Department of Agriculture (USDA). That agronomist determined that although the studies provided evidence that heavy metal accumulation in plants, the study's limitations prevented estimation of a human exposure dose. The agronomist further concluded:

> Many of the species sampled in this study are not eaten, and when edible species were sampled, the edible portions were not. Human exposures from locally grown foods are best estimated from the edible portions of the food source. In general, the edible portions of plants are less likely to accumulate metals from soil because of normal plant processes (e.g., physiological barriers that prevent contaminants from getting to the tops of plants) (ATSDR 2001).

The study lacked the use of standard reference materials to demonstrate that the results were accurate. The study also lacked background corrections for lead, cobalt, nickel, and cadmium to eliminate the effect of light scattering by non-element materials in the samples.

The metals analysis did not indicate whether the metals were bioavailable. This is important because samples of vegetation include both contaminants taken up by the plant and soil particles from the growing media. Even with a thorough washing, small soil particles will adhere to the plant materials, and can actually carry more contaminants than those the plant takes up from the soil (ATSDR 2001). When the external soil contamination is ingested, the chemicals bound to soil are not usually in a form that the body can absorb. While methods are available to determine how much of a chemical concentration adheres as soil and how much is in the plant tissue, the researchers here did not employ such methods.

Because of these factors, ATSDR could neither quantify human exposures nor draw any health conclusions regarding whether consuming plants grown in Vieques would result in harmful health effects. As part of its evaluation, ATSDR recommended additional sampling of subsistence produce.

Table A-6. Vegetation samples

	Average Concentrations (µg/g dry weight)					
	Lead	***Cobalt***	***Nickel***	***Manganese***	***Cadmium***	***Copper***
Monte Carmelo, agricultural area						
C. procera (leaves)	62.16	15.54	20.76	469.58	4.87	28.78
A. farnesiana (leaves)	11.77	7.57	5.81	338.81	1.79	22.90
A. famesiana (stem)	13.04	7.47	5.14	33.26	1.10	14.70
U. maxima (leaves)	8.97	15.24	6.50	29.80	1.57	14.79
Pumpkin (leaves)	42.23	22.52	22.96	97.69	7.94	32.57
Pepper (leaves)	22.64	21.45	9.07	68.53	3.91	21.98
Pepper (stems)	11.85	24.55	12.31	21.66	5.73	41.68
Pigeon Peas (leaves)	10.59	14.81	10.28	193.36	2.72	15.44
Pigeon Peas (stem)	4.80	16.06	4.00	40.38	1.38	8.77
Bo. Monte Santo. Gobeo Sector						
Pepper (leaves)	39.52	6.04	12.90	30.48	2.59	34.11
Pepper (stem)	44.65	8.93	5.52	11.50	2.55	27.66
Mango (leaves)	12.40	3.51	3.00	372.90	0.75	6.26
Yucca (leaves)	29.23	10.22	3.49	104.46	2.23	39.27
Guama (leaves)	7.80	3.81	2.07	155.11	0.94	13.02
Banana (leaves)	29.99	6.06	2.25	507.53	0.64	16.57
Pigeon Pea (leaves)	49.14	6.03	6.65	74.60	1.10	25.81
Pigeon Pea (stem)	63.17	14.58	11.35	37.58	3.57	22.67
Pumpkin (leaves)	17.02	9.86	13.99	110.49	3.58	33.71
U. maxima (leaves)	8.58	2.75	4.79	57.97	0.69	11.48
Pineapple (leaves)	40.46	6.78	14.48	98.85	2.78	17.64
Quenepa (leaves)	18.68	8.33	5.25	34.78	1.06	14.61

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References

ATSDR. 2001. Summary Report for the ATSDR Expert Panel Meeting on Tribal Exposures to Environmental Contaminants in Plants. Division of Health Assessment and Consultation; Atlanta, Georgia. March 23, 2001.

Department of Agriculture. 2001. Letter from Luis Colón to the Undersecretary of Agriculture concerning heavy metal tests on Vieques bovine livestock. October 17, 2001.

El Nuevo Día. 2001. They declare suitable the agricultural products in Vieques. December 13, 2001.

Category:	Environmental Data – Plants
Date of Publication:	2003
Title:	**Trace Element Composition in Forage Samples from a Military Target Range, Three Agricultural Areas, and One Natural Area in Puerto Rico**
Author(s):	Elba Díaz and Arturo Massol-Deyá
Affiliation:	University of Puerto Rico, Mayagűez
Publication Status:	Caribbean Journal of Science 2003;39(2):215–220
Peer Review Status:	Peer reviewed
Cited Previously by ATSDR:	No

Study Findings

This paper discusses the same data that the authors published in April 10, 2000 – plant samples collected February and March 2000 and analyzed for heavy metals. The paper compares concentrations found on Vieques with concentrations detected on the main island of Puerto Rico. The 2000 sampling included plant stems and leaves, but it did not include the edible portions of the plants sampled. This peer-reviewed publication did, however, provide more information on sampling, preparation, and analytical techniques than did the April 2000 report.

In addition to the 2000 sampling data, this paper included new samples of *C. cajan* (pigeon peas) leaves and fruit taken August 18, 2001 and discussed the ratio of contamination detected in the fruit to contamination detected in the leaves.

In Table 1 of the study, leaves and stems of four plant species were compared. When these findings were compared with samples from mainland Puerto Rico, the Vieques plant samples contained more lead, cobalt, and manganese, but less chromium and copper. For nickel and cadmium, results were mixed.

TABLE 1. Elemental analysis of leaf samples collected at two locations in Puerto Rico.

	Chemical element (µg/g dry weight)[1]						
	Pb	Co	Ni	Mn	Cr	Cd	Cu
Guánica, State Forest							
Calotropis procera	1.29 (1.10)	2.59 (0.00)	5.17 (1.46)	17.85 (1.10)	9.31 (0.73)	1.03 (0.00)	5.69 (1.46)
Syringodium filiforme	5.57 (1.90)	4.19 (0.06)	14.64 (4.74)	251.44 (1.39)	27.93 (1.60)	2.79 (0.04)	15.39 (4.16)
Sporobolus virginicus	0.60 (0.32)	3.11 (2.06)	5.36 (0.59)	7.54 (0.44)	7.35 (0.71)	0.79 (0.05)	2.76 (0.40)
Panicum maximum	nd	4.69 (0.16)	10.50 (2.95)	35.19 (1.24)	12.24 (3.71)	0.60 (0.85)	12.93 (2.11)
Vieques, AFWTF							
Calotropis procera	30.05 (3.63)	68.40 (9.08)	18.08 (2.50)	287.94 (3.39)	12.68 (2.82)	3.11 (2.09)	4.63 (4.25)
Syringodium filiforme	33.32 (10.77)	29.60 (5.51)	28.66 (1.58)	58.23 (1.86)	2.78 (1.33)	2.78 (1.33)	30.48 (3.63)
Sporobolus virginicus	30.45 (5.36)	34.81 (1.80)	nd	670.57 (267.85)	5.32 (0.99)	nd	nd
Panicum maximum	10.25 (1.27)	46.65 (8.91)	5.08 (3.25)	135.02 (31.72)	5.99 (0.58)	1.57 (0.14)	4.03 (3.34)

[1]Average (standard deviation; n = 2); nd = not-detectable.

Table 2 of the study compared leaves and stems of two plant species (pigeon peas and squash). As in Table 1, the Vieques samples had more lead and cobalt, but these two samples contained more cadmium and copper and less nickel and manganese.

Both sample sets showed that the Vieques plants contained more lead and cobalt. For the other metals, results were mixed depending on plant species.

TABLE 2. Trace element composition in forage from the civilian area of Vieques and mainland Puerto Rico.

	Chemical element (µg/g dry weight)[1,2]					
	Pb	Co	Ni	Mn	Cd	Cu
Mainland, Puerto Rico[3]						
Cajanus cajan	4.93 (0.28)	4.31 (0.30)	7.44 (0.56)	137.43 (6.68)	0.90 (0.13)	11.04 (0.41)
Cucurbita moschata	4.51 (1.54)	– (–)	17.43 (0.23)	113.03 (55.73)	0.65 (0.32)	21.16 (3.71)
Vieques, Puerto Rico[4]						
Cajanus cajan	29.14* (17.61)	10.42** (5.15)	7.38 (3.05)	126.32 (54.85)	1.79** (0.78)	20.58* (5.53)
Cucurbita moschata	33.08* (19.06)	7.95 (2.44)	10.32 (4.27)	92.54 (20.92)	2.34** (1.44)	29.76* (5.25)

[1]Average (standard deviation; n = 4 to 6); Dash = not available.
[2]Significant differences *$p < 0.05$; **$p < 0.10$.
[3]Mainland, includes samples from Mayagüez and Las Marías.
[4]Vieques, includes samples from Monte Carmelo, Villa Borinquen and Monte Santo.

Table 3 of the study compares concentrations detected in pigeon pea fruit and leaf samples collected August 18, 2001. For some metals, higher concentrations were found in the fruit (zinc, nickel, cadmium and cobalt). For other metals (i.e., copper and lead), higher concentrations were found in the leaves.

TABLE 3. Chemical elements in leaves and fruits of *Cajanus cajans* collected at [1]Monte Carmelo (Vieques, Puerto Rico) [µg/g dry weight].

	Cu	Zn	Ni	Pb	Cd	Co
Leaves	10.72 (6.06)	32.12 (9.95)	2.26 (0.28)	15.12 (0.59)	2.42 (0.23)	17.46 (0.95)
Fruits	8.22 (1.69)	34.00 (1.86)	4.80 (0.75)	6.35 (2.06)	3.17 (1.36)	25.75 (3.48)
Fruits/leaves[2]	0.8	1.1	2.1	0.4	1.3	1.5

[1]Samples were collected from all available plants (aprox. 20 individuals) on August 18, 2001.
[2]Fruits/leaves = 1.0, equal metal concentration in both plant tissue samples. Fruits/leaves > 1.0, higher metal concentration in the fruits. Fruits/leaves < 1.0, lower metal concentration in fruits.

How ATSDR Addressed the Study Findings

ATSDR obtained this study in May 2009. Because pigeon peas are a species Vieques residents eat, ATSDR has undertaken a review of the new pigeon pea data as part of its analysis of Vieques data (see Chapter 5).

Category:	Environmental Data – Plants
Date of Publication:	August 14, 2005
Title:	**Trace Elements Analysis in Forage Samples from a U.S. Navy Bombing Range (Vieques, Puerto Rico)**
Author(s):	Arturo Massol-Deyá, Dustin Pérez, Ernie Pérez, Manuel Berrios, and Elba Díaz
Affiliation:	University of Puerto Rico
Publication Status:	Int J Environ Res Public Health 2005;2(2):263–266
Peer Review Status:	Peer reviewed
Cited Previously by ATSDR:	No

Study Findings

This paper discussed the same data the authors published in April 10, 2000, and 2003 – plant samples collected February and March 2000 and analyzed for heavy metals. The authors note that "distinctive profiles are observed within the studied species thus reflecting differences in their physiological properties."

In addition to the 2000 sampling data, this paper included new samples of *Syringodium filiforme* (Manatee grass) taken from the Atlantic Fleet Weapons Training Facilities (AFWTF) in 2004 and from Guánica State Forest in 2003 and 2004. Concentrations of heavy metals were higher at AFWTF. The authors advanced the hypothesis of bioaccumulation through the food chain: manatee grass to crustaceans and fish to humans. A similar food chain scenario was presented for migratory birds.

The authors evaluated temporal variations of the *S. filiforme* samples. Table 2 of their study shows heavy metal concentrations in 2004 (after bombing activities stopped) were approximately ½ the concentration levels found in 2000 (when the bombing range was active). Similar results were found in the control population—plants from Guánica State Forest located approximately 120 miles from the bombing range.

Although not specifically stated by the authors, their data pointed out that "safe concentrations" detected in one species do not imply that other plant species have the same physiological properties and are also safe for consumption.

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Table 2: Elemental analysis of *Syringodium filiforme* collected at the AFWTF and Guánica State Forest

	[1]*Chemical Element (g/g dry weight)*						
	Pb	*Co*	*Ni*	*Al*	*As*	*Cd*	*Cu*
AFWTF-2001 (Active)	33.32 (10.77)	29.60 (5.51)	28.66 (1.58)	na na	na na	0.28 (0.13)	30.48 (3.63)
AFWTF-2004 (Non-active)	8.14 (3.15)	10.61 (4.21)	3.43 (2.13)	154.3 (67.7)	0.61 (0.46)	0.15 (0.15)	17.42 (1.83)
Guánica-2001	5.58 (1.90)	4.19 (0.06)	14.64 (4.75)	na na	na na	0.28 (0.01)	15.39 (4.16)
Guánica-2003/2004	2.33 (2.24)	1.82 (0.43)	3.75 (1.85)	341.2 (112.9)	1.04 (0.25)	0.28 (0.22)	12.16 (2.83)

[1]Average (standard deviation; n = 2 to 10);
na = not available.

How ATSDR Addressed the Study Findings:

Manatee grass is not a species Vieques residents eat. These data might provide ecologically important information, but they are not directly relevant to evaluating human health. ATSDR has recommended additional sampling of subsistence produce.

Category:	Environmental Data - Plants
Date of Publication:	January 2008
Title:	**Evaluación de Metales Pesados en Productos Agrícolas de una Finca con Prácticas Orgánicas en el Bo. Luján Sector Destino de Vieques (Puerto Rico).** Evaluation of Heavy Metals in Agricultural Products of a Farm with Organic Practices in the Bo. Lujan Sector Destination of Vieques (Puerto Rico) [trans.]
Author(s):	Elba Díaz de Osborne, Pablo Acevedo-Acevedo, and Arturo Massol-Deyá
Affiliation:	University of Puerto Rico
Publication Status:	Self-published
Peer Review Status:	None
Cited by Previously ATSDR:	No

Study Findings

In January 2008, researchers visited an organic farm in Vieques and collected leaves from cilantro, spinach, beans, peppers, and papaya plants. The researchers also analyzed for cadmium, chromium, copper, and lead. The researchers selected an organic farm because they believed the soil would be representative of naturally occurring soil and local airborne contamination. The researchers found concentrations of lead and cadmium in the Vieques vegetation significantly higher than concentrations detected in the reference plants in Mayagűez, a town on the western end of Puerto Rico, approximately 120 miles from Vieques.

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Table A-7. Heavy metal levels in vegetation and health guideline from 1999 study

	mg/kg, dry weight			
	Cadmium	***Chromium***	***Copper***	***Lead***
Health Guideline cited in Study*	1.0	None given	None given	None given
Vieques	0.52 – 19.50	0.98-2.69	30.18 – 112.48	3.43 – 16.43
Mayagűez	0.74 – 1.70	No samples	44.05 – 54.24	1.88 – 5.10

*Food, Safety & Health of the European Union (1999)

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Using these data, the researchers demanded that

1. The Navy compensate Vieques residents for the impact on agricultural activities: contaminated soil from Navy activities was windborne to the civilian areas of Vieques where it was taken up by agricultural plants.
2. Cleanup of the Live Impact Area (LIA) should include not only unexploded bombs, but contaminated soil as well: winds still blow contaminated soil toward the civilian area.
3. Cleanup of the Live Impact Area (LIA) should include removal of unexploded bombs in the sea adjacent to Vieques.
4. The Puerto Rico Environmental Quality Board should deny the Navy's application for a waiver to conduct open burning of vegetation: plants have taken up contamination in the soil, and burning plants would release that contamination to the air where it would ultimately reach Vieques residents.

How ATSDR Addressed the Study Findings

ATSDR requested and received this study from the author in October 2008. ATSDR found the concentrations, as reported, would be harmful to humans. Thus ATSDR consulted with the U.S. Department of Agriculture (USDA) about the study (Rufus Chaney, USDA, personal communication, October 28–30, 2008). USDA noted that

- The reported levels of copper would be phytotoxic to the plants.
- The soil might have been experimentally enhanced to increase metal uptake. The measurements reported were consistent with plants grown in soils artificially enhanced to increase metal uptake in plants.

Because of the limitations of this study, ATSDR recommended additional sampling of subsistence produce (see Chapter 5, Section 5.3.2).

Category:	Environmental Data – Pineapples and Soil
Date of Publication:	2005
Title:	**Determination of Arsenic (As), Cadmium (Cd), Chromium (Cr), Cobalt (Co) and Lead (Pb) in "Smooth Cayenne" Pineapple Fruit, Leaves Tissue and Soil Using Inductively Coupled Plasma Optical Emission Spectrometry (ICP-OES)**
Author(s):	José L. López Morales
Affiliation:	University of Puerto Rico, Mayagüez Campus
Publication Status:	Masters in Chemistry Thesis
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

In 2004, researchers from the University of Puerto Rico's Agricultural Extension Service in Vieques and the Land Authority of the Commonwealth of Puerto Rico collected study specimens. They collected a total of 72 samples of smooth cayenne pineapple fruit, leaf tissues, and soil from two pineapple plantations (one located in Barrio Luján in the civilian zone in Vieques and a control located in Barceloneta on the mainland of Puerto Rico). Inductively coupled plasma with optical emission spectrometry, together with U.S.EPA Method 200.7, were used to determine the total concentration of arsenic, cadmium, chromium, cobalt, and lead in the samples.

The author concluded that samples from neither plantation showed contamination in the pineapple fruit or leaves. In all the fruit samples, arsenic, cadmium, cobalt, and lead were either below levels of detection or not detected. No contamination was observed in the pineapples from Vieques when compared with those from the Barceloneta plantation. Higher levels of metals were found in the soil at the Barceloneta plantation, which the author attributed to the soil's natural composition. The author concluded that these results were in agreement with those of ATSDR and that all values were within accepted regulatory levels.

Table A-8. Heavy metals in pineapple and soil

Maximum Pineapple Fruit Concentration (mg/kg)					
	Arsenic	***Cadmium***	***Chromium***	***Cobalt***	***Lead***
Vieques	below LOD or ND	ND	2.3*	ND	ND
Barceloneta	below LOD or ND	ND	6.4	ND	ND
Maximum Soil Concentration (mg/kg)					
Vieques	169.7	3.9	6.7	9*	32.2
Barceloneta	268.4 or 331.4†	6.3	64.2 or 77.4†	12.7	76.5

LOD = limit of detection

mg/kg = milligrams per kilogram

ND = not detected

*Exact number not reported—value estimated from a bar graph.

†Both numbers are reported as the maximum detection.

How ATSDR Addressed the Study Findings:

As part of its analysis of Vieques data, ATSDR evaluated the level of chromium—the only metal detected—in the pineapple fruit samples (see Chapter 5, Section 5.2.3). Using a standard intake rate for fruit consumption,[36] the estimated exposure dose ATDSR calculated was below levels of health concern.

[36] About 7 ounces of fruit a day (mean total fruit intake; USEPA 1997)

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Category:	Environmental Data – Livestock
Date of Publication:	2001
Title:	**Herbivorous: Additional Evidence of Heavy Metal Mobilization through the Food Web**
Author(s):	Arturo Massol-Deyá
Affiliation:	Casa Pueblo de Adjuntas and University of Puerto Rico
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Soil Pathway Evaluation)

Study Findings

On August 18, 2001, researchers from Casa Pueblo de Adjuntas collected hair samples from goats grazing in Vieques' Mount Santos and Santa María areas. As a reference, samples were also collected from goats that pasture in the main island of Puerto Rico.

The publication reported that hair from goats grazing in Vieques contained higher levels of metals than did similar samples from goats on the main island. No mercury was detected in any goat hair (detection limit was 0.03 µg/g).

Table A-9. Metals detected in goat hair

Metals Detected in Goat Hair (µg/g)					
	Lead	***Cadmium***	***Cobalt***	***Nickel***	***Aluminum***
Main Island - Adjuntas, average	0.12	0.02	0.07	0.14	27.8
Vieques – Monte Santo, average	0.35	0.04	0.11	0.21	17.0
Vieques – Santa María, average	2.87	0.09	0.24	0.18	89.0
Monte Santo/Adjuntas	2.9	2.1	1.5	1.5	0.6
Santa María/Adjuntas	23.9	4.5	3.4	1.2	3.2
Maximum Ratio*	49.2	7.0	5.5	2.3	5.0

The authors concluded that their study confirmed the "spread of dangerous levels of contaminants through the Viequense's food web." Yet the study provided no comparison values.

Additional information about this study is found in *Science and Ecology: Vieques in Environmental Crisis*, pages 52–53, by the same authors. They noted that goats and cattle that grazed on guinea grass and that other plants consumed by humans had higher concentrations of lead and cadmium.

How ATSDR and the Puerto Rico Department of Agriculture Addressed the Study Findings

The Soil Pathway Evaluation (see pages 57–58 in the PHA) addressed this issue as a community concern. Because this and other studies by the same researchers reported elevated levels of metals in plants and livestock, on August 7, 2001, the Puerto Rico Department of Agriculture placed an embargo on Vieques produce and livestock. The Puerto Rico Department of Agriculture, in cooperation with the Farmers Association of Puerto Rico, sampled grass, fruit-bearing trees, and bovine livestock from Monte Carmelo, Martineau, Monte Santo, Esperanza, Lujan, Gubeo, and western Vieques for cadmium, cobalt, copper, lead, manganese, and nickel. the Department of Agriculture and the Farmer's Association concluded that the agricultural products from Vieques were suitable for consumption and did not contain toxic levels of these contaminants (Department of Agriculture 2001). Because the Department of Agriculture and the Farmer's Association could not duplicate Massol's results, the embargo was lifted. (El Nuevo Día 2001)

References

Department of Agriculture. 2001. Letter from Luis Colón to the Undersecretary of Agriculture concerning heavy metal tests on Vieques bovine livestock. 2001 October 17.

El Nuevo Día. 2001. They declare suitable the agricultural products in Vieques. 2001 December 13.

Category:	Environmental Data – Sea Life
Date of Publication:	December 6, 1999
Title:	**Draft Findings in Vieques, Puerto Rico (AKA Heavy Metals in Reefs Where Bombs Are)**
Author(s):	James Porter
Affiliation:	University of Georgia, Consultant to King and Spalding
Publication Status:	Letter to law firm, King and Spalding
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Fish and Shellfish Evaluation)

Study Findings

On November 26–30, 1999, Dr. Porter conducted a field survey to examine the health of the coral reefs to the south of the Live Impact Area (LIA).

The study reported on four objectives:

1. Collect samples of chemicals leaking from the bombs. The letter reported that analysis should have been available in late December, 1999.
2. Determine the origin of craters in the area. Magnetometer readings suggested that metal objects (i.e., bombs) were imbedded in the crater walls. The study concluded that detonations caused the craters.
3. Determine whether bombing occurred after 1993. Dr. Porter used draft cards data and serial numbers to identify dates. But no data or conclusions regarding when bombing occurred were presented in the letter.
4. Determine the extent of chemical contamination. Analyses were not available at the time of the letter, but visual observations were reported:

   a. Coral in contact with one of the leaking bombs was diseased and highly discolored.

   b. 100–150 barrels and one compressed gas cylinder were located near a sunken barge.

   c. 900–1000 barrels were located near a second barge that was broken in two. (The second barge was later identified as the former USS Killen, sunk during target practice.)

Dr. Porter's letter recommended

1. Identifying additional undersea areas with barrels—local fishers reported two additional areas.
2. Sampling barrels and gas cylinders to determine contents, management, and removal options.
3. Sampling coral in the area to determine the coral's age and hence the date when the barrels were sunk. [In a University of Georgia (UGA) press release dated December 13, 1999, Dr. Porter estimated the barges had sunk about 10 to 12 years previously.]

In a December 13, 1999, UGA press release, Dr. Porter reported seeing the sunken vessel and hundreds of 55-gallon drums. Dr. Porter reported these observations in a 1999 press release from the University of Georgia. This press release is no longer available at the UGA website (http://news.uga.edu/)

Note: Dr. Porter published his results in 2004. Dr. Porter then concluded that "concentrations of explosive chemicals found in fish and lobster collected in the vicinity of the USS Killen are below the EPA's Risk-Based Concentrations for commercially edible seafood."

How ATSDR Addressed the Study Findings

Dr. Porter's 1999 press release regarding leaking barrels prompted ATSDR's exposure investigation team to sample fish in Vieques coastal waters. In 2001, ATSDR and U.S.EPA collected 104 fish and 42 shellfish from six locations in and around Vieques, including the area that Dr. Porter sampled near the former USS Killen. To determine other sampling locations and species of fish to collect, ATSDR relied on research by Universidad Metropolitana, discussions with the person who had petitioned ATSDR for an assessment of public health conditions on Vieques, residents of Vieques, and information provided in the Vieques Special Commission Report. ATSDR collected grouper, snapper, parrotfish, grunt, goatfish, land crabs, queen conch, and spiny lobster. In addition, to address a specific community concern, ATSDR collected one honeycomb cowfish from a local fish market.

In 2003, ATSDR concluded the following in its Fish and Shellfish Evaluation:

- Explosive compounds were not detected in any of the edible fish and shellfish sampled from Vieques.
- Metals were detected in the fish and shellfish.
- ATSDR evaluated several consumption scenarios, including eating fish every day. ATSDR found that it is safe to eat a variety of fish and shellfish from Vieques on a daily basis, including snapper (the most desirable and commonly consumed species). ATSDR concluded in 2003 that fish and shellfish caught at any of the locations are safe to eat, including fish and shellfish from areas in and around the former LIA and USS Killen.

ATSDR is reevaluating mercury in fish and shellfish. The results from our evaluation of Vieques are in this report's Chapter 2.

Category:	Environmental Data – Crabs
Date of Publication:	January 12, 2000
Title:	**Biomagnification of Carcinogenic Metals in Crab Tissue, Vieques, Puerto Rico**
Author(s):	Arturo Massol-Deyá and Elba Díaz
Affiliation:	Casa Pueblo de Adjuntas and University of Puerto Rico, Mayagüez
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Fish and Shellfish Evaluation)

Study Findings

In November 1999, researchers from Casa Pueblo de Adjuntas, in cooperation with the Biology Department of Recinto Mayagűez at the University of Puerto Rico, sampled male fiddler crabs from Icacos Lagoon in the Live Impact Area (LIA). As a control, the researchers took samples from Puerto Mosquito, just east of Esperanza. Icacos Lagoon is adjacent to Anones Lagoon; the researchers sampled soil in Anones Lagoon on February 12, 2000. The purpose of the research was to assess the potential transport of metals from soil in the LIA to other ecosystems.

At each location, researchers collected close to 35 fiddler crabs. They analyzed the extremities (i.e., levers and legs) separately from the body (i.e., shell and internal contents) for cadmium, chromium, cobalt, copper, lead, manganese, nickel, and zinc.

The researchers then compared the levels of the heavy metals detected in fiddler crabs in Icacos Lagoon with the levels in the soils. They reported that with regard to cadmium, biomagnification was occurring; cadmium concentrations in the fiddler crabs were 10–20 times higher than in the soils.

The researchers further reported that the average cadmium concentration (8.05 µg/g) of crab bodies (i.e., shell and internal contents) exceeded the World Health Organization's (WHO) weekly maximum allowable concentration (0.007 µg/g) (approximately 60 µg/person/day for a 60 kg person) and the U.S. Food and Drug Administration's (USFDA) daily critical concentration for persons older than 2 years (6 µg/g).

Table A-10. Metals detected in fiddler crabs

Metals detected in fiddler crabs (µg/g dry weight)								
Location-Sample	***Copper***	***Manganese***	***Zinc***	***Lead***	***Chromium***	***Cadmium***	***Nickel***	***Cobalt***
Puerto Mosquito – extremities	49.94	86.56	62.99	n/d	25.48	1.74	17.15	12.17
Icacos Lagoon - extremities	51.18	37.17	61.91	n/d	23.07	4.77	7.02	9.82
Puerto Mosquito - bodies	381.75	199.96	120.80	n/d	40.27	2.24	21.81	10.32
Icacos Lagoon - bodies	499.91	97.75	75.59	n/d	40.70	8.05	18.95	35.69

nt detected/d = no

How ATSDR Addressed the Study Findings

Dr. Massol's study established that fiddler crabs contained evidence of heavy metals. The study raised the question of what metal concentrations would be found in the crabs humans eat.

But the study could not answer that question. It involved fiddler crabs, which humans do not eat. And the study did not separate the crab meat and shell—his analysis was of the shell and body contents combined.

Dr. Massol's report of high fiddler crab contamination did prompt ATSDR to sample land crabs, which humans do eat. In 2001, during the Fish and Shellfish Evaluation, ATSDR and U.S.EPA collected land crabs from three locations on Vieques, including the former LIA (see Sections IV and V and Appendix D of the PHA). No explosives compounds were detected in the edible portions of land crabs from any of the locations. Although metals were detected in land crabs, the levels were too low to be of health concern for people eating them.

ATSDR also reviewed the U.S. Fish and Wildlife Service's (USFWS) 2001 ecological study in the Fish and Shellfish Evaluation of land crabs and fiddler crabs from west Vieques (see pages 12–13 of the 2003 PHA). The USFWS research established that fiddler crabs and land crabs contained some heavy metals and pesticides. Because the USFWS samples were analyzed as whole body, the data from the report were useful to evaluate ecological contamination, but could not be easily converted to evaluate human health.

In 2006, an ATSDR health consultation assisted the USFWS to determine whether selected refuge areas could be opened to harvest land crab. In 2005, NOAA sampled 74 land crabs from 14 locations, analyzing them for explosive compounds, PCBs, organochlorine pesticides, and trace elements:

- The levels found in the land crabs were much lower than levels the scientific literature reported as causing harmful health effects.

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- Explosive compounds were not detected in any crab sample.
- No association was found between sampling location and land crab contaminant levels.

Category:	Environmental Data – Sea Grass
Date of Publication:	June 2000
Title:	**Evaluación Preliminar de las Condiciones Químicas y Físicas de las Hierbas Marinas *Thalassia testudium* y *Syringodium filiforme* en Vieques.** Research on the Physical and Chemical Conditions of Sea Grasses *Thalassia testudium* and *Syringodium filiforme* in Vieques [trans.]
Author(s):	Fernando L. Herrera, Brenda Alicea Lopez, Blanca Díaz Perez, Siomara Cardona Vilella, Neritza Guerra Villanueva, and Maria C. Ortiz Rivera
Affiliation:	Metropolitan University, School of Environmental Affairs; Universidad de Puerto Rico, Arecibo
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

The survey's purpose was to perform a demographic and morphological assessment of *Thalassia testudium* and identify and determine the heavy metal content in *Thalassia* and *Syringodium* tissues. In sea grasses around Vieques, Dr. Herrera found "high concentrations" of aluminum, arsenic, iron, nickel, zinc, cadmium, cobalt, and lead. He also noted a close relation between past military activities and the pollution found in Vieques marine plants.

How ATSDR Addressed the Study Findings

Dr. Herrera's report of heavy metals in sea grass—a substance humans do not eat—prompted ATSDR to sample conch, which humans do eat and which are sea-grass habitués. In 2001, ATSDR and U.S.EPA

collected 20 conchs from four sea grass beds around Vieques—north of the LIA, near the USS Killen, south of Esperanza, and west of the Monte Pirata Conservation Zone. These locations were chosen to represent productive fishing areas around the island. The conch meat was analyzed for explosive compounds and heavy metals:

- No explosive compounds were detected in conch from any of the locations.
- Metals were detected in conch, but those levels were too low to be of health concern for people who eat conch.

Category:	Environmental Data – Fish
Date of Publication:	June 2000
Title:	**Estudio Toxicológico de Metales Pesados en Poblaciones de Peces en la Isla de Vieques.** Toxicological Study of Heavy Metals in Fish Populations at Vieques Island [trans.]
Author(s):	Doris Caro PhD, Mei-Ling Nazario, and Noel Díaz
Affiliation:	Metropolitan University
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Fish and Shellfish Evaluation)

Study Findings

Between December 1999 and April 2000, researchers from Metropolitan University collected fish from fish markets on the northern and southern coasts of Vieques and from the Parguera fish market in Lajas on the western side of the Puerto Rico mainland.

To determine fish consumption rates and which species to sample, researchers used a questionnaire to ask Vieques residents about their dietary habits. Fifty-one residents responded.

The researchers concluded that “based on the data obtained . . . we were not able to verify our hypothesis of potential bioaccumulation in the fish . . . there is no clear relationship between fish weight and size and their metal content.”

In the 78 fish samples analyzed, no cadmium was detected, and no lead was detected in the fish fillets. But the authors did report “high concentrations” of arsenic, mercury, selenium, and zinc.

Table A-11. Heavy metals in market fish

Heavy Metals in Market Fish—Fish Fillet Samples (ppm)						
	Lead	***Cadmium***	***Arsenic***	***Mercury***	***Selenium***	***Zinc***
Vieques, north	n/d	n/d	0.325-1.676	0.019-0.048	0.123-1.290	1.2-9.1
Esperanza	n/d	n/d	0.290-0.658	0.022-0.048	0.168-0.713	3.3-8.2
Parguera, Main Island	n/d	n/d	0.000-2.236	0.000-0.008	0.103-0.502	0.7-5.9

n/d, none detected

Cadmium detection limit, 0.005 ppm

Lead detection limit, 0.050 ppm

How ATSDR Addressed the Study Findings

In the 2003 Fish and Shellfish Evaluation (see pages 11–12 of the PHA), ATSDR evaluated whether the concentrations reported by the authors would result in harmful health effects for people consuming fish. ATSDR determined at the time that it was safe to eat fish from the sampled fish markets on a daily basis (i.e., all of the concentrations reported by Universidad Metropolitana were too low to be of health concern). The study did, however, provide valuable information on dietary habits: specifically, how often Vieques Islanders ate fish and what species they ate. ATSDR used that dietary information throughout the PHA.

In Chapter 2 of this report, ATSDR uses information from Dr. Caro's survey and its own fish data to evaluate local fish consumption. The conclusions and recommendations from this evaluation are presented in Chapters 2 and 9.

Category:	Environmental Data – Sea Life
Date of Publication:	March 8, 2004
Title:	**Radiological, Chemical, and Environmental Health Assessment of the Marine Resources of the Isla de Vieques Bombing Range, Bahia Salina del Sur, Puerto Rico**
Author(s):	James Barton and James Porter, PhD
Affiliation:	Underwater Ordnance Recovery, Inc. and University of Georgia, respectively
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

This report describes research undertaken to determine the radiological, explosive, and chemical safety of the marine coastal waters in the vicinity of the USS Killen submerged wreck site. The report provided the following findings and conclusions:

Radiological

- Normal ambient radiological readings were found around the USS Killen and around the 55 gallon drums.
- The readings indicate no health threat.
- The wreck of the USS Killen and 55 gallon drums should be left undisturbed to provide marine habitat.

Explosives

- Measurable readings of explosive residues were detected in some biota from the vicinity of the some bombs, with concentrations decreasing with increasing distance from the source.
- The readings from the decaying ordnance are localized, and pose a minimal toxicological threat to humans.
- The fish and lobster collected in the vicinity of the USS Killen are safe to eat. The concentrations of explosive chemicals are below the U.S.EPA's risk-based concentrations for commercially edible seafood.

- As increased human access and environmental leaching patterns increase the risk of detonation, spread, and bioaccumulation in the immediate vicinity of UXO and related debris, all such items located from the high water mark on shore to the bottom of Bahia Salina del Sur should be removed.

Metals

- Arsenic concentrations in fish (0.76 and 1.05 ppm) are above the U.S.EPA Risk-Based Concentration Guide (0.26 ppm).
- The authors noted that concentration of total arsenic in lobster collected from Site 1 (11.0 ppm) is within the normal range for Atlantic spiny lobster (10–20 ppm). The report text states that the arsenic levels are above the U.S.EPA Risk-Based Concentration Guideline (0.13 ppm) and may warrant a limited consumption advisory.
- Concentrations of barium, cadmium, chromium, and selenium in fish and lobster were below the U.S.EPA Risk Based Concentration Guidelines and within the ranges reported in the 2001 ATSDR fish sampling event.
- Uranium and lead were not tested for fish collected from Sites 1 and 2.

Table A-12. Sampling results of marine coastal waters

Matrix	Site 1(-fore)		Site 1(-aft)		Site 2		Site 3		Hatillo, P.R. (control)	
	As	Se	As	Se	As	Se	As	Se	As	Se
Water	2.51 µg/L	30.8 µg/L	1.15-1.38 µg/L	19.0 - 27.4 µg/L	0.93-1.02 µg/L	17.1-25.0 µg/L	1.30-1.42 µg/L	21.8-25.5 µg/L	---	13.75 µg/L
Sediment	5.24 mg/kg	<2.70 mg/kg	1.75 mg/kg	<2.70 mg/kg	3.17-5.99 mg/kg	<2.70 mg/kg	1.28-1.90 mg/kg	<2.70 mg/kg	4.22 mg/kg	<2.70 mg/kg
Drums	---	---	---	---	2.55-42.2 mg/kg	<2.70 mg/kg	---	---	---	---
Fish	0.76 mg/kg	<0.90 mg/kg	---	---	1.05 mg/kg	1.06 mg/kg	---	---	---	<0.9 mg/kg *
Coral	<0.20 mg/kg	< 2.3 mg/kg	<0.20 mg/kg	< 2.3 mg/kg	<0.20-0.91 mg/kg	<2.3 mg/kg	0.41 mg/kg	<2.3 mg/kg	<1.13 mg/kg	<2.3 mg/kg
Lobster	38.4 mg/kg	11.0 mg/kg	---	---	---	---	---	---	---	---

** Control is fish from local Kroger, Athens, GA*

Table 4.4. Summary of As and Se concentration detected in samples analyzed. The EPA Drinking Water Maximum Contaminant Level (MCL) value for As is 50 µg/L, and for Se is 50 µg/L. The EPA Risk-Based Concentration (RBC) for As in fish is 0.26 mg/kg and 20 mg/kg for Se.

How ATSDR Addressed the Study Findings

Although the data were not published until 2004, in 1999 the University of Georgia issued a press release reporting that Dr. Porter discovered leaking barrels off the LIA coast. This report prompted ATSDR's 2001 exposure investigation during which it sampled fish, crabs, lobster, and conch around Vieques. In 2001, ATSDR and U.S.EPA collected 104 fish and 42 shellfish from six locations on Vieques, including the area that Dr. Porter sampled near the former USS Killen. In 2003, ATSDR's Fish and Shellfish Evaluation evaluated data from the agency's 2001 sampling event and concluded the following:

- Explosive compounds were not detected in any of the edible fish and shellfish sampled from Vieques.
- Metals were detected in the fish and shellfish.
- ATSDR evaluated several consumption scenarios, including eating fish every day. ATSDR found that it was safe to eat a variety of fish and shellfish from Vieques on a daily basis, including snapper (the most desirable and commonly consumed species).
- Fish and shellfish caught at any of the locations were safe to eat, including from areas around the former LIA and the USS Killen.

As part of its analysis of Vieques data, ATSDR evaluated the level of mercury in fish collected from the reefs surrounding Vieques and from a fish market on the island. The results from our evaluation of Vieques can be found in Chapter 2.

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Arsenic levels in fish and shellfish are often reported as total arsenic. It is important, though, to distinguish between inorganic arsenic, which is harmful to humans, and arsensobetaine, which is an arsenic-containing compound that is not harmful to humans. Arsenobetaine is not harmful because it is not metabolized by humans and is easily and quickly excreted within 2 to 3 days of ingestion. Most of the arsenic (usually less than 1%) in fish and shellfish, such as lobster, is the non-toxic arsensobetaine (Sloth 2005, Fabris 2006, Green 2006). One particular study reported the percent inorganic levels in the spiny lobster (*Panulirus* species) was 0.01 to 0.2 percent (Peshut 2008). *Panulirus* is the genus of lobster found near Vieques and other parts of the Caribbean.

References

Sloth JJ, Larsen EH, Julshamn K. 2005. Survey of inorganic arsenic in marine animals and marine certified reference materials by anion exchange high-performance liquid chromatography-inductively coupled plasma mass spectrometry. J. Agric Food Chem 53;6011-6018.

Fabris G, Turoczy NJ, Stagnitti F. 2006. Trace metal concentrations in edible tissue of snapper, flathead, lobster, and abalone from coastal waters of Victoria, Australia. Ecotoxicology and Environmental Safety 63;286-292.

Peshut PJ, Morrison RJ, Brooks BA. 2008; Arsenic speciation in marine fish and shellfish from American Samoa. 71(3);484-492.

Greene R, and Crecelius E. 2006. Total and inorganic arsenic in mid-Atlantic marine fish and shellfish and implications for fish advisories. Integrated Environmental Assessment and Management 2(4);344-354.

Category:	Environmental Data – Sea Life
Date of Publication:	February 21, 2009
Title:	**Carcinogens Found in Marine Life in Island of Vieques in Puerto Rico**
Author(s):	James Porter, PhD
Affiliation:	University of Georgia
Publication Status:	Press Release
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

This press release announced that Dr. Porter would present the findings of his 1999 sampling event at the Second International Dialogue on Underwater Munitions on February 25–27, 2009 in Honolulu, Hawaii.

Dr. Porter's 2004 report recommended leaving the unexploded bombs in place. According to the new press release, he now recommended ordnance removal using an ordnance recovery system designed by Underwater Ordnance Recovery, Inc., and using the technical expertise of machinists at the University of Georgia instrument shop.

The press release noted that carcinogenic material was detected up to 2 meters from the bombs and quoted Dr. Porter as saying a future step will be "to determine the link from unexploded munitions to marine life to the dinner plate."

How ATSDR Addressed the Study Findings

The Centers for Disease Control and Prevention (CDC) participated in the panel presentation at the Second International Dialogue on Underwater Munitions. ATSDR considered Dr. Porter's new information as part the agency's analysis of Vieques data.

Category:	Environmental Data - Air
Date of Publication:	May 1999
Title:	**Contaminación Producida por Explosivos y Residuos de Explosivos en Vieques, Puerto Rico.** Contamination Produced by Explosives and Explosive Residues in Vieques [trans.]
Author(s):	Rafael Cruz Pérez, PE
Affiliation:	College of Engineers
Publication Status:	Dimension magazine
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2003 Public Health Assessment: Air Pathway Evaluation)

Study Findings

Dr. Cruz-Pérez modeled air quality effects resulting high explosives used at Vieques.

Dr. Cruz-Pérez concluded that

- Detonation of a single artillery shell (i.e., a 105 mm mortar projectile) could emit 400 kilograms of soil and explosive residue into the air.
- Ambient air concentrations of pollutants from military training exercises are inversely proportional to the downwind distance raised to the 1.5 power.
- A single explosion of an artillery shell would cause ambient air concentrations of particulate matter in the residential areas of Vieques to increase by 33 $\mu g/m^3$ over the course of 15.9 minutes.
- Including contributions from background, an unspecified level of military training exercises would lead to particulate matter concentrations of 197 $\mu g/m^3$ in the residential areas of Vieques.

How ATSDR Addressed the Study Findings

When ATSDR first began its evaluation of the air pathway, two dispersion modeling studies were available—one prepared by Navy contractors and the other by Dr. Cruz-Pérez, whose air modeling of particulate matter disagreed with the Navy's modeling. This prompted ATSDR to hire three modeling experts to review both studies critically. After examining the reviewers' findings, ATSDR decided to conduct an independent modeling study that did not suffer from some of the previous studies' shortcomings. Note that given the limited air sampling that occurred at Vieques during the time when the Navy used live bombs, modeling was necessary.

The Air Pathway Evaluation conclusions were based largely on ATSDR's modeling effort. The Air Pathway Evaluation also included ATSDR's evaluation of Dr. Cruz-Pérez's air modeling analysis (see pages D-11 through D-15 of the PHA).

Dr. Cruz-Pérez's article provided no hard evidence (e.g., references, equations, input parameters) to defend the estimated air emission rate of 400 kilograms of soil per single artillery shell. In Appendix D of the PHA, to estimate emissions, ATSDR used a published model and documented all input parameters and assumptions.

Dr. Cruz- Pérez based the estimated ambient air concentrations on an assumed decay rate with downwind distance. All three expert reviewers questioned Dr. Cruz-Pérez's estimated decay rate, with one expert reviewer citing peer-reviewed publications suggesting that pollutants from instantaneous plumes—like those associated with bombing activity—would decay much more quickly than Dr. Cruz-Pérez's calculations suggested. Expert reviewers identified at least one error in Dr. Cruz-Pérez's calculations, and ATSDR noted that the approach did not take into account parameters known to affect atmospheric dispersion (e.g., initial cloud height, atmospheric stability, wind speed, wind direction, mixing height). To estimate ambient air concentrations at Vieques, ATSDR used a U.S.EPA-recommended model (CALPUFF) with options for modeling "puff" (i.e., instantaneous) sources, such as the bombing events at Vieques. ATSDR's modeling predicted the air quality effects of more than 75 pollutants—not just a single pollutant. And in its PHA, ATSDR fully documented all model assumptions, input parameters, and run-time options.

By contrast, Dr. Cruz-Pérez estimated particulate matter ambient air concentrations on days when military training exercises occurred. Expert reviewers noted, however, that these estimates do not document the averaging time for the concentrations (e.g., 24-hour, annual average) nor do they specify the particle size (e.g., PM10, PM2.5, TSP), both of which are important health evaluation considerations. ATSDR's modeling analysis accounted for these shortcomings by specifying the particle sizes considered and by evaluating both acute and chronic exposure durations. The PHA documents all ambient air concentrations ATSDR estimated during the modeling effort.

Category:	Environmental Data - Water and Soil
Date of Publication:	May 11–16, 1978
Title:	**Vieques Litigation Support: Explosives Analyses of Water and Soil Samples Taken on Vieques Island, Puerto Rico**
Author(s):	JC Hoffsommer and DJ Glover
Affiliation:	Navy, Naval Surface Weapons Center, Explosives Chemistry Branch, MD
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2001 Public Health Assessment: Drinking Water Supplies and Groundwater Pathway Evaluation and 2003 Public Health Assessment: Soil Pathway Evaluation)

Study Findings

From May 11 to May 16, 1978, the Naval Surface Weapons Center (NSWC) obtained and analyzed water and soil samples from inside and outside the Vieques Island live impact area (LIA). The samples were collected 1 week after a military exercise in which live ordnance was used at the LIA. Samples were analyzed for TNT, RDX, and Tetryl. The study also analyzed for the biotransformation products of TNT: 4-amino-2, 6-dinitrotoluene, and 2-amino-4, 6-dinitrotoluene. NSWC detected no biotransformation products.

NSWC did find that concentrations of TNT and RDX were below the toxicological hazard levels suggested by the U.S. Army Medical Bioengineering Research and Development Laboratory in 1977. No guideline was given for Tetryl.

NSWC took 26 water samples: 15 within the LIA (one drinking water) and 9 in Navy-controlled areas west of LIA (one drinking water) including the Naval Ammunition Facility (NAF) and Camp García, drinking water in Isabel Segunda, and drinking water in Esperanza.

Table A-13. NSWC water sample results

Station	*Station Description*	*TNT, ppm**	*RDX, ppm*	*Tetryl, ppm*
	Health Guideline cited in study	<0.01	<0.03	NA
	Drinking Water Samples			
5	Pump house water, NAF magazine Area	Absent	0.00006	Absent
6	Drinking Water, Esperanza	Absent	0.00004	Absent
7	Drinking Water, Isabel Segunda	Absent	0.00004	0.0005
OP-1	Drinking water Cerro Matias (within LIA)			
	Surface Water Samples			
1	Magazine water run-off NAF Area (maximum detected off LIA)	<0.00004	0.001	<0.0009
19	Crater A run-off water (maximum TNT detected within LIA)	0.00003	0.00045	Absent
20	Lagoon water (maximum RDX detected within LIA)	Absent	0.0059	Absent

*ppm = parts per million

NSWC collected six soil samples on land under Navy control; no samples were taken in residential areas. Like the water samples, these samples were analyzed for TNT, RDX, Tetryl, and biotransformation products of TNT. In addition, the soil samples were analyzed for acetone extractables. No Tetryl or biotransformation products were detected.

Table A-14. NSWC soil sample results

Station	*Station Description*	*TNT, ppm**	*RDX, ppm*	*% Acetone Extractables*
	Health Guideline cited in study	<0.01	<0.03	NA
8	Soil near Bahia de la Chiva, Maneuver Area, Camp García (maximum detected TNT)	0.0060	0.0030	0.04
24	Soil from Crater b, Demolition Range #6	0.0071	0.0054	0.14
20	Lagoon soil (maximum % acetone extractables detected)	Absent	0.024	0.31

*ppm = parts per million

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How ATSDR Addressed the Study Findings

ATSDR evaluated the data from this report as part of the Drinking Water Supplies and Groundwater Pathway Evaluation (see page 32 and Appendix E of the drinking water PHA) and the Soil Pathway Evaluation (see Section IV of the PHA).

The concentrations of explosive compounds reported in both drinking water and lagoon water were well below levels considered harmful to human health. Past exposure to these compounds did not pose a public health hazard.

The drinking water samples (4) and the chemicals analyzed in this study were both limited. To arrive at its drinking water health call, ATSDR used additional data. In the drinking water PHA, Appendix B lists sampling events and Appendix C lists chemicals for which analyses were performed.

Other sampling events detected no RDX or tetryl in the drinking water supplies. The study noted, "a completely positive identification was not possible due to the extremely low concentrations found." It further noted, "if these explosives are present, the concentrations do not exceed the values reported here." The Navy laboratory that analyzed the samples had developed sensitive techniques to test for explosives in seawater and particularly to detect explosive compounds. These methods were more sensitive than the current U.S.EPA laboratory methods now considered the standard for environmental work. The reported soil levels were below comparison values and did not indicate a health hazard.

The number of soil samples (6) was limited. Thus for its soil health call, ATSDR used additional data. For example, in 1999 and 2000, the Navy (at ATSDR's request) and Servicios Científicos y Técnicos collected soil samples from areas where the protestors lived. Protestors who lived on the LIA for a year (1999–2000) endured the longest exposure to contaminants in the LIA soil. ATSDR reviewed the data and determined that the protestors were not exposed to harmful levels of soil-embedded chemicals.

Category:	Health Outcome Data – Heart Disease
Date of Publication:	January 2001
Title:	**Vibroacoustic Disease Induced by Long-Term Exposure to Sonic Booms**
Author(s):	R Torres, G Tirado, A Roman, R Ramirez, H Colon, A Araujo, F Pais, W Maciniak, J Nobrega, A Bordalo e Sa, JMC Lopo Tuna, M Alves-Pereira, NAA Castelo Branco
Affiliation:	Virtual Research Group and Center for Human Performances
Publication Status:	Draft manuscript
Peer Review Status:	None
Cited Previously by ATSDR:	Yes (2001 Vieques Heart Study Expert Panel Review)

Study Findings

In January 2001, a pilot study comparing the echocardiograms of commercial fishers in Vieques and in Ponce Playa, Puerto Rico reported large proportion of Vieques fishers with substantial valvular abnormalities and pericardial thickening—conditions not seen in Ponce Playa. The possible abnormalities noted in the Vieques fishers were attributed to "vibro-acoustic disease" (VAD), which Portuguese investigators had described in the medical literature. VAD was said to occur as the result of noise and vibrations caused by naval exercises on Vieques Island. By Presidential directive, the Department of Health and Human Services undertook an investigation of the issues raised by the study. The Department, in turn, referred this request to ATSDR, then currently investigating environmental public health issues in Vieques. The Cardiovascular Diseases Branch of Centers for Disease Control (CDC) provided considerable assistance in this investigation.

Concurrent with this request, the Ponce School of Medicine (PSM), led by President and Dean Dr. Manuel Martínez Maldonado, began a more definitive study of possible cardiac abnormalities among Vieques residents. This study sought to overcome methodological problems in the earlier pilot study. On March 29–30, 2001, scientists from ATSDR and CDC met with the PSM investigators and agreed that their review and interpretation of the findings should include an independent opinion of recognized practitioners and scientists.

How ATSDR Addressed the Study Findings

In July 2001, ATSDR, PSM, and CDC co-sponsored an expert panel to review PSM's follow-up study. The panel's principal conclusion was that neither the PSM nor the Mayo Clinic readings contained information indicating a cardiac health problem in the commercial fishers from either location. The initial report of gross valvular pathology from the pilot study was not replicated. All reviewers agreed that contrary to pilot study reports, no clinically relevant difference in pericardial thickness surfaced between Vieques and Ponce Playa subjects. Moreover, neither the PSM nor the Mayo Clinic measurements showed any subject's pericardial thickness to be larger than 2 mm—a metric the published literature considered the upper limit of normal.

The PSM study does not support findings of cardiac pathology among Vieques fishers. Because of the inability of trans-thoracic echocardiography to measure reliably the small differences found, those reported are likely due to measurement error—intrinsic to the technique, not to scientists who use it. This in all likelihood accounts for the different results obtained when Mayo Clinic readings of pericardial thickness were used in place of PSM readings.

Category:	Human Biomonitoring – Hair and Stool
Date of Publication:	circa 2000
Title:	**Investigación Científica de los Metales Tóxicos habidos en el Terreno Biológico de niños y adultos viequenses y sus efectos sobre los Minerales Nutrientes.** Scientific Investigation of Toxic Metals Present in the Biological Terrain of Vieques Children and Adults and Their Effects on Nutrient Minerals [trans.]
Author(s):	Carmen Colón de Jorge, Edgardo Santiago, John Brooks, Francisco Lopez Perez, Jaime Rivera, Rafael Valle
Affiliation:	College of Physicians and University of Puerto
Publication Status:	draft manuscript (unpublished)
Peer Review Status:	None
Cited Previously by ATSDR:	Not cited, but basis for the 2001 Hair Analysis Panel Discussion

Study Findings

In 1999 and 2000, Dr. Colón de Jorge collected hair and stool samples from Vieques residents. The doctor stated that 45–50% of these nonrandomly selected people were poisoned with mercury. Using results from stool samples, the doctor found that 3 out of the 6 children tested in Vieques had metal concentrations in stools above the reference range provided by the clinical laboratory (i.e., Doctor's Data). The doctor also reported that 5 out of 6 children tested had antimony and arsenic in stool samples that exceeded the lab's reference range. And these were children who, unlike adults, might not be exposed to metals in paint, cigarette paper, tobacco, old pots, makeup, and hair dye.

Dr. Colón de Jorge reported that 30 hair samples were collected from Vieques residents with self-reported health conditions. The doctor also concluded that 1) 50% of the people tested were contaminated with antimony compared with 29% from a control population, 2) 50% of the people tested were contaminated with arsenic compared with 29% from a control population, and 3) 50% of

the people tested had a selenium imbalance. Dr. Colón de Jorge pointed out that antimony levels were high because it was used in ballistics on the island, thus making the case that naval bombardment activity was the reason antimony levels were high in children.

Dr. Colón de Jorge also used the following statements to summarize the results of seven control patients:

- None of the seven patients showed total toxic levels of metals. Two cases had antimony above the reference range established by the laboratory compared with 29% of those investigated outside Vieques. Selenium was normal for exogenous contamination.
- Two cases had arsenic above the reference range compared with 29% of the cases outside Vieques. Selenium was normal for exogenous contamination.
- Because their selenium levels were normal and within the reference range, the seven control patients did not have endogenous heavy metals contamination.
- Dr. Colón de Jorge collected hair samples at random from seven persons in Rio Piedras, on the main island of Puerto Rico. None of the results showed toxic levels of heavy metals. By contrast, tests of 21 of 30 persons (i.e., 63%) from Vieques with known health conditions (and thus not randomly selected) detected various metals at toxic levels.
- Dr. Colón de Jorge's report contains several appendices with additional information, mostly focused on how to evaluate and interpret elemental results in hair. One of the appendices contained a Doctor's Data report entitled *Comprehensive Interpretations for Hair Elements from Al to Zn* (Quiq 1998).

How ATSDR Addressed the Study Findings

To determine the relationship between hair data and the potential for adverse health effects, ATSDR sought the assistance of external experts. In June 2001, ATSDR convened an expert panel to discuss the state of the science relating to analyzing hair for environmental exposure (ATSDR 2001). The panel comprised persons from state and federal government agencies, academia, and private practice whose expertise, interests, and experience covered a wide range of relevant technical disciplines.

The panelists agreed that

> . . . for most substances, insufficient data currently exist that would allow the prediction of a health effect from the concentration of the substance in hair. The presence of a substance in hair may indicate exposure (both internal and external), but does not necessarily indicate the source of exposure.

The panel noted that—provided external contamination can be ruled out—a relationship between contaminant concentrations in hair and any kind of measurable outcome have only been established for methylmercury and, to a limited extent, for arsenic.

The panel recognized that laboratory methods are available to measure the levels of some environmental contaminants in hair, but commented that procedures needed to be standardized to help

ensure more accurate and reliable results. They also identified several factors that limited the interpretation of even the most accurate, reliable, and reproducible laboratory results.

The community concern about hair analysis was briefly addressed in the 2001 Soil PHA (see page 58).

References

Agency for Toxic Substances and Disease Registry. 2001. Summary Report, Hair Analysis Panel Discussion: Exploring the State of the Science. June 12–13, 2001. Atlanta: Division of Health Assessment and Consultation and Division of Health Education and Promotion; 2001 December. Available from: http://www.atsdr.cdc.gov/hac/hair_analysis/ [accessed 2010 Jan 13].

Category:	Human Biomonitoring – Hair
Date of Publication:	January 29, 2002
Title:	**Heavy Metal Exposure and Disease in the Proximity of a Military Base**
Author(s):	Carmen Ortiz Roque
Affiliation:	Private practice, GINECO, San Juan, PR
Publication Status:	draft manuscript (unpublished)
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

From January 2000 to July 2001, Dr. Carmen Ortiz Roque collected hair samples from 203 Viequenses. Table A-15 shows hair data for various metals.

Table A-15. Results of metal analysis of hair samples collected from Viequenses from January 2000 to July 2001.

Metal in ppm	*% Elevated*	*Average (95% CI)*	*Females*	*Males*	*Children <10 years old*
Mercury, N= 205	33% > 1 ppm	2.07 (0 – 8.9)	2.87	1.08	1.89
Aluminum, N=145	56% > 17 ppm	25.74 (10.52-25.75)	26.45	24.18	34.94
Cadmium, N=205	26% > 0.47 ppm	0.65 (0-5.0)	0.75	0.55	Not available
Lead, N=205	2.9% > 25 ppm	8.07 (0-19.0)	4.28	12.47	Not available
Arsenic, N=205	0 > 1 ppm	0.18 (0 – 3.0)	0.141	0.233	Not available

Dr. Ortiz Roque stated that elevated levels of mercury (33%), aluminum (56%), cadmium (26%), and lead (6%) were found in the participants. Hair arsenic levels were not elevated.

Dr. Ortiz Roque further stated that 22% of women and 60% of children sampled in Vieques had mercury levels above what the United States considers the 90th percentile.

A subset of 22 Vieques matched pairs of mothers and their children 5 years or younger was analyzed further. Dr. Ortiz Roque reported a significant correlation between mercury hair levels in mothers and their children (Pearson's correlation 0.93, p = 0.0001). Average aluminum hair concentration for Viequenses was 25.74 ppm, and this concentration exceeded the upper limit of a standard human

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population. Dr. Ortiz Roque pointed out that aluminum levels in children's hair are normally higher than in adults.

The doctor concluded that the data showed Viequenses had internal exposure to heavy metal, and that the data were supported by the high percentage of women and children with hair mercury levels above the 90th percentile compared with the continental United States. While none of the women in the NHANES 1999 survey had mercury hair levels above 12 ppm, 3 of 45 Viequenses women had hair levels above 12 ppm (i.e., 15.41, 25.26, and 101.3 ppm).

Dr. Ortiz Roque concluded that the correlation between mother and offspring hair showed that mercury exposure occurred *in utero,* that Viequenses exposure to mercury was too high to be considered safe, and that mercury was used in the detonators.

How ATSDR Addressed the Study Findings

As mentioned previously, ATSDR convened a panel of experts who agreed that

> . . . for most substances, insufficient data currently exist that would allow the prediction of a health effect from the concentration of the substance in hair. The presence of a substance in hair may indicate exposure (both internal and external), but does not necessarily indicate the source of exposure.

Nevertheless, Dr. Ortiz Roque documented several mothers with elevated mercury levels in hair and her findings are described in Chapter 3. Viequenses who remain concerned about exposure to mercury, cadmium other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing.

Category:	Human Biomonitoring – Hair
Date of Publication:	2004
Title:	**Mercury Contamination in Reproductive Age Women in a Caribbean island: Vieques**
Author(s):	Carmen Ortiz Roque, Yadiris López-Rivera
Affiliation:	Private Practice, GINECO, San Juan, Puerto Rico
Publication Status:	J Epidemiol Community Health 2004;58:756–57
Peer Review Status:	Yes
Cited Previously by ATSDR:	No

Study Findings

Dr. Ortiz Roque published the mercury data from the 2001 hair study of Vieques residents. That study investigated hair mercury levels in women 16 to 49 years old who lived on the island of Vieques. The doctor compared those levels with women from San Juan and Ceiba, Puerto Rico. Women were excluded whose hair had been chemically treated within 3 months of the test. A 1.5-cm proximal hair sample was collected and analyzed by one of two licensed laboratories.

Dr. Ortiz Roque reported statistics on 41 Viequenses women together with information about seafood consumption, and compared these data with mainland Puerto Rico and the United States. Table A-15 summarizes the data. Dr. Ortiz Roque used margin of exposure (MOE) to define unsafe exposure levels.

Table A-16. Mercury hair results in women published by Dr. Ortiz Roque in 2004

	#	Age	*Mercury in ppm*			*Margin of Exposure*	*Seafood Consumption*			*% Women> RfD*
			Median	*Mean*	*90th Percentile*		*Total*	*Local*	*Local fish*	
Vieques	41	31.8	0.66	4.4	8.96	1.3	4.9	2.9	1.9	26.8
PR	45	29.9	0.38	0.4	1	12	2.8	1.1	0.7	6.6
US	702	NA	0.2		1.4	8.6	NA	NA	NA	7

Dr. Ortiz Roque's MOE approach originated in a 2001 MMWR article and with the National Research Council. The NRC recommended a 5% effect level derived from the 95% lower confidence limit of a benchmark dose (BMDL). The basis for the 5% effect level is abnormal scores on cognitive function tests in children exposed *in utero* to methylmercury. The NRC recommended that the U.S.EPA derive their Reference Dose using a BMDL of 58 ppb methylmercury in cord blood, which was identified as the 5%

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effect level from a Faroe Islands study using statistical models. The 58-ppb cord blood translates to 12 ppm in hair (Ortiz Roque 2004; NRC 2000).

Results showed that the 90th percentile mercury hair concentration in Vieques women was 8.96 ppm compared with 1 ppm in women from Puerto Rico and 1.4 ppm in women from the United States. Dr. Ortiz Roque concluded that Viequenses women of reproductive age were exposed to mercury concentrations unsafe to their developing fetuses.

How ATSDR Addressed the Study Findings

Dr. Ortiz Roque documented several mothers with elevated mercury levels in hair. Her findings are described in Chapter 3.

Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing.

References

[NRC] National Research Council. 2000. Toxicological Effects of Methymercury. Washington (DC): National Academy of Sciences, National Research Council. Pubs: National Academy Press.

Ortiz Roque C, López-Rivera Y. 2004. Mercury contamination in reproductive age women in a Caribbean island: Vieques. J. Epidemiol Comm Health 58:756–57.

Category:	Human Biomonitoring – Hair, Nails and Urine
Date of Publication:	2004 and 2006-2008
Title:	**Arsenic Detected in Urine, Nails, and Hair Study (Exact title not available)**
Author(s):	Carlos Rodríguez Sierra
Affiliation:	University of Puerto Rico, Department of Environmental Health
Publication Status:	El Vocero (newspaper article)
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

Dr. Carlos Rodríguez Sierra apparently conducted two urine arsenic studies in Vieques. The first was before 2004 and the results were presented at several public health conferences, including the 2004 8th International Symposium on Metal Ions in Biology and Medicine. The second arsenic study was from 2006 to 2008 and was reported in *El Vocero* in March 2009. Neither study has been published in a peer-reviewed journal. ATSDR has requested that Dr. Rodríguez Sierra share a manuscript or summary statistics but have not received a response. Dr. Rodriquez Sierra is with the Department of Environmental Health, Site Medical Sciences, University of Puerto Rico.

The following information comes from a March 2009 newspaper article in *El Vocero* describing arsenic exposure in Vieques residents from eating fish. The article stated that inorganic arsenic was found in a representative sample of Viequenses with high fish consumption.

Dr. Rodríguez Sierra identified two groups in Vieques: one consisted of 30 persons who consumed substantial quantities of fish. Another consisted of 22 persons who consumed very little fish. As a biomarker of short-term exposure, the researchers measured the levels of inorganic arsenic in urine. As a biomarker of long-term exposure, they measured arsenic in nails and in hair.

The article stated that the researchers measured arsenic in urine and found that, on average, levels in urine, hair and nails were generally low. Nevertheless, some people, and particularly some men, who ate fish frequently had higher concentrations of *inorganic* arsenic in their nails—an indicator of long-term exposure.

How ATSDR Addressed the Study Findings

Because the data were not available, ATSDR considered the results qualitatively in Chapter 3. Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing.

Category:	Human Biomonitoring – Blood
Date of Publication:	November 2006
Title:	**Executive Summary of the Prevalence of Heavy Metals in Vieques**
Author(s):	Juan Alsono Echanove and Luis Manuel Santiago
Affiliation:	Commonwealth of Puerto Rico, Department of Health
Publication Status:	draft manuscript
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

Beginning in 2004, the Puerto Rico Department of Health (PRDOH) conducted a two-phase, 5-year, 500 person, epidemiological study of heavy metals in residents' blood, urine, and hair. The results were presented in a draft manuscript in 2006. In the first phase, the PRDOH study conducted interviews and laboratory analysis on 1) arsenic in hair and urine, 2) nickel in hair and urine, 3) cadmium in hair and urine, 4) mercury in blood, 5) lead in blood, 6) aluminum in blood, and 7) uranium in urine. The second phase of the investigation involved collecting and analyzing blood samples from Phase I participants whose tests showed metals at toxic levels. Phase II was carried out from December 2005 to March 2006.

The PRDOH manuscript reported the main findings of this study as

- Detectable levels of at least one heavy metal were found in over 90% of the population.
- The levels of aluminum in blood, arsenic in urine, and nickel in hair were over the laboratory threshold in more than 20% of the study participants. Arsenic results in urine were for total arsenic, not inorganic arsenic.
- Geometric means for uranium in urine, mercury in blood, lead in blood, aluminum in blood, nickel in hair, and cadmium in urine were significantly higher than geometric means from the 1999 NHANES survey.
- None of the geometric means identified by the laboratory was over the laboratory's toxicity threshold.
- For arsenic, cadmium, and nickel, the use of cigarettes, consumption of seafood, and use of hair dye were identified as risk factors for levels above the laboratory threshold.

- Fifteen persons (3%) were identified with levels above the toxicity threshold for aluminum in blood (10), lead in blood (2), mercury in blood (1), and cadmium in urine (2).
- In the follow-up study on 10 of the 15 persons, only one person with levels above the toxicity threshold was identified.

The PRDOH manuscript suggested establishment of a voluntary health registry of Vieques residents. A health registry would 1) allow for systematic documentation of factors related to potential risks, 2) identify people with early signs and symptoms that may be associated with illness, and 3) facilitate early care and health service access needs.

The PRDOH manuscript pointed out that blood lead levels were unknown for the 1,000 Vieques children 6 years of age and younger. This high-risk group is more vulnerable to lead exposure than are adults. The PRDOH manuscript stated that an epidemiologist would be appointed to work towards establishing this program, which will follow CDC guidelines for case management. Whether at this time this program has been implemented is unknown.

How ATSDR Addressed the Study Findings

The PRDOH biomonitoring study provided much insight into metal exposure for Vieques residents. Some results from the PRDOH study suggested elevated levels of various metals, thus raising concerns about excessive exposure on the island. The PRDOH study is described in detail in Chapter 3. Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing.

Category:	Human Biomonitoring – Hair
Date of Publication:	None
Title:	**Title not determined**
Author(s):	Dr. John Wargo
Affiliation:	Yale University, Connecticut
Publication Status:	draft manuscript (not available)
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

Yale University's Dr. John Wargo considered analyzing data and preparing a paper about heavy metals in Viequenses hair. However, he has informed ATSDR that he no longer plans to do this analysis because of quality assurance/quality control issues with the hair data and because of problems with using hair to measure most metals.

How ATSDR Addressed the Study Findings

No study findings will be available.

Category:	Health Outcome Data
Date of Publication:	November 7, 2006
Title:	**Cancer Incidence and Mortality in Vieques 1990–2001**
Author(s):	Nayda R. Figueroa, MD, MPH, Erick Suárez, PhD, Taina De La Torre, MS, and Mariela Torres, MS
Affiliation:	Commonwealth of Puerto Rico, Department of Health
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

From 1973 to 1989, the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program in part funded the Puerto Rico cancer registry (PRCR). After 1989, the contract between the PRCR and SEER was not renewed, and until 1997, PRCR operated on local funding. In 1997, the PRCR applied for and received funding from the Centers for Disease Control and Prevention (CDC) through a cooperative agreement via the National Program of Cancer Registries. In calendar year 1998, beginning with cancer cases diagnoses, the PRCR provided data to the CDC .

In this report, cancer incidence and mortality in Vieques during 1990–2001 for the most common anatomical sites (i.e., breast, colorectal, cervical, oral cavity and pharynx, lung and bronchus, and prostate) and a few other cancers potentially associated with environmental exposures (i.e., liver and intrahepatic bile duct, kidney and renal pelvis, bladder, lymphoma and leukemia) were assessed for two periods (1990–1995 and 1996–2001). Cancers were also assessed by sex where applicable. To present 5-year cancer incidence and mortality rates is standard practice, as noted in publications such as SEER's *Cancer Statistics Review*, the North American Association for Central Cancer Registries' *Cancer in North America*, and joint publications such as the *United States Cancer Statistics*.

After age adjustments, the analysis identified increased cancer *incidence* in Vieques for some specific anatomical sites as well as in all cancers combined. Significant increases were only identified in the 1990–1995 time period. Statistically significant, age adjusted standardized incidence ratios (SIRs) with corresponding 95% confidence intervals (95% CIs) were

- All cancers for both sexes combined (SIR=1.19; 95%CI 1.02, 1.39);
- Lung and bronchus for both sexes combined (SIR=2.25; 95%CI 1.35, 3.52);

- Lung and bronchus for men (SIR=2.24; 95%CI 1.19, 3.83); and
- Prostate (SIR=1.47; 95%CI 1.03, 2.03).

After age adjustments, the analysis identified an increased cancer *mortality* in Vieques for some specific anatomical sites as well as for all cancers combined. Statistically significant, age adjusted standardized mortality ratios (SMRs) with corresponding 95% confidence intervals (95% CIs) were

- All cancers for both sexes combined, 1990–1995 (SMR=1.26; 95%CI 1.01, 1.57);
- All cancers for women, 1990–1995 (SMR=1.43; 95%CI 1.01, 1.96);
- All cancers for both sexes combined, 1996–2001 (SMR=1.25; 95%CI 1.01, 1.52);
- Breast cancer, 1990–1995 (SMR=0.26; 95%CI 0.03, 0.92); and
- Oral cavity and pharynx for men, 1990–1995 (SMR=3.96; 95%CI 1.08, 10.00).

As mentioned briefly in the document and as confirmed by Dr. Figueroa (personal communication, July, 2009), some follow up was necessary to identify cases in Vieques that were not processed uniformly with the rest of PR. One example relates to documentation of age for cases in the registry. For this assessment, on the island of Vieques all cases indicating residence had their ages confirmed. But this practice was not followed on the PR main island. If a case did not have a known age, it was omitted from any rate calculations. Given the small number of Vieques cases, the age was confirmed to ensure that all potential cases could be included.

How ATSDR Addressed the Study Findings

Health outcome data was not included in previous ATSDR reports about Vieques. This study's results are now discussed in Chapter 4.

Category:	Health Outcome Data
Date of Publication:	March 2000
Title:	**Exploratory study of health and other conditions in Vieques**
Author(s):	Yadiris Lopez, and Crisarlin Carrosquillo
Affiliation:	Scientific and Technical Services and the University of Puerto Rico Graduate School of Planning
Publication Status:	Self-published
Peer Review Status:	Unknown
Cited Previously by ATSDR:	No

Study Findings

This document describes a cross sectional study of residents in four Vieques neighborhoods—Lujan, Puerto Rico Reconstruction Administration settlement (PRRA), Esperanza, and Santa Maria. Reportedly, fieldwork for this study occurred between April and November 2000.[37] The authors attempted to include all residents in each neighborhood and allowed the head of a household—typically the matriarch—to provide proxy responses for all household members. Beyond the demographic, employment, and dietary habit information collected, the survey also gathered self-reported disease prevalence data.

Cancer, heart disease, hypertension, diabetes, and asthma were self-reported at a higher prevalence than referent data from the main island of Puerto Rico. Specifically, the reported case rates were 1.18–2.69 times the reported case rates from a 1994 Puerto Rico study. All data were self-reported or reported via proxy. No attempt was made to access medical records to validate the collected information.

The four neighborhoods were collectively described as representative of the Vieques population. Lujan had 132 reported residents with a reported age range of 5–25 years, PRRA had 306 reported residents with a reported age range of 59–70 years, Esperanza had 388 reported residents, and Santa Maria had 217 reported residents.

[37] This time frame as reported appears inconsistent with the March 2000 publication date.

How ATSDR Addressed the Study Findings

Health outcome data was not included in previous ATSDR reports about Vieques. This study's results are now discussed in Chapter 4.

Category:	Health Outcome Data
Date of Publication:	November 25, 2009
Title:	**Incidence and Mortality of Cancer in Vieques 1990–2004**
Author(s):	Nayda R. Figueroa, MD, MPH, Erick Suárez, PhD, Taina De La Torre, MS, Mariela Torres, MS, and Javier Perez, MPH,CTR
Affiliation:	Commonwealth of Puerto Rico, Department of Health
Publication Status:	Self-published
Peer Review Status:	None
Cited Previously by ATSDR:	No

Study Findings

From 1973 to 1989, the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program in part funded the Puerto Rico cancer registry (PRCR). After 1989, the contract between the PRCR and SEER was not renewed, and until 1997, PRCR operated on local funding. In 1997, the PRCR applied for and received funding from the Centers for Disease Control and Prevention (CDC) through a cooperative agreement via the National Program of Cancer Registries. In calendar year 1998, beginning with cancer cases diagnoses, the PRCR provided data to the CDC .

In this report, cancer incidence and mortality in Vieques during 1990–2004 for the most common anatomical sites (i.e., breast, colorectal, cervical, oral cavity and pharynx, lung and bronchus, and prostate) and a few other cancers potentially associated with environmental exposures (i.e., liver and intrahepatic bile duct, kidney and renal pelvis, bladder, lymphoma and leukemia) were assessed for three periods (1990–1994, 1995-1999, and 2000–2004). Cancers were also assessed by sex where applicable. To present five-year cancer incidence and mortality rates is standard practice, as noted in publications such as SEER's *Cancer Statistics Review*, the North American Association for Central Cancer Registries' *Cancer in North America*, and joint publications such as the *United States Cancer Statistics*.

After age adjustments, the analysis identified increased cancer *incidence* in Vieques for some specific anatomical sites as well as in all cancers combined. Significant increases were identified in 1990–1994 and 1995-1999. Statistically significant, age adjusted standardized incidence ratios (SIRs) with corresponding 95% confidence intervals (95% CIs) were identified for:

1990–1994

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- Lung and bronchus for both sexes combined (SIR=2.34; 95%CI 1.36, 3.75 and ;
- Lung and bronchus for men (SIR=2.43; 95%CI 1.25, 4.24);

1995–1999

- All cancers for both sexes combined (SIR=1.26; 95% CI 1.08, 1.47)
- All cancers for men (SIR=1.31; 95% CI 1.07, 1.60)
- Prostate (SIR=1.53; 95%CI 1.09, 2.09).

After age adjustments, the analysis identified an increased cancer *mortality* in Vieques for some specific anatomical sites as well as for all cancers combined. Statistically significant, age adjusted standardized mortality ratios (SMRs) with corresponding 95% confidence intervals (95% CIs) were identified for:

1990 – 1994 - Colorectal cancer for women (SMR=2.75; 95% CI 1.11, 5.67)

1995 – 1999 – All cancers for both sexes combined (SMR=1.35; 95% CI 1.08, 1.67)

2000 – 2004

- All cancers for men (SMR=1.40; 95% CI 1.07, 1.80)
- Prostate cancer (SMR=2.62; 95% CI 1.66. 3.93)

As mentioned briefly in the document and as confirmed by Dr. Figueroa (personal communication, July, 2009), some follow up was necessary to identify cases in Vieques that were not processed uniformly with the rest of PR. One example relates to documentation of age for cases in the registry. For this assessment, on the island of Vieques all cases indicating residence had their ages confirmed. But this practice was not followed on the PR main island. If a case did not have a known age, it was omitted from any rate calculations. Given the small number of Vieques cases, the age was confirmed to ensure that all potential cases could be included.

How ATSDR Addressed the Study Findings

Health outcome data was not included in previous ATSDR reports about Vieques. This study's results are now discussed in Chapter 4.

Category:	Environmental Data – Coral Reef Ecosystem
Date of Publication:	June 2002
Title:	**Ex-USS Killen Site Investigation and Biological Characterization, Vieques Island, Naval Station Roosevelt Roads, Puerto Rico**
Author(s):	Ken Deslarzes, Robert Nowajchik, and David Evans
Affiliation:	Geo-Marine, Inc
Publication Status:	Self-published
Peer Review Status:	No
Cited Previously by ATSDR:	Yes

Study Findings

In November 2001, marine scientists mapped the remains of the former USS Killen (in two pieces in Bahia Salina del Sur), and assessed the submerged aquatic vegetation, fish population, and coral population around the sunken vessel. Given the amount of superstructure missing from the Killen, the hypothesis was that the drums were used as ballast to redistribute the weight of the target ship. They also found that several of the drums had been sealed empty to provide buoyancy to certain of the ship's compartments. The submerged aquatic vegetation around the two areas was comparable to the control site and did not exhibit signs of environmental distress. The vessel supported fish and coral populations similar to the control site, and was not found to be negatively affecting the ecosystem. The study concluded that the sunken vessel was acting as a productive artificial reef habitat.

How ATSDR Addressed the Study Findings

ATSDR used the results of this study in the 2003 Fish and Shellfish Evaluation to address the Viequenses' concerns about the former USS Killen and the drums associated with it. As part the agency's evaluation of Vieques, a summary of the study was added to the section discussing the effects former military activities on Vieques have had on the surrounding marine environment (See Chapter 2, Section 2.1.4).

Category:	Environmental Data – Coral Reefs
Date of Publication:	July 2008
Title:	**A Tale of Germs, Storms, and Bombs: Geomorphology and Coral Assemblage Structure at Vieques (Puerto Rico) Compared to St. Croix (U.S. Virgin Islands)**
Author(s):	Bernhard Riegl, Ryan P. Moyer, Brian K. Walker, Kevin Kohler, David Gilliam, and Richard E. Dodge
Affiliation:	National Coral Reef Institute
Publication Status:	Journal of Coastal Research 24(4):1008–1021
Peer Review Status:	Yes
Cited Previously by ATSDR:	No

Study Findings

This study compared the variability of coral assemblages of reef zones between Vieques and St. Croix to evaluate the differences and the influence of natural and manufactured factors. The researchers established 18 sites at Vieques (12 inside the former LIA and 6 in the former EMA) and 6 at St. Croix for evaluation of coral assemblage structure. No differences in living benthic coral reef cover or composition of coral assemblages between inside and outside the LIA were found at Vieques. Also, coral assemblages at Vieques and St. Croix were similar in composition and biotic coverage. The researchers considered the lack of differentiation within the coral communities of Vieques and between Vieques and St. Croix the most pronounced outcome of the study. Because the reefs at St. Croix were not in better condition than reefs at Vieques, the authors suggest that natural disturbances (disease and storms) had a greater impact on coral communities than former military bombing activities.

How ATSDR Addressed the Study Findings

Because Viequenses are concerned about the effects that bombing activities have had on the marine environment, ATSDR reviewed this new study as part the agency's evaluation of Vieques. A summary of the study was added to the section discussing ecological impacts to the coral reefs around Vieques (see Chapter 2, Section 2.1.4).

Category:	Environmental Data – Fish and Shellfish
Date of Publication:	November 2001
Title:	**Field Data Summary, Vieques Fish Assessment, Vieques, Puerto Rico**
Author(s):	Daniel Cook, Steven Clapp, and Alan Humphrey
Affiliation:	Lockheed Martin/REAC and U.S. Environmental Protection Agency/Environmental Response Team
Publication Status:	Self-published
Peer Review Status:	No
Cited Previously by ATSDR:	Yes

Study Findings

The U.S. Environmental Protection Agency's Environmental Response Team (EPA/ERT) collected and analyzed 104 fish and 38 shellfish (representing 20 different edible species) from the coastal waters and near shore land on Vieques in July 2001. Fiddler crabs from the LIA were also collected. EPA/ERT caught fish and shellfish from five preferred fishing locations (north of the LIA, south of the LIA, south of Esperanza, north of Isabel Segunda, and west Vieques) as well as from a local fish market in Isabel Segunda. Several heavy metals were detected in the fish and shellfish. No explosive compounds were detected in the fish and shellfish, with the exception of low levels of cyclotetramethylene tetranitramine (HMX) and a chemical *similar* to cyclotrimethylene trinitramine (RDX)[38] in fiddler crabs and a chemical *similar* to RDX in one trunkfish from the fish market. Because the fiddler crabs had not been rinsed prior to analysis, the detection may have been the result of soil contamination. Explosive compounds were not detected in any of the other 142 edible fish or shellfish samples. EPA/ERT statistically determined that the chemical concentrations in fish and shellfish collected from south of the LIA were similar to those collected from west Vieques. The divers qualitatively noted that the reefs were in good condition and the fish appeared healthy. They documented the presence of unexploded ordnance to the north and south of the LIA.

[38] A conclusive identification could not be confirmed by the laboratory that conducted the analyses.

How ATSDR Addressed the Study Findings

EPA/ERT conducted the study to assist ATSDR with their evaluation of whether commonly consumed fish and shellfish from Vieques contain levels of heavy metals and explosives compounds that would adversely affect public health. The results of the study were thoroughly evaluated and discussed in the 2003 Fish and Shellfish Evaluation. As part the agency's evaluation of Vieques, a brief summary of the general reef conditions found during the study was added to the section discussing ecological impacts to the coral reefs around Vieques (See Chapter 2, Section 2.1.4).

Category:	Environmental Data – Sea Life and Sediment
Date of Publication:	May 2010
Title:	**An Ecological Characterization of the Marine Resources of Vieques, Puerto Rico Part II: Field Studies of Habitats, Nutrients, Contaminants, Fish, and Benthic Communities**
Author(s):	Bauer, L.J. and M.S. Kendal (eds.)
Affiliation:	National Oceanic and Atmospheric Administration (NOAA)
Publication Status:	Government Publication, NOAA
Peer Review Status:	Yes
Cited Previously by ATSDR:	No

Study Findings

The purpose of this study was to provide a spatially comprehensive characterization of the marine ecosystem surrounding Vieques. It builds on NOAA's previous efforts by investigating fish fauna, benthic communities, nutrient levels, and chemical contaminants in the marine environment around Vieques. The data were grouped and analyzed according to former land use. The key findings are summarized below:

- A stratified random design was used to select 75 sites for benthic and fish community surveys around Vieques. The researchers found that Vieques was similar in terms of benthic cover, total fish abundance, and biomass to other nearby locations (southwest Puerto Rico, St. Croix, and St. John). Differences in fish and benthic communities could not be conclusively linked to former land use patterns.
- A stratified random design was used to select 78 sites for sediment (lagoon and offshore) and 35 sites for coral tissue sampling. The sediment and coral tissue samples were analyzed for 150 chemical contaminants including metals, pesticides, and explosives. Overall, contaminant concentrations were below established sediment quality guidelines, sediments from lagoons typically had higher concentrations than offshore sites, and sediments had higher concentrations of trace and major elements than corals. Sediment samples analyzed for 14 explosive compounds yielded no confirmed detections. DDT (at four sites) and chromium (at one site) were detected in sediment samples above established sediment quality guidelines.

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However, no sites had contaminant concentrations that were likely to affect sediment-dwelling biota. Sediment concentrations of polycyclic aromatic hydrocarbons were significantly higher in the land-use zone that included the former Naval Ammunition Support Detachment. The concentration of cadmium was significantly higher in the former LIA.

- Water samples from 40 stratified random designed sampling stations were analyzed for nitrate nitrite, silicate, orthophosphate, ammonium, urea, total nitrogen, and total phosphorus. No evidence of anthropogenic over-enrichment of nutrients was discovered. Nutrient concentrations were generally low and similar in magnitude to those measured elsewhere in Puerto Rico. Nitrogen and phosphorus concentrations were below published threshold values considered threatening for macroalgal overgrowth on coral reef ecosystems.

The authors concluded that there was little evidence of any difference in marine resources, nutrients, or contaminants around Vieques compared to other coral reef ecosystems in Puerto Rico and the U.S. Virgin Islands. The reef ecosystem appears to be shaped primarily by regional-scale processed rather than local factors. The results of the study supported neither of the following hypotheses: 1) naval activities negatively affected the marine environments around Vieques and 2) the lack of development on two-thirds of the island was a positive influence by preventing human activities documented elsewhere to be harmful to marine environments.

How ATSDR Addressed the Study Findings

Because Viequenses are concerned about the effects that bombing activities have had on the marine environment, ATSDR reviewed this new study as part the agency's evaluation of Vieques. The results of the heavy metal sampling in sediment and coral are discussed in Chapter 1, Section 1.3.3. A summary of the study was also added to the section discussing ecological impacts to the coral reefs around Vieques (see Chapter 2, Section 2.1.4). ATSDR notes that the scope of the study was to characterize the entire marine ecosystem around Vieques; it was not intentionally directed toward characterizing the impact from bombing activities.

Appendix B Summaries of Previous ATSDR Documents

This appendix contains summaries of ATSDR's previous documents evaluating environmental data related to Vieques. The appendix also includes summaries of two panel reviews funded by ATSDR—one concerning heart echocardiograms and the other concerning elemental hair analysis.

The question is often raised of peer reviews for public health assessments. Public health assessments are generally not peer reviewed; Congress specifically directed that "all studies and results of research conducted under this section (other than health assessments) shall be reported or adopted only after appropriate peer review." See 42 U.S.C. §9604(i)(13) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund), as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA).

Although public health assessments are generally not peer reviewed, they are released for public comment. In addition, some public health assessments are in fact peer-reviewed. For those relevant Vieques public health assessments that were peer reviewed, this appendix notes the public comment date and the peer reviewers' names.

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Category:	Environmental Data – Drinking Water Supplies and Groundwater
Publication Date:	February 20, 2001: Public Health Assessment released for public comment; October 16, 2001: Final Public Health Assessment
Title:	**Isla de Vieques Bombing Range Public Health Assessment: Drinking Water Supplies and Groundwater**
Author:	ATSDR
Publication Status:	Published after public comment and peer review
Peer Review Status:	The document was technically reviewed by • Dr. Fernando Díaz-Barriga, Universidad Autonoma de San Luis Potosi, Mexico • Robert E. Faye, P.E., Robert E. Faye and Associates, Inc. • Hernan Horta Cruz, MS, Puerto Rico Department of Health (PRDOH), • Mike Glogower, U.S. Environmental Protection Agency (EPA) Region II, • Sigfredo Torres-Gonzales, U.S. Geological Survey (USGS)

A Vieques resident petitioned ATSDR to investigate whether contaminated groundwater might move from beneath the Live Impact Area (LIA) to the populated areas of Vieques. ATSDR studied the hydrogeology of the island and evaluated chemical levels in drinking water and in groundwater samples collected from Vieques wells and tanks. ATSDR thereafter concluded that the water from the current public water supply system is safe to drink.

Most Vieques residents receive their drinking water through an underwater pipeline from the Puerto Rico mainland. In 1999 and 2000, the U.S. Environmental Protection Agency, the Puerto Rico

Department of Health, and the U.S. Navy tested the public water supply system drinking water for volatile organic compounds, inorganic compounds, and explosive compounds. After an evaluation of the test results, ATSDR concluded that Navy activities did not affect the public drinking water supply and that the water was safe to drink.

The water from most groundwater wells on the island is safe to drink

In the past, residents drew from groundwater wells in the Esperanza and Resolucion valleys. Smaller private wells in the residential areas also supplied drinking water. Many such wells currently provide supplementary drinking water whenever the mainland public water supply system is interrupted.

From 1995 to 2000, the U.S. Environmental Protection Agency, the Puerto Rico Department of Health, the U.S. Geological Survey, and the U.S. Navy sampled groundwater wells on the island for volatile organic compounds, inorganic compounds, pesticides, herbicides, polychlorinated biphenyls, and explosive compounds. Explosive compounds and their residues were not found in any of the wells. ATSDR evaluation included whether, for people drinking water from the wells, detected chemicals were at harmful levels.

ATSDR concluded that during public water supply interruptions, water from all but one of the wells was safe to drink. But ATSDR also found that the groundwater wells had naturally high sodium content—thus residents on sodium-restricted diets were advised to consider limiting their intake of water from the island's groundwater wells. One private well (Well 3-7) also showed high levels of nitrates/nitrites. Thus the water from Well 3-7 was not safe to drink, especially for children and pregnant women. The Puerto Rico Department of Health issued an appropriate advisory, and the department's staff personally informed residents that water from Well 3-7 was not safe. Given Vieques hydrogeology, ATSDR did not believe that contamination was a consequence of bombing range activities; rather, it was in all likelihood the result of area agricultural activities or septic systems.

Vieques Island's geology and topography prevent groundwater movement from the Live Impact Area into the area where groundwater wells are located

ATSDR evaluated the hydrogeology of the island to determine whether hazardous substances from the Live Impact Area could groundwater-migrate to the drinking water wells area. ATSDR determined that the wells were within isolated aquifers and therefore not geologically connected to the groundwater on the island's eastern end. In addition, between the Live Impact Area and the wells' location, the island's bedrock and westward topography sloped upward. Thus rather than migrating toward the wells, groundwater at the Live Impact Area moved slowly downhill toward the lagoons and, eventually, toward the ocean.

If good sanitation practices are followed, water from rainfall collection systems is safe to drink

Because no sampling studies characterized the quality of water in rainfall collection systems on Vieques, no site-specific sampling data were available on which to base firm conclusions. Nevertheless, if accepted sanitation practices were followed, ATSDR expected rainfall collection systems on Vieques would provide clean water that posed no health hazards.

In the past, Vieques water was safe to drink

In 1978, the Navy reported very low levels of explosive compounds in drinking water samples from Vieques. The laboratory that analyzed the water samples stated some uncertainty in the results. ATSDR reviewed those data, as well as the sampling and analytical procedures, to evaluate whether those reported detections posed a potential health hazard. To be protective of public health, ATSDR analyzed the results assuming that the explosive compounds were present. ATSDR concluded that the concentrations reported were well below harmful levels. The water did not pose a health hazard to anyone drinking it in the past. And recent analyses of drinking water samples using updated sampling and analysis methodologies did not detect any explosive-related contamination.

Category:	Environmental Data – Soil
Publication Date:	October 23, 2001 – Public Health Assessment released for public comment February 7, 2003 – Final Public Health Assessment
Title:	**Isla de Vieques Bombing Range Public Health Assessment: Soil Pathway**
Author:	ATSDR
Publication Status:	Published after public comment and peer review
Peer Review Status:	The document was technically reviewed by • David Smith, USGS • Sigfredo Torres-Gonzalez, USGS • Rufus Chaney, Ph.D., U.S. Department of Agriculture (USDA)

Community members expressed concern that contaminants generated by bombing and other Navy training activities might have been windborne from the Live Impact Area and deposited on soils of Vieques residential areas. To address this concern, ATSDR evaluated roughly 600 soil samples collected by the U.S. Geologic Survey, the Puerto Rico Department of Natural Resources, the U.S. Navy, and Servicios Científicos y Téchnicos, Inc. ATSDR analyzed the samples for metals, other inorganic compounds, and explosive compounds, and concluded that

Residents of Vieques are not exposed to harmful levels of chemicals in the soil.

ATSDR compared the levels of chemicals found in Vieques soils to levels public health professionals consider safe. ATSDR also conducted detailed analyses to determine the amount of chemicals to which people were expected to be exposed over a lifetime. The analyses showed that for either adults or children living on Vieques, incidental ingestion of soil or contact with soil would not result in harmful health effects.

The protestors who lived on the Live Impact Area for a year were not exposed to harmful levels of chemicals in the soil.

From April 1999 to May 2000, to protest the U.S. Navy's presence on Vieques, adults and children camped in the Live Impact Area. The Navy and Servicios Científicos y Téchnicos, Inc., collected soil samples from the areas where the protestors encamped. ATSDR analyzed the data and determined that for anyone incidentally ingesting or touching the soil, all of the chemicals were at levels below those that might cause harmful health effects.

In comparison with soil elsewhere in the region, some of the metals detected in Vieques soil were moderately elevated.

ATSDR compared Vieques soil quality with sediment on the Puerto Rico mainland and with soil in the United States. ATSDR found that the maximum level of some of the metals detected in Vieques soil was moderately elevated when compared with soil in Puerto Rico and in the United States. ATSDR also analyzed the chemical characteristics of soil on Vieques to determine whether metals were at unnaturally high levels. To do this, ATSDR grouped soil samples throughout the island according to their underlying rock (the geologic units), and compared the general chemical characteristics of those soils. ATSDR found that the Vieques soils were strongly influenced by the type of rock from which they were formed—in other words, soils developed on different underlying rock had different levels of metals. The levels of metals detected on Vieques were consistent with those normally found in soils underlain by the type of rock found on Vieques (e.g., volcanic rocks) and were not at levels of health concern.

The concentrations of metals in the soils of the Live Impact Area appeared moderately elevated but not at harmful levels.

According to ATSDR's analysis, Live Impact Area soils appeared to have been influenced by Navy training activities and contained elevated metals levels. ATSDR determined, however, that the metals concentrations in the soil were not at harmful levels.

ATSDR's spatial analysis showed that metals were apparently not moving from the Live Impact Area into Vieques residential areas.

ATSDR examined the soil data for spatial trends that might show movement of metals from the Live Impact Area to the residential areas of Vieques (i.e., a pattern of high to medium to low concentrations from east to west). To do this, ATSDR created spatial maps showing the locations of metal concentrations detected on Vieques. None of the spatial maps showed any patterns beginning with high concentrations in the Live Impact Area and decreasing concentrations tapering off to the western parts of the island. Thus the soil data collected from across the island indicated that contaminants from the Live Impact Area were not air-transported in substantial quantities and deposited in the island's residential areas.

Category:	Environmental Data – Fish and Shellfish
Publication Date:	November 14, 2002 – Public Health Assessment released for public comment June 27, 2003 – Final Public Health Assessment
Title:	**Isla de Vieques Bombing Range Public Health Assessment: Fish and Shellfish**
Author:	ATSDR
Publication Status:	Published after public comment and peer review
Peer Review Status:	The document was technically reviewed by • Felix Lopez, U.S. Fish and Wildlife Service (FWS) • Carlos Ramos, U.S.EPA Region II • Jerry Stober, U.S.EPA Region IV

Previous studies reported elevated levels of metals in fish and shellfish that are eaten by the residents of Vieques. To address this concern, ATSDR worked with the U.S. Environmental Protection Agency's Environmental Response Team to collect and analyze fish and shellfish from the coastal waters and near-shore land on Vieques to determine whether fish and shellfish muscle tissues contain levels of metals and explosive compounds that would be harmful to human health.

From July 16th to 20th, 2001, fish and shellfish were collected from six locations on Vieques. ATSDR decided to collect grouper, snapper, parrotfish, grunt, goatfish, blue land crab, spiny lobster, and queen conch because they were identified by several sources as types of seafood that are commonly caught and eaten. These fish and shellfish were collected from reefs and near shore areas at the following six locations: (1) north of the Live Impact Area, (2) south of the Live Impact Area, near a sunken Navy vessel, (3) south of Esperanza, (4) north of Isabel Segunda, (5) a fish market in Isabel Segunda, and (6) west of the Laguna Kiani Conservation Zone on the west end of Vieques. Fillet and muscle tissues were analyzed for metals and explosive compounds. All sampling and analysis procedures were conducted in accordance with established U.S. Environmental Protection Agency protocols.

During the sampling event, the divers noted that all sample locations supported diverse, healthy populations of marine organisms and that all reefs were in good condition. They also noted that, with very few exceptions, the organisms collected appeared to be healthy.

ATSDR reached the following conclusions:

- Explosive compounds were not detected in any of the edible fish and shellfish from Vieques.
- Metals were detected in the fish and shellfish from Vieques; however, the levels were too low to cause harmful health effects for people eating the seafood.
- It is safe to eat fish and shellfish from Vieques every day.

According to a local consumption study, almost half of the residents of Vieques eat seafood one or two times a week. However, some people responded that they eat seafood five or more times a week. To be protective of all residents, ATSDR estimated exposure by determining the amount of metals people would most likely be exposed to over their lifetime if they ate fish or shellfish every day for 70 years. ATSDR then compared the levels to those that are considered to be safe by public health professionals. ATSDR found that it is safe to eat a variety of fish and shellfish from Vieques on a daily basis.

Because of ATSDR's current evaluation of Vieques, some of these conclusions have changed. Please see Chapter 9 for new conclusions and recommendations concerning fish and shellfish.

Category:	Environmental Data – Air
Publication Date:	November 22, 2002 – Public Health Assessment released for public comment August 26, 2003 – Final Public Health Assessment
Title:	**Isla de Vieques Bombing Range Public Health Assessment: Air**
Author:	ATSDR
Publication Status:	Published after public comment and peer review
Peer Review Status:	See footnote #38

Several Vieques residents asked ATSDR if the air on the island was safe to breathe. The residents were most concerned about contaminants released into the air during the Navy's military training exercises. These concerns included the Navy's past live bombing exercises as well as the more recent practice bombing exercises. The residents also had questions about whether contaminated dusts from the bombing range might blow into their neighborhoods.

ATSDR concluded that

Given the results of ATSDR's modeling analysis, the Navy's "live bombing" exercises did not pose a health hazard to residents.

Three air-sampling studies were conducted during the time when the Navy used live bombs on Vieques. The Puerto Rico Environmental Quality Board conducted two of these studies, and the Navy conducted the other. None of the measurements in these studies found air pollution to be at levels of health concern. However, because original documentation of these studies has not been located, ATSDR could not rest its health conclusions on these studies alone.

ATSDR estimated air quality impacts from live bombs using a modeling analysis. This analysis considered nearly 100 different contaminants that are known to be released to the air when ordnance explodes. The modeling analysis found that chemicals released to the air in smoke by the bombs dispersed to extremely low levels as the smoke traveled from the bombing range toward where people live. For most contaminants, the predicted air quality impacts where residents live were so low that even highly sensitive air sampling devices would likely not be able to measure them. In the case of particulate matter, for example, emissions from live bombing exercises were predicted to account for less than 1 percent of the concentration of particulate matter currently measured in the residential areas of

Vieques. This comparison suggests that emissions from the bombing range have extremely small impacts on the air quality in the residential areas of Vieques. Based on this modeling analysis, ATSDR concluded that emissions from live bombing exercises did not cause air pollution to reach levels known to be associated with health effects.

The Navy's practice bombing exercises did not pose a health hazard to residents.

From May 2000 through May 2003, the Navy's military training exercises were conducted with so-called practice bombs. On days when practice bombs fell on the bombing range, the Puerto Rico Environmental Quality Board collected more than 50 particulate matter samples. In every sample the particulate matter levels were lower than health concern levels. In fact, in the residential part of the island no clear relationship appeared between the number of practice bombs dropped and the levels of air pollution measured. Using these observations and estimating air concentrations of other contaminants, ATSDR found that on days when practice bombing occurred, levels of air pollution did not present a health hazard to the island's residents.

Wind-blown dust from the bombing range does not pose a health hazard to residents.

The air quality effects of windborne dust are typically evaluated by measuring the levels of particulate matter in the air. The term "particulate matter" refers to solid particles and liquid droplets in the air that we breathe. For the last 3 years, the Puerto Rico Environmental Quality Board has measured levels of particulate matter at two locations in Vieques residential areas. By the time the ATSDR air evaluation was complete, nearly 450 air samples had been collected. In every measurement, the amounts of particulate matter were well below health concern levels.[39]

[39] The public health assessment in which this air sampling was discussed was presented at the Air and Waste Management Association's 96th Annual Conference and Exhibition in San Diego, June 22–26, 2003.

Category:	Environmental Data – Land Crab
Publication Date:	February 22, 2006 – Final Health Consultation
Title:	**Isla de Vieques Bombing Range Health Consultation: Land Crabs**
Author:	ATSDR
Publication Status:	Self-Published
Peer Review Status:	None

The National Oceanic and Atmospheric Administration (NOAA) asked ATSDR to determine whether eating land crabs from various Vieques locations was safe. In June 2005, NOAA collected land crabs (*Cardisoma guanhumi*) from 14 locations (13 on Vieques and one on the Puerto Rico mainland). The crabs were analyzed for explosive compounds, polychlorinated biphenyls (PCBs), organochlorine pesticides, and trace elements. After reviewing the analyses, ATSDR concluded that

Land crabs are safe to eat

The levels of PCBs, organochlorine pesticides, and trace elements found in land crabs were much lower than levels reported to cause harmful health effects. The levels were also below the U.S. Food and Drug Administration (USFDA) regulatory limit for shellfish consumption. Only a few land crab samples contained PCBs and pesticides, indicating these chemicals were not widespread. ATSDR did not expect harmful health effects to occur in people who ate Vieques land crabs.

Children should not eat the land crab's internal organs

Because of the amount of copper in land crab internal organs, children who ate those internal organs could experience stomach issues. As for the land crab's muscle meat, children can safely eat that every day. Prudent public health practice would dictate, however, that children reduce their exposure to copper by not eating land crab internal organs.

Explosive compounds were not detected in any crab sample, and no association was found between land crab sampling location and contaminant levels

In some of the areas sampled, land crabs contained higher average concentrations for certain chemicals than did land crabs in other areas. As for other chemicals, no remarkable differences were found between the locations. ATSDR found that people could safely eat every day one meal of land crab from any of the sampled locations.

Category:	Human Biomonitoring - Hair
Publication Date:	June 2001
Title:	**Hair Analysis Panel Discussion: Exploring the State of the Science – June 2001**
Author(s):	• Robert Baratz, DDS, Ph.D., MD, Internal Medical Consultation Services, Inc. • Thomas Clarkson, Ph.D., University of Rochester • Michael Greenberg, MD, MPH, MCP, Hahnemann University • Michael Kosnett, MD, University of Colorado Health Sciences Center • Dan Pascal, Ph.D., Centers for Disease Control and Prevention • Sharon Seidel, Ph.D., California Department of Health Services • LuAnn White, Ph.D., Tulane School of Public Health
Publication Status:	Published by ATSDR
Peer Review Status:	Not applicable. Document is a panel discussion summary.

Executive Summary

In Atlanta, Georgia, on June 12 and 13, 2001, the Agency for Toxic Substances and Disease Registry (ATSDR) convened a seven-member panel to review and discuss the then-state of the science related to hair analysis; specifically, its use in assessing environmental exposures. ATSDR invited a cross-section of scientific experts in the fields of hair analysis, toxicology, and medicine to participate in 1½ days of

discussion on a variety of topics, including analytical methods, factors affecting the interpretation of analytical results, toxicologic considerations, and data gaps/research needs.

Background

ATSDR convened this panel in response to 1) a then-growing number of inquiries from community members looking for assistance in interpreting hair analysis results, and 2) agency interest in learning more about the utility of hair analysis when evaluating exposures and health effects at hazardous waste sites. The agency anticipated using the input received from this effort to develop guidance for agency health assessors on the use and interpretation of hair analysis data.

The general questions that ATSDR sought to answer included

- For what substances are reliable hair analysis methods available?
- When is it appropriate/inappropriate to consider hair analysis in assessing human exposures to environmental contamination?
- What if any data gaps limit the interpretation and use of hair analysis in the assessment of environmental contaminants?

This summary report presents the findings of the panel discussions. Principal discussion points are highlighted.

Discussions Overview

Panelists engaged in a series of discussions to address ATSDR's questions, pointing to several limitations with the then-state of knowledge on the usefulness of hair analysis in environmental exposure assessments. Discussions focused primarily on metals and trace elements in scalp hair. Panelists considered the distinct differences between using hair analysis to identify exposures (e.g., Is the substance reaching people? Does a competed pathway exist?) and using it to predict, diagnose, or treat disease (e.g., What do hair concentrations tell us about the likelihood of harmful health effects?). Panelists noted that the latter question pointed up the largest data gaps that then existed.

Although not required to reach consensus, the panelists did agree on the following summary statement related to the overall usefulness of hair analysis in evaluating environmental exposures:

> For most substances, insufficient data were available that would allow the prediction of a health effect from the concentration of the substance in hair. The presence of a substance in hair may indicate exposure (both internal and external), but does not necessarily indicate the source of exposure.

For what substances are reliable hair analysis methods available?

The group agreed that then-available laboratory methods could measure the levels of some environmental contaminants in hair. But to help ensure more accurate and reliable results, procedures needed to be standardized. That would include ensuring samples were collected by a trained person and would include but not be limited to establishing consistent sampling protocols, washing protocols, and

quality control/quality assurance procedures. The panel further agreed that testing should be targeted to the specific element of interest.

When is it appropriate/inappropriate to consider hair analysis in assessing human exposures to environmental contamination?

In general, panelists agreed that before determining whether hair analysis was an appropriate assessment tool, assessors should consider

1. *The exposure type and period.* To understand the likelihood that a particular substance will be in the hair at the time of testing and to identify other exposure sources (e.g., hair treatments), take exposure histories.
2. Because the growth rate of hair is on average 12 centimeters per year, the panel concluded that hair analysis is not generally useful for evaluating very recent exposures or those longer ago than 1 year. Segmental analysis of hair (i.e., looking at concentration trends along the length of the hair) may have a role in documenting exposures over time (e.g., identification of a high-dose acute exposure). But this would require consideration on a subject-, substance-, and situation-specific basis.
3. *The type of substance and its behavior in the body.* Determine the biological plausibility that a particular substance will be present in hair and whether it is a marker of external contamination.
4. The group agreed that little is known about the transfer kinetics of substances into hair.
5. *The clinical relevance of a negative or positive finding.* Determine the dose-response relationship, if any, between chemical concentrations in hair and target organ effects/illness. Without an understanding of a dose-response relationship, useful interpretations would not be possible.

The panelists agreed that a relationship between contaminant concentrations in hair and any kind of measurable outcome has only been established for two substances: methyl mercury (e.g., the relation between maternal hair levels and observed developmental neurological abnormalities in offspring) and, to a limited extent, arsenic (e.g., segmental analysis for forensic analysis)—provided external contamination can be ruled out. Unique forensic settings for other substances could, however, provide similar relationships.

The group also indicated the need to evaluate, on a substance- and exposure-specific basis, the extent to which hair analysis might be more advantageous than other biological sampling, such as blood or urine analysis.

What if any data gaps limit the interpretation and use of hair analysis in the assessment of environmental contaminants?

The group identified several factors that limited the interpretation of even the most accurate, reliable, and reproducible laboratory results. These include

- *The lack of reference (or background) ranges in which to frame the interpretation of results.* In the absence of environmental exposures, assessors need a greater understanding of what is

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expected to be in hair to determine whether detected levels are elevated as a result of environmental releases, including possible geographical or regional differences in background levels.

- *Difficulties in distinguishing endogenous (internal) from exogenous (external) contamination in hair.* Making this distinction is important in evaluating internal doses of the substance of interest. The group voiced different views on the effectiveness—before analysis—of washing hair to eliminate external contamination. Some felt that the then-current literature suggested no reliable washing method capable of separating external contamination from internal deposition of elements. One suggestion was that where possible, identifying metabolites (or other unique markers of internal exposure) for substances of interest was most helpful in distinguishing internal from external contamination.
- *A lack of understanding of how and to what extent environmental contaminants are incorporated into the hair.* At the time of the conference, little scientific information was available on the uptake or incorporation of environmental contaminants into hair. For metals or for environmentally relevant organic compounds, neither kinetic models nor metabolite data were known or fully understood.
- *The lack of correlation between levels in hair and blood and other target tissues, as well as the lack of epidemiologic data linking substance-specific hair levels with adverse health effects.* Before hair analysis results could be used as a diagnostic tool or to predict health endpoints, these correlations must be understood. The panel noted that hair analysis was not likely to play a role in evaluations of some of the more common health concerns associated with hazardous waste sites (e.g., cancer, birth defects).
- *Little information was available that was pertinent to the study of environmentally relevant organic compounds in hair.*
- The panel recommended taking advantage of what was known about hair analysis in testing for drugs of abuse.

The panelists encouraged standardization of sampling protocols and identified possible research areas. Yet hair analysis could only become a valid tool for any particular substance if research

- Could establish better reference ranges,
- Gained a better understanding of hair biology and pharmacokinetics,
- Further explored possible dose-response relationships,
- Established whether and when hair might serve as a better measure or predictor of disease than other biological samples (e.g., blood or urine), and
- Learned more about organic compounds in hair.

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Category:	Human Biomonitoring – Heart Study
Publication Date:	July 2001
Title:	**Expert Review of the Vieques Heart Study**
Author(s):	• Henry W. Blackburn, Jr., MD, University of Minnesota • Richard Devereux, MD, Cornell University • W. Dana Flanders, MD, D.Sc., MPH, Emory University • Miguel Ángel García-Fernández, MD, Hospital General Universitario Gregorio Marañón • Mauricio Hernández-Avila, MD, MPH, Ph.D., National Institute of Public Health • Elizabeth Ofili, MD, MPH, Morehouse School of Medicine • Dr. Manuel Posada de la Paz, Instituto de Salud Carlos III • Jesús Vargas-Barrón, MD, Instituto Nacional de Cardiología "Ignacio Chávez"
Publication Status:	Published by ATSDR
Peer Review Status:	Not applicable. Document is a record of peer review comments on the Ponce School of Medicine Heart Study

In January 2001, a pilot study comparing the echocardiograms of residents in Vieques and in Ponce Playa, Puerto Rico reported substantial valvular abnormalities and pericardial thickening in a large proportion of the Vieques residents—findings not seen among Ponce Playa residents. The possible abnormalities noted in the Vieques residents were attributed to "vibro-acoustic disease" (VAD), previously described in the medical literature by Portuguese investigators. VAD was said to occur as the

result of noise and vibrations caused by naval exercises on the Island of Vieques. By Presidential directive, the Department of Health and Human Services investigated the issues raised by the study. The Department, in turn, referred this request to the Agency for Toxic Substances and Disease Registry (ATSDR), which was already investigating environmental public health issues in Vieques.

Concurrent with this request, the Ponce School of Medicine (PSM) led by President and Dean Dr. Manuel Martínez Maldonado had begun a more focused study of possible cardiac abnormalities among Vieques commercial fishers. This study sought to overcome methodological problems (e.g., sampling frame, lack of blinding) found in the earlier pilot study. On March 29–30, 2001, scientists from ATSDR and CDC met with the PSM investigators and agreed to invite the assistance of recognized practitioners and scientists in reviewing and interpreting the findings. The chosen reviewers were experts with international reputations in echocardiography and environmental or cardiovascular epidemiology. Because of its extensive experience, the echocardiography "core" laboratory at Mayo Clinic, directed by Dr. Jae K. Oh, was selected to review the echocardiograms.

Eight accomplished physician-scientists accepted invitations to participate. They were principally from academic institutions: four panelists were from U.S. universities, two from Mexico, and two from Spain. Half of the panelists were specialists in cardiology and echocardiography; others were epidemiologists. Additional meeting participants included personnel or consultants of PSM and ATSDR, Dr. Jae Oh of Mayo Clinic, and Dr. John Rullán, Secretary of Health of Puerto Rico. The meeting took place July 12–13, 2001, in the Condado Plaza Hotel in San Juan. Dr. Martínez Maldonado and Dr. David Fleming, then Deputy Administrator of ATSDR, co-chaired. Participants reviewed the methods, results, and public health significance of the Vieques Heart Study, considering both PSM and Mayo Clinic data.

Dr. Martínez Maldonado and Dr. Carlos Ríos presented the Vieques Heart Study. The study objective was to determine any association between place of residence (Vieques or Ponce Playa) and morphological cardiovascular changes among commercial fishers.

Investigators sampled randomly from the lists of licensed commercial fishers from Vieques and from Ponce Playa and obtained 53 and 42 subjects from the two areas, respectively. Investigators measured height, weight, blood pressure, and other physical parameters, collected questionnaire data on demographics and possible confounders, and recorded subjects' echocardiographic images. The echocardiograms were read "blindly" (i.e., without knowledge of the site of residence of the particular subject) for pericardial thickness by a group of several experienced PSM cardiologists, with caliper placement done by consensus and by using magnified images.

By PSM's measurements, the average pericardial thickness was slightly greater among Vieques fishers than among those from Ponce Playa (1.20 mm vs. 1.05 mm), and this difference was statistically significant ($P = 0.03$). The values for pericardial thickness measured by Mayo were within the same range as those measured by PSM, but did not achieve statistical significance when Vieques and Ponce fishers were compared (0.78 mm vs. 0.82 mm, respectively).

Panel Conclusions

The panel's principal conclusion was that neither the Ponce Playa nor the Mayo readings contained information indicating a cardiac health problem in the fishers. The initial report of gross valvular pathology from the pilot study was not replicated. All reviewers agreed that no clinically relevant difference between Vieques and Ponce Playa subjects in pericardial thickness appeared, contrary to that reported in the pilot study. Moreover, neither the PSM nor the Mayo measurements showed any subject's pericardial thickness to be larger than 2 mm—a value the published literature considered in the upper limit of normal.

The PSM study got generally high marks from the panelists regarding study design and statistical analysis. The sampling frame (lists of registered fishers) was regarded as appropriate, and reviewers generally felt that the response rate was adequate. The fact that reasonably clear-cut hypotheses had been developed beforehand largely obviated concerns about the problem of multiple comparisons. In general, panelists felt that the statistical tests used were appropriately chosen and employed. Panelists noted that echocardiographic readings were performed with appropriate blinding—including masking of dates—at both PSM and Mayo.

Summary Conclusion

The PSM study did not support a finding of cardiac pathology among Vieques commercial fishers. Because of the inability of trans-thoracic echocardiography to measure reliably the small differences found, the differences reported were likely due to measurement error (intrinsic to the technique, not the scientists who used it). This fact almost certainly accounts for the different results obtained when Mayo readings of pericardial thickness replaced PSM readings.

The Vieques Heart Study represented a valuable contribution to scientific knowledge regarding the use of echocardiography. The panelists recommended the study's publication in the peer-reviewed scientific literature.

Appendix C Peer Review Comments and ATSDR Responses

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Introductory note: We submitted the same six questions to all six peer reviewers. Some of those questions included requests for comments to specific sub questions. We reproduce here each of the questions and sub questions we asked, each reviewer's comments to the questions and sub questions, and our responses to their comments. Also, throughout our responses we refer to An Evaluation of Environmental, Biological, and Health Data from the Island of Vieques, Puerto Rico as simply "the Report."

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Reviewer 1

Overall, has ATSDR's re-evaluation of public health issues associated with historical bombing exercises at Vieques been appropriate?

[Comment] The document as a whole is well written and relatively easy to follow. However, it suffers from a significant lack of consistent focus. While the ostensible question addressed by the document is whether the historical bombing exercises at Vieques have impacted public health, the document only sporadically focuses on those environmental health impacts that are specifically linked to the bombing and related military activities and often shifts focus to general environmental exposure and health considerations. Given the stated purpose of the review, one would expect that (at least for the environmental exposure/risk assessment portion of the review) there would be a logical structure that first defines the activities with a potential to impact public health, then identified the environmental contaminants that could be anticipated to result from those activities, then identified the environmental/biological media in which those contaminants are likely to be found, then presented the sampling results for those media and contaminants, and then compared those results to appropriate background data to make a determination of whether the exposures and potential exposures related to the sampling data were likely to be associated with the bombing and related activities. This logical progression should have been driven by one or two focused and explicit hypotheses to be tested. These hypotheses would take a form similar to, "The bombing and related military activities in Vieques have resulted in exposures to specific environmental contaminants above non-bombing-related background levels," and "Elevated levels of the specific bombing-related contaminants pose a significantly elevated health risk to the exposed population." While such an approach is implied in the review, it is only intermittently followed.

The review does refer to specific contaminants that were identified as resulting from the bombing activities - TNT, RDX, aluminum powder and "86 contaminants" including 15 metals that were identified in the BangBox test series. However, the relationship between the prevalence of these contaminants in the explosives and their likelihood of occurrence in environmental/biological media is not presented. This lack of prioritization not only makes hypothesis formulation and testing unclear, but also results in large uncertainty in distinguishing bombing-related contamination from background.

In contrast, the review seems to present information about environmental contamination that often appears to have no clear potential relationship with the bombing-related activities either with respect to the contaminants discussed or the spatial relationship between the impacted areas and the location of the samples. While this disconnect is not always the case in the review, it is the case sufficiently often that the reader loses the sense of a clear focus and context for the information presented. This is most notably the case in the sections on the Fish Pathway and Biomonitoring, although similar considerations also apply to the document's treatment of other pathways. For example, in the Biomonitoring and Fish sampling portions of the document, there is a large focus on mercury. Given the extent of that focus, one could conclude that mercury contamination in fish and seafood is highly anticipated to result from the bombing-related activities and is a major focus of the environmental health impact associated with

those activities. However, I have not found any place in the document where the relationship between those activities and mercury contamination in the environment in general, or in marine species in particular, is established. A more focused and useful formulation and discussion of the potential impact of the bombing-related activities on mercury/methylmercury impact on the Vieques population could proceed along the following lines:

- Is mercury/methylmercury contamination expected to be a major outcome of bombing-related activities?
- If so, where would the impact be expected?
- Are the fish that are likely to be affected by bombing-related mercury, the fish that are consumed by Vieques residents?
- What are the consumption patterns of Vieques residents relative to the likely impacted fish?
- What is the background dietary exposure to methylmercury in Vieques (i.e., from non-impacted fish) and how does it compare to the exposure from the likely impacted fish?

Given the ubiquity of at least low-level methylmercury contamination of fish worldwide, it should be anticipated that mercury would be found to some extent in all fish caught and/or consumed in Vieques. Furthermore, without a clear understanding of the background methylmercury exposure and the Vieques-specific fish consumption patterns, there is no clear way to distinguish the potential impact of bombing-related mercury exposure from the background in Vieques.

ATSDR generated few focused data specific to the assessment of bombing-related impacts in Vieques, but instead had to rely on existing data, much of which were not generated specifically for the purpose of this assessment and/or were not collected or analyzed in a systematic fashion designed for the appropriate hypothesis testing. This results in the classic situation where ATSDR largely ends up looking for its “keys” (i.e., relevant data) next to the lamppost because that’s where the light is. The quality of the overall assessment needs to be viewed in light of these limitations. Nonetheless, the use of the existing data without a clearer focus on the goal of the assessment and without a clear discrimination of the data relative to its quality and its relevance to the that goal has resulted in a document that is not clearly focused and often confusing in important parts.

> **ATSDR Response:** All reviewers commented on contaminants that could be linked to military exercises on Vieques. We therefore provided additional clarification within the summary report and provided references from one section to other sections. For example, when we discussed substances measured in fish, we included references to explosive ordnance constituents and soil measurements. We also provided some perspective about those media constituent levels we expected to be higher than others. The Report (Executive Summary and Chapters 1; 2; 3; 6; and 9) has been revised to reflect greater clarity between substances found in the Viequense environment and those associated with activities related to military exercises involving live ordnance.

ATSDR recognizes that the Report discusses both pathway-specific exposures to military exercise-related contaminants and to more general public health exposures. In that regard, ATSDR evaluated its previous work in response to continuing concerns about Viequenses' general health. The above-referenced revisions do identify possible links found between substances in the environment and military exercises. While most of our data on contaminants in soil, water, air, and seafood are relatively recent, the releases that might or might not have contributed to them are historical.

To establish conclusive links or specific pathways between the two is very challenging, except in the rare case where contaminants are uniquely associated with a site, such as RDX and TNT and bombing activities. This situation is not unique to Vieques—it is an inherent problem at any site where environmental contaminants have many potential sources, including some associated with past activities at that site.

Regarding mercury, scientists visited ATSDR in November 2009. They requested that ATSDR take another look at mercury levels in fish in light of the National Academy of Sciences' statements on mercury's harmful effects. We have added information to Chapter 2 to make this point more clearly.

Finally, ATSDR does not typically collect environmental samples. We rely on available data from other sources.

Has ATSDR adequately addressed the pathways of human exposure to bombing range-specific contaminants?

[Comment] ATSDR has identified and addressed the appropriate pathways of exposure. However, as discussed above, with obvious exceptions (e.g., TNT, RDX) it has not clearly identified those environmental contaminants that are bombing range-specific, nor has it identified the extent of exposures to contaminants in the environment that are potentially associated with bombing range activities. The exceptions to this are the air and soil contact pathways where ATSDR has made a strong case that significant exposure to bombing-related contaminants is unlikely.

ATSDR Response: We provided additional clarification within the Report and provided references from one section to other sections. We included references to explosive ordnance constituents when we discussed substances measured in fish. The Report (Executive Summary and Chapters 1; 2; 3; 6; and 9) has been revised to reflect greater clarity between substances found in the environment and those associated with military exercises involving live ordnance.

Please include specific comments on:

Consumption of seafood from reefs near Vieques

[Comment] While the very limited data on contaminants unequivocally associated with the bombing-related activities (e.g., TNT, RDX, Al) suggests that there is little or no significant accumulation of these chemicals in Vieques fish and little or no potential for accumulation, the great bulk of the assessment of consumption of bombing-related contaminants in fish is focused on mercury (Hg). As discussed above, there is no clear rationale provided for this focus. The nature and extent of the relationship between the bombing-related activities and mercury is not discussed and it is not even clear whether an accumulation of methylmercury (MeHg) in fish would be expected as a result of the bombing-related activities. I suspect that the focus on mercury in fish results more from the availability of data related to more general concerns about MeHg exposures than from a specific connection to the bombing-related activities.

> **ATSDR Response**: Because scientists who visited ATSDR in November 2009 raised mercury in fish as a health concern, ATSDR reviewed its findings for mercury in fish. The scientists were concerned that ATSDR did not consider the National Academy of Sciences' (NAS) evaluation of mercury in fish and the NAS's Reference Dose recommendations to the U.S.EPA. Moreover, residents are still concerned that the fish they eat are contaminated with mercury. We have added information to Chapter 2 to make this point more clearly.
>
> Iron, aluminum, copper, manganese, zinc, and lead are the metals that are most likely to be elevated in LIA soils from bombing activity, though other metals were also found in the bombs. It should be noted that all of these metals are also naturally present in the soils and rocks of Vieques (ATSDR 2003a, Learned 1973, USGS 1997 and 2001).
>
> We also associated aluminum, iron, copper, and explosive compounds in some LIA-vicinity seafood with LIA-related military exercises. But these associations did not include mercury or other substances. Nonetheless the Report (Executive Summary and Chapters 1, 2, 3, 6, and 9) has been revised to clarify any relationship between substances found in the Viequense environment and those associated with military exercises involving live ordnance.

[Comment continued] There were few data available on the fish actually consumed by Vieques residents. The primary source of species-specific consumption data appears to be the data collected by Dr. Caro in 2000. However, those data were based on a sample size of only 51 consumers. This sample size is unlikely to be representative of the population as a whole. No information is presented about the demographic representativeness of that sample, of the geographic location of the sample relative to the locations of concern, or of the fishing habits of the subjects relative to the population of Vieques in general. Furthermore, as presented, the data provide no information about the relative consumption frequency by species or of the variability in frequency among consumers. The document states that ATSDR "used multiple information sources to identify the preferred types of fish and shellfish for collection" including the data of Dr. Caro. However, the other sources are not specified and the manner in which the data in total were used is not specified. These data gaps make it very difficult to create a

reasonable model of fish consumption in Vieques and, in turn, to assess the exposure to contaminants in fish in general in Vieques, and especially in fish potentially impacted by bombing-related activities.

ATSDR Response: ATSDR agrees with the comments about Dr. Caro's 2000 survey. This is why we supplemented Dr. Caro's data with information gathered from 1) discussions with the residents of Vieques, 2) information provided in the Vieques Special Commission Report, 3) visits to local fish markets, and 4) PRDOH's 2006 report. These discussions confirmed Dr. Caro's findings. Dr. Caro's data suggested 34% of Viequenses ate fish more than 3–4 times a week. PRDOH's more extensive and random 2004 survey supported these findings (PRDOH 2006). We added this fact to Chapter 2.

Yet to refine our exposure estimates from eating reef fish, we do need additional information. We know that a significant proportion of Viequenses eat fish regularly. Still, more information about Viequense dietary habits would be useful, such as the frequency of consumption by sex and age for

- Reef fish, ocean-going fish, and canned fish,
- Various fish species,
- Portion size, and
- Reef location.

This additional information would allow more refined dose estimates and subsequent risk evaluations. In any event, we have made changes in Chapter 2 to highlight the lack of information about frequency of fish consumption by species and that having this information would allow more refined estimates of dose and subsequent risk evaluations.

Use of biomonitoring tools to assess exposure to metals and other hazardous chemicals in blood, urine, or hair that are related to historical bombing exercises

[Comment] Biomonitoring of exposure is potentially a very powerful tool. However, the occurrence of contaminants in biological media integrates exposures from all sources. Thus, the interpretation of biomonitoring data relative to the exposure and public health implications for a specific population from a specific source is highly reliant on both the understanding of the sources of exposure and the background of the exposure from other sources in that population. The biomonitoring studies considered in the review have significant limitations for the assessment of the public health impact of the bombing-related activities. The sample sizes in the studies other than the PRDOH study were quite small (n = 30, 52), the relationship of the subjects to potential sources of the specific exposures in question were not presented, and there was no appropriate referent population from Vieques that could be used to distinguish background sources of exposure from bombing-related sources of exposure. Information about background levels of exposure were either from Puerto Rico, US-NHANES or from laboratory reference ranges. The relationship between the background exposures in the Puerto Rico and NHANEs data and those in Vieques were not discussed and the nature of the laboratory reference ranges was likewise not discussed. Furthermore, based on the ATSDR assessment of the PRDOH study, the data were not finalized and were of uncertain reliability. Elevations of biomonitoring levels relative to the mean or median levels in a dataset are to be expected due to normal statistical

variation. To interpret those elevations in an exposure context requires either a statistically valid reference population and/or a separate measure of exposure (e.g., air, soil, water, food) that is comparable in time and space to the biomonitoring data and that can be used to elucidate the exposure context of those elevated biomonitoring levels.

Much of the biomonitoring data was for mercury relative to methylmercury exposure from fish consumption. However, I was unable to discern information from those data that allowed levels of mercury in those data to be related to potential bombing-related exposure (or lack of exposures). In any fish-consuming population, one would expect to find measurable concentrations of mercury in hair and blood and it would not be surprising to find a fraction of the population with levels that exceeded the levels corresponding to the USEPA RfD. This would be the case for populations consuming store-bought fish caught in remote locations and for populations consuming locally caught fish. Elevated levels of mercury in such datasets cannot be attributed to specific sources of contamination without additional data: mercury levels in a comparable population without exposure to locally impacted fish; the specific types of fish consumed; the locations those fish were caught; and the sources of contamination in those sources.

> **ATSDR Response**: We agree that the Vieques human biomonitoring studies are limited. We also agree that using biomonitoring data can be problematic, especially when attempting to determine whether residents have been exposed to contaminants in explosive ordnance. For clarification, we have made changes in Chapter 3.
>
> We dedicated a large portion of the Report to describing mercury exposures. Many of the Report details were absent from the original PHAs. We were concerned that such disproportionate discussion of mercury might lead reviewers to conclude that we found an association between mercury and military exercises on Vieques. On the contrary, our assessment focused only on that chemical exposure scenario posing the greatest human health risk. Chemical results for soil, air, water, and biota did not reveal extensive contamination. And biomonitoring studies are unlikely to reveal whether residents were or are exposed to contaminants from explosive ordnance. We clarified these conclusions by revising Chapters 1, 2, and 3. In summary, chemical results for soil, air, water, and biota did not reveal extensive site-related contamination. Soils from the LIA, however, were shown to contain metals above typically occurring background levels and some of these metals are associated with bombing activities (e.g., iron, copper zinc, and lead). None of the levels detected in soil are harmful to humans. Although biomonitoring is unlikely to establish any link between human biomonitoring data and military contaminants on Vieques, some biomonitoring might identify unusual exposures. We agree that for some exposures, no reliable, accepted biomonitoring detection/measurement methods are available. We further agree that all biomonitoring has some limitations.

Consumption of food (produce, meat, dairy, eggs) grown on the island

[Comment] Because endogenous mercury in hair and blood almost exclusively reflects methylmercury exposure and because methylmercury exposure comes almost exclusively from fish/seafood consumption, mercury biomonitoring data (other than urine data) can be related to fish consumption

even if the additional data needed to relate that fish consumption to a specific source of mercury are not available. For other contaminants, where there is a unique or nearly unique source of exposure, positive biomonitoring results for those contaminants can provide clear evidence of exposure to those sources. However, for the biomonitoring of contaminants with multiple sources of exposure, including all of the metals reported for biomonitoring results from Vieques residents, biomonitoring data without additional exposure context provide little or no useful information about specific sources of exposure. Furthermore, to relate reliably biomonitoring levels to exposure from single source or from a combination of discrete sources, there has to be a known and established relationship between the concentration of the contaminant in the given biological medium (e.g., hair) and the exposure to that contaminant. Not all media have a valid concentration-exposure relationship for a given contaminant. Specifically, cadmium (Cd), arsenic (As), nickel (Ni), antimony (Sb) and lead (Pb) in hair are, to the best of my knowledge, not established or directly interpretable biomarkers of exposure.

For these reasons I do not believe that the biomonitoring data presented by ATSDR can be interpreted relative to exposure to produce grown on the island, much less to exposure to contaminants from the bombing-related activities through consumption of food grown on the island.

> **ATSDR Response**: ATSDR agrees with the reviewer's comment about the irrelevancy of the mercury exposure source when evaluating the association between mercury and seafood consumption. In summary, chemical results for soil, air, water, and biota did not reveal extensive site-related contamination. Soils from the LIA, however, were shown to contain metals above typically occurring background levels and some of these metals are associated with bombing activities (e.g., iron, copper zinc, and lead). None of the levels detected in soil are harmful to humans.

[Comment continued] With respect to the direct sampling of locally grown food, ATSDR correctly points out the limitations in much of the available data. These include sampling of inedible portions of food plants and sampling of non-food plants, lack of appropriate analytical standards, and lack of consideration of bioavailability. Considerable discussion is focused on the cadmium levels in pigeon peas. While I agree with ATSDR's analysis of the potential health impact of consumption of the sampled peas, that discussion is presented without a context for the results. It is unclear how, if at all, the sampled peas reflect exposure to pigeon peas island-wide, or the extent to which pigeon peas figure in the diet of the Vieques population in general. For the peas in particular and for the sampled produce in general, there is no basis for connecting data on contaminant levels in those samples to the bombing-related activity.

> **ATSDR Response**: Text in Chapter 5, Section 5.2.2 has been modified to address the reviewer's comments. At this time, no data (i.e., consumption survey or further info on intake of pigeon peas relative to total legumes) are available on consumption patterns. Thus for this preliminary analysis we used conservative assumptions that modeled high-end intake rates. Further refinement of these exposure estimates could make them more realistic and more representative of the Viequense population.
>
> ATSDR agrees with the lack of evidence to support any assertion of a viable transport pathway of metals contamination from the Live Impact Area to distant areas where food is grown. Any

detected metals are more likely to reflect other sources, such as soil background concentration or some other local source. This is an important distinction; yet the community did cite food intake as a health concern. Thus as a public health measure we used available data to conduct a conservative, preliminary evaluation of this pathway.

Exposure to air, soil, and water on Vieques

[Comment] My conclusions presented above for interpretation of biomonitoring data relative to locally grown food apply equally to exposures from air, soil, and water on Vieques.

With respect to direct assessment of the air pathway, this is the strongest section and the one most clearly relevant to assessment of the potential impact of the bombing-related activities. In lieu of real time air monitoring data, the modeling approaches evaluated by ATSDR are the best approach to assessing this pathway. The conclusions from these analyses are robust and clearly support a conclusion that the bombing-related activities did not result in a significant exposure to residential areas.

With respect to the assessment of bombing-related contaminants in soil, although the sampling of on-site soil was necessarily limited, the conclusions of little historical health risk to the on-site protestors appear reasonable. This conclusion is based on the observations that the samples were taken at relevant and representative areas, that the bombing-related activities appear to have been widespread in that part of the island, and that essentially none of the soil sampling results indicated significant risk with long or short-term exposure. With respect to the impact of the bombing-related activities on the more remote, residential areas, ATSDR is correct in pointing out that potential transport of contaminants from the bombing areas to the residential areas would only have occurred by the same pathways that would have resulted in residential inhalation exposure to those contaminants. Thus, the modeled lack of significant airborne contamination from the bombing sites at the residential areas likewise, indicates lack of deposition of those contaminants to residential soil.

With respect to the assessment of drinking water, the provision of drinking water through the pipeline system remote from the bombing locations ensures that there is no exposure through drinking water for the great majority of the Vieques population. The lack of clear data on the extent of private well water and rain collection basin use and the associated lack of data for the quality of those sources creates some uncertainty about exposure from drinking water. Nonetheless, the hydrogeologic findings regarding the direction of groundwater flow appear to preclude contamination of the relevant aquifers. However, there are no data presented to substantiate that claim and these would be helpful (e.g., groundwater flow contours).

ATSDR Response: Thank you for your comments. We have added a topographical map showing groundwater flow contours as Figure 1.3 in the Report.

Does the Vieques summary report adequately present and describe the limitations and uncertainty of assessing human exposure to bombing range-specific contaminants?

[Comment] For the most part, ATSDR appears to be aware of the limitations of the available data for assessing human exposure to the bombing-related contaminants. The consideration of the air exposure, although somewhat short on detail, appears to be both site-specific, and appropriate in scope. The drinking water data are largely straightforward and are presented with the appropriate limitations largely arising from uncertainties about the extent of private well use. For the other exposures, despite awareness of the limitations associated with those data, data of limited or doubtful relevance are presented and considered in the overall assessment. I believe that more stringent criteria for inclusion of data in the review would have been appropriate for those exposures. In addition, particularly, in the sections on fish consumption and biomonitoring, ATSDR seems to repeatedly lose sight of the distinction between exposure to contaminants in general and exposure to contaminants specifically arising from the bombing-related activities. Specific instances of these points are presented in my text-specific comments.

> **ATSDR Response**: As for the specific example of mercury, visiting scientists in November 2009 requested that ATSDR review again its findings on mercury levels in fish, especially in light of the National Academy of Sciences' statements on mercury's harmful effects. As stated, the disproportionate focus on mercury might mislead reviewers into concluding that we found an association between mercury and the military exercises on Vieques. We could not show an association. On the contrary, in our assessment we focused heavily on the chemical exposure scenario that posed the greatest risk. Chemical results for soil, air, water, and biota did not reveal any such extensive mercury contamination.
>
> Describing all the environmental and health data available for Vieques is important—even when some of the data could not be verified or validated. The hair data results were presented for numerous metals. Some scientists believed the data were valid and drew conclusions from them. We described the limitations of these data in detail, pointing out the inability to distinguish between external contact and internal ingestion for most metals, except mercury. To this extent, ATSDR has attempted to report fairly the available data and to provide insight into their usability in making health decisions. In our conclusions and recommendations the data that could not be verified or validated were either not used (e.g., metals in hair, except mercury) or were used qualitatively (e.g., arsenic in urine reported in a Webinar).
>
> All reviewers commented on Report discussions about possible links between contamination and the military exercises on Vieques. We thus provided additional clarification within the Report and provided references from one section to other sections. For example, when we discussed substances measured in fish, we included references to the explosive ordnance constituents and soil measurements. We also provided some perspective about various media constituents we expected to be higher than others. The Report (Executive Summary and

Chapters 1, 2, 3, 6 and 9) has been revised to clarify further those substances found in the environment and those associated with military exercises.

In that regard, ATSDR recognizes the Report discusses both pathway-specific exposures to explosive-ordnance contaminants and more general public health exposures. This resulted from the request ATSDR received to review its previous work in light of Viequenses' general health concerns. The revisions referenced above provide information on possible links between substances found in the environment and military exercises on Vieques. Yet to prove links or specific pathways between current detections and historical releases of contaminants from multiple potential sources is impossible. This is not a Vieques-specific problem—it is inherent at many sites where historical activities possibly resulted in environmental contamination.

Are independent studies utilized appropriately in the determination of potential health hazards?

[Comment] As indicated above, many of the independent studies were not conducted specifically to address the issue of the impact of the bombing-related activities. Some of the other studies that attempted to focus more directly on those activities were poorly designed or too limited in scope to be useful. ATSDR did not set a particularly high standard for discriminating among those studies in terms of incorporating data into its overall assessment. To some extent this is understandable given the necessity of addressing the communities concern and the limited pool of data with which to work. However, the net effect is one of a very mixed and loosely woven patch quilt of data sources. I address more specific comments below.

ATSDR response: We agree that the data are of varying quality, and we described their limitations. That said, only in very few instances did poor data underlie potential exposure assessments. Mercury in some fish—the public exposure scenario that posed the greatest health risk—was based on very high quality data. But even those data were limited.

In response to the concerns about independent studies, it is important to describe all the environmental and health data that are available for Vieques. And this is true even when some of the data might be questionable. For instance, hair data results were presented for numerous metals. Some scientists believed these data were valid and drew conclusions on them. But ATSDR described the limitations of these data in detail. We pointed out the inability to distinguish between external contact and internal ingestion for most metals, except mercury. To this extent, ATSDR has attempted to report fairly the available data and to provide insight into their usability in making health decisions. In our conclusions and recommendations, these questionable data were either not used (e.g., metals in hair, except mercury) or were used qualitatively (e.g., arsenic in urine reported in a Webinar).

On the basis of ATSDR's re-evaluation of historical bombing activities in the Vieques summary report, has ATSDR reached the appropriate conclusions and recommendations?

[Comment]

Fish pathway. I believe that the conclusions about mercury (Hg) levels by species and the health risk (relative to the methylmercury RfD) of consuming those fish at given rates of consumption are appropriate. Nonetheless, the connection between those conclusions and the impact of the bombing-related activities remains a salient gap. I therefore agree with ATSDR's conclusion that, "ATSDR cannot be certain of the extent that military activities did or did not contribute to the mercury levels found in fish." However, given this conclusion, the discussion of methylmercury (MeHg) exposure from fish consumption in Vieques is likely to be misleading as a connection to the bombing-related activities is implied by that discussion.

> **ATSDR Response**: We agree that the mercury discussions are extensive. We further agree that some readers might assume that mercury is related to explosive ordnance used during military exercises on Vieques. We clarified this possible misconception by revising Chapters 1, 2, and 3.

[Comment continued]

Biomonitoring. ATSDR did not actually reach conclusions regarding the biomonitoring or its relationship to the bombing-related activities. Other than to express caution about the use of biomonitoring using hair due to potential exogenous contamination (and not, however, because of the lack of a clear relationship between endogenous hair concentration and internal exposure as discussed above), ATSDR merely reiterated the conclusions of the individual investigators and did not attempt and overall synthesis. I believe that the appropriate conclusion is that the biomonitoring results do not permit any conclusions about exposure to the bombing-related contaminants.

> **ATSDR Response**: We agree. Biomonitoring studies do not reveal whether residents were or are exposed to contaminants from live ordnance used during military exercises. We clarified this point by revising Chapter 3.

[Comment continued]

Health Outcome Data. Here too, ATSDR did not state clear conclusions about the relationship between the health outcomes data and the bombing-related activities. I agree, however, with the ATSDR's conclusions about the methodological limitations of the Vieques disease incidence data.

> **ATSDR Response**: Thank you for your comment. ATSDR did not state clear conclusions about any potential relationship due to the various methodological limitations in the limited, historical work.

[Comment continued]

Local Produce and Livestock Pathway. I agree with ATSDR's overall conclusion for this area that, " Currently available plant and livestock data are not adequate to evaluate thoroughly the extent of

exposure to heavy metals in people who eat locally grown Vieques produce." I also agree with the conclusion that (as discussed above) the conclusions for the air pathway would appear to preclude significant off-site contamination of produce either by direct deposition onto plants, or deposition to soil followed by uptake by plants.

ATSDR Response: Thank you for your comment.

[Comment continued]

Air Pathway. I agree with ATSDR's conclusion that "…airborne contaminants from historic uses of the Vieques Naval Training Range would have been essentially nondetectable in the residential areas of Vieques and unlikely to have resulted in harmful effects."

ATSDR Response: Thank you for your comment.

[Comment continued]

Soil Pathway. I agree with ATSDR's conclusion that, "Those who occupied the LIA from 1999–2000 were not at increased risk of adverse health effects from exposure to surface soil contaminants." I also agree with the conclusion that, "Limited available data from other locations and air pathway considerations suggest that the military exercises in the LIA did not result in current contamination of residential soils with inorganic or explosive compounds at levels considered harmful," and the related conclusion that, "Modeling described in the air pathway discussion has suggested that airborne transport of contaminants during past military exercises would not have been heavy enough to have affected soils in the island's residential area." It is not clear to me that the additional qualifiers that appear in the Chapter 9 conclusions are necessary.

ATSDR Response: Thank you for your comment. Because we wanted to identify clearly all possible uncertainties regarding our evaluation, we kept the qualifiers.

[Comment continued]

Drinking Water Pathway. I agree with ATSDR's conclusion that consumption of the pipeline source of drinking water precludes exposure to drinking water contaminants from the bombing-related activities. I am somewhat confused, however, by the apparent contradiction between ATSDR's conclusion on the one hand about the direction of groundwater flow precluding groundwater contamination westward of the bombing sites, and its conclusion on the other hand that an insufficient number of wells have been sampled to draw firm conclusions about the potential impact of bombing-related activities on drinking water wells.

ATSDR Response: We added clarifying text to Chapter 8, Section 8.3.1.2. Our evaluation indicates that bombing activities on Vieques would not affect the former drinking wells. Still, limited sampling in the 1970s showed two detections of explosives; thus we could not exclude the possibility of an unknown, past mechanism. But even conceding that the contaminants might have been extant in the past, they have not been detected in recent years.

Select the appropriate category below:

List recommended changes (or reasons for not recommending)

- Recommend ( )
- Recommend with Required Changes (X)
- Not Recommended ( )

Additional Questions:

Are there any comments on ATSDR's peer review process?

No comment from the reviewer.

Are there any other comments?

Text-Specific Comments

Pg. 15, last par. - The FDA's risk benefit analysis has only been circulated in draft form and has been broadly criticized by the USEPA. While there is clearly a risk benefit trade off with respect to fish consumption and mercury (Hg), there is no clear basis for quantifying the nature of that trade-off at this time.

ATSDR Response. We agree. Thank you for the comment

[Comment continued]

Pg. 17, par. 2 - "…the authors concluded that the results show that mercury concentrations are higher in fish from markets in Vieques compared with the control population from Parquera." There is no statistical analysis presented to support this assertion. Furthermore, the comparison between Vieques and Esperanza does not indicate a likely significant difference. Without more information and contextual description, no conclusion can be drawn from these data.

ATSDR Response: This conclusion is based on averages calculated much differently than proposed by the U.S.EPA or FDA. Some of Dr. Caro's conclusions were based on two samples, analyzed at separate times, and treating a nondetected value differently from that suggested by U.S.EPA. Of the few species that had sufficient samples for comparison (hind, parrot, and grunt), only grunt had statistically higher levels of mercury in Vieques. The highest mercury level found by Dr. Caro of 0.052 ppm was found in Parguerra and this was 37% higher than the highest level found in Vieques. Complete data tables from Dr. Caro's appendix 10 have been added to Chapter 2 (see Table 2-2).

[Comment continued]

Table 2-2 - This table requires sample numbers (n's) to be valid.

ATSDR Response: We have added the sample numbers to Table 2-2.

[Comment continued]

Pg. 18, par. 1 - What is the source of the fish referred to here as "market fish?"

> **ATSDR Response**: In Chapter 2, Section 2.2.1, we clarified that the market fish in this statement refers to Vieques fish markets.

[Comment continued]

Pg. 22, par. 1 - The goal here is to characterize consumption, not the species *per se*. Therefore, discussion about combining species should have been driven by consideration of which species were most commonly consumed.

> **ATSDR Response**: Insufficient data are available to evaluate contaminant levels by species; we had to combine fish by families. In most cases, this resulted in data by genus rather than by species. Grouping fish by families did allow an evaluation of mercury levels in grunt, hind, and snapper.

[Comment continued]

Last paragraph - If, as stated "*…the data were not adequate for comparison by species,*" then why does the preceding discussion compare grunt/hind with other fish.?

> **ATSDR Response**: We have analyzed the data further and found that some species could be compared. Previously we grouped species in each family (e.g., hind, grunt, snapper) together to compare concentrations at locations. We have since determined that some species (e.g., rock hind and red hind) can be individually compared. These comparisons are described in Chapter 2, Section2.5.

[Comment continued] Pg. 26, #5 - There is no basis for this assertion. There is no LOAEL or NOAEL for MeHg

> **ATSDR Response**: We have modified the sentence.

[Comment continued]

Last paragraph - The NAS report did <u>not</u> support a lowest observed effect level (LOAEL) for MeHg. Pg. 27, par. 1 - The notion that the NAS report identified a LOAEL or (as stated here, a "lowest level known to cause harmful effects," is incorrect. The value endorsed by the NAS report and ultimately adopted by the USEPA is based on benchmark dose modeling. It is an estimate of an effect level (i.e., doubling of the population in the lowest 5% of performance) not a lowest or no-effect level. Pg. 28, par. 3 - See language in the previous comment regarding the nature of the basis for the USEPA MeHg RfD.

> **ATSDR Response**: We have modified the language in Chapter2, Section 2.2.5 to be consistent with terminology used by the NAS and USEPA.

[Comment continued]

Pg. 29, second bullet – *"… whether eating those fish and shellfish could make people sick…"* This is not the appropriate outcome or terminology – particularly with respect to MeHg. Rather, the issue is increased risk of an adverse effect.

ATSDR Response: We have changed the wording in Chapter 2, Section 2.3.1.

[Comment continued]

Pg. 34, limitations - It appears that the choice of fish samples was not based on any direct data about actual consumption by species. This should be added as a significant limitation of the sampling strategy.

ATSDR Response: We used the direct data collected by Dr. Caro and supplemented these data with information gathered from discussions with the residents of Vieques, information provided in the Vieques Special Commission Report, and visits to local fish markets.

ATSDR agrees that additional information is needed to refine exposure estimates from eating reef fish. We know that a significant proportion of Viequenses eat fish regularly. However, more information about Viequenses dietary habits would be very useful. It would be useful to know the frequency of consumption by gender and age for:

- Reef fish, ocean-going fish, and canned fish,
- Various fish species,
- Portion size, and
- Reef location.

This additional information would allow a more refined risk estimate. In any event, we have made changes to Chapter 2.

[Comment continued]

Pg. 49, par. 2 - All biological media are not equally appropriate for all analytes because of a lack of a demonstrable exposure-concentration relationship. In general, hair is considered a standard biological medium for Hg only. I am not aware of any established method for biomonitoring of Sb in hair. In NHANES, CDC quantified Sb in urine.

ATSDR Response: ATSDR agrees. While antimony levels in hair can be measured, interpreting the results is difficult. Distinctions are impossible between oral intake of antimony (internal exposure) and hair contact with antimony-containing products (external exposure).

[Comment continued]

Par. 2 - *"… Dr. Ortiz Roque's identification of three residents with hair mercury levels above 12 ppm is an important finding."* Other than possibly from a clinical standpoint, why is this an important finding? Without information on sources of exposure (e.g., canned tuna), no inference for environmental exposure can be drawn.

ATSDR Response: We agree. The findings are important from a clinical perspective but do not indicate an association to military activities on Vieques. Regarding the finding that some women

and children with mercury hair levels above a level that might cause harmful effects to a developing fetus or a young child indicates excessive mercury exposure for at least some residents. While the assumption is that this excessive exposure comes from eating fish, only follow up can confirm this. The mercury exposure might have resulted from a high frequency of eating a certain fish (e.g., canned tuna, certain reef fish, or certain ocean-going fish). The Chapter 2 dose estimates indicate that exposure to mercury can be excessive in some people who frequently eat fish that contain moderate to high levels of mercury.

[Comment continued]

Pg. 51, Table 3-2 - The summary information on the studies of mercury in blood is 3-5 years out of date.

ATSDR Response: The studies referred to in Table 3-2 for mercury are the Faroe Islands study, the Seychelles study, and the New Zealand study. These are the principal studies the National Academy of Sciences, the U.S.EPA, and ATSDR use to develop health guidelines. The studies have resulted in numerous peer-reviewed journal articles that we consulted when evaluating the toxicologic significance of the methylmercury exposure. ATSDR is not aware of more recent peer-reviewed studies.

[Comment continued]

Pg. 52, par. 5 and ff. - "…when… a person is found to have an elevated metal or chemical in a biological matrix a follow-up investigation should be considered."

Why? From an individual chemical standpoint, an elevated biomonitoring result warrants follow-up only if there is a potential for adverse effects. From a population-based standpoint, a single elevated level provides little or no useful inferential data.

ATSDR Response: ATSDR agrees that a single elevated level in one person provides very limited information when it comes to making broad statements about inferring exposure to bomb-related chemicals and elements. We have changed the text accordingly. Prudent public health practice, however, requires follow-up investigations when persons have metals in biological matrices at levels of health concern. A prime example is elevated blood lead levels.

[Comment continued]

Pg. 57 - I suggest adding the following to the end of the paragraph: "and whether the results will be informative about health risk. It is not, for example, clear that elevated levels of Al or Zn provide useful information about health risk."

ATSDR Response: We agree and have changed the text.

[Comment continued]

Pg. 69, par. 3 - In this context, "expected" has not been defined. If it is based on NHANES data, it is not clear that it is appropriate to assess the distribution of these biomarkers in a Puerto Rico, or Vieques (as opposed to U.S.) population based on NHANES data.

ATSDR Response: We reported the findings as stated in a 2006 draft manuscript prepared by PRDOH as part of a biomonitoring study in Vieques.. We cannot say what is meant by the use of

"expected." Given the study context, the PRDOH study could have meant comparison to statistics generated by NHANES. We agree with the comment that the U.S. NHANES may not be wholly applicable to a Puerto Rican or Viequense population. In Chapter 3 we discuss the need to include a control or reference population from the Commonwealth of Puerto Rico.

[Comment continued]

Pg. 73 - Table 3A-3 appears to be mislabeled based on its identification on pg. 67 where it is described as the Phase 2 results (i.e., follow-up on elevated levels in Phase 1), whereas the table, itself, is labeled as referring to the Phase 1 results.

ATSDR Response: We have corrected the numbering of Table 3A-2 (Phase 1 results) and Table 3A-3 (Phase 2 results) in Chapter 3, Appendix 3-A.

[Comment continued]

Pg. 76, par. 4 - It is not clear that it is appropriate to combine data from head, pubic and maxillary hair.

ATSDR Response: ATSDR agrees. We have added a caution to Chapter 3, Sections 3.7.2.1 and 3.7.8.1.

[Comment continued]

Pg. 77, table - The table does not have a number assigned to it. Also, the data presented for males and females separately are not labeled. Are these *average* levels by sex?

ATSDR Response: The table is labeled Table 3A-5. We have modified the heading to state clearly that the values reported are averages for various groups.

[Comment continued]

Pg. 79, par. 1 - Data on matched pair correlations are not presented. In addition, the children's ages are not given. The conclusion about mother-child correlation and in-utero exposure are not substantiated.

ATSDR Response: Dr. Ortiz Roque's manuscript does not provide data on matched paired correlations. As stated in the text, Dr. Ortiz Roque reported that in one subset of 22 matched pairs, the age of children was less than 5 years. As stated in the text, Dr. Ortiz Roque reported that the Pearson's correlation was 0.93 and the p value was 0.0001. No Report changes are warranted.

[Comment continued]

Pg. 84, par. 2 - Where did the 52 subjects reside? What were the criteria for their recruitment? What was the dichotomous criterion for dividing the subjects into high and low fish consumption categories?

ATSDR Response: We have modified the text to indicate that the 52 subjects were Viequenses. Because this information came from a Webinar, we cannot currently answer the other questions. This information is likely to be available when Dr. Rodríguez Sierra publishes his data.

[Comment continued]

Pg. 85, table - Given the previous, brief description of the 2009 Rodríguez Sierra study, I would have expected to see the data stratified by high and low fish consumption rates.

> **ATSDR Response**: Because this information came from a Webinar, we cannot currently answer the question. This information is likely to be available when
>
> Dr. Rodríguez Sierra publishes his data.

[Comment continued]

Pg. 97, par. 3 - The levels of 85 and 58 µg/L are somewhat misleading. Although 58 µg/L is the lower 95% CL on the dose corresponding to a 5% decrease in performance, the value of 5.8 µg/L to a significant extent reflects the population variability in the intake dose (µg/kg/day) required to reach 58 µg/L. Therefore, the factor of 10 reduction from 58 µg/L is not simply an arbitrary default.

> **ATSDR Response**: The paragraph cited does not discuss 5.8 µg/L or the use of an uncertainty factor of 10 to derive 5.8 µg/L from 58 µg/L. The paragraph comes from a CDC Web site and is provided for informational purposes. No report changes are warranted.

[Comment continued]

Pg. 102, par. 2 - It is not clear how "cancer registries typically represent the best population-based dataset for any chronic health condition."

> **ATSDR Response**: Our intent was to communicate that the infrastructure for reporting cancer and the legal requirements for doing so are more rigorous than for most noninfectious chronic diseases and typically produce good data. We have clarified the text.

[Comment continued]

Pg. 103, par. 1 - The sentence starting, "One initiative…" is not a complete sentence.

> **ATSDR Response**: We revised the sentence in the fourth paragraph of Chapter 4, Section 4.1 as listed below.
>
> > The Selected Metropolitan/Micropolitan Area Risk Trends (SMART) project was initiated to develop prevalence estimates at the local level.

[Comment continued]

Pg. 108, #2 and ff. Given the latency for most cancers, how do temporal aspects of cancer incidence relate to military activities on Vieques?

> **ATSDR Response** The U.S. Navy has occupied some part of Vieques since 1941. Considering the latency of most cancers, evaluating cancer incidence and mortality from 1990–2004 is a reasonable timeframe.

[Comment continued]

Pg. 109, #4 - It is important to discuss the socio-economic status (SES) of Vieques relative to Puerto Rico.

ATSDR Response: ATSDR added a discussion of SES in Chapter 1.

[Comment continued]

Pg. 115, first bullet - Where are the locations of the reference populations given here? It would be useful to have a map at this point.

ATSDR Response: We agree a map would be helpful. Unfortunately, Figure 2 of Massol-Deya 2000 contains only the study areas. Other than the name, no details of the reference area and sampling locations are available.

[Comment continued]

Pg. 116, first complete bullet - Do the "toxic" levels of "all metals" referred to here refer to plant toxicities or human toxicities? Also, why are "absorption rates" for Pb and Cd referenced here instead of concentrations?

ATSDR Response: We have modified the text in Chapter 5, Section 5.2.1 of the Report to address these comments.

[Comment continued]

Last bullet. Are the goats in Puerto Rico that are being compared to those in Vieques the same species of goat? Are they the same age? What were the levels in Vieques and Puerto Rico goats? Was the difference statistically significant?

ATSDR Response: This information was not included in the publication (Massol-Deya 2002) that presented the hair sampling data. Among other things, the lack of this information precluded ATSDR from using these data to evaluate human exposure from consuming local-livestock meat and milk products.

[Comment continued]

Pg. 121, Table 5-1 - If the childhood exposure duration (ED) is assumed to be 6 yr, the adult ED should be 70 - 6 = 64 yr rather than the 70 yr value used here for adults if a 70 yr lifetime is assumed. Also, why do the ingestion rate (IR) assumptions differ for the MRL calculation (0.10, 0.05 kg/day) versus the NOAEL/LOAEL calculations (0.05, 0.01 mg/kg/day)?

ATSDR Response: Table 5-1 has been modified to show that exposure duration had no effect on the estimated daily dose. The ingestion rates used for comparison to the MRL of 0.10 and 0.05 kg/day reflect 95th percentile intake rates for adults and children, respectively. The rates for the NOAEL and LOAEL comparisons reflect 95th and 50th percentile intakes for children only.

[Comment continued]

Pg. 124, par. 2 - How many pineapple samples were taken? Was it only 1? If so, how can this observation be used to support modeling?

ATSDR Response: Lopez Morales (2005) does not overtly state the sample number, but apparently a total of 72 samples were taken: 36 from the study site and 36 from a reference plantation.

[Comment continued]

Pg. 126, par. 2. I do not see that these very limited data support any larger assessment of exposure or risk.

ATSDR Response: Thank you for your comment. ATSDR agrees that limitations in the current sampling data make it difficult to evaluate the extent of public health impact from exposure to metals in locally grown produce. Given the uncertainties in the data, ATSDR believes prudent public health practice requires further investigation of this potential exposure pathway. To address this data deficiency, ATSDR has recommended further sampling.

[Comment continued]

Pg. 164, par. 1. The sentence starting, "Examining results from background locations not directly affected by military operations…" does not make logical sense. If the samples are, in fact, background samples, this implies that they are assumed not to be impacted by specific activities.

ATSDR Response: In Chapter 7, Section 7.2.4, we inserted parentheses around the phrase to indicate that background locations are by definition not affected by military operations.

[Comment continued]

Pg. 165, par. 2 - This is a reasonable assumption for the sites of the actual explosions. However, when considering off-site transport, surficial material becomes more important.

ATSDR Response: Most of the surface soil samples used in this evaluation were collected directly from the LIA. We recommend that any future sampling conducted outside the LIA consider surficial (top 3 inches) contamination.

[Comment continued]

Pg. 190, 1b - Hair is not an appropriate medium for biological monitoring for most contaminants. Blood is generally more appropriate. But for blood too, the determination of the applicability of its use for biological monitoring is contaminant-specific.

ATSDR Response: We agree that with the exception of mercury, hair is of limited use for environmental exposure monitoring. Numerous statements in the Report convey this message. We also agree that if other metals are considered as part of a biomonitoring study, developing a scientifically rigorous and valid protocol is important. This includes determining whether the results will inform about health risk.

[Comment continued]

Pg. 192, #2 - The review has not provided any basis to conclude what this single observation of Cd levels in the pigeon peas implies for exposure. This observation should not be presented and discussed without proper context.

ATSDR Response: Text in Chapter 5, Section 5.2.2 of the Report has been modified to address these comments.

[Comment continued]

Pg. 193, #5 - Add “and false low values” after, “To avoid false high values”

ATSDR Response: Text in Chapter 5, Section 5.3 of the Report has been modified.

Reviewer 2

Overall, has ATSDR's re-evaluation of public health issues associated with historical bombing exercises at Vieques been appropriate?

[Comment] The ASTDR document: A Fresh Look at Environmental, Biological, and Health Data from the Island of Vieques, attempts to explain in readily accessible language, the potential for human health effects as a result of decades of use of portions of the island as a U.S. Navy training and testing area. The document has been developed using data collected by related agencies specifically for the purposes of this risk assessment as well as data collected by external sources. The risk assessment discussion has been organized based on individual pathways as a means to both structure the information and explain the potential risks in a systemized manner.

In evaluating this document, I've approached the information as a scientist versed in bioavailability of contaminants in soil systems as well as someone familiar with Vieques. Before graduate school, I worked in Vieques for two winters during the mid-1980s. At this time the Navy still had an active presence on the island. I remember being able to both feel and hear some of the munitions testing during my time on the Island. I read about protests by Viequenses that were a factor in the decision to end the testing operations. I have also been back since the Navy ceased operations and the former Navy bases became wildlife areas managed by U.S. Fish and Wildlife.

Understandably, the residents of Vieques are concerned about potential negative health effects as a result of the decades long use of the island as a training and testing area for the Navy. Certain factors, including high cancer rates and increased incidences of select medical conditions would suggest to concerned residents that there is likely a link between the testing operations and these observed health effects. The risk assessment document attempts to ascertain any potential relationship between the testing and these observed effects by going through each exposure pathway and discussing all available evidence linking any human health effects to the testing results for each pathway.

This effort is handicapped by a number of factors. The testing began several decades before any research efforts to document health effects were started. It is likely that the records of testing re: chemical composition, quantity and final fate of the ordinance used, is not complete. Some of the data used for the current effort was compiled by external scientists, without the appropriate quality assurance, quality control, information on methods, and control populations that are general considered essential for the data to be considered appropriate for use. Finally, the necessary studies to provide quantitative guidance on contaminant concentrations in different human tissues, soils, fish that are likely associated with health effects are not available for all of the contaminants considered in this document.

In general, this review is very sympathetic to the concerns of the residents of Vieques. Data is included in this analysis that is likely inappropriate for use in a more rigorous exercise. Examples of this include web casts and unpublished data on health of residents. The desire to address concerns of residents has also resulted in credence and attention being given to certain pathways that are not realistically supported by the data as being a significant threat.

ATSDR Response: Thank you for your summary comments.

Has ATSDR adequately addressed the pathways of human exposure to bombing range-specific contaminants?

[Comment]

The document divides the potential for harm to have resulted from Navy activities into the following categories:

Consumption of fish

Terrestrial food chain pathway

Air pathway

Soil pathway

Drinking water pathway

The pathway approach is appropriate. In addition, all relevant pathways have been considered.

ATSDR Response: Thank you for your comments.

Please include specific comments on:

Consumption of seafood from reefs near Vieques

Use of biomonitoring tools to assess exposure to metals and other hazardous chemicals in blood, urine, or hair that are related to historical bombing exercises

[Comments combined by reviewer]

The primary conclusions of the report are that the pathways that have the potential to be of concern are the consumption of fish and the terrestrial food chain pathway. Mercury is the primary contaminant of concern for the consumption of fish and Cd is the primary concern for the terrestrial food chain pathway. The authors cite data from a number of studies that show elevated Hg concentrations in certain fish species caught in the vicinity of testing impacted reefs in Vieques. They also cite less credible studies that indicate elevated Hg concentrations in hair for, depending on the study, a significant number of Viequenses in comparison to control populations (Dr. Colón de Jorge and Dr. Rodríguez Sierra). The elevated Hg in humans is related to the observed high Hg content in fish. The authors of the current report conclude that reducing fish consumption and avoiding certain species of fish are sensible options to reduce any risk associated with elevated Hg in seafood. These conclusions are reasonable based on the Hg concentrations in certain of the fish species sampled as well as the availability of other fish species that have been shown to have low levels of Hg in edible tissue. The authors of this report present the findings of Dr. Colón de Jorge and Dr. Rodríguez Sierra while also pointing out the flaws or unknowns for both studies. It does not make sense to spend additional resources to test human Hg body burdens when a clear option for reducing Hg in diets while maintaining fish as a staple source of protein has been described. The one factor that is not considered in this discussion is the potential for the

observed excess Hg in fish to be related to Naval activities. There is no discussion in the document on the quantities of Hg that were likely used in Naval activities or whether elevated Hg concentrations have been detected in reefs impacted by Naval activities. So while the conclusions and recommendations of the ASTDR report re fish consumption are reasonable, a clear link has not been made between the observed elevated concentrations in certain fish species and Navy activities on the island.

> **ATSDR Response:** The Report focused heavily on mercury in fish because it poses the greatest risk of those scenarios assessed. Chemical results for soil, air, water, and biota do not reveal extensive contamination and do not identify an association between mercury and the bombing activities. We clarified this point by revising the Executive Summary and Chapters 1, 2, 3, 6, and 9.

Consumption of food (produce, meat, dairy, eggs) grown on the island

Exposure to air, soil, and water on Vieques

[Comments combined by reviewer] The other main concern outlined in the report is the potential for high Cd consumption from eating pigeon peas. This recommendation is based on a single study that included samples of about 20 pigeon pea plants from a single area on the island. This recommendation is also based on the assumption that a high quantity of pigeon peas will be part of a daily diet and that these peas will be grown on the island. For this reviewer, this conclusion and recommendation is not supported by the available data. There are a number of reasons for this.

The report concludes that there is no basis for concerns re direct ingestion of soils (the soil pathway) or from airborne contaminants. Both the available data and modeling efforts show no evidence of or potential for significant hazards associated with either the soil pathway or the air pathway for residents in the middle portion of the island. This is the inhabited portion and has always been exempt from military testing. For pigeon peas to have excess Cd as a result of the Naval testing, there would also need to be associated soil contamination. As modeling and data show no hazard from the soil pathway or the airborne pathway, it is also highly unlikely that soils would have become sufficiently contaminated with Cd as a result of the Naval activities to result in consistently elevated plant Cd concentrations in the edible portions of plant tissue.

As was stated earlier with Hg, there is no evidence provided of high Cd use or Cd concentrations in any of the munitions used in the testing. Cadmium was historically used as a pigment. It is not clear that any more than trace amounts of this metal were involved in any of the Naval activities. Soils can be naturally elevated in Cd- for examples soils in Salinas, California, derived from Monterrey shale have Cd concentrations ranging from 0.05-10.1 mg kg^{-1} (Burau et al., 1973)[40]. Sediment samples collected from the LIA show Cd concentrations ranging from 60-110 mg kg^{-1}. This was in an area where metal concentrations were elevated through the sampling depth as a result of direct impact of explosives. While these sediment Cd concentrations are clearly very high, they are directly in the impacted area. It is highly unlikely that agricultural soils will show any detectible increase in soil Cd as a result of the Navy activities.

[40] The peer reviewer did not provide a full citation in the comments sent to ATSDR.

009777

There are other factors that make the high Cd concentrations in this single study an insufficient basis for concern. Plant uptake of Cd will likely vary based on specific soil and plant factors. The study used as a basis for concern was from a single site. It is highly likely that Cd concentrations for peas grown in different areas of the island would vary significantly. A mean value, from a range of sites, showing highly elevated Cd, would be much more significant.

Bioavailability of Cd will also vary based on the nutritional status of the individuals. A study showed significantly higher adsorption of Cd in cases where Ca and Fe were below required levels (Reeves and Chaney, 2001). Sufficient Zn in diets was also observed to protect against excess Cd adsorption. The most studied cases of adverse health effects as a result of excess diet Cd were in Japan where milled rice was the staple grain. This occurred after WW II when diets were poor in Japan. Pigeon peas are likely a far superior source of essential elements than milled rice. In addition, diets in Vieques are likely superior to those where Cd in food resulted in adverse health effects.

Finally, it is highly unlikely that enough pigeon peas are grown in Vieques to feed a significant portion of the population for any amount of time. Although historically sugar cane was an important crop economically, agriculture in Vieques is limited. Families will have a few citrus trees, some banana and other sundry plants, however, there are very few large- scale farms in Vieques. During my time there, there was one attempt to grow tomatoes for export that failed. There isn't sufficient rainfall to consistently grow crops commercially without irrigation.

The above comments can be applied to the entire food chain transfer model (excluding seafood). Looking at the soil pathway section, there is no data presented in this chapter. However, based on the airborne discussion and the conclusions in the soil chapter, it seems pertinent to revisit the conclusions reached on metal uptake from home grown vegetables. That chapter did not include data on metal concentrations in soils where plants were grown. The chapter on soil pathways suggest that the potential for elevated soil metals as a result of Navy activities in the populated portion of the island, the portion that might be used for agricultural activities, is minimal. Airborne spread of metals would be the primary pathway for metal contamination of these soils. Metals would persist in the soil over time, unlike the organic contaminants that were also measured. The absence of any increase in soil metals suggests that the elevated Cd content in the pigeon peas is not likely related to soil contamination as a result of previous Naval activities. There are many factors that can lead to elevated plant metal concentrations, including a high soil electrical conductivity level, low soil Zn concentrations, and there may be a relation with low soil phosphorus. Many of these soil factors can be controlled with proper management. Appropriate soil testing and agricultural practices are suggested as a means to address concerns.

> **ATSDR Response:** Thank you for your comments. Relevant text in Chapter 5, Section 5.2.2 has been modified.

009778

Does the Vieques summary report adequately present and describe the limitations and uncertainty of assessing human exposure to bombing range-specific contaminants?

[Comment] Yes

Are independent studies utilized appropriately in the determination of potential health hazards?

[Comment] In the interest of including as much information as possible with a particular goal of including any information generated by researchers from Puerto Rico, information is included in this report that would not be considered of sufficient scientific integrity to be included in most studies. There are a range of reasons for doing this, and the inclusion of these studies has not resulted in unreasonable recommendations. The recommendations from the report are likely to be more broadly accepted because this data has been included.

ATSDR Response: Thank you for your comment

On the basis of ATSDR's re-evaluation of historical bombing activities in the Vieques summary report, has ATSDR reached the appropriate conclusions and recommendations?

[Comment] See discussion on plant Cd concentrations above, however general response is yes.

ATSDR Response: None required.

Select the appropriate category below:

List recommended changes or reasons for not recommending

- Recommend (X)
- Recommend with Required Changes ( )
- Not Recommended ( )

Additional Questions:

Are there any comments on ATSDR's peer review process?

[Comment] No

Are there any other comments?

[Comment] In general, the report is potentially too lenient in its use of any and all appropriate data. The report expresses concerns with minimal basis. The information presented suggests that the history of

naval activities on Vieques has not resulted in any measurable human health impacts. With that said, it is likely that if full and unrestricted access was granted to the former naval areas including home construction, the conclusions of this risk assessment would need to be revisited. The area is likely to require extensive remediation before unrestricted access can be permitted. In addition, the limited data presented in this report on contamination both in the waters around Vieques and for portions of the impacted areas suggest that a full ecosystem risk assessment would reach very different conclusions than this limited human health risk assessment.

ATSDR Response: Thank you for your comment. We changed the text (in the Executive Summary, Chapters 1, 2, and 3 to improve the discussion of contaminants in bombs and their possible contribution to various pathways. Iron, aluminum, copper, manganese, zinc, and lead are the metals most likely to be elevated in LIA soils from bombing activity, though other metals were also found in the bombs. It should be noted that all of these metals are also naturally present in the soils and rocks of Vieques (ATSDR 2003a, Learned 1973, USGS 1997 and 2001).

Although associations with these samples and military activities are not strong, the samples were not designed for a thorough ecological assessment. Thus they should only serve as indicators of further study.

Also, in Chapters 7 and 9 we clarified that ATSDR recommends environmental assessment and remediation activities continue at the LIA and at other potentially contaminated sites on Vieques. Restricted access to those areas should continue until the areas are cleared for unrestricted access.

Reviewer 3

Overall, has ATSDR's re-evaluation of public health issues associated with historical bombing exercises at Vieques been appropriate?

[Comment] Yes, the re-evaluation has been appropriate. The exposure pathways have been adequately considered, local studies that were made available were included and analyzed and proposals have been made for further work. My answer is qualified by only two things. First, there seems to be a degree of ignorance on local conditions, which are reflected in the report: the recommendation for Viequenses to follow the U.S.EPA/FDA's national fish advisory and its three recommendations for selecting and eating fish and shellfish, and the lack of knowledge concerning the public water supply of Vieques. My second qualification has to do with the multiplicity of samplings and data gathering activities that are recommended to fill current data gaps. Considered individually, I generally agree with them. However, taken as a whole, they constitute a huge and expensive undertaking likely to take many years. It is suggested that priorities be established and the most important gaps be filled first in order to establish within a reasonable period of time whether the health of Viequenses is at risk or not.

Details of this are given below.

> **ATSDR Response**: Thank you for your comments. We have modified the text in Chapter 2 to better reflect local fish consumption habits. We also modified Chapter 8 to address concerns about the public water supply. ATSDR considers education about choosing fish with lower mercury levels to be the highest priority public health recommendation and plans to work with our partners and the community to prioritize other recommendations.

Has ATSDR adequately addressed the pathways of human exposure to bombing range-specific contaminants?

[No Comment]

Please include specific comments on:

Consumption of seafood from reefs near Vieques

[Comment]

1. I agree with recommendations #1-3. However, ATSDR should reconsider recommendation #4 (Viequenses follow the U.S.EPA/FDA's national fish advisory and its three recommendations for selecting and eating fish and shellfish). These recommendations are hardly applicable at Vieques.

Consider that Viequenses eat fish provided by local fishers, which consist mainly of those shown in Table 2-1. The EPA/FDA's recommendations are for them to eat canned light tuna, or three other species (salmon, pollock and catfish) that are non-existent in the area. This is not going to happen, and instead

the recommendation should be to eat local species that are low in mercury, and a table should be provided.

Viequenses are asked to check local advisories about fish safety. To my knowledge no such advisories are made in Puerto Rico. This same recommendation makes reference to "local lakes, rivers…" of which there are none in Vieques. Using these references gives the impression that ATSDR is not aware of the local situation.

Viequenses are told not to eat shark, swordfish, king mackerel, or tilefish, species that are not listed in Table 2-1.

> **ATSDR Response**: We agree that portions of the EPA/FDA national fish advisory do not apply to Viequenses. We have modified this recommendation in Chapter 2,Section 2.3.2.
>
> Using all the data from Dr. Caro's survey, we expanded the list of fish that Viequenses eat (see Table 2-1 in Chapter 2, Section 2.2.1). Table 2-1 now shows the 22 fish species and two shellfish (i.e., conch and lobster) that the 51 respondents reported to Dr. Caro that they eat. Note that residents reported eating shark, tuna, and marlin—species that can have very high mercury levels. In addition, residents reported eating salmon, a low-mercury containing species. That Viequenses eat canned tuna seems a reasonable assumption. Therefore, some portions of the EPA/FDA national advisory apply while other portions clearly do not.
>
> During the November 2009 scientific meetings, a scientist also criticized the agency for 1) not citing the EPA/FDA national fish advisory and 2) contradicting the national advisory in ATSDR's previous, 2003 Fish PHA findings. That Viequenses know which reef fish have higher mercury levels is important, as is knowing which reef fish have lower mercury levels. ATSDR now recommends that the agency develop a health education program specific to Vieques. This health education program should empower Vieques residents to choose to eat fish that are lower in mercury.

[Comment]

2. Dr. Caro's study:

   Table 2-2 appears to be incomplete (it only shows three fish species). Caro characterizes this three fish as "…an example, the average contents of the metal may be mentioned in the following fish…." She adds that: "Additional data in Annex 10 illustrate these results." However, this and many other annexes were not included in the copy of the Caro report that was sent by ATSDR.

   ATSDR Response: We added the additional information from Annex 10 to Table 2-2 in Chapter 2, Section 2.2.1.

[Comment]

I agree that not knowing whether the results are dry weight or wet weight prevents comparison with other datasets and estimation of human doses from eating fish, but assuming dry weight would provide

a conservative estimate and allow for qualified comparisons and estimation of doses. If wet weight is used the actual tissue concentrations could be higher.

ATSDR Response: We provided additional analysis of Caro's data. See Chapter 2, Section 2.2.1

[Comment]

The correct spelling for Parquera is Parguera. The Caro report appears to make the same mistake.

ATSDR Response: The spelling of this town is corrected.

[Comment]

It is said the ATSDR is currently renewing its efforts to determine whether the data was dry weight or wet weight. Please note that Dr. Caro passed away in 2002.

ATSDR Response: We tried to contact the laboratory. We revised the sentences in the Chapter 2, Section 2.2.1 to reflect our efforts accurately.

[Comment]

3. Fish and Seafood Data from ATSDR

   It is indicated that ATSDR used standard body weights for adults and children. If this refers to a mainland standard, it should be noted that generally Puertoricans are smaller in size and weight, which could under estimate calculations on tissue concentration.

   ATSDR Response: In ATSDR's 2003 Fish PHA, we used standard body weights of 70 kg for adults and 16 kg children. The point that Puerto Ricans typically weigh less also was raised by scientists in our November 2009 discussions. Therefore, in the current Report, we used a range of body weight from 4.5 to 100 kg (9.9 to 220 pounds) to represent the body weight of various age groups (see Chapter 2, Section 2.6, Table 2A-1 in Appendix 2A-2). We also included a range of daily fish ingestion rates to cover daily intakes up to the 99th percentile. Therefore, the estimated doses of mercury in this report include a much wider range of doses than the 2003 Fish PHA.

[Comment]

4. There is a poster presentation titled "Health Risk Assessment of Arsenic from Fish in Coastal Waters of Vieques, PR" by Acevedo et al, which measured arsenic concentrations in eight edible fish in Vieques in 2001. Concentrations ranged from 0.3 to 3.53 mg/Kg wet. The study considered different exposure scenarios for inorganic As and found that concentrations exceeded EPA's reference dose of 0.0003 mg/Kg/d in six or more fish species for systemic effects in adults and children. The probability of excess lifetime cancer for exposure to As by adults was estimated to exceed the acceptable cancer risk of 10^{-6}. I do not believe that this study has been published but the poster may be obtained from Dr. Carlos Rodríguez Sierra at the School of Public Health of the Medical Sciences Campus of the University of Puerto Rico.

   ATSDR Response: ATSDR has attempted to locate this information but has not been successful. Average total arsenic in fish samples collected by ATSDR in 2001 ranged from 1.85 ppm (or

mg/kg) to 2.84 ppm (wet weight), with one trunkfish sample showing 8.3 ppm total arsenic. The median total arsenic concentration in fish was 2.8 ppm. Conch and land crabs had similar levels (0.3 ppm and 3.9 ppm, respectively). These levels are typical for total arsenic concentrations in seafood, and in particular for fish (WHO 2001, Rodríguez Sierra 2002, ATSDR 2007) and are similar to the levels reported by the reviewer in the poster (0.3-3.53 ppm).

Numerous studies have shown that the predominant form of arsenic in fish is arsenobetaine, an organic arsenic (ATSDR 2007). Arsenobetaine is not harmful to humans because humans rapidly excrete this chemical unchanged within 24 to 48 hours of ingestion. The remaining small amount of inorganic arsenic in fish is not harmful (WHO 2001).

Spiny lobster from Vieques reef were shown to have total arsenic levels ranging from 27 ppm to 48 ppm with an average of 33 ppm. Like fish, most of the arsenic in spiny lobster (genus *Panulirus*) is arsenobetaine and only about 0.05% is inorganic arsenic (Peshut et al. 2007). No changes were made in the report.

Use of biomonitoring tools to assess exposure to metals and other hazardous chemicals in blood, urine, or hair that are related to historical bombing exercises

[Comment]

1. The chapter provides a good review of six Vieques human biomonitoring studies. I was surprised to learn that ATSDR's previous public health assessments did not review biomonitoring data from Vieques.

   ATSDR Response: Thank you for your comment. The biomonitoring studies were not readily available when the public health assessments for Vieques were being written or, in the case of the PRDOH study, had not yet been conducted. When ATSDR began its evaluation of Vieques in 2009, we knew that PRDOH had conducted a study, but were not aware that a draft manuscript was available. ATSDR was provided a draft copy of the PRDOH study in late 2009

[Comment]

2. The Puerto Rico Department of Health (PRDOH) appears to have conducted the most comprehensive biomonitoring study, in which it collected hair, urine or blood samples from 500 randomly selected Viequenses. However, no public report was ever released and it was not until August 2009 that ATSDR obtained a draft, unpublished manuscript. Quite frankly, the way this is written makes one wonder why it took three years for ATSDR to obtain a copy, and how it was obtained.

   ATSDR Response: When ATSDR began its evaluation of Vieques in 2009, we learned that PRDOH had conducted a study, but were not aware that a draft report was available. Shortly before the November 2009 meeting with invited scientists, ATSDR received a draft copy of the PRDOH study.

[Comment]

3. The discussion in Chapter 3.3 of the strengths and limitations of previous human biomonitoring studies for Vieques is comprehensive and balanced. It is recognized that NHANES biomonitoring data applies to the entire U.S. population and that due to regional, cultural, and ethnic differences they can only cautiously apply to Puerto Rico. I completely agree that when looking for a Vieques comparison group, a control population from another town in Puerto Rico should be more appropriate.

   ATSDR Response: Thank you for your comment.

[Comment]

4. The conclusions from Dr. Colón de Jorge's investigations indicate that children could not be exposed to metals in paint as adults might be. As EPA states in http://www.epa.gov/lead/pubs/leadpdfe.pdf (accessed 25 February 2013:

   "Lead-based paint may also be a hazard when found on surfaces that children can chew or that get a lot of wear-and-tear. These areas include

   Windows and window sills.

   Doors and door frames.

   Stairs, railings, and banisters.

   Porches and fences."

My own experience with lead paint studies indicates that Puerto Ricans tend to paint their houses using layer upon layer of paint, and that the inner layers (which are the older) are the ones that have lead paint. Children will chew all the layers, thus exposing them to the lead paint.

   ATSDR Response: We agree that children can be exposed to lead from lead-based paint and have added a comment to Chapter 3, Section 3.7.9.1.

[Comment]

5. "Dr. Colón de Jorge pointed out that antimony levels were high because antimony was used in explosive ordnance on Vieques, thus making the case that military exercises was the reason antimony levels were high." This conclusion appears to have no supporting data or analysis and the review by ATSDR of the report should so state.

   ATSDR Response: Dr. Colón de Jorge states that antimony levels in bombs were high and this is the reason that some children have high antimony levels. We have modified the text in Chapter 3, Sections 3.2 and 3.7.9.1 to include Dr. Colón de Jorge justification. We also have pointed out that Dr. Colón de Jorge did not describe how residents might have been exposed to antimony.

[Comment]

6. The discussion of the investigations by Dr. Carlos Rodríguez (this is the same person as the one referenced in comment 4 of the section on consumption of seafood from reefs near Vieques above) is based on a presentation made in a webinar. Although it is stated that Dr. Rodríguez intends at some point to publish his data, he has apparently not done so, which raises an overall concern that I have about the lack of peer review of many of the local studies that are presented in the ATSDR report.

   ATSDR Response: ATSDR agrees that using peer-reviewed data is preferable. But data collected and analyzed using valid quality assurance/quality control procedures is also acceptable. In an effort to consider all available Vieques data, ATSDR chose to include the non peer-reviewed data in the Report. In the Report appendix, ATSDR summarizes all the available data and designates whether the data are peer-reviewed. In e-mail conversations with Dr. Rodríguez Sierra, he stated that his biomonitoring data were submitted for publication and are currently in the journal's peer-review process. Those data will be available to ATSDR after publication

Consumption of food (produce, meat, dairy, eggs) grown on the island

[Comment]

1. This chapter includes an analysis of several studies dealing with contaminants detected in locally raised garden produce and livestock. All the studies present serious limitations. For example, one included plant stems and leaves but did not include the edible portions of the plants sampled. It appears that the locally grown pigeon peas could have a high cadmium level, but, again, there are insufficient data.

   ATSDR Response: ATSDR concurs. Thank you for your comment.

[Comment]

2. Reference is made to USDA's suggestion of several simple methods for reducing metal uptake when soils are contaminated. It is stated that most home gardeners use many of the suggested practices. However, the referenced website goes to a very general page on plants and crops. A more specific reference should be provided, insuring that the suggested practices are applicable to Vieques.

   ATSDR Response: No specific reference was available. We defaulted to USDA recommendations, which are generally applicable and relevant to gardening and agriculture.

Exposure to air, soil, and water on Vieques

[Comment]

1. *Air Pathway* The chapter makes no mention of the possibility of Saharan dust contributing contaminants to the air or soil in Vieques. For example, see Garrison et al. (Rev. Biol. Trop. 54 (Suppl. 3): 9-21. Epub 2007 Jan. 15.) who sampled in the Virgin Islands where trace metal concentrations in the Saharan dust were found to be similar to crustal composition. Although I

think that metals concentrations in Saharan dust are not going to be high enough to make a significant contribution to metals found in Vieques, the issue should be addressed.

ATSDR Response: *African Dust Storms*: The 2003 Air PHA evaluated the public health implications of exposure to airborne particulates from African Dust Storms (pages 59–61) and found that PM10 (and PM2.5) from such storms on Vieques were not at levels of health concern. However, no contaminant specific data on dust from African Dust Storms were available at the time of the Air PHA (which recommended further evaluation if such contaminant specific data became available). Contaminant specific dust data are presented in a recent study by Gioda et.al. (2007) and a discussion of this information has been added to Chapter 6, Section 6.2.3.

[Comment]

2. The discussion of post-Air PHA sampling results and modeling studies as well as recent BIP detonations is comprehensive and leads to the stated conclusion that in the residential areas of Vieques, airborne contaminants from past military operations at the Vieques Naval Training Range would have been essentially nondetectable and unlikely to have resulted in harmful effects.

   ATSDR Response: Thank you for your comment.

[Comment]

1. *Soil Pathway* This chapter evaluated potential direct soil exposures in two situations: people who stayed on the LIA property during 1999-2000 protests and people who live in the island's residential areas. It concluded that people who lived on the LIA during protests were not exposed to soil contaminants at levels high enough to cause adverse health effects. This conclusion is well supported by the available data as discussed in the report. Concerning the island's residential areas, no soil data are deemed adequate to characterize potential exposures fully. Although current or reasonable recent data supports the conclusion that the existing contaminant levels at residential areas are not likely to cause adverse effects, there is a lack of historical data that would support a wider conclusion to the effect that it never has. The report calls for additional sampling to resolve existing uncertainties, which would be appropriate, but the wider question is now impossible to resolve.

   ATSDR Response: Thank you for your comment.

[Comment]

Drinking Water Pathway

1. The Puerto Rico Aqueduct and Sewers Authority (PRASA), a public corporation in existence since 1945, is charged with providing drinking water and wastewater treatment for the Commonwealth of Puerto Rico. It is the only entity in the United States with a statewide mandate, and as such is one of the largest water and wastewater utilities in the United States. In 2005 PRASA ran 130 different water systems throughout Puerto Rico, which processed 500 million gallons of water a day. It also operated 29 sewage disposal plants and 1,600 pump

stations. Vieques is part of its service area. While it is correct that in recent years (for about eight years until 2004) PRASA had part of its operations partially conducted by private corporations such as the mentioned Compañía de Aguas or at another time by ONDEO de PR, it has always been regulated by the Safe Drinking Water Act as a public water system. This regulatory function has been delegated by EPA to the Puerto Rico Department of Health (DOH). PRASA is required to annually produce and distribute to its users Consumer Confidence Reports where the quality of its water is reported and explained in non-technical language. Such reports for the years 2004-2009 can be obtained at http://www.acueductospr.com/AMBIENTE/ccr_reports.htm

(the system that feeds the Vieques pipeline appears under 5386-Humacao Urbano and then as Río Blanco in the pdf file) and reports for earlier years should easily be available at PRASA or the DOH. This data should be enough to settle the question concerning the quality of the pipeline water. For example, the data for 2009 shows that the lead action level of 15 ppb was exceeded four times.

ATSDR Response: We have modified text in Chapter 8, Section 8.1 to correct any inaccuracy regarding the current operators of the public water supply. We did not include detailed descriptions of the water supply system; it could detract from our evaluation of possible exposures from all water supplies, past and present.

The water quality reports from 2004–2009 referenced in the comment indicate that the pipeline supply has occasionally exceeded drinking water standards for microbial contaminants, turbidity, and lead. Elevated lead levels, detected particularly in 2006 and 2007, were not related to military exercises on Vieques. But regardless of source, lead in drinking water is an important public health issue. ATSDR has reworded text and added discussion to address these points in Chapter 8 and Chapter 9, Sections 9.8.1, and 9.8.2 of the Report.

[Comment]

2. The ATSDR report also addresses wells that had been and are now occasionally used for drinking water. These wells are very likely to belong to PRASA, who should have historical data on the quality of their water. Also concerning the wells, the presence or absence of a sanitary landfill in Vieques that might be impacting groundwater and the use of septic tanks for sewage disposal should be discussed.

   ATSDR Response: We added discussion about historical data to the Report in Chapter 8, Sections 8.2.2, 8.2.4.2, and 8.3.1.2. We did not attempt to gather additional historical information on the water quality of these wells. Even if available, questions about sampling, analytical, and quality control procedures from the 1970s and 1980s render unlikely the possibility that such reports would give us sufficient information to alter our current conclusions. Similarly, a lack of adequate historical data precludes any conclusion about more remote (1970s and 1980s) public supply well exposures.

   Thus, in Chapter 8, Section 8.3.1.2 we reworded text and added a statement about possible nonmilitary sources of contaminants, including sanitary landfills and septic systems. While sanitary landfills and septic systems could affect groundwater resources, whether they have was

not a focus of our evaluation. Instead, ATSDR based its conclusions on actual sampling of supply wells, private wells, and taps.

[Comment]

Although it would be good to have data on the rainwater systems, their overall use and effect is likely to be very small.

ATSDR Response: Thank you for the comment.

[Comment]

ATSDR should consider rewriting this whole chapter to better reflect the role of PRASA in supplying the drinking water at Vieques.

ATSDR Response: We have modified text in Chapter 8, Section 8.1 to correct any inaccuracy regarding the current operators of the public water supply. We did not include detailed descriptions of the water supply system. We were concerned that it might detract from our evaluation of possible exposures from all water supplies, past and present.

Does the Vieques summary report adequately present and describe the limitations and uncertainty of assessing human exposure to bombing range-specific contaminants?

[Comment]

I believe that the report is not as clear as it should be, particularly from the point of view of a lay person. The limitations and uncertainty are adequately presented, for a person that carefully reads the report in its entirety. Just reading the summary report does not present the same picture.

ATSDR Response: In the Report, we provided additional information regarding environmental data available for assessing any exposures associated with the military exercises on Vieques. We identified that those chemicals found in environmental samples were also found in explosive ordnance. Changes were made to the Report's Executive Summary and Chapters 1, 2, 3, 6, and 9.

Are independent studies utilized appropriately in the determination of potential health hazards?

[Comment]

Yes, but they are being given the same weight as other peer-reviewed studies.

ATSDR Response: The principal consideration for independent studies is the importance of describing all the environmental and health data available for Vieques—even when some of the data were not peer reviewed. We considered hair data results for numerous metals. Some scientists believed these data were valid and drew conclusions from them. ATSDR described the

limitations of these data in detail, pointing out the inability to distinguish between external contact and internal ingestion for most metals, except mercury. To this extent, ATSDR attempted to report fairly the available data and to provide insight into their value for health decisions. In our conclusions and recommendations, these data of questionable quality either were not used (e.g., metals in hair, except mercury) or were used qualitatively (e.g., arsenic in urine reported in a Webinar).

On the basis of ATSDR's re-evaluation of historical bombing activities in the Vieques summary report, has ATSDR reached the appropriate conclusions and recommendations?

[Comments]

Yes as to their quality, but I have the following comments regarding the presentation:

1. It is confusing that there are conclusions and recommendations at the end of each chapter and in Chapter 9, and they are different. For example, at the end of Chapter 5 there is a nine-row paragraph with recommendations, while in Chapter 9 these recommendations are expanded to ten items over one full page. Also, as another example of confusion, Chapter 5 is called Terrestrial Food-Chain Pathway in the text of the report while in Chapter 9 it is referred to as Local Produce and Livestock Pathway. A similar thing happens with Chapter 2.

   ATSDR Response: To address this comment, we changed "Terrestrial Food-Chain Pathway" to "Local Produce and Livestock Pathway" in the Executive Summary and Chapter 5" The conclusions and recommendations in Chapter 2 are now consistent with Chapter 9.

[Comment]

2. Consider the first recommendation in Chapter 9 about Chapter 4 Health Outcome Data:

   Assess the feasibility of applying the SMART BRFSS methods for generating stable Vieques specific prevalence estimates on asthma, diabetes, hypertension, and other chronic diseases.

   In Chapter 4 the complete Recommendations paragraph reads:

   At this time, ATSDR recommends performing additional analyses to quantify cancer and noncancer morbidity and mortality, assessment of primary and specialty health care needs, and alternative approaches for delivery of health services.

   So, in Chapter 9 specific methods come up without any explanation as to what they are, why they are better or even needed at all.

   ATSDR Response: Assessing the feasibility of SMART to overcome the limitations of developing prevalence estimates in a small population should be included as a recommendation or next steps in chapter 9. We have made the recommendations in Chapter 4 consistent with the

recommendations listed in Chapter 9. The language local level versus small population is interchangeable.

Comment]

In Chapter 9 some of the previous chapters have Summary and Conclusions while others only have Conclusions. There should be consistency on this to avoid further confusion.

ATSDR Response: Revisions have been made so that the heading reads conclusions.

Select the appropriate category below:

List recommended changes or reasons for not recommending)

- Recommend ( )
- Recommend with Required Changes (X)
- Not Recommended ( )

[Comment]

Rewrite the Drinking Water Pathway chapter to better reflect the role of PRASA in supplying the drinking water at Vieques.

ATSDR Response: We have modified text in Chapter 8, Section 8.1 to correct any inaccuracy regarding the current operators of the public water supply. We did not include detailed descriptions of the water supply system. We were concerned it would detract from our evaluation of possible exposures from all water supplies, past and present.

Additional Questions:

Are there any comments on ATSDR's peer review process?

[Comment]

None, except for Question 6 above.

Are there any other comments?

[Comment]

None.

Reviewer 4

Overall, has ATSDR's re-evaluation of public health issues associated with historical bombing exercises at Vieques been appropriate?

[Comment] This reviewer recognizes and applauds the strong efforts done by the people and community leaders of Vieques and of PR to access the health status of its community. In addition, congratulates all the researchers that have contributed to the generation of scientific knowledge in this area. Overall, the re-evaluation done by ATSDR seems appropriate. This report supports the need for additional studies and public health interventions in Vieques, PR, as although some results are still not conclusive, evidence supports potential environmental exposures in this population, as well as health disparities as compared to the mainland of PR and the United States. Most studies have been included in the discussion, although additional efforts should be made to contact researchers whose results have not been published. This reviewer has highlighted several issues that merit consideration before this manuscript is published (see below).

ATSDR Response: We appreciate all the comments.

Has ATSDR adequately addressed the pathways of human exposure to bombing range-specific contaminants?

Please include specific comments on

Consumption of seafood from reefs near Vieques

[Comment] Chapter 2. The ATSDR concludes that children who consumed fish from the reefs near Vieques and those born to mothers who also had this consumption were at increased risk for several health conditions. Nonetheless, the report highlights that this conclusion about the risk of harmful effects to the fetus and to children is somewhat uncertain because a person's mercury response may vary by sex, genetics, health and nutritional status. It is the belief of this reviewer that, despite some limitations, the observed association between fish consumption in Vieques and health hazards in these population sub-groups is very relevant and should not be minimized. ATSDR should add to its recommendations in this area the need for an assessment of the health and nutritional status of children in Vieques and how these factors can influence the impact that fish consumption could have on the health of children in Vieques. Some information regarding nutritional status of adults and children in Vieques could be collected from the BRFSS and the Youth Risk Behavior Survey (YRBS) of PR.

ATSDR Response: Thank you for your comments.

Children and pregnant women are advised by the U.S. Food and Drug Administration (FDA) to avoid eating those fish with the potential for the highest level of mercury contamination (e.g., shark, swordfish, king mackerel or tilefish); to eat up to 12 ounces (two average meals) per week of a variety of fish and shellfish that are lower in mercury (e.g., canned light tuna, salmon, pollock, catfish); and check

local advisories about the safety of fish caught in local lakes, rivers and coastal areas. Since fish are highly consumed in Vieques, we believe that fish consumption among this population should be addressed.

[Comment]

Table 2-2 (page 17): Efforts should be made to contact Dr. Caro and determine if the data came from dry or wet weight fish.

> **ATSDR Response:** Dr. Caro is deceased. The laboratory that conducted the fish analysis for her report has not responded to phone calls or e-mails. We have provided additional analysis to Dr. Caro's data in Chapter 2, Section 2.2.1.

[Comment]

Chapter 2. Given that the authors highlight lack of knowledge regarding the estimation of combined doses of exposure (from single and multiple pathways) regarding possible mixture effects for residents eating seafood, ATSDR should add to the recommendations of this chapter (and of the overall report) that risk from possible mixtures effects for residents eating seafood should be further studied.

> **ATSDR Response:** At this time, evaluation of possible mixtures effects from chemicals in fish is not possible because of limitations in the science. This topic is discussed in Chapter 2, Section 2.2.6.

[Comment]

Chapter 2.3.2. The list of recommendations on Chapter 2.3.2 could be more specific and describe in more detail some of the recommendations previously mentioned in chapter 2. In addition, the authors should add to the recommendations of this chapter "Collecting additional fish samples (within each family and by reef location) from Vieques."

> **ATSDR Response:** We provided additional information in Chapter 2 specifying the need to collect additional fish samples to allow analysis by species and location.

Use of biomonitoring tools to assess exposure to metals and other hazardous chemicals in blood, urine, or hair that are related to historical bombing exercises

[Comment] Chapter 3 (page 50). The authors highlight that the levels of inorganic arsenic in Viequenses urine were higher than those reported in the U.S. general population according to the NHANES survey. To facilitate the discussion in this section, I suggest adding a table with the values of metals observed in the general population of the US according to NHANES.

> **ATSDR Response:** In Tables 3A-3 and Table 3B-1, ATSDR has included data from NHANES showing the average concentration of metals in urine and blood.

[Comment]

Chapter 3.2 (page 50, fourth paragraph). To complement this first sentence, the authors should include a brief summary of the conclusions of the studies by Dr. Colón de Jorge and Dr. Ortiz-Roque.

ATSDR Response: The conclusions from Dr. Colón de Jorge's and Dr. Ortiz Roque's studies are presented in the second, third, fifth, and sixth paragraphs in Chapter 3, Section.3.2. No changes were made in the Report.

[Comment] Chapter 3. Section 3.5.1 Conclusions: Section does not acknowledge that metals were also detected in feces. In addition, sentence 6 of this paragraph starts by saying *"Another study weakness…"* Given that this is the only weakness discussed, the sentence should start by saying "A study weakness…."

ATSDR Response: ATSDR has included feces in the list of biological matrices where metals were detected and rephrased the discussion about study weakness.

[Comment] Section 3.5.2 (page 56). In the conclusions of this chapter, the authors recommend that additional biomonitoring studies should measure mercury levels in blood and hair of Viequenses; these studies should also consider other metals and substances in the assessment. The authors should stress in this paragraph why additional studies are needed, as presented evidence already suggests high mercury levels (and other metals) in hair and blood of Viequenses-suggesting a health hazard in this population.

ATSDR Response: ATSDR has added additional information to the Report conclusions in Chapter 3. While Dr. Ortiz Roque found several women with elevated levels of mercury in hair, the PRDOH study did not show high mercury levels in the blood of 500 Viequenses. Some uncertainty therefore remains regarding whether high mercury exposure is prevalent on the island.

[Comment] Section 3.7.1. Summary of Human Biomonitoring, (page 61, third sentence): "These studies are briefly mentioned in the main text (not only in this appendix) for Chapter 3 and are described in more depth…" The word "depth" should be added to the sentence.

ATSDR Response: ATSDR has reworded this sentence.

[Comment] Section 3.7.2.2. PRDOH Discussion Phase 1 (bottom of page 65-top of page 66)- Given that the authors mention that "At the total population level, the average concentration of some metals was above expected norms as established by Quest Laboratories but not above toxic levels as also established by Quest Laboratories. The PRDOH manuscript stated that this suggests the need for further investigation concerning the various risk factors, with particular emphasis on how smoking contributes to metal body burden." Thus, it is the recommendation of this reviewer that future studies should do a complete assessment of tobacco consumption of persons in Vieques. Although data presented in some of the analyzed reports does not suggest a higher prevalence of smoking in Vieques than in the rest of PR, this data should be analyzed in depth and included in this report. Given that it is of great relevance for various topics within this assessment, it should be included or recommended for inclusion in future studies in this population?

ATSDR Response: ATSDR agrees that future studies should include detailed information about tobacco use for study participants. Since PRDOH's 2006 study is confidential, ATSDR is unable to

obtain the raw data. We are consequently unable to provide more detailed information about tobacco use in Vieques.

[Comment] Section 3.7.2.2 (page 66, line 5)- Regarding the following statement "PRDOH concludes that eating fish within 3 days of a test or smoking could partly explain some but not all of the elevated metal body burdens in participants.", the authors should add to the text a discussion of what other potential explanations does the PRDOH provide for this result.

ATSDR Response: ATSDR agrees and has added text to Chapter 3, Sections 3.7.2.2 and 3.7.4.

[Comment continued] The authors highlight in this appendix that PRDOH data show that "In over 90% of the population, detectable levels were found of at least one heavy metal. In more than 20% of the study participants, the levels of aluminum in blood, arsenic in urine, and nickel in hair were over the laboratory reference threshold. Geometric means for uranium in urine, mercury in blood, lead in blood, aluminum in blood, nickel in hair, and cadmium in urine were significantly higher than the geometric means from the 1999 NHANES survey." Nonetheless, the conclusions of chapter 3 recommend additional research in this area. A statement highlighting the reasons for these additional studies is warranted in the main conclusions/recommendations of this chapter.

ATSDR Response: ATSDR has added clarifying text in the Executive Summary for its recommendations about future biological sampling and in particular the need for an appropriate comparison group.

[Comment] Section 3.7.5 (page 70). According to the referenced manuscript, the correct sample size of the study was 500 participants (not n=499).

ATSDR Response: The draft PRDOH report uses both numbers as the sample size: 499 and 500. Which is the correct sample size is unclear. We will use 500.

[Comment] Section 3.7.7.1 (page 76). Results of Dr. Roque regarding the high levels of mercury in the hair of Viequense women as compared to those of women in the mainland of PR and the US should be highlighted as evidence in the main text. Particularly, given that evidence exists that mercury in hair of pregnant mothers is an indicator of disease risk in the fetus.

ATSDR Response: Dr. Ortiz Roque's findings are highlighted in the main text of Chapter 3 in statements such as these:

Chapter 3, Section 3.2. Dr. Ortiz Roque identified several residents with hair-mercury levels above 12 ppm—the level the National Research Council identified as causing a 5% increase in neurological effects in children who were exposed *in utero* (NRC 2000).

Chapter 3, Section 3.7.8.3. Dr. Ortiz Roque's identification of three residents with hair mercury levels above 12 ppm is an important finding.

[Comment] Section 3.7.8.3. Dr. Ortiz Roque's identification of three residents with hair mercury levels above 12 ppm is an important finding.

ATSDR Response: ATSDR agrees that Dr. Ortiz Roque's findings are important. We have made this point several times in Chapter 3. In particular, biomonitoring results from Dr. Ortiz Roque

showed that some Viequenses had elevated mercury in hair above the level identified by the National Academy of Sciences to cause harm in 5% of fetuses exposed *in utero*.

[Comment] Section 3.7.6.2. The manuscript mentions that the PRDOH will develop a protocol for the management of persons with suspected acute poisoning of heavy metals using CDC guidelines. What is the status of this protocol? Has it been already developed and implemented? The authors could contact the PRDOH and include this information as part of the manuscript.

ATSDR Response: ATSDR has been unable to determine whether PRDOH has developed and implemented a blood lead survey of Viequense children.

[Comment] Section 3.7.6.3 (page 73). Table 3A-3 would be enriched by the inclusion of levels of heavy metals found in the U.S. population according to NHANES.

ATSDR Response: As suggested, ATSDR has included data from NHANES in Tables 3A-3 and 3B-1.

[Comment] Section 3.7.8.2 (page 78, line 8). When discussing Dr. Ortiz-Roque's study, the authors say that the non-random participant selection might account for some confounding factor elevating the rates. Nonetheless, it should be highlighted that this procedure could also have reduced them.

ATSDR Response: ATSDR agrees that a nonrandom survey could be a confounding factor that increases or decrease rates. We have made the suggested change in the text.

[Comment] Section 3.8.3 (page 94). Add reference for the following statement: "Several population studies of persons residing in areas with higher cadmium soil concentrations or with cadmium pollution have reported mean blood and urine cadmium levels as much as 10 times higher than control groups or representative U.S. data."

ATSDR Response: The paragraph with this statement has been deleted from the report; and, the reader is referred to the 4th Report, which contains toxicological information about metals.

[Comment] Section 3.8.3 (page 95, first line)- Please include references regarding the impact that cadmium levels in blood or urine could have on human health.

ATSDR Response: The paragraph with this statement has been deleted from the report; and, the reader is referred to the 4th Report, which contains toxicological information about metals.

[Comment] Chapter 4 Summary (page 101). The authors emphasize that "As a result of the small population, all studies suffer to some degree from a lack of statistical power and methodological limitations that make interpretation difficult. Despite these limitations, the studies are valuable for describing the health status of Viequenses. And in Vieques relative to the rest of Puerto Rico, the studies indicate elevations in the prevalence of chronic disease, cancer incidence and mortality, and infant mortality." This reviewer disagrees with the statement that interpretation of these studies is difficult. I suggest rephrasing this statement.

ATSDR Response: Quantifying differences in rates of morbidity and mortality is statistically challenging in small populations. The limitations previously described also introduce additional uncertainty. Despite these limitations, the studies are valuable for describing the health status of Viequenses. Relative to the rest of the population in Puerto Rico, these studies indicate

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elevations in the prevalence of chronic disease, cancer incidence and mortality, and infant mortality in Vieques.

Consumption of food (produce, meat, dairy, eggs) grown on the island

[Comment] Chapter 5. We agree with the authors in that evidence shows that "Levels of cadmium in pigeon peas could be potentially harmful for preschool children who eat more than four 6-ounce portions of pigeon peas each week for several years; possibly resulting to health problems later in life."

ATSDR Response: Thank you for your comment. Relevant text in Section 5.2.2 has been revised based on comments received.

Exposure to air, soil, and water on Vieques

[Comment] Section 6.2.1 (page 133, third paragraph). Regarding the Air PHA conclusions, the authors mention that the *"The Navy's ordnance exercises at the LIA did not pose a health hazard in the residential areas of Vieques."* This statement should be clarified and highlight that it is referring specifically to the hazard of residential areas specifically by air pollution (other hazards may still be possible through other pathways).

ATSDR Response: The statement referenced summarizes the conclusions of our previous Air Public Health Assessment. The current report clarifies this issue (see Chapter 6).

[Comment] Section 6.2.1 (page 134, first bullet). The authors highlight that levels of contaminants in air were low, and not at levels that would be associated to disease risk. Regarding the Air PHA conclusions, the authors mention that the ***"****Wind-blown dust from the explosive-ordnance range did not and does not pose a health hazard to residents."* Comment should be more specific, as contaminants were measured in the air, and air quality was not associated to disease risk. This is of particular relevance given that cumulative exposure has not been evaluated.

ATSDR Response: As there was no significant transport of airborne contaminants from the LIA to the residential areas of Vieques, there was no significant deposition to soils or subsequent leaching of contaminants from soil to groundwater. Consequently, the exposure pathways for soil and groundwater contaminants (from military operations) are incomplete and there is no basis for assessing cumulative exposures for these pathways. The Report does discuss the issue of cumulative exposures (as chemical mixtures in fish consumption) in Chapter 2, Section 2.2.6 and in Chapter 9, Section 9.9.

[Comment] Section 6.2.3 (page 140). Regarding the following statement in the text: "In addition to the recent evaluation of BIP detonations, the Navy has proposed prescribed burning of LIA vegetation to access safely and remove remaining unexploded ordnance. In support of this proposal, the Navy has conducted additional air modeling to assess the potential effects of such prescribed burning (CH2MHill 2008a). Although the Air PHA did not address vegetation burning, it did address past operations of open burning/open detonation (OBOD) of excess or recovered ordnance. As the mass of ordnance involved in past OBOD events was small relative to the mass involved in then-ongoing Naval operations, the 2003 Air PHA found that air emissions from past OBOD events did not present a public health hazard to the residents of Vieques. A permit to allow LIA vegetation burning is pending before the U.S.EPA and the

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Puerto Rico Environmental Quality Board. If so requested by these agencies or by any concerned citizen, ATSDR will further evaluate this issue." Have any requests been done? Do we need to wait for a request to do this or is ATSDR recommending that it should be done anyway?

Besides the data presented on Chapter 8 (Drinking Water Pathway), have any effort be made to determine the effect of airborne contaminants to water sources?

> **ATSDR Response:** Although ATSDR has not received a specific request to evaluate the pending air quality permit, Chapter 6 does include a summary of the air modeling and monitoring studies that support the permit application. Those studies indicate that vegetation burning in the LIA will not result in harmful inhalation exposures in the residential areas of Vieques and are in agreement with the results of the Air PHA. There is no public health basis for further evaluation of this issue. We have revised the Report by deleting the sentence indicating that ATSDR will further review this issue if so requested.

[Comment] Chapter 7 Summary (page 150). We agree with the authors in the "…need for continuing to restrict access to the LIA and to other potentially contaminated military areas until environmental assessment and remediation clear the way for unrestricted public access. To address remaining uncertainties about residential soil contamination issues, ATSDR recommends surface soil sampling in the island's residential areas." Particularly as some of the data evaluated is old (from the 1970's) and may not represent the reality of the soil today. Nonetheless, this summary paragraph of chapter 7 should include the recommendation of soil cleanup (as it does in the chapter-section 7.2.1).

> **ATSDR Response:** Thank you for this comment. We reworded the relevant section of the Report, Chapter 7 Summary and in Chapter 9, Section 9.7.2, to clarify that we do recommend continuation of environmental assessment and remediation activities.

[Comment] Chapter 7 Section 7.2.1 (page 158). The authors mention that a limitation of the study by García (2000) (study that collected soil samples at the LIA and in areas where people lived during 1999-2000) was that it was missing information on 1) the exact location, depth of sampling, and a 2) complete presentation of study results. Did the authors do an effort to contact García and colleagues to clarify this missing information? This information is important in this assessment, as the authors are suggesting that García's findings may be biased high (getting this information will clarify if this assumption is in fact correct).

> **ATSDR Response:** ATSDR has attempted to contact Dr. García on two different occasions by e-mail but has not yet received a response. The authors reported only the highest and next-to-highest detections, so that would by definition high-bias the concentrations.

[Comment] Section 7.2.1 (page 159). Even though the authors conclude that there is no indication that protesters living in the LIA in 1999-2000 were exposed to harmful levels of surface soil contaminants, they mention that a limitation is that conclusions were based on a small number of samples (making the conclusion not valid if future/representative analyses showed something different). This is not consistent with their sentence in the Summary section of this chapter (page 149) that says that *"Sufficient data are available to conclude that people who lived on the LIA during the protests were not exposed to soil contaminants at levels high enough to cause adverse health effects."* Although at this

point evidence does not support risk for this group, the limitation of small sample size should be included in the summary statement.

> **ATSDR Response**: The limitation mentioned in Chapter 7, Section 7.2.1 is a common limitation for any environmental characterization. Despite this limitation, the soil data collected for protesters living in the LIA were sufficient to characterize potential exposures during that time. Samples collected from the protester camp were designed to represent actual exposures to those living there. Samples collected from the LIA were collected from areas likely to have higher concentrations of contaminants. These samples, although limited in number, are reasonable to use as protective descriptors of exposures that might have occurred to protesters.

[Comment] *Section 7.2.4 (page 164, last 2 sentences). The authors mention that* "Yet detection of explosive residues in the background samples also suggested that all areas of the island, including the residential area, might have been affected by explosive compounds from past bombing activities. Although residual levels are low today, it is impossible to say what past levels were." The authors should highlight that although this cannot be corroborated at this point, these exposures may have had an impact in the past, this should be highlighted in the conclusions, as right now they are suggesting that residential exposure was likely minimal.

> **ATSDR Response:** No changes were made in the report. Although verification is impossible regarding past levels of explosives in residential areas, the modeling work described in Chapter 6 and the levels of residues in background areas on the land adjacent to the LIA do suggest that any past residential exposures were small.

[Comment] Chapter 8. As recommended by ATSDR, it is the opinion of this reviewer that additional studies of the pipelines, wells and rainwater collection systems are needed to confirm the safety of the drinking water consumption of Vieques. For groundwater, we agree with the authors in that current results do not exclude the possibility for other groundwater contamination caused by military and nonmilitary activities hydrogeologically connected to the groundwater that supplies water wells. Thus, supporting the need for additional research in this area.

> **ATSDR Response:** Thank you for the comment.

[Comment] Section 8.2.2 (page 172, line 4). The authors mention that "Even assuming the detections were present at the level in drinking water, the levels of explosives were too low to result in adverse effects (ATSDR, 2001)." What about cumulative exposure? Its possibility and potential harm should be acknowledged in the text.

> **ATSDR Response:** No significant transport of airborne contaminants occurred from the LIA to the residential areas of Vieques. No significant deposition occurred in soils. Consequently, the exposure pathways for air and soil were incomplete; thus assessment of cumulative exposures for these pathways was without basis. For drinking water, the health guideline values used to compare with the possibly detected levels of explosives in this section were chronic guidelines. They would have assumed regular exposure of more than 1 year. But the historical data were extremely limited. Using such data to draw definitive public health conclusions about past exposures, or about past cumulative exposures, is impossible. The Report does discuss the issue

of cumulative exposures (as chemical mixtures in fish consumption) in Chapter 2, Section 2.2.6 and in Chapter 9, Section 9.9.

[Comment] Section 8.4. Given that several contaminants were found at levels that would make groundwater unsuitable for consumption, the need for further studies and cleanup of this sources should be highlighted in the summary and recommendations sections of this chapter, as it has been done within the chapter.

ATSDR Response: We added recommendations that environmental assessment and cleanup of groundwater affected by military operations continue to Chapter 8, Section 8.4.2 and Chapter 9, Section 9.8.2 of the Report.

Does the Vieques summary report adequately present and describe the limitations and uncertainty of assessing human exposure to bombing range-specific contaminants?

[Comment] Overall, strengths and limitations of studies seem appropriately discussed in the report. Besides the recommendations already provided in this regard, additional specific recommendations are provided below.

Chapter 3 (page 81). Given the relationship between metal toxicities and various diseases and health conditions, the report should also include recommendations for additional studies of the effect of metal toxicities to: cardiac conditions, cardiovascular, emotional disturbances, violence, poor absorption and cancer.

ATSDR Response: Until more information is available that shows contamination of the environment with specific metals or explosive compounds and exposure of Viequenses, additional recommendations are not warranted to study the effects of metals on various health conditions.

[Comment] Chapter 4, Section 4.1. The correct term is the "Puerto Rico Central Cancer Registry (PRCCR)," not the Comprehensive Cancer Registry (PCCPR)

ATSDR Response: We have corrected Chapter 4, Section 4.1 to reflect the Puerto Rico Central Cancer Registry.

[Comment] Chapter 4. When describing the PR Cancer Registry, the following statement/reference should be added to the text:

> "In the year 2003, a CDC audit concluded that 95.3% of all cancer cases diagnosed or treated in hospital facilities in PR were appropriately reported to the PRCCR; a result comparable to the US median (95%)."

REFERENCE: ORC MacroSM. *National Program of Cancer Registries, Technical Assistance and Audit Puerto Rico Central Cancer Registry 2000, Case Completeness and Data Quality Audit. Centers for Disease Control and Prevention.* Department of Health and Human Services; 2003. pp. 1–32.

ATSDR Response: The statement was added to the Report text as the closing statement in the paragraph below:

> The data reported by the RCCPR have several strengths. Cancer registries typically represent the best population-based dataset for any chronic health condition. Most cancer cases are captured by registries due to legal reporting requirements, redundancies in the reporting system (i.e. reporting by labs, clinics, hospitals, and specific oncologists), and the clinical course of most cancers that requires substantial follow up and repeated visits. The analysis of cancer registry data should then represent the most accurate assessment of cancer incidence in Vieques. CDC has continuously funded the cancer registry through a cooperative agreement since 1998 and the cancer registry has steadily improved its data accuracy. Finally, "in the year 2003, a CDC review concluded that 95.3% of all cancer cases diagnosed or treated in hospital facilities in PR were appropriately reported to the PRCCR; a result comparable to the US median (95%)."

[Comment] Section 4.1 (page 103). Although not mentioned, another limitation of the BRFSS is that it is based on self-reported information. Nonetheless, given that the National Health and Nutrition Examination Survey (NHANES) of the US does not collect data for PR, the BRFSS is the best available data of population-based morbidity and health behaviors in PR. In order to generate consistent population-based data of the health status of people living in PR, efforts should be made to include PR (and Vieques) in the US NHANES survey. ATSDR could help in these efforts.

ATSDR Response: NHANES is not intended to provide national prevalence estimates. BRFSS would thus continue as a better population-based data option for the health status of PR residents.

[Comment] Section 4.2.1 (page 105). The report highlights that all cancer cases in Vieques were age-confirmed, an activity that could not be done with all cancer cases in PR, where if a case did not have a known age, it was not included in any rate calculation. The authors suggest that this could have inflated standardized incidence ratios. Is there indication on the PRCCR reports of the percent of cases excluded from analyses from the PR mainland due to this reasons? This could give an idea if the potential for bias is small or high. If the reports do not specify, this information can be obtained by contacting the PRCCR and Dr. Nayda Figueroa. Despite this limitation, mortality rates would not be affected by this, and in fact they show higher mortality from certain cancer types in Vieques as compared to PR. This should be given higher relevance in the report. In addition, results from this analysis could be better described in tables/graphs in order to give the reader a sense of the excess risk observed in Vieques as compared to PR.

ATSDR Response: We contacted Dr. Figueroa to obtain information on the percentage of cases in PR that were excluded from analyses because of the lack of age confirmatory data. We have not heard back from Dr. Figueroa, and therefore have nothing to add to this response at this time. We are continuing to follow up.

The magnitude of the combined effect of excluding cases from Puerto Rico and actively seeking out cases in Vieques and not in Puerto Rico would make the SIR higher than if the limitations mentioned were not present. The magnitude of this bias is unclear—it may or may not change the overall conclusions. We agree that mortality for certain cancer types is higher in Vieques than in Puerto Rico. Although mortality is an important measure of health status, we believe that disease incidence is a better measure than is mortality for assessing whether military activities affected Viequenses' health.

[Comment] Section 4.2.2 (Page 105). The authors say that the demographics in 2 of the 4 communities studied by Lopez and Carrosquillo (2002) in the study appear unusual. On what basis the authors conclude this and why is this a limitation of the study? The demographics of Vieques are in fact different from those of PR, a reference from the Census that supports this statement should be included.

ATSDR Response: A review of the 1990 and 2000 census data showed that the selected sample population was older than the population in Vieques. Also, only persons ages 5–25 were selected from Lujan, and only older persons ages 59 –70 were selected from Puerto Rico Reconstruction Administration PRRA. The percentage of people in the sample population who were 60 years of age or older appears to be higher than the percentage of people in that same age category based on data from the 1990–2000 census report. This is not a limitation, but an inconsistency that we observed.

[Comment] Table 4.1 could be strengthened by including in the table the percent of persons that were affected with each of the health conditions (Lopez and Carrosquillo 2002).

ATSDR Response: The percent of persons affected was added to Table 4.1 as suggested.

[Comment] Section 4.2.4 (page 107). A better description of the results obtained in the study by Roque (2002) could be included in this section. On this page, the following statement should be highlighted as a strength: *"Typically, mortality data are population-based, with near universal coverage, and are less prone to bias."*

ATSDR Response: Our statements about the strengths of mortality data were intended to apply for both the cancer registry and for Dr. Ortiz Roque. We modified the paragraph to make this point clearer. Mortality data reported by RCCPR and Ortiz Roque may also provide meaningful insight into the health status of Viequenses and have several strengths. Typically, mortality data are population-based, with near universal coverage, and are less prone to bias. Analyses of mortality patterns in a population can be useful for hypothesis generation. But using these data to quantify potential relationships in exposure-disease relationships has limitations. Many potential confounding variables are not susceptible to assessment (e.g., access to care, lifestyle factors, and dietary habits).

[Comment] Section 4.2.2 (page 108). Was an effort made to contact the study researchers of the Lopez Carosquillo study and get information of the confidence intervals for the risk ratio estimates to identify differences? Despite the lack of confidence intervals, the magnitude of the excess risk observed in this study should be considered a strength that supports the greater disease burden in Vieques as compared to PR.

ATSDR Response: We did not attempt to contact researchers to obtain confidence intervals for this review. We relied on the reports as written and commented on strengths and weaknesses of the methods used to produce results.

[Comment] Section 4.2.4 (page 108, line 15)-The authors say that "Finally, whether the Puerto Rico disease prevalence data was used as a reference from the PRCMS or some other source is unclear—the PRCMS was based on systematic self-reporting mechanisms (e.g. , the Behavioral Risk Factor Surveillance System)." This statement is not clear to this reviewer. The PRCMS data is based on the Continuous survey of PR (See page 15 of the Lopez and Carosqullo study, 2002).

ATSDR Response: This statement was deleted. At the time of our initial draft of this report, we did not have a copy of the 1994 Annual Survey.

[Comment] Section 4.3 Findings (page 108). Please add to this section information on the PRCCR report of 2009, as it does not seem to have been addressed.

ATSDR Response: The findings reported in 5-year intervals (1990–1994, 1995–1999, 2000–2004) are from the 2009 report. We added tables to make this more apparent.

[Comment] Section 4.3 (page 109). Authors suggest that the fact that 36% of participants from the Lopez and Carrosquillo (2002) study were unemployed may have implications for lack of insurance coverage and access to appropriate medical care, thus affecting morbidity and mortality data. Even though this may be in part true, the authors should acknowledge that approximately 90% of the population of PR are insured, and close to 40% of the population of PR are covered by *Reforma* (the public, government funded health insurance program). Thus, most unemployed people will in fact have access to health care through *Reforma*. Nonetheless, physical barriers (such as lack of proximity to health care providers) does exist for residents of Vieques.

ATSDR Response: Of the participants in the Lopez and Carrosquillo (2002) study, 36% were unemployed. Although these participants may have access to health care through the government funded health program, their implications may be due to the lack of access to specialty care, thus affecting morbidity and mortality data.

[Comment] Section 4.4.2 (page 110). Contrary to what is expressed by the authors "The limitations associated with these analyses, particularly the methodological concerns discussed in this report, introduce considerable uncertainty and make interpretation difficult. These findings can nonetheless serve as a guide for future investigations of Viequenses health status." This reviewer believes that the data provided suggests evidence of a higher burden of chronic disease in the population of Vieques as compared to PR. The recommendations for the section of "Health Outcomes" seem vague. What are the specific recommendations in this area? There is enough evidence to support higher cancer incidence and particularly mortality among people residing in Vieques as compared to PR. What are the specific recommendations regarding future studies in this area? Also, additional future studies of chronic disease prevalence in Vieques as compared to PR should be added to the list of recommendations in this section.

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ATSDR Response: We agree that the health outcome data suggest a higher burden of disease in the population of Vieques as compared with Puerto Rico. But we also believe that the limitations in the available data reduce the strength of these conclusions. The limitations include the completeness of the Registry for much of the period of analysis and the self-reported data from the symptom prevalence survey. We believe that the next steps should focus on studies that better define exposure and that better characterize the occurrence of cancer and noncancer morbidity and mortality. ATSDR plans to consult with the community in Vieques, scientists in Puerto Rico, public health officials, and other external stakeholders to determine recommended, specific studies.

[Comment] Chapter 6. Page 131 (second paragraph). Could the air-sampling studies performed in the 1970's by the Navy be biased given that they were not corroborated or validated by an independent group? Is there any way to validate this data?

ATSDR Response: The 2003 Air PHA included a comprehensive review of historic data, including a determination of their utility as a basis for public health determinations. The Air PHA determined an adequately *post facto* data quality assessment was impossible. We consequently developed an air modeling study to assess historic exposures. The results of the air modeling study are consistent with all of the air monitoring studies and collectively provide an adequate basis for the public health conclusions.

Are independent studies utilized appropriately in the determination of potential health hazards?

[Comment] Overall, independent studies have been used appropriately in the determination of health hazards. Most studies have been included in the discussion, although additional efforts should be made to contact researchers whose results have not been published. In addition, as it has been already mentioned in this evaluation, additional efforts should be made to get specific unavailable information from some of these studies, as it is very relevant for a complete assessment. Also, as previously stated, results from the section of Health Outcomes seem strong, and should be acknowledged as so in that Chapter. Additional recommendations are described below.

Even though on section 2.5.1 (page 34) the authors say that "By collecting only larger species, the average concentrations might have been biased high, which might have resulted in a mercury overestimate" , they later says (page 35) that the "School of environmental matters collected 52 fish of varying sizes.." These statements seem inconsistent, please clarify.

ATSDR Response: Chapter 2 discusses two fish sampling plans, one from ATSDR and the other from Dr. Caro. ATSDR attempted to capture larger fish because larger fish tend to have higher contaminant concentrations. ATSDR's sample of various fish species might therefore show high-biased mercury levels. We intended this bias so that ATSDR would be sure to sample fish with higher contaminant levels. Dr. Caro reported that she could not find an association between mercury content and fish size. She attributed this lack of association to the predominance of

smaller fish in her study. We provided additional information about Dr. Caro and ATSDR's fish data in Chapter 2, Sections 2.2.1 and 2.2.2.

[Comment] Section 2.5.6 (page 38). Authors mention that "Table C1 in Appendix C of U.S.EPA (2001a) includes a complete summary of all comparisons between locations, parametric and nonparametric, for all species." For the convenience of the readers, please consider including this table as an appendix to this manuscript.

ATSDR Response: As requested, Table C1 from U.S.EPA's analytical report of ATSDR's 2001 fish sample has been added to the Report, Chapter 2, Section 2.5.6.

[Comment] Chapter 4. PRCCR data and the Lopez Carrosquillo study could be further discussed in this chapter (see previous comments). In addition, other references cited in this paper by Lopez and Carrosquillo (2002) seem relevant to the assessment in discussion, and should be considered for inclusion in this report.

ATSDR Response The references mentioned in the Lopez and Carrosquillo paper did not provide any additional information on morbidity and mortality other than what we obtained previously from other reports.

Chapter 4. The 2009 report of the PRCCR, which evaluated cancer incidence and mortality from 1990–2004, is not summarized in the Appendix of this manuscript. Please include.

ATSDR Response: We added a summary of the 2009 PRCCR report to Appendix A.

[Comment] The study by Dr. Carlos Rodríguez Sierra regarding arsenic levels in people of Vieques (page 272) is very relevant for biomonitoring. Further efforts should be made by the authors to contact Dr. Rodríguez Sierra and get a copy of his results for further discussion in this report.

ATSDR Response: On September 23, 2010, Dr. Rodríguez Sierra responded to our request for additional information about his arsenic studies with this statement:

> "Regarding the results of arsenic in Vieques, we just submitted this work as a research manuscript to a journal to be evaluated for publication (waiting time is 6-10 weeks)."

If the study is accepted for publication, it should be available in 2011. Dr. Rodríguez Sierra has repeatedly declined to share his data with ATSDR—that he wishes to publish the data first is understandable. Until his data are available, ATSDR will use the results he presented in his Webinar for qualitative purposes only.

On the basis of ATSDR's –re-evaluation of historical bombing activities in the Vieques summary report, has ATSDR reached the appropriate conclusions and recommendations?

[Comment] *Despite some limitations characteristic of research studies, previous research suggests a high burden of environmental health risks and of chronic conditions in the Viequense population. This*

reviewer overall agrees with recommendations/conclusions made in Chapter 9 of this manuscript. Nonetheless, specific suggestions are described below.

Fish pathway:

Given that this manuscript shows potential health risks associated with the consumption of large amounts of fish in Vieques, this reviewer agrees with the proposed recommendations of additional conclusive studies in this area. These future studies should consider fishes of different sizes, different classes of fish within the same family species and from different reef locations.

> **ATSDR Response:** We added the concept of looking at fish species and locations to the recommendation for an additional fish study. See Chapters 2 and 9.

[Comment]

Biomonitoring:

Increased blood levels of mercury in blood and hair of Viequenses has already been documented. Given that some limitations have been discussed in the text regarding this data, the authors should clarify in the recommendations of this chapter (Chapter 9) why they are suggesting additional studies.

> **ATSDR Response:** We have recommended that a survey of Vieques residents be conducted to determine the types, frequency, and quantity of fish consumed. The results of this survey could be used to conduct additional risk assessments and statistical analyses to validate our concerns about mercury in Vieques reef fish. ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time because we found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation.
>
> We have recommended that if individual Viequenses remain concerned about exposure to mercury, cadmium, other metals, or metalloids, they should consult their healthcare provider to discuss the need for and cost of testing. A qualified laboratory should do the testing and analysis.

[Comment]

Health Outcome Data

Increased burden of chronic diseases, cancer incidence and cancer mortality are documented in Vieques as compared to PR. The authors suggest that the confidence intervals are too wide. To what study are they making reference when doing this comment?

ATSDR Response: We are referring to the last line of paragraph 2 on page 56 of the 2009 Cancer Incidence and Mortality report, which refers to low precision in the confidence intervals. We added language to clarify our reference to cancer incidence and mortality in Chapter 9, Section 9.4.1. The revised response refers to cancer incidence and mortality**.** Uncertainty is also evident in the wide confidence intervals reported indicating imprecise estimates of cancer incidence and mortality ratios in Vieques relative to the rest of Puerto Rico (RCCPR 2009).

[Comment]

Section 9.4.2 (page 191, 4th recommendation). Why do they suggest point 4?? The data already provide evidence of increased disease risk in this population. Please provide more specific recommendations of the reasons for future studies in this area, and the suggested qualities/focus that these studies should have.

ATSDR Response: Although the data suggest increased prevalence of chronic diseases, we believe the findings contain considerable uncertainty. The conclusions for the prevalence of noncancer chronic diseases are based solely on one report. We believe that the next steps should focus on studies that better define exposure and that better characterize the occurrence of cancer and noncancer morbidity and mortality.

[Comment]

Local Produce and local Livestock Pathway

Given that evidence suggests potential contamination of foods (i.e. pigeon peas), potential for uptake of metals from soil into local food crops, and lack of definite data on contamination of edible portions of produce and livestock, this reviewer agrees with the recommendation of further sampling and research in this area. The splitting of samples between labs for further validation of results is an important suggestion.

ATSDR Response: Thank you for your comment.

[Comment]

Air Pathway:

Is there any way to confirm the U.S. Navy records of the types and amounts of ordnance used at the training range? Fluctuations in this records could impact the models developed to access population exposure

ATSDR Response: The 2003 Air PHA included a comprehensive review of the Navy's ordnance usage data and metals and explosive by-products emissions estimates that ATSDR derived from them. While there is no way to provide an ordnance usage estimate independent of naval records it should be noted that the Navy does not keep such records for environmental assessment purposes. The Navy maintains ordnance usage data for the purpose of monitoring training activities and ultimately because they must replace expended ordnance. Consequently, there is little rationale for the Navy to underestimate ordnance usage.

[Comment]

The report concludes no potential exposure to residential areas and thus, no further research in this area. Given that this reviewer is not an expert in Air pathways, the feedback of another reviewer in this area is recommended.

ATSDR Response: Comment noted.

[Comment]

Soil Pathway:

The results presented in this chapter suggest that contaminant levels at the LIA may have in the past been high, and are potentially high in the present, and thus may represent a harm for human exposure. Thus, this reviewer agrees with the need of future studies in this area. Validation of samples between laboratories should be done during these efforts.

ATSDR Response: Thank you for your comment.

[Comment]

Drinking Water Pathway:

I agree with the recommendation of additional research to further sample drinking water in Vieques to determine the safety of its consumption.

ATSDR Response: Thank you for your comment.

[Comment]

Section 9.9- Final Summary

The authors suggest the need for a well-conceived and well-conducted biomonitoring study. They should further elaborate on the proposed characteristics for this study, including that it should have the capacity to study mixtures and cumulative exposures and their impact on human health in Vieques.

ATSDR Response: In this type of document, elaboration is not appropriate regarding the study design of any future biomonitoring study in Vieques. A more appropriate approach is first to gather buy-in from scientists and health professionals who would be interested in such a study. After identifying such a group or groups, much discussion would be needed to determine the metals and other compounds that could be monitored and that could provide insight into whether residents are being exposed to contaminants from past military exercises. Adding mixtures to such a study would require a great deal more thought and insight. Many reviewers commented that one failing of the previous studies and work on Vieques was the inability to relate contaminants to bombs and munitions. Thus identifying which metals are military exercise-related is important, as is whether a pathway of exposure can be identified that warrants a biomonitoring study. ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time because we found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from

eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation. The Report does discuss the issue of cumulative exposures (as chemical mixtures in fish consumption) in Chapter 2, Section 2.2.6 and in Chapter 9, Section 9.9. No change was made in the Report.

Select the appropriate category below:

List recommended changes or reasons for not recommending)

- Recommend ( )
- Recommend with Required Changes (X)
- Not Recommended ( )

Additional Questions:

Are there any comments on ATSDR's peer review process?

[Comment] This report acknowledges the commitment of ATSDR in its continuing involvement in Vieques and in continuing to work with local community and health and environmental officials to implement the recommendations of this report. This reviewer thinks that this commitment is essential, not only in expertise, but also in other recourses, such as financial matters (as a future, well-rounded study will be costly).

ATSDR Response: Thank you for your comment.

Are there any other comments?

[Comment] The following terms (and others) should be abbreviated on their first use in the text, then the abbreviation should be used consistently throughout the document.

Live impact area (LIA)

Puerto Rico Department of Health (PRDOH)

National Research Council (NRC)

Nuclear regulatory commission (NCR)

Eastern Maneuver Area (EMA)

National Health and Nutrition Examination Survey (NHANES)

ATSDR Response: Thank you for your comment. Changes have been made as appropriate.

[Comment] Section 2.2.1 (page 16). The correct term is Parguera (not Parquera), an area located at the west of Puerto Rico's mainland.

ATSDR Response: We have corrected the spelling.

[Comment] Section 3.3 (page 52). A description of the NHANES survey is done on page 52, although the study is mentioned previously in the text. Its description should come earlier.

ATSDR Response: The use of NHANES has been corrected.

[Comment] Section 3.4.1 (page 55). When the authors suggest that before hair analysis could become a valid diagnostic tool for any particular substance, research needed to *"*to"*…4) learn more about organic compounds in hair."* Please specify what kind of knowledge is suggested in this area.

ATSDR Response: This type of discussion is beyond the scope of this report.

[Comment] Section 3.4.2 (page 55). Add reference for the report of the 2005 German Federal Environment Agency report.

ATSDR Response: The reference is included in the reference section to Chapter 3 and in the text (see GFEA 2005 in Section 3.4.2). No change in the report is needed.

[Comment] *The results of this manuscript should be discussed with researchers, community leaders, the Department of Health of Puerto Rico and other people interested in the health of Vieques for further evaluation and recommendations. Multidisciplinary and collaborative efforts should be developed to further study and improve the health of this community.*

ATSDR Response: Thank you for your comment. ATSDR plans to work with our partners to discuss the implementation of the Report's recommendations.

Reviewer 5

Overall, has ATSDR's re-evaluation of public health issues associated with historical bombing exercises at Vieques been appropriate?

ATSDR Note: Peer Reviewer 5 did not answer the six questions that were sent, but rather provided comments in the format below. ATSDR has provided responses to the comments after each comment where appropriate.

[Comment]

Critique of Air Pathway Analysis:

1. Failure to Collect and Manage Air Pollution Data: ATSDR concluded that air pollution data was mismanaged and therefore provides unreliable information regarding the magnitude and distribution of air contaminants during high activity periods on the Live Impact Area.

   The Navy's 1979 Environmental Impact Statement (EIS) for continued use of the bombing range documents results from a 2-month air sampling program (TAMS 1979 provided no information on the sampling methods used or on data quality. No documentation can be found describing the sampling methods used or the quality assurance measures taken.
   ATSDR Response: Quality of data used in the assessment: The Air PHA included a comprehensive review of all of the available air monitoring data, modeling studies, and meteorological data. While historic data and data reports do not include all of the data quality assessments that are required in current practice, this does not preclude the use of such historic data. Such data, however, should be interpreted with caution. The Air PHA used the historic data in conjunction with recent monitoring data and modeling studies. Although in isolation each of these information sources includes aspects of uncertainty, collectively they provide a comprehensive assessment of the potential airborne exposures in the residential areas of Vieques.

[Comment]

Exposures to Releases from Military Training Exercises Using "Live" Bombs

Averaging Periods: A problem is identified with respect to air pollution modeling. Pollution levels were averaged over two periods, one year and 24 hours. This may be relevant for chronic respiratory disease prevalence, however it neglects the potential for short term bursts of pollution to exacerbate existing respiratory problems such as asthma, allergies and chronic bronchitis. Averaging pollution over 24 hours could make temporal bursts caused by periodic explosions disappear, while these episodes may be quite relevant to estimating respiratory distress among the sensitive. This is especially problematic for young children who have immature and narrower airways than adults.

ATSDR Response: The reviewer is correct in noting that there was no assessment of PM10 concentrations for time periods fewer than 24 hours, and that military operations could have resulted in higher PM10 concentrations for time periods of fewer than 24 hours. The U.S.EPA

and the California Air Review Board have established recommended procedures for converting between various air loading averaging periods (USEPA 1992; CA ARB 1994). The recommended conversion to estimate a 1-hour averaging period from a 24-hour period is to multiply the 24-hr value by 2.5 (Figure H.1/ Appendix H, CA ARB, 1994).

Using the upper-bound assumptions for contaminant emissions, daily ordnance use, and meteorological transport conditions, the ATSDR Air PHA estimated that "live bombs caused the 24-hour average PM10 concentrations in residential areas to increase by 10.2 $\mu g/m^3$ " (page 40, Air PHA). Using the above conversion procedure, the maximum 1-hour increase in PM10 would be 25.5 $\mu g/m^3$ (10.2 $\mu g/m^3$ x 2.5). Adding the calculated maximum 1-hour increase of 25.5 $\mu g/m^3$ to the maximum 24 hour measured PM10 concentration (94 $\mu g/m^3$; Isabel Segunda, page C-9, Air PHA) results in a maximum short-term air PM10 concentration of 119.5 $\mu g/m^3$. This value is below the 24-hour PM10 standard of 150 $\mu g/m^3$ and unlikely to result in respiratory distress or other adverse health effects. As shorter averaging periods do not change the health determination, no changes to document have been made as a result of this comment.

[Comment]

2. Particle Size: Low diameter particles (less than 2.5 microns in size) were not measured. These fine and ultrafine particles may stay suspended for longer periods of time, move longer distances and may become more deeply embedded in the lungs.

   ATSDR Response: Chapter 6, Section 6.2.2 (of the summary document explicitly addressed the issue of PM2.5. No further revisions are necessary.

3. Wind Blown Dust: It was concluded that wind-blown dust from the LIA on days when bombing did not take place is not a health hazard.

   ATSDR Response: Thank you for your comment.

4. Total Suspended Particulates (TSP) are primarily a measure of larger diameter airborne particles that would likely settle out in close proximity to the location of their generation. Particles of smaller diameter—PM 2.5 microns in diameter and smaller—are far more likely to remain in the atmosphere for longer periods of time and travel longer distances before deposition. These finer particles were not measured by ATSDR, the Navy or EPA. These particles may also act as nuclei for other hazardous VOC's.

   ATSDR Response: Chapter 6, Section 6.2.2 of the summary document explicitly addressed the issue of PM2.5. No further revisions are necessary.

[Comment]

5. *Chaff:* Chaff fibers typically are 25 microns (µm) thick and between 1 and 2 centimeters long. Chaff fibers are visible to the human eye and have the appearance of short, very fine, hair-like fibers.

a. ATSDR estimates that 266,000 pounds/year of chaff may have been deliberately dropped over or near Vieques.

b. Ground level concentrations were never monitored by the Navy or other government authorities.

ATSDR Response – Chaff: Regarding the issue of chaff emissions and exposures, the Air PHA explicitly addressed the issue of chaff emissions and potential exposures. Using health protective assumptions regarding emissions, exposure, and uptake, the Air PHA concluded that "…the usage of chaff at Vieques does not pose a public health hazard, whether the chaff particles are inhaled or deposited in the mouth and swallowed." The Report summarizes this finding regarding chaff and other air contaminants in Chapter 6, Section 6.2.1. No further revisions or assessment are necessary.

[Comment]

Critique of Drinking Water Analysis

[Comment]

1. Chemicals released to the environment of the island of Vieques by the U.S. Navy may have resulted in human exposure and associated health threats that may be dose dependent. A 35 year period between 1943 and 1978 is the most likely time when the island's population was exposed via drinking water to possible hazardous compounds released to the environment by the Navy. Yet this is also a period when government testing of environmental quality on the island was minimal.

ATSDR Response: The current conclusion that a lack of historical data prevents a definitive conclusion about distant-past exposures to drinking water from wells is consistent with this comment; therefore, no changes have been made. We believe, however, it is important to indicate what limited past information and data do tell us about the relative likelihood of past exposures through the drinking water pathway. As discussed in Chapter 1, Section 1.2, ordnance training on Vieques was not scaled up until the 1970s, particularly after 1975, when training on the island of Culebra was discontinued. Therefore, it is not unreasonable to presume that contamination of public supply wells or cisterns was very unlikely before the mid-1970s.

2. [Comment] The poor history and quality of water testing makes it difficult to precisely reconstruct a history of exposure. Water supplies were not routinely tested for chemicals that were released to the environment by the Navy.

ATSDR Response: The problem of limited historical environmental monitoring data is not specific to Vieques. It is common at many sites that operated in the past. ATSDR evaluates public health effects from potential past exposures in such situations by reviewing available

contaminant fate and transport data and, using professional judgment, making assumptions about past exposure and identifying areas of uncertainty.

3. [Comment] The most probable routes of exposure to chemicals released to the Vieques environment by the Navy include: 1) contamination of drinking water wells from airborne chemicals that drifted and settled in the watersheds surrounding municipal wells; 2) contamination of cisterns from airborne chemicals that drifted and settled into the tanks; 3) contamination from Naval use of pesticides and herbicides; 4) contamination from airborne and surface fuel releases; 5) waste disposal practices.

   ATSDR Response: Thank you for this comment. ATSDR's drinking water PHA and Chapter 8 of this report discuss contamination of drinking water wells and cisterns (rainwater collection systems). Potential sources of contamination, including use of pesticides or herbicides, releases of fuels, and waste disposal practices, are evaluated by evaluating levels of associated contaminants in air, soil, or drinking water, as was done in past ATSDR PHAs and this report.

4. [Comment] Most reported studies are not peer reviewed, remain unpublished and are often based upon sampling designs and exceptionally small sample sizes (ranging between 1-12 samples). Degradation products were not tested or reported.

   ATSDR Response: ATSDR summarized the findings, strengths, and limitations of the reviewed studies in Appendix A of the Report. ATSDR was requested to review all available studies and data related to potential exposures at Vieques. While many of the studies were limited, they added to the body of knowledge about Vieques environmental exposures.

5. [Comment] Since the ATSDR drinking water study was completed prior to the air study the conclusions of this report are not conclusive. The most plausible pathway for drinking water contamination resulting from Navy activity on the islands is from airborne chemicals having settled in drinking water systems.

   ATSDR Response: As described in Chapter 6 and detailed in ATSDR's Air Pathway Public Health Assessment (ATSDR 2003b), evaluation of the air pathway suggested that no substantial levels of atmospheric contamination had reached the residential area of Vieques. ATSDR was unable to evaluate directly the potential for exposures from cistern water as no cistern water data are available.

6. [Comment] These studies do not provide a reasonable estimate of the drinking water contamination that may have occurred prior to the completion of the municipal water supply line from the main island of Puerto Rico in 1978.

   ATSDR Response: As concluded in Chapter 8, Section 8.4.1 and Chapter 9, Section 9.8.1, ATSDR recognizes that the 1978 data are insufficient to make public health conclusions about exposures long past. But as detailed in Chapters 6 and 8, consideration of groundwater flow patterns, air modeling studies, actual bombing patterns, and recent sampling results suggest that past exposures from drinking water were not high.

7. [Comment] The EPA studies cited did not test for pesticides and herbicides. Also an analysis of military use of pesticides and herbicides not been conducted or presented.

   ATSDR Response: The Report does not consider pesticides and herbicides. As discussed in ATSDR's Drinking Water Pathway PHA (ATSDR 2001), 1995 sampling had shown no detections of a wide range of pesticides and herbicides, indicating that drinking water supplies had not been contaminated at any location by pesticides or herbicides. Pesticides and herbicides were not analyzed in many of the soil sampling events. Soil sampling at the NASD and recent EMA sampling related to remediation activities have found a few detections of DDT and its breakdown products. In all but one specific location, these detections were far below health-based comparison values; one location had detections higher than comparison values. ATSDR's recommendation to restrict the LIA and other potentially contaminated military areas and continue environmental assessment and remediation activities will protect the public from any harmful exposures to pesticides and herbicides remaining from military activities.

8. [Comment] It was reported the presence of RDX (0.04 ppb) and Tetryl (0.05) in the drinking water supplies of Isabel Segunda (0.5 ppb) and RDX (0.04 ppb) in the drinking water of Esperanza in May of 1978, referencing a Naval Surface Weapons Center report (Hoffsommer and Glover 1978; Lai 1978).

   ATSDR Response: ATSDR discussed these findings in Chapter 8, Sections 8.2.2 and 8.3.1.2 of the Report.

9. [Comment] The studies interpreted by ATSDR do not demonstrate the absence of health threats associated with naval activities. Instead, they demonstrate the absence of proper testing of the community's drinking water supplies at a time in its history when it was most vulnerable.

   ATSDR Response: As concluded in Chapter 8, Section 8.4.1 and Chapter 9, Section 9.8.1, ATSDR recognizes that the 1978 data are insufficient to make public health conclusions about distant past exposures.

Critique of Soil Analysis:

1. [Comment] Failure to Collect and Manage Soil Contamination Data: The Navy consistently failed to collect data on soil contamination associated with training and operations. The absence of these data prevented the understanding when and where possible soil contamination might have posed a public health threat. This could occur from particles exploding into the atmosphere, drifting along the island into the breathing zone of inhabitants, settling on soils, infiltrating water supplies, being absorbed by plants, some of which might have been consumed by animals, in turn consumed by humans. By failing to monitor pollution air pollution, patterns of deposition to soil and other sediments, this in turn prevented an understanding of possible food chain dynamics.

   ATSDR Response: We agree that the issue of food-chain dynamics has not been thoroughly investigated on Vieques. Long-range transport of certain contaminants (e.g., aerial deposition of mercury) has been observed elsewhere under the right conditions (i.e., continuous emissions of large quantities from a single large source or multiple smaller sources, combined with

appropriate meteorological conditions). Nevertheless, actual sampling data and air dispersion modeling on Vieques do not indicate that environmental transport of contamination originating from the bombing areas would be expected to affect areas distant from the range. It appears that any environmental effect from past range use activities is localized.

2. [Comment] Grazing Animals and their Products: The Navy, EPA and ATSDR did not do research on grazing activities by cattle, goats, sheep, pigs and chickens. Their importance to the diet of Viequenses is also poorly understood, but could potentially be an important additional pathway of contaminant exposure. The community members expressed concern over the possibility that livestock are accumulating heavy metals by grazing on contaminated plants.

   ATSDR Response: Thank you for your comment. At the time of the original public health assessment discussing the soil evaluation pathway, little information or data were available pertaining to the potential for heavy metal accumulation in local livestock. The Puerto Rico Department of Agriculture, in cooperation with the Farmers Association of Puerto Rico, sampled grass, fruit-bearing trees, and bovine livestock from Monte Carmelo, Martineau, Monte Santo, Esperanza, Lujan, Gubeo, and western Vieques for cadmium, cobalt, copper, lead, manganese, and nickel. They concluded that the agricultural products from Vieques did not contain toxic levels of these contaminants and were suitable for consumption (El Nuevo Día 2001).

   Since that time, additional plant data have been collected. But due to data limitations, uncertainty remains regarding the degree of potential exposures from consuming local produce. The Report contains a thorough discussion of currently available sampling data from locally produce and livestock. Although the results of sampling by the Puerto Rico Department of Agriculture is reassuring, ATSDR recommends additional sampling of locally grown produce to allow a more complete evaluation of this exposure pathway.

3. [Comment] Air, Soil and Water: The most plausible hypothesis regarding transport and fate of chemicals is that chemicals released to the atmosphere were eventually deposited on soils and plants and then washed into underlying aquifers by rains.

   ATSDR Response: To assess whether the dust releases present public health hazards, ATSDR evaluated whether dusts blow into the residential areas in appreciable quantities. The findings of the Air Pathway Evaluation, described in Chapter 6, suggest that substantial levels of atmospheric contamination had not reached the residential area of Vieques.

4. [Comment] Plant Contamination: ATSDR could not quantify exposures to contaminants from these reports nor draw any health conclusions about whether consuming plants grown in Vieques would result in harmful health effects.

   ATSDR Response: Most of the relevant studies sampled plant species typically not eaten by humans or sampled the parts not eaten. As a result, the data were not useful for interpreting the degree of exposure or representative of locally grown produce on the island. To address this data gap, ATSDR recommends further sampling.

Reviewer 6

Overall, has ATSDR's re-evaluation of public health issues associated with historical bombing exercises at Vieques been appropriate?

[Comment] According to my best knowledge, the ATSDR re-evaluation of public health issues associated with historical bombing exercises at Vieques is appropriate. This opinion is based on the evidence presented, including ATSDR's own data and the data generated by Puerto Rican scientists and researchers. As ATSDR recognizes, there is a huge degree of uncertainty in this type of analysis and the recommendations made are directed to reduce that uncertainty. Vieques was used as a military target and ammunition-explosive storage for more than 60 years and, as expected, these military activities are a major source of contamination, which constitutes a human health concern. Past and present conditions of Vieques reflect the difficulties of approaching this type of human health assessment based on environmental data that was not necessarily developed to answer the type of questions associated with public health issues. In addition, Vieques social and environmental conditions are very complex. Thus, some degree of uncertainty will always remain. Nevertheless, military activities were always considered the major environmental and social stressor of Vieques' residents. Some connections between military activities and human health concerns in Vieques have been established by local scientists and researchers and this report very well examines the scientific strengths and weaknesses of those connections. This report also incorporates new environmental, biomonitoring and health outcome data and revisits the previous ATSDR reports. Therefore, new recommendations and findings are integrated into this report.

ATSDR Response: Thank you for your comments.

Has ATSDR adequately addressed the pathways of human exposure to bombing range-specific contaminants?

Please include specific comments on:
Consumption of seafood from reefs near Vieques

[Comment] This subject was addressed adequately. ATSDR recognized the limitations of the data in measuring mercury levels in fish and shellfish. It also indicates that some children born to women who frequently ate fish from waters surrounding Vieques were at increased risk of adverse health effect and some children who frequently ate fish from waters surrounding Vieques the same fish were also at risk of harmful effects. Based on the information available, ATSDR could not make any connection with the military activities and the mercury level found in fish. Then, the best recommendation is to make a risk assessment around the LIA to determine the effect that military activities have had on the marine ecosystem.

ATSDR Response: Thank you for your comments. The National Oceanic and Atmospheric Administration's (NOAA) National Centers for Coastal Ocean Science (NCCOS), in consultation with NOAA's Office of Response and Restoration (OR&R) and other local and regional experts,

conducted a characterization of coral reef ecosystems, contaminants, and nutrient distribution patterns around Vieques (NOAA 2010).

Use of biomonitoring tools to assess exposure to metals and other hazardous chemicals in blood, urine, or hair that are related to historical bombing exercises

[Comment] This subject was addressed adequately in some extent. ATDSDR reviewed the PRDOH, and some local researcher studies, and found elevated levels of various metals in residents' blood, urine and hair. ATSDR emphasized that the weakness of all these studies was the inability to investigate each person's environment, thus making it impossible to identify the source for high metal concentration. The PRDOH identified cigarette use, hair dyes and seafood consumption as possible sources for high metal concentration. Dr. Ortiz Roque showed that some residents had elevated levels of mercury in hair and that the most likely source was fish consumption. The best recommendation to address this issue is to make a survey of Viequenses to determine the type and quantity of fish consumed as well as a biomonitoring study to measure mercury and other metals in blood and hair. ATSDR should use the preliminary data developed by PRDOH and Dr. Ortiz to enforce a more specific study, which include an association between residents' habits, their environmental conditions and metal concentration in different parts of their body.

ATSDR Response: Thank you for your comment. We have recommended that a survey of Vieques residents be conducted to determine the types, frequency, and quantity of fish consumed. The results of this survey could be used to conduct additional risk assessments and statistical analyses to validate our concerns about mercury in Vieques reef fish. ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time because we found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation. Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing. A qualified laboratory should do the testing and analysis. Consumption of food (produce, meat, dairy, eggs) grown on the island

[Comment] This subject was addressed adequately. The main ATSDR conclusion about this subject is that the overall data is insufficient to adequately determine human exposure or to draw any valid health conclusion. ATSDR evaluated Dr. Díaz and Dr. Massols (2003) research, which found cadmium levels in pigeon peas potentially harmful for preschool children. ATSDR cannot be certain of the extent to which military activities contribute to the cadmium level found in pigeon peas. Recommendations suggested by ATSDR encouraged additional sampling and collaborative data collection for plants, livestock and soil. Based on the data collected by Dr. Díaz and Dr. Massol, ATSDR should enforce more specific studies about this subject and its association with military activities.

ATSDR Response: Thank you for your comment. In fulfilling its role as a nonregulatory science advisory agency, ATSDR cannot direct other entities to conduct further studies, nor collect samples of local produce. However, we hope that recommendations for a collaborative sampling effort will help resolve uncertainties and limitations in data on locally grown produce.

Exposure to air, soil, and water on Vieques

[Comment] This subject was addressed adequately to some extent. The main objective of this section was to determine whether airborne contaminant could potentially expose Viequenses in residential areas to harmful levels of metals and particulates that might result in adverse health effects. ATSDR used all the climate and meteorological information available as well as the ordinance detonation and the residential area air quality data. ATSDR reviewed and adapted a new version of a dispersion model and applied it to Vieques. Using this model ATSDR concluded that in residential areas of Vieques airborne from past military operations would have been essentially nondetectable and unlikely to have resulted in harmful effect. This conclusion is very critical in the sense that most of Vieques population receive the winds coming from the east (where the LIA was located) and they were not located far away from this military activities. This model should incorporate measurement of 2.5pm that is proved to be harmful for humans. This model should also include measurement of particulate matter coming from the Sahara desert, which is very well documented that affects human health throughout the Caribbean region. A conclusion sustaining that there is no air pollution reaching soil or water is somewhat uncertain. The rest of the data, conclusion and recommendations are very well documented.

ATSDR Response: Chapter 6, Section 6.2.2 of the summary document explicitly addressed the issue of PM2.5. No further revisions are necessary. The 2003 Air PHA evaluated the public health implications of exposure to airborne particulates from African Dust Storms (pages 59–61) and found that PM10 (and PM2.5) from such storms on Vieques were not at levels of health concern. But no contaminant-specific data on dust from African Dust Storms were available at the time of the Air PHA (which recommended further evaluation if such contaminant specific data became available). Contaminant-specific dust data are presented in a recent study by Gioda et.al. (2007) and a discussion of this information has been added to 6.2.3.

Does the Vieques summary report adequately present and describe the limitations and uncertainty of assessing human exposure to bombing range-specific contaminants?

[Comment] Yes, the summary report, in general, adequately describes the limitations and uncertainties of assessing human exposure to military activities. As the report recognized, the data used has strengths and weaknesses and cannot address the effect of mixture and cumulative exposure on the health of Viequenses.

ATSDR Response: Thank you for your comment—no revision required.

Are independent studies utilized appropriately in the determination of potential health hazards?

[Comment] Yes, independent studies contained in this report are properly used to determine the potential health hazard caused to the population of Vieques by past military activities. Most of these studies bring out new information and data to support new evidence and conclusions.

ATSDR Response: Thank you for your comment.

On the basis of ATSDR's reevaluation of historical bombing activities in the Vieques summary report, has ATSDR reached the appropriate conclusions and recommendations?

[Comment] Based on the data and information available ATSDR, in general, reached appropriate conclusions and recommendations. As stated in the report, more studies could be done in order to complete the whole picture of a really complex environmental and human health problem. At this moment, the information collected represents a lot of fragmented pieces of an environmental health puzzle**.** Therefore, an integrated and holistic approach could be developed for future studies.

ATSDR Response: ATSDR agrees and has made recommendations in the Report to 1) conduct a scientific survey of Viequenses to assess their seafood consumption and other food consumption, and 2) reanalyze the existing data using information obtained from the survey. ATSDR is not recommending a comprehensive, systematic biomonitoring effort at this time because we found little evidence of current exposure to contaminants from past military activities. Many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Public health officials could consider a targeted human biomonitoring investigation following the release of this report, for example, by testing for excessive mercury exposure from eating fish. If a biomonitoring investigation is conducted, it should include a comparison group from mainland Puerto Rico. If requested, CDC/ATSDR subject matter experts will provide technical assistance and support to public health officials or scientists in planning and conducting such an investigation. ATSDR also recommends that Viequenses who remain concerned about exposure to mercury, cadmium, other metals, or metalloids should consult their healthcare provider to discuss the need for and cost of testing.

Select the appropriate category below:

List recommended changes or reasons for not recommending)

- Recommend ( )
- Recommend with Required Changes (X)
- Not Recommended ( )

Additional Questions:

Are there any comments on ATSDR's peer review process?

[Comment] The peer review process is very well documented and guided.

ATSDR Response: Thank you for your comment.

Are there any other comments?

[Comment] Sometimes science is not sufficient to find answers to some environmental problems. Particularly, when dealing with the human health dimension of these problems. What is critical is to find the best possible response to the particular environmental health problem. In the case of Vieques, I think that a necessary approach is to develop a comprehensive epidemiological study of its population. This study should consider all human health conditions and diseases. It should also consider peoples' habits, cultural activities and personal and social environmental conditions. This study should be complemented with several studies recommended in the different sections of this report. But, instead of approaching the problem from an environmental perspective, it will be better to approach the analysis from a human health dimension. I completely agree that a future comprehensive study should investigate the combined and cumulative effects of exposure to military activities in Vieques.

ATSDR Response: ATSDR is mandated to focus on the environmental associations with adverse health effects. Also, it is not methodologically feasible to consider "all human health conditions and diseases." Understanding these two facets, the agency's approach is typically to identify environmental exposures of concern and then evaluate biologically plausible health effects possibly associated with these exposures.

References

[ATSDR] Agency for Toxic Substances and Disease Registry. 2001. Public health assessment, drinking water supplies and groundwater pathway evaluation, Isla de Vieques bombing range. Atlanta: US Department of Health and Human Services; October.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003a. Public health assessment, soil pathway evaluation, Isla de Vieques bombing range. Atlanta: US Department of Health and Human Services; February.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003b. Public Health Assessment, Air Pathway Evaluation, Isla De Vieques Bombing Range Vieques, Puerto Rico. Atlanta: US Department of Health Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry; 2003 Aug 26.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007 March. Toxicological profile for arsenic (update). Atlanta: US Department of Health and Human Services, Public Health Service.

[RCCPR] Commonwealth of Puerto Rico Department of Health. 2009. Cancer incidence and mortality in Vieques 1990–2004.

[CA ARB] California Air Resources Board. 1994. ARB memorandum dated 4/11/94 from A. Ranzieri to J. Brooks on the subject, "One-hour to Thirty-day Average Screening Factor."

Díaz E and Massol Deyá A. 2003. Trace element composition in forage samples from a military target range, three agricultural areas, and one natural area in Puerto Rico. Caribbean Journal of Science 39;2:215–20.

El Nuevo Día. 2001. They declare suitable the agricultural products in Vieques; 13 December.

García N, Lopez AM, Soto M, Rosado S, Berrios B. 2000. Environmental impact of Navy activities in Vieques. San Juan, PR: Servicios Cientificos y Tecnicos, Inc.; July.

Gioda et al. 2007. Particulate matter (PM_{10} and $PM_{2.5}$) from Different Areas of Puerto Rico. Fresenius Environmental Bulletin v. 16 No.8; 2007.

Hoffsommer JC and Glover DJ. 1978. Vieques litigation support: explosives analysis of water and soil samples taken on Vieques Island, Puerto Rico, May 11 through May 16, 1978. Silver Spring, MD: Naval Surface Weapons Center, Explosives Chemistry Branch.

Lai MG. 1978. Explosion products content of water and soil samples taken on Vieques Island, Puerto Rico, May 11 through May 16, 1978. Silver Spring, MD: Naval Surface Weapons Center; July.

Learned RE, Grove GR, Boissen R. 1973. A geochemical reconnaissance of the Island of Vieques, Puerto Rico. US Geological Survey and Puerto Rico Department of Natural Resources. Open-file report 73-155 (order number 1866).

López Morales JL. 2005. Determination of arsenic (As), cadmium (Cd), chromium (Cr), cobalt (Co) and lead (Pb) in "smooth cayenne" pineapple fruit, leaves tissue and soil using inductively coupled plasma-optical emission spectrometry (ICP-OES). Mayagüez, PR: University of Puerto Rico. Available at: http://www.conucopr.org/ViewRecord.do;jsessionid=9352FA03CF29511445F3F457499BD7F1?id=11161028 [accessed 2012 Aug 28].

Lopez Y, and Corrosquillo C. 2002. Exploratory study of health and other conditions in Vieques.

Massol Deyá A. 2002. Herbivorous: additional evidence of heavy metal mobilization through the food web. *In:* Ciencia y Ecología: Vieques en Crisis Ambiental, Third edition. Casa Pueblo de Adjuntas, PR.

Peshut PJ, Morrison RJ, Brooks BA. 2008. Arsenic speciation in marine fish and shellfish from American Samoa. Chemosphere 71(3);484-92.

[PRDOH] Commonwealth of Puerto Rico, Department of Health. 2006. Executive summary of the prevalence study of heavy metals in Vieques. San Juan: PR DOH; November.

Rodríguez Sierra CJ, Jimenez B. 2002. Trace metals in striped majarra fish (*Diapterus plumier*) from Puerto Rico. Marine Pollution Bulletin 44;1039-1045.

[USEPA] U.S. Environmental Protection Agency. 1992. Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised, October 1992, EPA-454/R-92-019. U.S. Environmental Protection Agency, Research Triangle Park, NC.

[USEPA] U.S. Environmental Protection Agency/Environmental Response Team (EPA/ERT). 2001. Field Data Summary, Vieques Fish Assessment, Vieques, Puerto Rico. November 8, 2001.

[USGS] U.S. Geological Survey. 1997. Collecting rocks. RM Barker. June 24, 1997. Available at http://pubs.usgs.gov/gip/collect1/collectgip.html, [accessed 2012 Aug 28].

[USGS] U.S. Geological Survey. 2001. Understanding our planet through chemistry. Compiled by JE Taggart. Edited by CA Bales and SK Runberg. Composition by ML Johnson. Graphics by RP Walker, ST Smith, and SM Smith.

[WHO] World Health Organization. 2001. Arsenic and arsenic compounds. Environmental Health Criteria 224, Geneva.

Appendix D Public Comments and ATSDR Responses

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009829

Introduction

ATSDR received comments from four individuals or groups regarding An Evaluation of Environmental, Biological, and Health Data from the Island of Vieques, Puerto Rico. Their comments are shown below along with ATSDR's response to their comments. Also, throughout our responses we refer to An Evaluation of Environmental, Biological, and Health Data from the Island of Vieques, Puerto Rico as simply "the Report."

Commenter 1

[Comment]

A 90 day period had been given for comments to the aforementioned document which took more than two years to prepare. The following critique is done only for those priority areas in which the College of Physicians and Surgeons of Puerto Rico has been directly involved.

During 2009, the College of Physicians and Surgeons of Puerto Rico expressed our opinion about the risk of exposure to military contaminants in Vieques and increased morbidity and mortality. As scientists, physicians, epidemiologists and as public health advocates, we identified the following priorities:

1. The need to obtain a full disclosure of ALL the contaminants (PHYSICAL, CHEMICAL, and/or BIOLOGICAL) utilized in Vieques' military activities by the USA, NAVY and NATO for more than 40 years.
2. Once the most potentially hazardous exposures are identified, the corresponding biological monitoring (in humans) has to be done in order to avert disease for this and the future generations of Viequenses.

To date, a full disclosure has not been produced. Thus, any evaluation of the exposure to military agents in Vieques is seriously limited by the lack of the most critical information.

With the aforementioned stated, we proceed to offer our professional opinion about some aspects of the ATSDR Evaluation.

a. Identification of Exposures by ATSDR

   The ATSDR did not formally nor systematically identify hazardous agents (chemical, physical or biological) utilized by the military forces in Vieques. Available information was not utilized and is hereby presented for their benefit.

   i. Since 1994, there is USA government documentation (Environmental Protection Agency's (EPA) Discharged Monitoring Reports) that identify excessive concentrations of military contaminants (NAVY had 102 discharge violations) in the coastal waters of Vieques. Docket no. RCRA-02-2000-7301. In particular, MERCURY was among the hazardous violations found by EPA.

      Page 5: D009 a solid waste exhibiting the toxicity characteristics of mercury pursuant to CFR 261.24

ATSDR Response: Docket no. RCRA-02-2000-7301 is a Consent Order that outlined the responsibilities of both the U.S. EPA and the Navy with regard to the Atlantic Fleet Weapons Training Facility. The Navy conducted a RCRA Facility Investigation (RFI) to determine fully the nature and extent of any release of hazardous wastes, solid wastes, or hazardous constituents at or from the facility. The U.S. EPA reserved the right to perform any and all work the Order required, including but not limited to any additional site characterization, feasibility study, or response or corrective action deemed necessary to investigate the facility thoroughly, or to protect human health or the environment.

ATSDR reviewed data on many contaminants at the Atlantic Fleet Weapons Training Facility. ATSDR's 2003 Soil public health assessment (PHA) reviewed the sample results of explosive residues and over 40 other chemicals. Sample results for over 300 different chemicals were considered as a part of the 2001 Drinking Water PHA, while the 2003 Air PHA modeled the potential exposure to nearly 100 different contaminants. This information was reviewed again as a part of the Report, which also considered information from studies published since completion of the 2001–2003 PHAs.

While the Consent Order does not contain the details of the 102 discharge violations, ATSDR was able to review correspondence between the EPA and the Navy that does discuss the Discharge Monitoring Reports and the exceedances of effluent limits set forth in the Navy's National Pollution Discharge Elimination System (NPDES) permit. ATSDR reviewed a list of 126 effluent violations that occurred between February 1994 and April 1999. Only one of the violations involved mercury. The violation for mercury was a reported value of 0.0014 mg/L, which was above the permitted limit of 0.001 mg/L but below the maximum contaminant level (MCL) of 0.002 mg/L. The Consent Order does not add to the understanding of the levels of mercury or other contaminants present in the different environmental media on the island or the amount to which persons were or are exposed.

[Comment]

3. On February 11, 2005, EPA's Region 2 documented once again the presence of mercury as one of the many contaminants in Vieques originated by the Atlantic Fleet Training Area (Vieques).

Threat of contaminants

Extensive amounts of unexploded ordinance [sic] and remnants of exploded ordinance [sic] have been identified in the range areas of Vieques, and in the surrounding waters. Hazardous substances associated with ordinance [sic] may include mercury, lead, copper, magnesium, lithium, perchlorate, TNT, napalm and depleted uranium among others. At the Eastern Maneuver Area, and in the NASD, the hazardous substances present a range of chemicals such as PCB, solvents and pesticides.

ATSDR Response: The U.S. EPA's Site Narrative of February 11, 2005 lists only the *possible* contaminants associated with unexploded ordnance. The Site Narrative does not contain any sampling results or other environmental data. Consequently, it does not add to the understanding of mercury levels or other contaminants in the island's environmental media or to the understanding of mercury levels to which persons were or are exposed.

In the Report, ATSDR staff reevaluated the list of possible contaminants in unexploded ordnance, particularly mercury. The following sections in Chapter 1.3 (Chemicals in Munitions and Detonation Byproducts) provide detailed information about organic and inorganic components in various ordnance types in the Vieques environment and in human exposure pathways:

1.3.1. Organic Compounds in Munitions and Detonation Byproducts

1.3.2. Inorganic Compounds in Munitions and Detonation Byproducts

1.3.3. What Bomb-related Constituents Were Found in Vieques

1.3.4. Linking Contaminants in Vieques with Human Exposure Pathways

With regard to mercury in military ordnance, the Report states:

> "Because mercury was historically used in certain detonators, mercury has been mentioned as a contaminant of concern (García et. al. 2000). Bomb casing composition data originally provided by the Navy indicated that casings did not contain mercury, which is consistent with information on Material Safety Data Sheets available for steel. The 2003 Air PHA indicated that the total annual estimated mercury emissions from high explosive ordnance used at the [Live Impact Area] (LIA) were very low (i.e., less than 0.5 kg per year). Following detonation, the mercury was presumably a trace constituent in the explosive charge and in the soil ejected into the air. Therefore, the live ordnance dropped on the LIA does not appear to be a significant mercury source in the Vieques environment".

To provide perspective, ATSDR has added information from the mercury National Emission Standard for Hazardous Air Pollutants (NESHAP) for allowable mercury air emissions from chlor-alkali plants. Developed in 1973 and last amended in 2000 by US EPA, this standard allows a chlor-alkali plant to release 2.3 kg of mercury daily into the atomosphere. Therefore, the annual estimated mercury emissions from high explosive ordnance in the LIA (less than 0.5 kg per year) can be compared with the NESHAP standard for chlor-alkali plants (844 kg per year). More information is available at: http://www.gpo.gov/fdsys/pkg/CFR-2011-title40-vol8/xml/CFR-2011-title40-vol8-part61.xml#seqnum61.52.

In addition, if military ordnance were a significant source of mercury in the environment, soil in the LIA should have significantly higher mercury levels than surrounding soils. The Report states in Section 1.3.3, however,

> "Mercury was also considered in this data comparison. The average mercury levels in LIA soils from the 2000 dataset was 0.02 ppm, and the highest level was 0.086 ppm (CH2MHILL 2000). Only one of 29 soil samples had a mercury level that could be considered above naturally occurring levels when compared with the background upper tolerance levels (UTLs) (0.057–0.31 ppm; CH2MHILL 2007)".

[Comment]

1. To this date, no other local sources of mercury, uranium nor napalm contamination have been identified in Vieques other than military activities.

   ATSDR Response: While no civilian uses of napalm have been documented on Vieques, both the military and civilians used gasoline—one of the main napalm ingredients (Reich and Sidel 1967; Department of the Army 1984).

   With regard to uranium, it is a naturally occurring radionuclide present in nearly all rocks. It is also present in soils, which are derived from rock erosion. Uranium is also a trace contaminant of phosphate fertilizers (ATSDR 1999). ATSDR's 2001 Drinking Water PHA addressed the incident involving the firing of 263 depleted uranium rounds onto the LIA. During June 6–15, 2000, the Nuclear Regulatory Commission's (NRC) conducted an inspection of Vieques to determine whether the depleted uranium rounds contaminated the environment. The NRC found that the only detections of uranium, at levels above the natural background level, were measured in five samples collected in the holes where DU rounds were found, and concluded that depleted uranium had not spread to areas outside the LIA. Thus the public had not been exposed to depleted uranium contamination or other radiation above normal background (i.e., naturally occurring) levels (NRC 2000). ATSDR reviewed the NRC report and concluded that the background levels of radiation detected on Vieques did not present a public health hazard.

   The Report also identifies other possible mercury sources. For example, case histories from the scientific literature show that broken mercury thermometers and reclaimed dental fillings can expose residents to mercury in the home. In addition, ATSDR has added religious rites using mercury-containing capsules as a possible source of mercury exposure from indoor air.

[Comment]

2. Military activities in Vieques were and are not limited to the east end of the island. The Naval Ammunition Storage Detachment (NASD) is in the west end of Vieques.

   ATSDR Response: As this commenter mentions in a later comment, ATSDR's fish sampling included fish samples from the west end of Vieques. The Report also references several studies that include data from environmental samples collected at the western end of the island. Some of these studies include the following:

   Baker Environmental, Inc. (Baker). 1999. Vieques groundwater sampling information. November 15, 1999.

   Bauer LJ and Kendall MS, eds. 2010. An Ecological Characterization of the Marine Resources of Vieques, Puerto Rico Part II: Field Studies of Habitats, Nutrients, Contaminants, Fish, and Benthic Communities. NOAA Technical Memorandum NOS NCCOS 110. Silver Spring, MD.

   CH2MHill 2002. Final soil, groundwater, surface water, and sediment background investigation report, US Naval Ammunition Support Detachment, Vieques Island, and Puerto Rico. Tampa, FL: CH2MHill; October.

   CH2MHill. 2007a. Remedial investigation report for solid waste management unit (SWMU) 6, CH2MHill; February.

CH2MHill 2007b. Remedial investigation report for area of concern (AOC) J, CH2MHill; May.

CH2MHill. 2007c. Remedial investigation report for area of concern (AOC) H, CH2MHill; July.

CH2MHill. 2008a. Remedial investigation report for solid waste management unit (SWMU) 7. Tampa, FL: March.

CH2MHill. 2008d. Remedial investigation report for area of concern (AOC) I. Tampa, FL: June.

CH2MHill. 2008e. Remedial investigation report for area of concern (AOC) E. Tampa, FL: July.

Hoffsommer JC and Glover DJ. 1978. Vieques litigation support: explosives analysis of water and soil samples taken on Vieques Island, Puerto Rico, May 11 through May 16, 1978. Silver Spring, MD: Naval Surface Weapons Center, Explosives Chemistry Branch.

[Comment]

3. Thus, in addition to mercury global contamination, for decades Vieques has been recipient of mercury contamination of military origin.

   ATSDR Response: Bomb casing composition data originally provided by the Navy indicated that casings did not contain mercury, which is consistent with information on Material Safety Data Sheets available for steel. However, the Bang Box studies showed that small amounts of mercury could be present in the explosive charge of some bombs. The 2003 ATSDR Air PHA indicated that the total, annual estimated mercury emissions from high-explosive ordnance used at the LIA were very low (i.e., less than 0.5 kg per year). To provide perspective, ATSDR has added information from the mercury National Emission Standard for Hazardous Air Pollutants (NESHAP) for allowable mercury air emissions from chlor-alkali plants. Developed in 1973 and last amended in 2000 by US EPA, this standard allows a chlor-alkali plant to release 2.3 kg of mercury daily into the atomosphere. Therefore, the annual estimated mercury emissions from high explosive ordnance in the LIA (less than 0.5 kg per year) can be compared with the NESHAP standard for chlor-alkali plants (844 kg per year). More information is available at: http://www.gpo.gov/fdsys/pkg/CFR-2011-title40-vol8/xml/CFR-2011-title40-vol8-part61.xml#seqnum61.52.

   The estimate of less than a kg of mercury per year took into account the small amount of mercury present in some explosive charges and the amount from soil ejected into the air. But most mercury levels in LIA soils appear to be at naturally occurring levels, with no significant spatial variation to suggest that mercury levels are higher at the LIA. Therefore, the small amount of mercury in some bombs was not sufficient to raise mercury soil levels in the LIA, the most heavily bombed area on Vieques. Additionally, ATSDR's fish sampling investigation did not find unusually elevated tissue concentrations of mercury in Vieques reef fish compared with levels reported by the FDA (FDA 2010). Therefore, the live ordnance dropped on or fired into the LIA does not appear to be a significant mercury source in the Vieques environment. (See the Report, Chapter 1, Section 1.3.)

[Comment]

4. After evaluating our 2004 publication, ATSDR concluded that women of reproductive age and children in Vieques are exposed to mercury levels that can be neurotoxic to their fetuses.

   We and the consensus of the ATSDR reviewers agree.

   a. Mercury contamination in women of reproductive age in Vieques is a fact. While only 6.6% of the similar women sampled in the larger island of Puerto Rico had mercury levels that could cause neurologic disease to the developing fetuses, in Vieques 26.8% had such levels. Samples were done on or before 2003, before bombing ceased. As a reference, in the continental USA only 7% of the sampled women exceed the [reference dose] (RfD). Margin of exposure analysis also revealed an unsafe mercury exposure for Viequenses. (Ortiz Roque and Rivera López 2004). This study has been published and internationally peer reviewed.

   b. The only variable that correlated with mercury hair levels in women was LOCAL FISH CONSUMPTION.

   c. Unpublished data from the same researchers revealed that children 5 years of age or younger in Vieques were also dangerously exposed to mercury. While the margin of exposure was 30 in USA children, the margin of exposure in Vieques children was 4.4. A margin of exposure of less than 10 is considered dangerous for a population (Ortiz Roque and López Rivera 2004).

   d. Thus, direct bio monitoring and hair mercury levels' sampling of the most susceptible humans in Vieques-using internationally validated methodology-demonstrated significant mercury internal exposure and potential neurologic human disease.

   Mercury findings exemplify the importance of identifying exposures and the proper human bio monitoring.

   An indirect approach, pathway analysis and use of sentinel species, requires extensive ecological knowledge and investigation which is not always available, as was demonstrated by the fish sampling studies done by ATSDR.

   ATSDR Response: ATSDR agrees that if some pregnant Viequense women are high fish consumers, their fetuses are at risk of harmful effects later in life. We presented this conclusion in the Report and recommended development of an educational program about mercury in fish that incorporated local habits and information about Viequense seafood consumption.

   The cited 2004 publication reported statistics on 41 Viequense women together with information about seafood consumption. The publication compared these data with the main island of Puerto Rico and with results from the 1999 NHANES survey in the United States. While this study showed that higher mercury levels were associated with Viequense higher fish consumption, the patients in this investigation were not randomly selected and the number of participants was very low (i.e., 41 women). Therefore, the prevalence of Viequenses with elevated mercury levels in hair is uncertain because a systematic survey of mercury levels in hair was not conducted

The 2006 PRDOH study did not find excessive mercury levels in blood. The study either did not measure for or did not report mercury level in hair. Therefore the Report concludes that some uncertainty remains concerning the prevalence of high Viequense mercury levels.

Finally, the Report concludes these biomonitoring results do not permit any conclusions about exposure to the bombing-related contaminants. (See Report, Chapter 3, Section 3.5.)

[Comment]

5. FISH sampling studies done by ATSDR:

    a. The hypothesis of the fish sampling study done by ATSDR was not clearly delineated.

    ATSDR Response: ATSDR's 2001 sampling and analysis activities focused on whether muscle tissues from commonly consumed fish and shellfish contained levels of heavy metals and explosive compounds that would result in health effects (see Section IV of ATSDR's 2003 Fish and Shellfish PHA). In designing the sample plan and in collecting the samples, ATSDR partnered with the U.S. EPA's Environmental Response Team, which has extensive experience in sampling and analyzing fish. In addition, ATSDR consulted with the petitioner and local residents to advise us on the sampling plan.

[Comment]

    b. It seems to be implicit in their analysis that if the mercury contamination was produced at the LIA, it will not sublimate, travel by air, wind or water to the rest of Vieques and surrounding waters.

    Fish were sampled at several locations that include LIA, Isabel Segunda, the market place and the west end of Vieques.

    ATSDR Response: The Report states that some migration of contaminants from the soils to the marine environment undoubtedly occurs at Vieques, just as weathering of soils naturally occurs in virtually any shoreline setting. Precipitation and wind can carry soils from the LIA—and can carry chemicals found in the LIA soils—either directly into marine waters or into inland lagoons. And those lagoons have the potential to overflow into the nearby ocean. Although migration of contaminants clearly occurs, the rate and amount of chemical migration are important factors in determining whether significant amounts of mercury are actually entering the marine environment. From our evaluation of LIA soils, significant amounts of mercury from LIA soils are not entering the marine environment. Nevertheless, to investigate this possible exposure pathway further, ATSDR collected fish from several reefs near Vieques. One of the strengths of ATSDR's fish sampling plan was that it collected fish from five different locations; and, land crabs were collected from three surface locations. The fish sampling investigation did not find unusually elevated tissue concentrations of mercury in Vieques reef fish.

[Comment]

c. ATSDR did not have knowledge (and still is devoid of knowledge) of the alimentary habits of Vieques women and children. In particular, the researchers did not know which species of fish and at which frequency these groups consume fish in Vieques.

ATSDR Response: ATSDR used multiple information sources to identify the most commonly caught and consumed species of fish and shellfish for collection during the 2001 fish sampling investigation. One important source was a Puerto Rican university report containing survey information about the frequency and types of fish eaten (Caro 2000). ATSDR substantiated the survey results with information from the petitioner and other residents, the Vieques Special Commission Report, and visits to the local fish markets on Vieques. Nevertheless, ATSDR agrees that more information about Viequenses fish-eating habits could either verify the findings of the Report or allow a more refined assessment of eating mercury-containing fish. The Report recommends a survey of Vieques residents to determine the types, frequency, and quantity of fish consumed. Information from this survey could then guide additional risk assessments and statistical analyses if necessary. But any findings that result from such assessments and analyses are not likely to change the fact that pregnant women who are high fish consumers are at risk of excessive mercury exposure, which might harm their developing fetuses (unborn baby).

[Comment]

d. ATSDR did not know (and still do not acknowledges) [sic] that military activities in Vieques are the only proven source of mercury contamination.

ATSDR Response: It is unlikely that military activities are a significant source of mercury in Viequenses. Bomb casing composition data originally provided by the Navy indicated that casings did not contain mercury, which is consistent with information on Material Safety Data Sheets available for steel. The Bang Box studies, however, showed that small amounts of mercury could be present in the explosive charge of some bombs. The 2003 ATSDR Air PHA indicated that the total, annual estimated mercury emissions from high-explosive ordnance used at the LIA were very low (i.e., less than 0.5 kg per year). To provide perspective, ATSDR has added information from the mercury National Emission Standard for Hazardous Air Pollutants (NESHAP) for allowable mercury air emissions from chlor-alkali plants. Developed in 1973 and last amended in 2000 by US EPA, this standard allows a chlor-alkali plant to release 2.3 kg of mercury daily into the atomosphere. Therefore, the annual estimated mercury emissions from high explosive ordnance in the LIA (less than 0.5 kg per year) can be compared with the NESHAP standard for chlor-alkali plants (844 kg per year).

The estimate of less than a kg of mercury per year took into account the small amount of mercury present in some explosive charges and the amount from soil ejected into the air. But most mercury levels in LIA soils appear to be at naturally occurring levels, with no significant spatial variation to suggest that mercury levels are higher at the LIA. Therefore, the small amount of mercury in some

bombs was not sufficient to raise mercury soil levels in the LIA, the most heavily bombed area on Vieques.

Mercury exposure from frequently eating fish could be a significant source of mercury for some Viequenses. The Report also points out other possible sources of mercury. For example, case histories from the scientific literature show that residents can be exposed in the home from broken mercury thermometers and from reclaimed dental fillings. In addition, ATSDR has added other sources that could increase metal exposure, such as cooking utensils, metal residues in foods (e.g., tea and vegetables), consumption of drinks with metallic packaging, religious rites using mercury-containing capsules, skin creams, antacid formulations and antiperspirant formulations.

[Comment]

e. ATSDR does not know what the typical portion of fish consumed by Viequenses women and young children is.

f. ATSDR is devoid of knowledge of the typical weight of Viequenses.

g. Ortiz Roque and Lopez Rivera's extensive questionnaire has information on Viequenses' weight, body mass index, diet, fish species preference, but it was not consulted by ATSDR researchers.

ATSDR Response: Because of suggestions during the November 2009 scientific consultation, ATSDR specifically considered lower than standard body weights in the Report. Then ATSDR used a range of age-specific meal portion sizes and a wide range of body weights to calculate mercury exposure doses. From these calculations, ATSDR concluded that women with a varied fish diet who typically ate more than 2 oz. of fish every day had estimated mercury doses that exceeded U.S. EPA's chronic RfD, and children with a varied fish diet who typically ate more than 0.5 oz of fish every day had estimated mercury doses that exceeded U.S. EPA's chronic RfD.

ATSDR also has requested that the commenter share his or her information about Viequenses' weight, body mass index, diet, and fish species preference.

[Comment]

h. ATSDR did not know what the food chain relations in Vieques are.

ATSDR Response: The food chain involving mercury cycling in the environment is well-known and is the most likely source of mercury in marine food surrounding Vieques. Nevertheless, even with knowledge of this well-established mercury cycle, ATSDR investigated whether bombing-related activities could have contributed significant amounts of mercury to the Vieques environment. Our evaluation shows that while small amounts of mercury were present in some military ordnance, the amount was not sufficient to affect mercury levels in the environment or in fish. Elevated mercury levels in LIA soil were not detected, and the amount of mercury introduced to the Vieques environment from bombs was very small.

[Comment]

i. ATSDR analysis of mercury reference dose was based on fish sampling that had multiple limitations which included no stated hypothesis, no rational criteria for the species sampled, no confirmation of market fish origin and sampling consisting of unique individuals for some species.

1. ATSDR arrived at the conclusion that the average mercury concentration in Vieques' fish was 0.1066 ppm. It is noteworthy to mention that this is 200% above the EPA screening level (0.049 ppm).
2. ATSDR does not know if the sampled fish were representative of Viequenses diet.
3. ATSDR did not find a significant difference between the mercury concentrations in fish in the different locations in Vieques. Most averaged above EPA's screening level.
4. Evidently, ATSDR has assumed knowing in which putative sites mercury can be found in Vieques as well as knowing about its environmental dispersion and bioaccumulation. ATSDR assumed that fish stay still in one coral reef.

ATSDR Response: ATSDR's 2001 sampling and analysis activities focused on whether muscle tissues from commonly consumed fish and shellfish contained levels of heavy metals and explosive compounds that might result in health effects. In designing the sample plan and in collecting the samples, ATSDR partnered with the U.S. EPA's Environmental Response Team, which has extensive experience in sampling and analyzing fish. In addition, ATSDR consulted with the petitioner and with local residents for sampling plan advice. The six sample locations were chosen to represent productive fishing areas surrounding the island, which also accounts for fish that might move from reef to reef. ATSDR used multiple information sources to identify the most commonly caught and consumed species of fish and shellfish, including the petitioner and other residents, a Puerto Rican university report (Caro 2000), the Vieques Special Commission Report, and visits to the local fish markets on Vieques. The Report does acknowledge that the agency's 2001 fish sampling had some limitations. Most notably, though the fish sampling investigation was designed using the best criteria of the time, the 2001 sample design was not consistent with some criteria found in current U.S. EPA and the State of California sample designs for developing fish consumption advisories. (See the Report, Chapter 2, Section 2.2.3.)

Even with these limitations, ATSDR's fish sampling data showed mercury in fish was highly dependent on fish family and on fish species. Mercury was higher in two fish families (i.e., grunt and hind, 0.18 ppm and 0.12 ppm, respectively) compared with other families sampled (e.g., parrotfish and snapper, 0.01 ppm, and 0.05 ppm, respectively). Mercury in hind was associated with fish weight; larger hind typically had higher concentrations of mercury. For the 104 fish samples collected from the marine areas near Vieques and from the commercial fish market, the average mercury level was 0.1 ppm and the range was nondetectable to 0.33 ppm. The mercury levels detected in

Vieques reef fish are similar to levels reported by the FDA (FDA 2010). The mercury levels are in the low (0.02–0.2 ppm) to mid (0.2–0.6 ppm) mercury range identified by FDA in its recent risk and benefit assessment (FDA 2009).

The average mercury level in all fish (0.1 ppm) exceeded EPA's screening level of 0.049 ppm, which EPA developed for subsistence fishers[41] (US EPA 2000). Note that exceeding a screening level does not mean that harmful effects are possible but rather that a more detailed health evaluation is needed. ATSDR conducted this more detailed health evaluation by estimating the amount of mercury consumed using a range of daily fish intakes and body weights. This approach allowed ATSDR to evaluate the possibility of harmful effects for Viequenses who are occasional fish eaters as well as those who are frequent fish eaters. The Report did identify mercury exposure from frequent consumption of marine seafood as a potential public health hazard. Women with a varied fish diet who typically eat more than 2 oz of fish every day have estimated mercury doses that exceed U.S. EPA's chronic RfD. And as portion size increases, the estimated doses approach the lowest level known to cause harmful effects to a developing fetus. ATSDR concludes that if women who are pregnant consume more than 2 oz of fish daily , their developing babies have a small increased risk of neurological effects later in life. The risk of harmful effects increases as portion size increases. Possible harmful effects identified from studies of non-Viequense children exposed *in utero* involve language, attention, and memory, and, to a lesser extent, visual/spatial and motor functions.

[Comment]

6. ATSDR ignores (does not know or refuses to acknowledge) EPA's reports on mercury contamination in Vieques.

   ATSDR Response: As stated earlier, ATSDR was able to review correspondence between the U.S. EPA and the Navy that does discuss the Discharge Monitoring Reports and the exceedances of effluent limits set forth in the Navy's National Pollution Discharge Elimination System (NPDES) permit. ATSDR reviewed a list of 126 effluent violations on Vieques that occurred between February 1994 and April 1999. Only one of the violations involved mercury. The violation for mercury was a reported value of 0.0014 mg/L, which is above the permitted 0.001 mg/L limit but below the MCL of 0.002 mg/L.

[Comment]

[41] U.S. EPA defines subsistence fish eaters as persons who rely on noncommercially caught fish and shellfish as a major source of protein in their diets. In developing their screening level for subsistence fishers, U.S. EPA used a daily fish consumption rate of 5 ounces (142 grams) per day. More information about U.S.EPA's screening levels is available at: http://www.epa.gov/waterscience/fish/advice/volume2/v2cover.pdf.

7. In addition to global mercury contamination, for many years Vieques has had a significant additional source of mercury contamination: military activities.

ATSDR, based on their fish sampling study, concluded that mercury in humans was most probably due to GLOBAL mercury contamination: *non sequitur*

ATSDR Response: ATSDR concluded mercury levels in fish in and around the LIA are most likely the result of the global reservoir of mercury circulating in the environment. This conclusion is supported by the low mercury levels in LIA soils, which appear to be at naturally occurring levels. Additionally, air modeling estimated that from the ordnance dropped on the LIA, less than 0.5 kg per year of mercury would have been emitted into the air. To provide perspective, ATSDR has added information from the mercury National Emission Standard for Hazardous Air Pollutants (NESHAP) for allowable mercury air emissions from chlor-alkali plants. Developed in 1973 and last amended in 2000 by US EPA, this standard allows a chlor-alkali plant to release 2.3 kg of mercury daily into the atomosphere. Therefore, the annual estimated mercury emissions from high explosive ordnance in the LIA (less than 0.5 kg per year) can be compared with the NESHAP standard for chlor-alkali plants (844 kg per year). (See the Report, Chapter 1, Section 1.3.)

[Comment]

8. ATSDR Educational document for the Viequenses cannot be followed by a highly educated human.

    a. ATSDR concludes that women of reproductive age in Vieques who eat 14 ounces of fish per week exceed the EPA reference dose of 0.1 microgram/kg/day (2 ounces per day). Thus, are exposing their fetuses to a neurologic disease.

    b. ATSDR recommended women of reproductive age in Vieques to consume up to 12 ounces of fish per week. (1.7 ounces per day).

    c. ATSDR does not know what the typical portion of fish per meal in Viequenses diet is, but assumed the portion is 4 ounces.

    d. ATSDR assumes that the typical human can establish the difference between 12 ounces and 14 ounces of fish per week OR between 1.7 ounces and 2 ounces portions of fish per day.

    e. There is no evidence that a highly educated human can follow these recommendations simply because the difference between healthy fish consumption and risky fish consumption is very close for Viequenses. Again, the margin of exposure is high for these susceptible groups (fetus and children).

    f. The FDA establishes a typical fish portion to be 6 ounces. Why use 4 ounces for Vieques?

    ATSDR Response: ATSDR agrees that much of this information is too technical for the public at large. Therefore, ATSDR has developed a fish consumption fact sheet that explains our findings, conclusions, and recommendations in plain language. The fact sheet also includes diagrams and pictures to help Viequenses understand the difference between risky and healthy fish consumption. We shared this fact sheet with the PRDOH. We are currently

awaiting their input on how to make the ATSDR fact sheet more relevant and useful to Viequenses.

ATSDR evaluated a range of age-specific meal portion sizes in the Report, including the 50th, 95th, and 99th percentiles. Table 2-6 shows the meal sizes for the various ages evaluated. Table 2A-1 shows the estimated doses, which were calculated on a continuous weight profile from 9.9 to 220 pounds and increasing meal size up to the 99th percentile (e.g., teenagers 15 to 18 years have a 99th percentile fish-meal portion of 20 ounces). This approach allowed ATSDR to evaluate the risk of harmful effects for Viequenses who are casual fish eaters as well as those with high fish consumption and to develop health messages for both groups.

[Comment]

9. ATSDR analysis of mortality studies 1991-1998.

   Standardized mortality studies were done utilizing death certificates' data universally collected by the Puerto Rico Department of Health. Death certificates are completed by licensed physicians in Puerto Rico. No significant disease coding changes took place during the study period. Thus, there is no reason to believe that this data collection was biased during 1991-1998.

   ATSDR Response: The Report does not claim that the data collection was biased due to changes in disease coding. The Report does point out, however, that the mortality data presented did not enumerate all causes of death—only those that were elevated.

[Comment]

10. Biological monitoring

    Again, as it has been the opinion of the College of Physicians and Surgeons of Puerto Rico for the last 2.5 years, appropriate knowledge of all the exposures and strategic biological monitoring are critical for human risk evaluation. Disease prevention is the objective of public health.

    ATSDR 2011 conclusions about lack of human internal exposure to military contaminants in Vieques is not evidenced based.

    ATSDR Response: Because the Report found little evidence of exposure to contaminants from the site, a comprehensive biomonitoring program is not justified at this time. In addition, many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Nevertheless, ATSDR is currently working with our partners in Puerto Rico to explore the value of conducting a targeted human biomonitoring program in Vieques. For example, residents are concerned about mercury exposure from eating local seafood. Therefore, measuring mercury in blood has public health value for Viequenses.

ATSDR References:

[ATSDR] Agency for Toxic Substances and Disease Registry. 1999. Toxicological profile for uranium. Atlanta: US Department of Health and Human Services, Public Health Service; September.

Caro DA, Nazario M, Díaz N. 2000. Toxicological survey of heavy metals in fish populations, Vieques Island. Universidad Metropolitana, School of Environmental Matters. June 2000.

Cooper KR, Burton DT, Goodfellow WL, Rosenblatt DH. 1984. Bioconcentration and metabolism

Department of the Army, Headquarters. 1984. Military Explosives. Department of the Army Technical Manual, TM 9-1300-214. September. Last accessed February 25, 2013 at: http://www.archive.org/stream/TM_9_1300_214_Military_Explosives_/Ia_tm9-1300-214MilitaryExplosives_djvu.txt .

[FDA] Food and Drug Administration. 2010. Mercury concentrations in fish: FDA monitoring program. Last accessed May 18, 2012 at: http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm191007.htm , and http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm115644.htm .

[FDA] Food and Drug Administration. 2009. Report of quantitative risk and benefit assessment of consumption of commercial fish, focusing on fetal neurodevelopmental effects (measured by verbal development in children) and on coronary heart disease and stroke in the general population, January 15, 2009. Last accessed May 18, 2012 at: http://www.fda.gov/Food/FoodSafety/Product-SpecificInformation/Seafood/FoodbornePathogensContaminants/Methylmercury/ucm088794.htm .

[NRC] Nuclear Regulatory Commission. 2000. Environmental Survey Inspection Report, Vieques Island, Puerto Rico: September 28, 2000.

Reich P and Sidel VW. 1967. Napalm. New England Journal of Medicine, 244: 86–88. July. Last accessed 2012 May 17: http://www.nejm.org/doi/full/10.1056/NEJM196707132770207. [USEPA] US Environmental Protection Agency. 1999. Region 2 Memorandum to C.E. Cassidy, Commander, CEC, US Navy. Re: Vieques Naval Atlantic Fleet Weapons Training Facility ('Navy"), Request for additional information ("Request 2") Relative to Permit PRG990001 ("Permit") and Multi-Sector General Permit Number PRR05A08F ("MSGP"), Attachment 3; September 29.

[USEPA] US Environmental Protection Agency, Region 2. (undated). Administrative Order on Consent, Docket No. RCRA-02-2000-7301. Last accessed 2012 May 18: http://www.epa.gov/region2/waste/3008ord.pdf.

[USEPA] US Environmental Protection Agency. 2000. Guidance for assessing chemical contaminant data for use in fish advisories. Fish sampling and analysis. Third Edition, Volume 1. Available at: http://water.epa.gov/scitech/swguidance/fishshellfish/techguidance/risk/upload/2009_04_23_fish_advice_volume2_v2cover.pdf. [accessed 2012 Aug 275].

Commenter 2

[Comment]

This letter serves as my formal comment on the report entitled "An Evaluation of Environmental, Biological, and Health Data From the Island of Vieques, Puerto Rico," which was released by the Agency for Toxic Substances and Disease Registry (ATSDR) in draft form on December 8, 2011.[1]

As you know, I represent the 3.7 million residents of Puerto Rico, including the nearly 10,000 residents of Vieques, in the U.S. Congress. Since taking office in January 2009, I have met with the Director or Acting Director of ATSDR on five occasions to discuss the health and environmental issues addressed by the draft report. Specifically, I have met with Dr. Howard Frumkin on September 22, 2009 and October 22, 2009; with Dr. Henry Falk on February 4, 2010; and with you on December 14, 2010 and December 7, 2011. During our most recent meeting, you briefed me on the conclusions and recommendations in the draft report. Upon receiving that briefing, I expressed concerns related to both process and substance. Now that I have had a chance to study the draft report more closely, I would like to elaborate on those concerns and to make six specific recommendations.

At the outset, it is important to make two points clear. First, my objective in providing comments on the draft report is to propose a path forward that will enable the federal government to fulfill its obligations to the people of Vieques—American citizens who were asked by the federal authorities to sacrifice a great deal to advance our nation's military readiness and security. Thus, my comments, even where critical, are intended to be constructive. The people of Vieques do not benefit one iota from attacks on ATSDR, the Navy or any other federal agency. What residents want—and what they deserve—is a realistic, evidence-based and fair-minded evaluation of the draft report that is designed to produce concrete action on the part of the federal government.

Second, since ATSDR issued its draft report, a three-judge panel of the U.S. Court of Appeals for the First Circuit, in a 2-1 vote, dismissed the tort claims that had been filed by over 7,000 residents of Vieques against the U.S. government.[2] The plaintiffs' claims are rooted in the argument that they have suffered harm to their health as a result of the Navy's negligence in emitting certain contaminants during training exercises conducted over the course of six decades.

The appellate panel dismissed the case on procedural, not substantive, grounds, concluding that the plaintiffs did not have standing under the Federal Tort Claims Act to pursue damage claims against the United States. The majority opinion explicitly stated that it took "no position on whether the Navy's operations on Vieques have had adverse health effects on the island's residents." In fact, the court said that the plaintiffs' pleadings, if true, "raise serious health concerns." By virtue of its ruling, however, the First Circuit has made it impossible for the merits of plaintiffs' claims and the Navy's defenses to be adjudicated by a judge or jury. A trial would presumably have required the Navy to provide a comprehensive account of its training activities—what weapons it used, where it used them, and for how long it used them. It would also have enabled both the plaintiffs and the defense to call scientific experts to testify about the prevalence of certain diseases on Vieques and the causal relationship between those diseases and past military training activities on the island.

Unless the panel's decision is reversed by the First Circuit sitting en banc or by the U.S. Supreme Court, the opportunity for residents of Vieques to establish facts and seek relief through the judicial system will be foreclosed. This means that the people of Vieques, and their elected leaders, have no alternative but to turn to the other two branches of their national government: the legislature and the executive. That explains why the First Circuit panel, at the close of its opinion, took the unusual but appropriate step of stating that the health issues raised by residents of Vieques in their pleadings "should be brought to the attention of Congress." Indeed, the judges instructed the clerk of the court to "send a copy of this opinion to the leadership of both the House and Senate."

On November 18, 2011, in anticipation of the release of the ATSDR report, I formally requested that the House Committee on Natural Resources hold a hearing "to assess the ongoing federal response to the significant land use, environmental and public health challenges facing the U.S. citizens of Vieques, Puerto Rico."[3] I intended to recommend to the Committee that it invite you or your designee to appear as a witness at this hearing, in order to provide testimony about the conclusions in the ATSDR report and to explain what action the agency intends to take—or recommend that other federal agencies take—going forward.

In summary, ATSDR's draft report expressly acknowledges that multiple human health studies performed since 1999 indicate that residents of Vieques have higher rates of cancer and other chronic illnesses than residents of mainland Puerto Rico. The principal questions that ATSDR seeks to answer in its report is whether there is evidence to show or suggest a causal relationship between these identified health problems and the military training exercises that were conducted on Vieques.

The draft report is a re-evaluation of the conclusions reached by ATSDR in a series of public health assessments that the agency conducted on Vieques between 2001 and 2003. In those assessments, ATSDR examined four "pathways" through which residents of Vieques could potentially have been exposed to training-related contaminants harmful to their health—air, soil, fish and shellfish, and groundwater. ATSDR concluded that, although there were contaminants attributable to the training exercises in certain of those pathways, they were not at levels expected to cause adverse health effects. The methodology used by ATSDR and the conclusions reached by the agency in those public health assessments were subject to significant criticism in Puerto Rico, in sectors of the scientific community, and in Congress.

In the draft report, ATSDR again examined these four pathways. It also examined a fifth pathway—the consumption of produce and livestock grown on Vieques. Although there are some modest differences, the overarching conclusion reached by ATSDR in the draft report is essentially the same as the conclusion reached by the agency in its earlier public health assessments—specifically, that the available scientific data does not establish or indicate that the contaminants in these pathways, certain of which can be linked to the military training activities, were at levels expected to cause the adverse health effects that residents of Vieques have been experiencing.

However, as ATSDR repeatedly acknowledged throughout the draft report, this conclusion is by no means definitive—or even close to it—because the available scientific data upon which the agency relies is incomplete and insufficient in many respects.

To its credit, ATSDR recommends in the draft report that further studies be conducted to fill in some of the identified data gaps so that more credible and reliable conclusions can be drawn, particularly in connection with the fish, local produce and livestock, and soil pathways. My fundamental criticism of the draft report is that the agency does not go nearly far enough. In a November 2009 release to the public, ATSDR stated it had "identified gaps in environmental data that could be important in determining health effects." ATSDR further stated that it "expects" to "recommend biomonitoring to determine whether persons living on Vieques have been exposed to harmful chemicals, and, if so, at what levels those chemicals may be in their bodies." Yet, in a startling move that appears both inconsistent with its November 2009 release and contrary to common sense, ATSDR states in the draft report that "it is not recommending a comprehensive, systematic biomonitoring effort at this time," although "public health officials could consider a targeted human biomonitoring investigation."[4] This recommendation is made despite the fact that ATSDR expressly acknowledges that the existing "biomonitoring data available for Viequenses cannot be used to determine whether residents of Vieques were exposed to past, military exercise-related constituents [i.e., contaminants]."[5]

I respectfully, but strongly, submit that this recommendation is inadequate. Given the reported health problems on Vieques, and the possible link between those problems and military training activities, such half-measures are misplaced. And it is certainly neither acceptable nor appropriate for ATSDR to suggest that such half-steps, if they are undertaken, ought to be conducted by local public health officials using primarily local funds.

In light of the foregoing, my six recommendations are as follows:

1. I recommend that ATSDR adhere to the letter and spirit of its November 2009 release and propose a comprehensive biomonitoring effort to determine whether, and to what degree, residents of Vieques have been exposed to harmful chemicals linked to training activities. Unless that data is obtained, this entire exercise seems like a hollow one, designed to reach a pre-determined conclusion of "no harm." If ATSDR does not amend its recommendations to propose comprehensive biomonitoring, I ask the agency to explain clearly why it does not believe this step to be justified and appropriate, and to explain why it has evidently reversed course from its November 2009 release.

   ATSDR Response: Because the Report found little evidence of exposure to contaminants from the site, a comprehensive biomonitoring program is not justified at this time. In addition, many chemicals are short-lived in the human body and thus cannot be measured through a biomonitoring program if the exposure occurred some time ago. Nevertheless, ATSDR is currently working with our partners in Puerto Rico to explore the value of conducting a targeted human biomonitoring program in Vieques. Because, for example, residents are concerned about mercury exposure from eating local seafood, a biomonitoring program to measure mercury in blood has public health value for Viequenses.

[Comment]

2. I support ATSDR's recommendations that additional studies should be performed to fill data gaps identified with respect to the fish, local produce and livestock, and soil pathways. I urge that additional studies, to the extent they could be useful, be performed to fill any data gaps that have

been identified in connection with the air and groundwater pathways. I fully understand that not all data gaps can be filled, but clearly some of them can—and the federal government should take all reasonable steps to ensure that they are.

ATSDR Response: Given our evaluation of the exposure pathways at the site, ATSDR has recommended a focused biomonitoring program in Vieques. If this biomonitoring program is undertaken with our partners in Puerto Rico, we would expect that concurrent sampling of soil, garden produce, and a food survey would occur. If the results from this focused biomonitoring program indicate, a more comprehensive biomonitoring program could be undertaken. If no additional biomonitoring program is undertaken, ATSDR could explore other possible ways to conduct further sampling, such as partnering with other federal agencies involved with environmental investigations in Vieques.

[Comment]

3. In general, I recommend that ATSDR and other federal agencies take a far more active and assertive role in designing, implementing and especially funding the additional health and environmental studies that are needed to determine, to a reasonable degree of confidence, the precise nature and potential causes of the health problems being experienced by residents of Vieques. It is dismaying that, more than a decade after ATSDR completed its first public health assessments on Vieques, so many fundamental questions about the safety of the island's environment and the health of its residents remain unanswered.

   ATSDR Response: Because of limited resources, ATSDR seldom collects environmental samples at hazardous waste sites. For this reason, we rely on our federal and state partner agencies to collect these samples. We will continue to work with the Navy, U.S. EPA and any other agency who may collect environmental samples from Vieques.

   Selected Metropolitan/Micropolitan Area Risk Trends (SMART) is a part of the Behavioral Risk Factors Surveillance System (BRFSS) and is designed to gather health information from smaller geographic units. We have recommended that public health agencies assess the feasibility of using SMART BRFSS methods to identify prevalence estimates on asthma, diabetes, hypertension, and other chronic diseases. Implementing such a system for Vieques would provide very valuable information about the health status of Viequenses.

   In addition, we have offered to assist Puerto Rico health officials should they decide to conduct a biomonitoring program. ATSDR is willing to provide technical advice and laboratory support for the effort.

[Comment]

4. Below, I quote at length from testimony provided by Dr. John Wargo, a Yale University professor, during a May 20, 2010 hearing held by the Subcommittee on Investigations and Oversight of the House Committee on Science and Technology. That testimony serves as an extensive, pathway-by-pathway critique of ATSDR's 2001-2003 public health assessments on Vieques. Some of the shortcomings that Dr. Wargo identified in 2010 with respect to the public health assessments have

been rectified in the 2011 draft report. For example, unlike the public health assessments, the draft report has been peer-reviewed[6] and contains a section on the "local produce and livestock" pathway. However, based on my review, it also appears that many of Dr. Wargo's most incisive criticisms and recommendations have not been addressed by the draft report. Therefore, I believe it would be helpful for ATSDR to include in its final report a section that explains whether, and to what extent, the report incorporates Dr. Wargo's recommendations and addresses his criticisms.

ATSDR Response: During our November 2009 scientific consultation, Dr. Wargo met with ATSDR to discuss his concerns about Vieques. We considered them as we evaluated historical and new data on Vieques. We respond below with specific responses to his concerns.

[Comment]

5. I urge ATSDR, as part of any broader response it furnishes to the criticisms and recommendations made by Dr. Wargo, to respond specifically to what I regard as one of Dr. Wargo's most perceptive critiques of ATSDR's public health assessments—namely, that the agency "routinely relied on studies previously prepared or data collected by others rather than designing new studies that are appropriate for local conditions and problems" and that the agency "rarely conducted its own research on environmental contamination, human exposure, and disease prevalence, and flaws in any available studies leads them to conclude there is no credible evidence of a causal relation between hazardous materials and disease…."

   It would appear that this critique applies with equal force in the case of the draft report. Because it relies exclusively on the scientific work of others, ATSDR's conclusions are only as good (or as poor) as the underlying work itself. By my count, 32 studies were analyzed by ATSDR in the draft report and used as the basis for the agency's conclusions. Only three of those studies were conducted by federal agencies: one by the Navy in 1978, one by the Environmental Protection Agency (EPA) in 2001, and one by the National Oceanic and Atmospheric Administration (NOAA) in 2010. The Navy study and the EPA study are not, in fact, studies at all, but rather simply collections of sampling data of contaminant levels—and thus were not peer-reviewed. And the NOAA study was not intended to be a human health study or, as ATSDR phrased it, to "characterize the impact from bombing activities."[7]

   The 29 non-federal studies appear to be of uneven quality and rigor. While some tremendous work has been done by local researchers—including Arturo Massol-Deyá, Elba Díaz, Carmen Ortiz Roque, and researchers from the Puerto Rico Department of Health—only four of the 29 studies were peer-reviewed and only three were published in scientific journals.

   In my view, this summary underscores the need for the federal government, working in partnership with experienced independent researchers, to assume a more prominent role in designing, implementing and funding additional studies on Vieques.

   ATSDR Response: ATSDR does not have the resources to conduct extensive site-related environmental investigations or human research. As such, we rely heavily on work prepared by other agencies and scientists. Also, CERCLA procedures require that the responsible party, the Navy

in this case, pay for and conduct all investigations and remediation. The U.S. EPA oversees these activities.

To ensure our conclusions are sound, we have made several recommendations about collecting more environmental and human data and are discussing these recommendations with our federal and commonwealth partners. To ensure that our evaluation of environmental and human data are sound, we gave the Report to six scientists for peer review and made numerous changes to the Report based on their comments.

[Comment]

6. Finally, during a recent meeting in my office, Dr. James Porter, an Associate Dean at the Odum School of Ecology at the University of Georgia, provided me with a copy of an article that he co-authored, entitled "Ecological, Radiological, and Toxicological Effects of Naval Bombardment on the Coral Reefs of Isla de Vieques, Puerto Rico." The nearly 60-page piece was published in 2011—evidently not in time to be cited by ATSDR in its draft report—as a chapter in G.E. Machlis et al. (eds.), Warfare Ecology: A New Synthesis for Peace and Security (The NATO Science for Peace and Security Programme).[8] According to the article, "Our data show unequivocally that toxic substances leaching from UXO [unexploded ordnance] have entered the coral reef food web. Since the concentration of explosive compounds is highest near unexploded ordnance, we recommend that surface UXO on the Vieques coral reef be picked up and removed." I respectfully ask ATSDR to analyze this article and to incorporate it into the agency's final report, because the article appears to provide information highly relevant to the fish and shellfish pathway.

   ATSDR Response: We have reviewed a copy of this article. It does not contain any new environmental data; it contains only the sample results already published by Dr. Porter, reviewed by ATSDR, and discussed in the Report. Consequently, we have made no changes to the Report as a result of Dr. Porter's 2011 article.

[Comment]

To ensure optimal inter-agency attention and coordination, I am providing a copy of this letter to key members of the President's Task Force on Puerto Rico's Status, including its (former and current) co-chairs and its representatives from the Department of Health and Human Services, the Environmental Protection Agency, and the Department of Defense. The Task Force's March 2011 report included a section on Vieques, which stated: "There is much that the Federal Government can do to improve the quality of life for the people of Vieques." I concur—and my recommendations are rooted in that premise. It seems evident that ATSDR cannot resolve the outstanding questions on its own, given the agency's limited funding and mandate.[9] Instead, a broader federal government-wide approach is necessary. If the Administration believes it requires additional funding or authority from Congress in order to timely implement the recommendations I have made, I urge the Administration to seek such funding and authority via an amendment to its budget request for Fiscal Year 2013. I, in turn, pledge that I will do everything within my power, working with my allies in the House and Senate, to provide the Administration with what it needs.

To place my recommendations in context, some background information is useful.

The Navy's Operations on Vieques[10]

In the 1940s, the federal government expropriated lands on the eastern and western portions of Vieques for use by the Navy, and residents of those areas who remained on Vieques were required to relocate to the central portion of the island. The Navy established the Vieques Naval Training Range is eastern Vieques, which consisted of two facilities: (1) the Atlantic Fleet Weapons Training Facility, which was used for ship-to-shore and aerial bombing exercises, and comprised a Live Impact Area and a Secondary Impact Area, and (2) the Eastern Maneuver Area, which was used primarily for ground-based training involving smaller munitions. The Navy also established the Naval Ammunition Support Detachment in western Vieques to store munitions used in its training in eastern Vieques and to dispose of obsolete or damaged munitions. The Navy reported that it had dropped between three million and four million pounds of ordnance on Vieques each year between 1983 and 1998.

The Closure and Transfer of the Navy's Facilities on Vieques

In the National Defense Authorization Act for Fiscal Year 2001 (P.L. 106-398), Congress directed the Navy to close its facilities in western Vieques and to transfer approximately 4,000 acres of that property to the Municipality of Vieques, approximately 3,100 acres to the Department of the Interior, and approximately 800 acres to the Puerto Rico Conservation Trust. In the National Defense Authorization Act for Fiscal Year 2002 (P.L. 107-107), Congress authorized the Navy to close its training facilities in eastern Vieques if equivalent training facilities were available elsewhere and directed the Navy, upon closure, to transfer administration of the roughly 15,000 acres of that property to the Department of the Interior. In January 2003, the Navy certified to Congress that alternative training sites had been identified and confirmed that training operations would cease on Vieques by May 2003.

The Ongoing Cleanup Effort on Vieques

The Navy continues to be responsible for administering and funding the cleanup of munitions and contamination that resulted from its past activities on Vieques, subject to oversight by the Environmental Protection Agency and the Puerto Rico Environmental Quality Board, an agency of the Puerto Rico government. As of April 2011, over 35,000 munitions had been recovered and destroyed on Vieques, including at least 19,000 live munitions. Through the end of the Fiscal Year 2010, the Navy had spent a total of $142.4 million to support the cleanup of its former facilities on Vieques, and estimated that an additional $380.6 million would be needed from Fiscal Year 2011 onward to complete all planned cleanup actions.

ATSDR Public Health Assessments on Vieques (2001-2003)[11]

Between 2001 and 2003, ATSDR conducted a series of public health assessments on Vieques. ATSDR examined four pathways through which residents of Vieques could have been exposed to contaminants harmful to their health: (1) air, (2) soil, (3) consumption of fish and shellfish, and (4) groundwater.

In its public health assessments, ATSDR concluded that there was "No Apparent Public Health Hazard" to island residents through any of these four pathways. This finding indicates that, while there may be the potential for exposure to a contaminant, such exposure is not likely to occur at a level that would cause adverse health effects, based on acceptable levels of exposure established by public health standards

for individual contaminants. This finding does not confirm or deny the existence of certain health effects within a population, but rather indicates that the potential for human exposure to the contaminants examined does not appear to be the cause of any reported health effects.

The air pathway has been a chief concern among the public because the residential population in the central portion of Vieques is located downwind of the former Live Impact Area on the eastern end of the island. However, ATSDR's assessment of the air pathway is inherently limited by the lack of historical data on the emissions that were released during training exercises over several decades. Given the absence of data on actual historical exposures through the air, ATSDR evaluated this pathway through the use of air quality modeling data—that is, by attempting to estimate past emissions based on more limited, recent sampling. Utilizing this methodology, ATSDR concluded:

- Residents of Vieques have been exposed to contaminants released into the air during the Navy's military training exercises. However, the estimated exposures are lower than levels known to be associated with harmful health effects. Airborne dust and contaminants released during training activities at the former Live Impact Area disperse to extremely low levels before reaching the residents of Vieques.

The soil pathway has also been a source of concern to residents of Vieques. This pathway is related to the air pathway, because contaminants from the Navy's training activities could have settled out of the air and been deposited into the soil. However, ATSDR concluded:

- Touching or incidentally ingesting the soil on Vieques would not cause harmful health effects.
- Training activities have elevated the levels of some metals in the soil on the former Live Impact Area. However, the levels are too low to be of health concern.

The safety of fish and shellfish regularly consumed by residents of Vieques has been an additional concern. The marine environment could have been contaminated by the leaching of chemicals from munitions that landed in underwater areas or from contaminated storm water runoff from the training range into the sea. This, in turn, could have contaminated edible fish and shellfish species and resulted in harmful exposure through human consumption. However, ATSDR concluded:

- It is safe to eat seafood from the coastal waters and near-shore lands on Vieques, including fish and shellfish from areas north and south of the former Live Impact Area.
- Several metals were detected in fish and shellfish collected around Vieques. However, the metal concentrations were too low to be of health concern, even if a person ate fish or shellfish every day for 70 years.
- Explosive compounds were not detected in any of the edible fish and shellfish that were sampled from Vieques.

Finally, residents of Vieques have expressed concerns that the island's drinking water supply could have been contaminated by the Navy's activities. ATSDR concluded that there did not appear to be the potential for the migration of contamination from the Navy's facilities to the middle of the island where the residential population is located. A 1999 study prepared by a contractor at the Navy's request

indicated that the groundwater primarily does not flow from the Navy's facilities to the residential portion of the island. In addition, the groundwater beneath the residential portion of the island has not been used as a primary source of drinking water on Vieques since 1978, because of increased saltwater intrusion within the aquifer. Instead, residents receive their drinking water primarily from the public water supply that is piped from mainland Puerto Rico. ATSDR evaluated the results of a sampling of the public water supply and concluded:

- The water was not affected by contaminants from the Navy's facilities and was safe to drink.
- A few public and private groundwater wells still exist in the residential portion of the island and are occasionally used when the public water supply is interrupted. Water from these wells was safe to drink, with the exception of one private well that contained water contaminated with nitrates and nitrites (most likely originating from agricultural sources or nearby septic systems, not contamination that migrated from the Navy's facilities).

The Response to ATSDR's Public Health Assessments

ATSDR's conclusions were controversial—not only among island residents who believe that the health effects they have experienced are caused by exposure to contamination from the Navy's operations on Vieques, but also among a number of independent (that is, non-federal) researchers who have studied environmental contamination on Vieques. Those researchers have asserted that the contaminant levels are higher in some cases than ATSDR reported, and that the potential health hazards are therefore likely to be greater overall than ATSDR concluded.

March 12, 2009 Hearing, House Committee on Science and Technology, Subcommittee on Investigations and Oversight

On March 12, 2009, the Subcommittee on Investigations and Oversight of the House Committee on Science and Technology held a hearing entitled "ATSDR: Problems in the Past, Potential for the Future."[12]

The Subcommittee's Majority staff, in a background report it prepared in connection with the hearing, noted that "[m]any independent scientists and health experts question" ATSDR's findings on Vieques. The staff report observed that Professor James Porter (reference above) had "presented findings at a conference last month that found unexploded munitions from the U.S. Navy around the island were, in fact, leaking toxic cancer causing substances into the ocean endangering sea life." The staff report further noted that, "[a]though Professor Porter cautioned that it is still unclear what sort of impact these toxins have had on the dinner plate," other studies had shown that residents of Vieques "have a 23 percent higher cancer rate than those on the main island of Puerto Rico" and that "plants on the island have high concentrations of lead, mercury, cadmium, uranium, cobalt, manganese and aluminum."

During the hearing, at which then-Director Frumkin testified, members of the Subcommittee argued that ATSDR's public health assessments on Vieques were flawed and urge the agency to re-evaluate its conclusions in light of the research conducted on island by independent scientists. In response to questioning, then-Director Frumkin stated: "I am very happy to pledge to you moving out of this hearing

to take a fresh look at the Vieques situation and to collect any data necessary to clarify the health situation for the people there."

November 13, 2009 ATSDR Press Release

On November 13, 2009, ATSDR issued a press release that it posted on its website.[13] In the release, ATSDR stated that it intended to issue a revised report that "modifies some of its earlier conclusions about health risks" and "changes some of its earlier conclusions regarding the safety of environmental exposures" on Vieques. According to the release, the decision "followed a thorough review of ATSDR public health assessments finalized in 2003 and other environmental studies of the island conducted in the intervening years." ATSDR said that its re-examination of the data comes as part of a "fresh look" that ATSDR pledged to island residents and to Congress.

The release quoted then-Director Frumkin as saying: "Much has been learned since we first went to Vieques a decade ago, and we have identified gaps in environmental data that could be important in determining health effects…. The gaps we found indicate that we cannot state unequivocally that no health hazards exist in Vieques. We have found reason to pose further questions."

Finally, ATSDR stated in the release that it "expects" to "recommend biomonitoring to determine whether persons living on Vieques have been exposed to harmful chemicals, and, if so, at what levels those chemicals may be in their bodies" and to "work with Puerto Rican health officials to conduct more in-depth evaluation of health outcomes."

The contents of ATSDR's November 13, 2009 public release were consistent with the message that Dr. Frumkin conveyed to me personally during our meetings on September 22, 2009 and October 22, 2009.

May 20, 2010 Hearing, House Committee on Science and Technology, Subcommittee on Investigations and Oversight

On May 20, 2010, the Subcommittee on Investigations and Oversight held a hearing entitled "Preventing Harm - Protecting Health: Reforming CDC's Environmental Public Health Practices."[14] In the "Hearing Charter" prepared by the Subcommittee's Majority staff, it was observed that ATSDR's public health assessments on Vieques had been "widely criticized."[15]

Dr. John Wargo's Testimony at the May 20, 2010 Subcommittee Hearing

One of the witnesses at the May 20, 2010 hearing was Dr. John Wargo, a professor of Risk Analysis and Environmental Policy at Yale University, who had conducted research on Vieques for a number of years. Dr. Wargo's written testimony constitutes one of the most comprehensive critiques of ATSDR's public health assessments on Vieques. As such, it merits close consideration here—principally as an aid to analyzing whether ATSDR's 2011 draft report displays any of the same shortcomings that Dr. Wargo identified in connection with the agency's earlier work on the island.

Dr. Wargo argued that military training activities on Vieques "have created a toxic soup, a mess that I have never seen anything similar to in my experience." Dr. Wargo testified that ATSDR's public health assessments on Vieques "contain flaws in scientific methods, analyses and interpretations of evidence, yet the agency consistently concludes that human health risks are insignificant."[16] Dr. Wargo noted that "[l]ead, mercury, cadmium, chromium, arsenic, and uranium have all been released into the Vieques

environment" as a result of military training activities, and that these elements "are well recognized to [be] hazardous substances, and they have the potential to be absorbed by plants, wildlife, fish and shellfish." Dr. Wargo opined that "[a] careful review of the ATSDR public health assessments reveals an agency determined to find no causal relation between the Defense Department's 60 year history of dropping nearly 100 million pounds of weapons on a small island, and the exceptional incidence of human illness among those that lived through this history."

ATSDR Response: We agree that bombing activities are likely to have increased contaminant levels in parts of the Vieques environment. We make this point in Chapter 1, Section 1.3 of the Report.

ATSDR's pathway analysis, however, is a determination of whether persons could be exposed to military activity-related chemicals at levels that could harm their health and requires that

- Persons have contact with those chemicals,
- The chemicals enter their bodies, and
- The amount of exposure (i.e., the dose) is at harmful levels.

In our review of soil, air, groundwater, and seafood data, we could not identify how persons might come into contact with military activity-related chemicals at levels of health concern:

- Contaminant migration in air is insufficient to result in inhalation exposure at levels of health concern.
- Residents do not have contact with contaminated soils in the LIA.
- Residents are not drinking contaminated groundwater.

The only chemical we found that was a public health concern was mercury in fish. And this mercury is likely from the global reservoir of mercury circulating in the environment—not from military activities on the island.

ATSDR staff who conducted the Report were not involved in previous ATSDR Vieques reports. And the Report went through extensive internal and external peer review. The Report's analysis and conclusions are based on the current, best available science.

[Comment]

Dr. Wargo's oral and written testimony also included the following conclusions:

- ATSDR "concluded that the absence of evidence of contamination is sufficient to conclude the absence of significant health threat. However, the poor quality of environmental monitoring and surveillance makes it impossible to justify the sweeping declarations of safety made by ATSDR."

ATSDR Response: After an evaluation of the best data available, ATSDR believes that no exposures of health concern occurred to Viequenses as a result of the military activities conducted on the island. Our recommendations about fish consumption for children and pregnant women are based on our concerns about mercury levels in fish that result from the global reservoir of mercury circulating in the environment.

[Comment]

- ATSDR "routinely relied on studies previously prepared or data collected by others rather than designing new studies that are appropriate for local conditions and problems."

- ATSDR "rarely conducted its own research on environmental contamination, human exposure, and disease prevalence, and flaws in any available studies leads them to conclude there is no credible evidence of a causal relation between hazardous materials and disease…."

- ATSDR "conducted no human testing on Vieques to determine whether hazardous chemicals released by the Navy were present in the tissues of island residents." Nor did ATSDR "conduct any original epidemiological studies to understand patterns of disease prevalence on the island." However, "[t]hese types of data are fundamentally necessary to understand the relations between hazardous chemicals and human illness."

  ATSDR Response: Because of limited resources, ATSDR seldom collects environmental samples at hazardous waste sites. We rely on our federal and state environmental partner agencies to collect these samples. We will continue to work with the Navy, U.S. EPA and any other agency who may collect environmental samples from Vieques.

  We have recommended that public health agencies assess the feasibility of using SMART BRFSS methods to identify prevalence estimates on asthmas, diabetes, hypertension, and other chronic diseases. Selected Metropolitan/Micropolitan Area Risk Trends (SMART) is a part of the Behavioral Risk Factors Surveillance System (BRFSS) and is designed to gather health information from smaller geographic units. Implementing such a system for Vieques would provide very valuable information about the health status of Viequenses.

  In addition, we have offered to assist Puerto Rico health officials should they decide to conduct a biomonitoring program. In addition to technical advice, ATSDR is willing to provide laboratory support for the effort.

[Comment]

- The Navy "has carefully controlled access to the bombing range in a manner that has precluded the conduct of scientific research by independent scientists…. When the government controls the science, they control the narrative risk to human health. There is a clear need to create an alternative [institution] to conduct these health assessments by independent and unbiased scientists."

  ATSDR Response: The site access is restricted because of concerns about unexploded ordnance, which may present an on-site physical danger. Even though site access is restricted, sampling at the LIA by independent parties has occurred:

  - Researchers from Casa Pueblo and the University of Puerto Rico sampled vegetation from the LIA in 2000 (Massol Deyá and Díaz 2000).

- Servicios Científicos y Técnicos, Inc. collected soil samples during the protest from May 1999 to April 2000 (García et al. 2000).
- ATSDR/EPA collected fish and shellfish from on and around the LIA in July 2001.
- NOAA collected land crabs and fiddler crabs from the LIA in June 2005 (Ridolfi and NOAA 2005).

The data ATSDR evaluated are available to any scientist who wishes to review them.

[Comment]

- With respect to the air pathway, “air pollution data was mismanaged by the Navy and therefore provides unreliable information regarding the magnitude of distribution of air contaminants during high activity training periods on the Live Impact Area.” As a result, ATSDR relied entirely on a modeling study to evaluate this pathway, which limits the persuasive value of its conclusion “that estimated exposures are lower than levels known to be associated with harmful health effects.”

  ATSDR Response: This comment appears to pertain to an air sampling study that the Navy performed in the 1970s. ATSDR concluded that "...the measured concentrations from this sampling effort are of an unknown quality, because no documentation can be found describing the sampling methods used or the quality assurance measures taken." It is unfortunate that further documentation is not available for this sampling effort that did not find unusually elevated air concentrations of particulate matter. However, in our experience, it is not uncommon to find incomplete documentation for sampling that occurred 30 years ago. On a related note, ATSDR made numerous attempts to acquire documentation for air sampling that the Puerto Rico Environmental Quality Board (PREQB) had conducted on Vieques in the 1970s; to date no documentation of that study has been provided to ATSDR. After investing considerable effort trying to locate and evaluate past air sampling events at Vieques, ATSDR eventually concluded that "no sampling programs extensively characterized air quality on Vieques during live bombing exercises."

  After making this conclusion, ATSDR had to make a decision regarding how to evaluate past air pollution levels. All live bombing exercises on Vieques had ceased by the time ATSDR became involved with the site. Therefore, it was impossible to conduct a new sampling study that would somehow quantify past air pollution levels. Because sampling was not an option, ATSDR's primary choices for assessing past air pollution levels were to either: (1) use models to estimate what air pollution levels might have been or (2) state that no data are available and conclude that the past air pollution levels could not be determined. ATSDR chose to pursue the modeling option in the interest of communicating scientific findings to the residents of Vieques. All air modeling evaluations have inherent uncertainties and limitations, and this was acknowledged throughout the 2003 Air PHA. However, for multiple reasons cited in the 2003 Air PHA, ATSDR believes that assumptions made in the modeling are conservative (protective) and typically erred on the side of overstating emissions and hence exposures, especially for metals and

explosives. ATSDR thoroughly considered these uncertainties and the magnitude of the estimated air concentrations (which were several orders of magnitude lower than concentrations of health concern for most pollutants) before concluding that the modeling analysis presented a reasonable account of exposures that occurred on Vieques and did not understate the exposures that residents might have experienced.

The Report carefully evaluates all assumptions and inputs into the previous modeling analysis and concludes the following: "We reviewed the data on airborne contaminants from military exercises at the former Vieques Naval Training Facility. This review confirmed our previous findings and indicated that airborne contaminants from past military operations were very unlikely to have had health effects on Viequenses." For more information on this, refer to the 2003 Air PHA and Chapter 6 of the Report.

A recent Gioda et.al. (2007) study examined particulate matter (PM), specifically PM_{10}, and metals concentrations from air samples for several Puerto Rican locations, including Vieques. This study makes some chemical-specific data available. Most of the metals analyzed in the Gioda et al. samples (cadmium, cobalt, copper, iron, nickel, lead, and vanadium) were well below their applicable health comparison values and do not represent a public health hazard. For more information about this topic see Chapter 6, Section 6.2.3 of the Report.

[Comment]

- With respect to the soil pathway, the Navy and ATSDR “failed to collect soil contamination data associated with military operations. The absence of these data prevented them and others from understanding when and where soil might pose a public health threat. This could occur from soil particles exploding into the atmosphere, drifting downwind in the atmosphere, eventually settling on plants, soils, and perhaps open cisterns.” Dr. Wargo questioned why ATSDR had not tested soil—as well as edible plant tissues and animal products—for hazardous compounds released by training activities.

  ATSDR Response: ATSDR reviewed all the soil sampling done on the LIA. The results show very few instances of contamination above background levels (see the Report, Chapter 1, Section 1.3.3). Air modeling results indicate that only small amounts of military activity-related contaminants could have migrated to the residential areas. Further, sampling for explosives was conducted along the western border of the EMA in 1999, and none were detected (CH2MHILL and Baker 1999). ATSDR has recommended collection of soil and produce samples from residential areas to confirm that these areas were not affected.

[Comment]

- With respect to the fish and shellfish pathway, “ATSDR’s conclusions that fish intake by Vieques residents poses no health threat is not supported by the data the Agency relied upon to reach the finding.” While ATSDR collected fish and examined them to identify the presence of hazardous chemicals, “their sampling designs were inappropriate and insensitive.”

ATSDR Response: ATSDR's fish sampling investigation was conducted in partnership with the U.S. EPA's Environmental Response Team, which has extensive fish sampling experience. The sampling effort design used the protocols available at the time the sampling was conducted (in 2001). In addition, ATSDR consulted with the petitioner and local residents to advise us on the sampling plan. ATSDR is no longer concluding that it is safe for everyone to eat fish. We revised our conclusions in the Report and made recommendations about fish consumption for children and pregnant women based on our concerns about mercury.

[Comment]

- In addition, ATSDR wrongly "assumes that fish constitute the only significant food that might carry contaminants of military origin to the dinner table." The Navy, EPA and ATSDR "neglected research on grazing activities by cattle, goats, sheep, pigs and chickens," which "could potentially have been a significant additional pathway of exposure." Because the Navy leased lands to those who grazed their stock, some in close proximity to the Live Impact Area, "it seems prudent to consider the potential for metals, explosives, and other contaminants of military origin to be taken up by plants that are in turn consumed by cattle." The "restriction of ATSDR attention to fish seems convenient rather than scientifically justified."

  ATSDR Response: The Puerto Rico Department of Agriculture, in cooperation with the Farmers Association of Puerto Rico, sampled grass, fruit-bearing trees, and bovine livestock from Vieques. The Department of Agriculture and the Farmer's Association concluded that the agricultural products from Vieques were suitable for consumption and did not contain toxic levels of metals. To confirm this conclusion, ATSDR has recommended local livestock sampling. (See the Report, Chapter 5, Section 5.2.)

[Comment]

- With respect to the groundwater/drinking water pathway, ATSDR's conclusion that public drinking water supplies pose no health hazard "is not supported by a statistically valid sampling design." The 35-year period between 1943 and 1978—when a public water supply from mainland Puerto Rico was completed—is "the most likely time when the island's population might have been exposed to hazardous compounds released to the environment by the Navy via drinking water." Yet, "this is also a period when government testing of environmental quality on the island was minimal." The "poor history and quality of water quality testing make it difficult to reconstruct a history of exposure with precision," because "[w]ater supplies on Vieques were not tested routinely for chemicals that were intensively released to the environment by the Navy" and "ATSDR did not conduct any tests of its own." Instead, the Agency relied on previous studies conducted by the Puerto Rico Department of Health, the EPA, the U.S. Geological Survey, and a consulting firm hired by the Navy. Although ATSDR reported the presence of the explosives RDX and Tetryl in the drinking water supplies, the Navy and ATSDR did not provide "a plausible explanation for these findings." The studies interpreted by ATSDR "do not demonstrate the absence of health threat associated with Naval activities. Instead, they demonstrate the absence of the Navy's testing of the community's drinking water supplies."

ATSDR Response: ATSDR has reviewed the available data about drinking water quality in Vieques. The Report notes that when historical data were not available, we cannot determine whether the water was safe to drink. Groundwater from the LIA does not flow towards former public water supply wells on the island. Therefore, military activities in the LIA could not have contaminated those wells.

Given the data we have reviewed, the water is currently safe to drink. Still, continued safety of the pipeline water supply relies on continued monitoring of the water supply according to federal regulations and, if necessary, on prompt action to address violations.

[Comment]

More generally, Dr. Wargo testified that ATSDR suffered "from an underlying cultural problem." Specifically, he argued that the agency had "misperceived its intended mission." He said that it was clear, based on the public health assessments, that ATSDR "believes its purpose is to search for conclusive evidence that hazardous chemicals have caused health loss." However, "[s]ince data necessary to demonstrate the cause of health loss … rarely exist, the agency normally finds 'no significant threat to human health,' and it declares the safety of surrounding communities. Yet these conclusions are illogical, and scientifically flawed." While "ATSDR may not have sufficient evidence to conclude community danger," Dr. Wargo testified, neither does it "have sufficient evidence to conclude 'safety."'

ATSDR Response: ATSDR's mission is to determine whether communities could be exposed to levels of hazardous substances that might be harmful. If communities are so exposed, the agency must determine what health effects might result. In the case of Vieques, given the available information, ATSDR believes that Viequenses are not likely to be exposed to harmful levels of contaminants related to the site.

ATSDR, however, has identified mercury exposure from frequent consumption of marine seafood as a potential public health hazard. Our conclusions and recommendations about eating fish from Vieques reefs are presented in Chapter 2 and in the Executive Summary. We could find no relationship between mercury in fish and military operations on Vieques. A more plausible explanation for the mercury levels found in fish is that they resulted from the global reservoir of mercury circulating through the environment.

[Comment]

Dr. Wargo recommended, among other things, that ATSDR conduct human tissue testing; evaluate disease prevalence; explicitly evaluate the quality and uncertainty of each data source; and establish rigorous standards before declaring safety.

ATSDR Response: As previously mentioned, ATSDR is currently working with our partners in Puerto Rico to explore the possibility of a targeted human biomonitoring program in Vieques. ATSDR has also recommended that health officials consider a SMART BRFSS to determine the prevalence of various health conditions in Vieques. ATSDR evaluated the quality and stated limitations of each data source in the Report.

Conclusion

On the basis of the foregoing, I respectfully urge ATSDR to implement the six recommendations I have made. I look forward to continuing to work with you on this matter.

ATSDR Response: Please see previous responses.

Footnotes:

[1] http://www.atsdr.cdc.gov/sites/Vieques/2011_report.html

[2] http://www.ca1.uscourts/gov/pdf.opinions/10-1648P-01A.pdf.

[3] http://pierluisi.house.gov/PDF/letters/2010/11.18.11%20Letter%20to%20Natural%20Resources%20Committee%20Regarding%20Hearing%20on%20Vieques%20PR.pdf.

[4] Draft Report, page xi.

[5] Draft Report, page 59. See also Draft Report, page 65 (stating that the five biomonitoring investigations conducted on Vieques to date by local officials "do not permit any conclusions about exposure to the bombing-related contaminants.")

[6] I am aware that federal law does not require peer review for public health assessments, but does require if to other types of ATSDR studies. See 42 U.S.C. 9604(i)(13). Of course, the statute does not prohibit ATSDR from seeking peer review of its public health assessments either.

[7] Draft Report, page A-73.

[8] http://www.springerlink.com/content/978-007-1214-0#section=902961&page=2&locus=19.

[9] I note that the Navy is responsible for the costs of ATSDR studies on Vieques. Thus, it is the adequacy of the Navy's funding that will be key to the implementation of my recommendations.

[10] This section, and the two sections that follow it, are drawn in large part from the Findings section of H.R. 1645, the Vieques Recovery and Development Act of 2011, introduce on April 15, 2011 and cosponsored by 21 members of Congress.

[11] This section is drawn largely from a 34-page report prepared by the Congressional Research Service for my office at my request, entitled "Cleanup of Former U.S. Navy Facilities on Vieques Island, Puerto Rico" and dated August 19, 2009.

[12] http://www.gpo.gov/fdsys/pkg/CHRG-111hhrg47718/pdf/CHRG-111hhrg47718.pdf. (hearing transcript).

[13] http://www.atsdr.cdc.gov/news/displaynews.asp?PRid=2455.

[14] http://www.gpo.gov/fdsys/pkg/CHRG-111hhrg57173/pdf/CHRG-111hhrg57173.pdf (hearing transcript).

[15] http://science.house.gov/sites/republicans.science.house.gov/files/documents/hearings/052010_charter.pdf.

[16] http://gop.science.house.gov/Media/hearings/oversight10/may20/Wargo.pdf.

ATSDR References:

CH2MHILL and Baker Environmental, Inc. (Baker). 1999. Results of the hydrogeologic investigation: Vieques Island, Puerto Rico. November 4, 1999.

García N, López AM, Soto M, Rosado S, Berríos B. 2000. Environmental impact of Navy activities in Vieques. Servicios Científicos y Técnicos, Inc. July 11, 2000.

Massol Deyá A and Díaz E. 2000. Heavy metals in the impact area prevailing vegetation, Vieques, Puerto Rico. Casa Pueblo de Adjuntas, PR: University of Puerto Rico, Recinto Universitario Mayagüez, Biology Department; 10 April.

Ridolfi, Inc and National Oceanic and Atmospheric Administration (NOAA). 2005. Draft data report for the Vieques Island Biota Sampling Project, Vieques Island, Puerto Rico. December 13, 2005.

Commenter 3

[Comment]

I'm commenting on the ATSDR draft document on Vieques, PR, and in particular on the concerns raised over mercury exposure to humans and fauna living on and around Vieques.

Despite the considerable concern expressed by individuals and agencies over allegedly elevated levels of mercury on Vieques, and the several hypotheses as to the source of these elevated mercury levels, there appears to be a glaring omission of a likely source of direct exposure to elemental mercury in the homes of inhabitants of Vieques, as well as to the larger terrestrial and aquatic environments.

Many Puerto Ricans put elemental mercury in a wide variety of magico-religious uses, in particular within the religions of Espiritismo and Santeria. The generic belief behind this use is that mercury (*azogue, asoque, mercurio*) attracts good and repels evil. Many of these esoteric uses expose mercury to indoor air, most problematically the practice of sprinkling mercury on the floors of homes. This mercury will remain in flooring for periods of several decades, while outgassing developmentally neurotoxic levels of mercury vapor.

All of the mercury put to these occult domestic uses eventually enters the larger terrestrial and aquatic environments.

Over the past two decades I have alerted EPA Region 2 about this issue, both here at their New York City headquarters, as well as their offices in Puerto Rico. I have also alerted numerous Puerto Rican health and environmental authorities and universities. Yet none of them have undertaken any substantive research to assess the levels of mercury in housing or botanicas in Puerto Rico. The only research on the topic that I am aware of was done by Dr. Nunez-Molina of the University of Mayaguez (attached) in the early 1990s. The Puerto Rico Department of Consumer Affairs made a (vain?) attempt at banning the importation and distribution of mercury for sale by botanicas in 1991.

1. What have the ATSDR and the Puerto Rican medical and environmental establishments done to assess magico-religious mercury use, and the domestic and environmental contamination it causes?

   ATSDR Response: Under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA, also known as Superfund), Congress provided ATSDR with the authority to conduct certain public health assessment activities. Note, please, that CERCLA provides ATSDR with the authority to evaluate residential exposure to environmental contaminants released from hazardous waste sites or facilities under a variety of exposure scenarios. These scenarios include potential exposure to contaminants that may be brought into the home by infiltration with the ambient air, attached with soil particles on shoes, or attached to worker clothing. But CERCLA does not provide ATSDR with the authority to evaluate potential residential exposure to hazardous substances that residents choose to bring into their homes.

That said, ATSDR recognizes the potential health effects of exposure to harmful levels of mercury vapor that residents may experience due to magico-religious uses of mercury (e.g., from the use of mercury-containing capsules). ATSDR also recognizes that the level of residential exposure to mercury vapor resulting from magico-religious use is not well quantified. Additional research could provide valuable information on this important topic. Again, CERCLA does not provide ATSDR with the authority to conduct the fundamental research necessary to quantify this type of exposure.

We suggest that the commenter contact health and environmental officials in Puerto Rico to determine what if any activities they have conducted concerning the magico-religious use of mercury in Puerto Rican homes. ATSDR's Mercury and Your Health topic page (http://www.atsdr.cdc.gov/mercury/) contains a public service announcement (*Don't Mess With Mercury*) and four informational fact sheets in both English and Spanish, as well as several other important resources that might be useful for the commenter.

[Comment]

2. Why does the document in question fail to even mention this certain source of direct human exposure to elemental mercury, and even more certain environmental mercury contamination?

   ATSDR Response: As pointed out by the commenter, some practitioners of certain Caribbean and Latin American religions, such as Voodoo, Santeria, Palo, and Espiritismo, use mercury ceremonially [USEPA 2002; Johnson 1999; Masur 2011; Newby et al. 2006; Wendroff 2005; Zayas and Ozuah 1996]. Ceremonial uses of mercury include applying it to the skin, adding it to candles, or sprinkling it around the home. These practices can potentially expose practitioners and their families. Because mercury contamination in the home can persist for years, ceremonial use of mercury in the home could expose future occupants and their children, contributing to health disparities in these populations. We have added religious rites using mercury-containing capsules that release mercury to the home environment as a possible source of mercury in some residents.

[Comment]

3. Why the concern of trivial amounts of mercury on uninhabited bombing ranges, yet no concern over gross domestic mercury contamination of housing with mean weights of some 9 grams of elemental mercury intentionally sprinkled on floors of Puerto Rican homes?

   The report as drafted needs to be amended to address the mercury input from the magico-religious use of mercury in Vieques.

   ATSDR Response: During ATSDR's meeting with Puerto Rican and other scientists, they raised concerns that mercury in bombs may have contaminated the Vieques environment and resulted in Viequenses' exposure to military activity-related mercury. In response to these concerns, the Report documents the review of environmental, biological, and health data.

   We have amended the Report to show that religious rites using mercury-containing capsules also may be a source of mercury exposure in Vieques.

References

Johnson C. 1999. Elemental mercury use in religious and ritualistic practices in Latin American and Caribbean communities in New York. Popul Environ 20(5):443–453.

Masur LC. 2011. A Review of the Use of Mercury in Historic and Current Ritualistic and Spiritual Practices. Altern Med Rev 16(4):314-320.

Newby AC, Riley DM, Leal-Almeraz TO. 2006. Mercury use and exposure among Santeria practitioners: Religious versus folk practice in northern New Jersey, USA. Ethn Health 11(3):287–306.

[USEPA] US Environmental Protection Agency. 2002. Task force on ritualistic uses of mercury report. Available from: http://www.epa.gov/superfund/community/pdfs/mercury.pdf [accessed 10 December 2012].

Wendroff AP. 2005. Magico-religious mercury use in Caribbean and Latino Communities: Pollution, persistence, and politics. Environ Pract 7:87–96.

Zayas LH, Ozuah PO. 1996. Mercury use in Espiritismo: A survey of botanicas. Am J Public Health 86(1):111–112.

Commenter 4

[Comment]

I do not trust the report on Vieques environmental situation. I also very much dislike that the Navy bombed there and then you let the Navy, hardly an independent source, do the tests on the pollution they left behind. I do not think the US public can trust this report. What possessed the authorities to let the same people who bombed the island report on the pollution left behind? Fact is the Navy and US military have left pollution all over this world and all over America. The pollution left behind at military installations is always enough to kill you. This report is bogus. It needs to be done again by an independent lab - maybe another country's lab.

> **ATSDR Response:** This site followed the procedures used for all U.S.-owned Superfund sites. While the owner of or responsible party for the site, the Navy in this case, pays for and conducts all investigative and remedial work, their work is overseen by the US EPA. In doing the Report, ATSDR used studies prepared by many other independent scientists, groups, and agencies in addition to reports prepared by the Navy.

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Review

Vibroacoustic disease: Biological effects of infrasound and low-frequency noise explained by mechanotransduction cellular signalling

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Abstract

At present, infrasound (0–20 Hz) and low-frequency noise (20–500 Hz) (ILFN, 0–500 Hz) are agents of disease that go unchecked. Vibroacoustic disease (VAD) is a whole-body pathology that develops in individuals excessively exposed to ILFN. VAD has been diagnosed within several professional groups employed within the aeronautical industry, and in other heavy industries. However, given the ubiquitous nature of ILFN and the absence of legislation concerning ILFN, VAD is increasingly being diagnosed among members of the general population, including children. VAD is associated with the abnormal growth of extra-cellular matrices (collagen and elastin), in the absence of an inflammatory process. In VAD, the end-product of collagen and elastin growth is reinforcement of structural integrity. This is seen in blood vessels, cardiac structures, trachea, lung, and kidney of both VAD patients and ILFN-exposed animals. VAD is, essentially, a mechanotransduction disease. Inter- and intra-cellular communication is achieved through both biochemical and mechanotranduction signalling. When the structural components of tissue are altered, as is seen in ILFN-exposed specimens, the mechanically mediated signalling is, at best, impaired. Common medical diagnostic tests, such as EKG, EEG, as well as many blood chemistry analyses, are based on the mal-function of biochemical signalling processes. VAD patients typically present normal values for these tests. However, when echocardiography, brain MRI or histological studies are performed, where structural changes can be identified, all consistently show significant changes in VAD patients and ILFN-exposed animals. Frequency-specific effects are not yet known, valid dose-responses have been difficult to identify, and large-scale epidemiological studies are still lacking.
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Keywords: Extra-cellular matrix; Actin; Tubulin; Collagen; Tensegrity

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1. Introduction

This review paper deals with the biological effects of infrasound (0–20 Hz) and low-frequency noise (ILFN) (20–500 Hz). For the past 60 years, there has been much controversy and acrimonious debate over whether or not acoustical phenomena can cause extra-auditory effects on living organisms (Alves-Pereira, 1999). At present, the only officially (and legally) recognized consequence of noise exposure is hearing loss, albeit noise-induced annoyance, sleep disturbances and hypertension have been gaining more recognition over the past several years.

The scientific understanding of non-auditory, noise-induced biological effects can only be achieved if several obstacles are overcome. These obstacles pertain to the way the scientific community, in general, and biological scientists, in particular, view noise pollution and cellular signalling: noise only causes hearing impairment and cellular signalling is accomplished only through biochemical pathways. These untenable positions are powerful (scientific) hindrances that have impeded valuable research efforts. There are other key obstacles related to the awareness and recognition of ILFN as an agent of disease, but these are associated with the political, financial and social features of our collective societies and are, therefore, beyond the scope of this report.

Much of the literature pertaining to this field of study has been produced by non-English-speaking authors. Although the majority possess an abstract in English, full translations of all these scientific papers (e.g., from Chinese, Russian, Slovenian, Japanese and Polish) have been difficult to obtain. Additionally, many of the early papers produced by this team (from 1980 through 1989) were published in Portuguese with abstracts in English. Hence, several scientific papers in this review are only referred to abstracts.

Herein will be demonstrated that excessive exposure to ILFN causes extra-auditory pathology, specifically, vibroacoustic disease (VAD), and that the physiological and biological basis for this disease can only be understood if the concept of mechanotransduction cellular signalling is taken into account.

2. Noise pollution

Historically, it is understandable that noise exposure has always been associated with hearing loss. According to the Epic of Gilgamesh, a Babylonian king who lived in 2700 BC, the Great Flood was brought to

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the planet Earth because the demi-gods were unable to sleep due to the noise produced by humans (Sandars, 1972). In Ancient Greece (600 BC), metalwork involving hammers was banned within city limits (Ward, 1973). In Ancient Rome, legislation existed pertaining to the noise associated with the iron wheels of wagons that disrupted sleep, while in certain cities of Medieval Europe, horse carriages were not allowed during night time (World Health Organization, 1999).

But with the advent of machines, a different kind of noise became ubiquitous. Sometimes, a low rumble from public transportation systems in urban and suburban settings, sometimes a hum from an air-conditioning unit, a refrigerator or a fan, this noise does not cause hearing impairment. But it may cause annoyance. As a definition of annoyance, the European Commission Noise Team (2000) maintains: "Annoyance is the scientific expression for the non-specific disturbance by noise, as reported in a structured field survey. Nearly every person that reports to be annoyed by noise in and around its home will also experience one or more of the following specific effects: Reduced enjoyment of balcony or garden; When inside the home with windows open: interference with sleep, communication, reading, watching television, listening to music and radio; Closing of bedroom windows in order to avoid sleep disturbance. Some of the persons that are annoyed by noise also experience one or more of the following effects: Sleep disturbance when windows and doors are closed; Interference with communication and other indoor activities when windows and doors are closed; Mental health effects; Noise-induced hearing impairment; Hypertension; Ischemic heart disease." Hence, the parameter "annoyance" is, in itself, of a subjective nature.

2.1. dBA versus dBLin

Quantifying noise based on the subjective awareness of humans has guided the vast majority of noise-related biomedical studies. In fact, the foundational unit of noise legislation—the dBA—is grounded solely in the acoustical phenomena that humans can perceive with their ears, i.e., sound. The "A" on the dB unit refers to the usage of a filtering or weighting network that simulates human hearing and, thus, its purpose is to measure the acoustical phenomena present in the environment that can cause hearing impairment. Humans are considered to perceive sound between 20 and 20 000 Hz, but non-uniformly, i.e., there is an acoustical window where the human ear is most susceptible: 500–8000 Hz. It is within these frequency bands that hearing impairment occurs—legal deafness is assessed at 4000 Hz. Using the A-filter de-emphasizes all values of acoustical energy that occur below 500 Hz, and ignores all acoustical energy below 20 Hz. Fig. 1 demonstrates the usefulness of the dBA unit.

Scientifically, the acoustical environments of the cockpit and train shown in Fig. 1 cannot be considered comparable because the distribution of acoustical energy throughout the different frequency bands is quite distinct. This distinction is not taken into account if the acoustic environments are solely described by a dBA value, as is clearly illustrated in Fig. 1. Nevertheless, it is this dBA value that most noise-related legislation requires to assess the risk of noise exposure, and it is this same dBA value that most biomedical scientists use to describe their experimental acoustical environments.

The usage of the dBA value for scientific study can only be justified if the purpose of the study is related to effects on, or via, the human auditory system. If the purpose is to study the biological effects of noise, then describing acoustical environments merely in terms of a dBA value is a scientifically unsound methodology. Example: Two different and independent teams of researchers expose the same animal population, in equal conditions, to an acoustical environment described as "80 dBA". Is it scientifically valid to compare the results of these two studies? As the example given in Fig. 1 clearly demonstrates, the answer is no, but the scientific community at large does so in its numerous published papers. At present, the general consensus of mainstream science is (still) that noise-induced extra-auditory pathology is a controversial, contradictory and inconclusive subject, hence non-existent and, therefore, not subjected to legislation (Alves-Pereira, 1999, 2005).

2.2. What you can't hear, won't hurt you

The title of this section is a quote, from Campanella (2001). There is no scientific evidence supporting this statement, and there is a colossal amount of scientific evidence indicating otherwise. Nevertheless, leading

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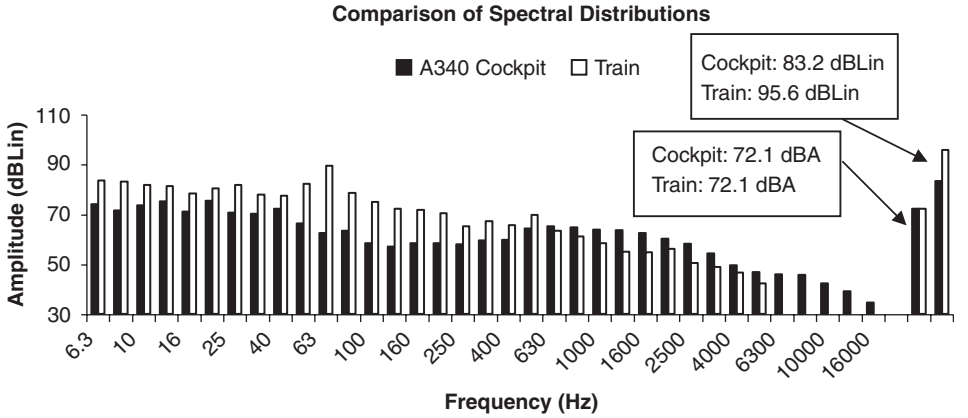


Fig. 1. Spectral distribution in 1/3 octave bands, without A-weighting (dB-Linear), obtained in (a) cockpit of the Airbus-340 (Alves-Pereira et al., 2001a), at cruise flight, and (b) in a Lisbon commuter train while in motion, in the passenger position (Alves-Pereira et al., 2004a). The two bars on the right compare the overall (Leq) values in both dBA and dBLin, i.e., with and without A-weighting, respectively. As can be observed, the dBA values are the same while the dBLin values differ by 12 dB. The value in dBA represents what the human can *hear* while the dBLin value indicates the amount of acoustic energy present in the environment. Hence, in both cockpit and train, what will be *heard* will be the same, but the individual in the train will be exposed to a larger amount of acoustical energy.

acousticians still opt to ignore this fact, and persist on perpetuating the notion that if hearing protectors are properly worn, no noise-induced extra-auditory effects will arise (von Gierke and Mohler, 2002).

In fact, over the past 25 years, research conducted in Portugal (and, independently, in other countries, such as the Soviet Union/Russia, Japan and China) has been showing that acoustical phenomena, whether it is perceived by the auditory system, or not, can indeed cause organic changes in biological tissue (Alves-Pereira, 1999). As a result of the efforts of a multidisciplinary team of scientists, including medical doctors, mathematicians, physicists, biologists, engineers, and acousticians, a pathological entity has been defined—VAD (see below) (Castelo Branco and Rodriguez Lopez, 1999a; Castelo Branco and Alves-Pereira, 2004a).

VAD is specifically caused by excessive exposure to ILFN (taken to be all acoustical phenomena occurring from 0 to 500 Hz). However, the acoustical energy responsible for VAD is never taken into account by standard noise measurements. As explained above, the scientific community and legislative bodies insist in accepting acoustical environments described merely in terms of a dBA value. Hence, they also perpetuate the erroneous notion that noise only affects the ear. The end result is a multitude of studies, focused on the biological effects of noise, but without the necessary methodology to allow them to be compared amongst each other. In the rare cases where information on the frequency distribution is provided, spectra are often measured in dBA units, which, once again de-emphasizes the amount of acoustical energy actually present in the environment, but gives a nice estimate of the noise being processed by the ear.

The scientifically unsubstantiated, but prevalent notion that noise only affects hearing has had a tremendous impact on individuals who develop ILFN-induced pathology. The gravity and magnitude of this issue will be further discussed in later sections.

2.3. Acoustic pollution

It is high time that scientists begin to view acoustical phenomena within a framework usually applied to electromagnetic phenomena. Within the electromagnetic spectrum, the human eye perceives light in a certain range of frequencies, just as within the acoustical spectrum, the human ear perceives sound in a specific range of frequencies. There exist electromagnetic phenomena that are not perceived by any of the human senses during the actual exposure (e.g. X-rays), and yet, excessive exposure to X-rays can cause severe biological damage. Without any subjective perception of the agent of disease, humans can nonetheless develop pathology caused by that unperceived agent of disease. While this is obviously true, it is apparently forgotten when one deals with acoustical phenomena, specifically ILFN. This may, in fact, be a unique case in the History of

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Medicine, whereby the agent of disease is considered to only have an effect on the host, if the host perceives that same agent of disease.

That specific electromagnetic frequencies influence specific types of tissues is a well-known fact, and is the basis for numerous medical diagnostic and therapeutic tools. Science has data regarding which frequencies cause which types of pathology, for example, ultraviolet radiation can cause ocular disease, such as cataracts. But in the acoustical spectrum, the only frequencies that are considered to pathologically affect humans are within the audible range (ultrasound is beyond the scope of this paper), and all of them are focused on the hearing apparatus. Acoustical phenomena within the ILFN range can affect several organs and tissues, but this depends also on the frequency of the acoustical event because every organ and tissue has its own acoustical properties (e.g., resonance frequency and acoustical impedance).

Noise protection is, of course, focused exclusively on avoiding hearing impairment and minimizing annoyance. Thus, in some work environments where ILFN values can reach up to 90 and 100 dB (Alves-Pereira et al., 2004b), the only protection provided are hearing protectors. Returning to the analogy with the electromagnetic spectrum, this would be equivalent to providing dark glasses to individuals who work with X-rays.

Clearly, a new attitude toward noise and noise pollution is urgently required. The term *acoustic pollution* reflects the real nature of acoustical phenomena and how it impacts on humans. Acoustic pollution encompasses those frequencies that, being audible to humans, can cause hearing impairment. But it also includes all the other acoustical phenomena, ILFN *and* ultrasound, which may, or may not, pathologically affect human beings. Acoustic pollution deals with the entire acoustical landscape, and not just with the acoustical phenomena that produce sound to the human perception. In fact, where ILFN is concerned, dosimetry studies cannot be adequately carried out if the notion of acoustic pollution is not well understood. (See below.)

3. Chemical and mechanical cellular signalling

When new biological models successfully explain a larger number of biological events, usually that model is adopted and older, less applicable models are discarded. While this may seem like a logical course of action, sometimes the inertia associated with human nature to accept change constitutes an impediment.

The conventional model of the biological cell assumes it to be an elastic cortex surrounding a viscous cytoplasm that contains an elastic nucleus at the centre. This is a "continuum" model and assumes that the load-bearing elements are infinitesimally small compared to the overall size of the cell. Although this model has successfully explained many cellular behaviours, it does not take into account the distinct functional contributions of the cytoskeleton network.

For the past 30 years, the Ingber Laboratory at Harvard Medical School has been demonstrating that the "balloon" model of the cell is inadequate. Instead, a cellular model based on tensegrity architecture has been proposed and has been successfully explaining many cellular and tissue behaviours, both during normal metabolic activity and in disease (Ingber, 2003, 2004a, b, among others). This "new" cellular model is crucial to understanding the type of pathology developed by ILFN-exposed biological organisms because only the tensegrity model adequately explains how mechanical signals are transduced over cells and tissues.

At present, cell and tissue regulation is considered to be largely mediated by molecular conformations, intermolecular interactions, and linear signal transduction cascades. But this is proving to be a reductionist approach because the neglected mechanotransduction cellular signalling also plays a key role in cell and tissue communication (Ingber, 2004a). Modern medicine focuses on the importance of genes and chemical factors to control and explain tissue physiology and disease development. The "genome euphoria" (Ingber, 2003) disregards the physical (structural and mechanical) properties of cells and tissues despite the fact that in order to maintain normal cell behaviour (motility, growth, apoptosis), the ability of cells to sense and respond to mechanical stresses is of critical importance (e.g., Wang et al., 1993; Matthews et al., 2004; Alenghat et al., 2004).

3.1. Tensegrity architecture

At the turn of the 20th century, mechanical interpretations of biological behaviour were a common methodology. Form and function of cells and tissues were of great importance to understanding biological

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processes. However, as the field of molecular biology developed, biochemicals and genes became the forefront of scientific interest. "Medicine went from a holistic view of describing the relation between form and function to a much more reductionist view of describing what life is made of. And the mechanics were thrown out like the baby with the bathwater" (Ingber, 2004b).

The term *tensegrity* (tensile integrity) was coined by R. Buckminster Fuller, "father" of the geodesic dome in Architecture, and of the bucky ball in Physics (Fuller, 1975). Tensegrity is a form of structural stabilization that minimizes weight by using discontinuous-compression and continuous-tension, as opposed to continuous-compression. To visualize the difference, compare a brick-on-brick type of construction (continuous compression) with a stick-and-elastic construction of a geodesic dome, where the sticks are the discontinuous-compression elements while the elastics provide the continuous tension. Anchoring points, or nodes are essential to tensegrity structures because it is through these points that mechanical forces are transduced throughout the constituent compression and tensile elements. Any local, external perturbation of a tensegrity structure will result in a well-organized redistribution of tensional forces throughout the entire structure, with the purpose of maintaining structural integrity.

3.2. Cellular tensegrity architecture

Cellular cytoskeletons (CSK) form isomeric networks of microtubules, intermediate tubules, intermediate filaments, and actin. Forces generated within the CSK are involved in cytoplasmic organelle transportation (mitochondria and synaptic vesicles), chromosome movement during mitosis, and tension generation in the muscle cell contraction process (Ingber, 2003a). The CSK receives signalling from other cells through cell–cell junctions, and from the extra-cellular matrix (ECM), through cell–matrix junctions. Table 1 summarizes the properties of both types of junctions.

In the CSK, microfilaments form a mesh network of fine cables that constitute the continuous-tension elements (elastics) of the cellular tensegrity model. The compression elements (sticks) are formed by microtubules that are anchored to the ECM through transmembrane proteins called integrins, at sites called focal adhesions. Integrins differ from other cell-surface receptors because they bind with relatively low affinity ($Ka = 10^6$–10^9 l/mol), and their highest concentration is on cell surfaces.

Previous studies have probed the functioning of focal adhesion integrin receptors through magnetic twisting cytometry (Wang et al., 1993) and magnetic microneedle manipulation followed by magnetic pulling cytometry (Matthews et al., 2004). At focal adhesions, CSK and ECM possess structural linkages in the form of integrin cell-surface receptors. Mechanical forces applied directly to integrin cell-surface receptors alter cell biochemical and gene expression in a stress-dependent way (Ingber, 2003, 2004a, b; Wang, et al., 1993;

Table 1
Properties of cell–cell anchoring junctions (adherens junction and desmosomes) and of cell–matrix anchoring junctions (focal adhesions and hemidesmosomes)

	Cell–cell		Cell–matrix	
Junction	Adherens junction	Desmosome	Focal adhesions	Hemidesmosomes
Intracellular (CSK) attachment filaments	Actin	Intermediate filaments	Actin	Intermediate filaments
Transmembrane adhesion protein	E-cadherin	Cadherin (desmoglein, desmocollin)	Integrin	Integrin ($\alpha_6\beta_4$, BP180)
Extracellular ligand	Cadherin (adjacent cell)	desmogleins, desmocollins (adjacent cell)	ECM proteins	ECM proteins
Intracellular anchor protein	α,β-catenins, vinculin, α-actinin, γ-catenin	desmoplakins, γ-catenin	talin, viniculin, α-actinin, filamin	Plectin, BP230

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Matthews et al., 2004; Alenghat et al., 2004). When the same forces are applied to other types of membrane receptors, there is no such effect.

External forces applied to integrins can activate intercellular signalling pathways, such as, protein tyrosine phosphorylation, ion fluxes, cAMP production, and G protein signalling (Ingber, 2003, 2004a, b; Wang, et al., 1993; Matthews et al., 2004; Alenghat et al., 2004). These integrin linkages allow for mechano-chemical transduction signalling that produce changes in cell form and function. This type of intracellular signalling is a critical regulator of cellular biochemistry, gene expression and tissue development (Ingber, 2003, 2004a, b; Wang, et al., 1993; Matthews et al., 2004; Alenghat et al., 2004). There is a large variety of mechanochemical-transducing integrin receptors molecules; each type of integrin only binds to one ECM macromolecule, and cell-type specificity modulates integrin binding activities, i.e., in fibroblasts, ligands bind specifically to collagen, fibronectin and laminin. Hemidesmosomes (connecting the basal lamina to adjacent cells) are integrin receptors but that do not bind to the CSK through the actin cortex. Instead, hemidesmosome integrins connect directly with intermediate filaments. In the CSK, these are responsible for helping individual microtubules from buckling under compression, and link the nucleus to the surface membrane. Desmosomes connect intermediate filaments from cell to cell. The biological explanation for some diseases based on mechanotransduction impairment has already been successfully advanced (Ingber, 2003, 2004a, b).

4. VAD

VAD is a systemic pathology, caused by excessive exposure to ILFN, and characterized by the abnormal proliferation of collagen and elastin, in the absence of an inflammatory process. VAD has been diagnosed in aeronautical technicians (Castelo Branco, 1999a), pilots and flight attendants (Araújo et al., 2001), as well as in an islander population exposed to environmental ILFN (Torres et al., 2001). Cases of VAD have also been documented among ship workers (Arnot, 2003) and in residential areas (Araújo et al., 2004, Monteiro et al., 2004).

4.1. Brief chronology of scientific enquiry over the past 25 years

In 1980, co-author Castelo Branco was appointed chief medical officer at an aircraft manufacturing, repair and rework facility (OGMA), owned and operated by the Portuguese Air Force, and employing around 3500 workers. The first step was to visit the workstations of all employees to assess the nature of the different occupational hazards, possible emergency situations that could arise, and types of required worker protection.

After maintenance is performed on an aircraft, Quality Control personnel carry out manufacturer's procedures while the aircraft is stopped on the tarmac, and has its engines test run at all possible speeds. During one of these run-up tests (EA3B, with afterburn) Castelo Branco observed a worker beginning to walk aimlessly, without purpose, and in the direction of the turbines. A co-worker grabbed him by the arm before he got too close, and the incident remained at that. After the run-up test, the co-worker was questioned about what had happened. Apparently it was not a rare occurrence, and in the 1960s someone *had not* been caught in time, which led to a fatality. The non-purposeful movements exhibited by the worker appeared to Castelo Branco to be of an epileptic nature.

OGMA was founded in 1918 and, since the 1960s, detailed medical records are kept for all workers (administrative and technical). Based on the observation during the run-up test, the second step was to survey all medical records to count how many technicians had previously been diagnosed with late-onset epilepsy, as detailed in their medical files. In the Portuguese general population, the incidence of epilepsy is 0.2%. In the group of 306 aircraft technicians employed at OGMA, 10% had been previously diagnosed with late-onset epilepsy (GIMOGMA, 1984a). Here began this team's enquiry into ILFN-induced pathology.

4.1.1. 1980

- Establishment that 10% ($N = 306$) of the aircraft technicians employed at OGMA had been previously diagnosed with late-onset epilepsy (GIMOGMA, 1984a).

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- Initiated neurophysiological examinations. The results from brainstem auditory evoked potentials (BAEP) were initially difficult to interpret, given their large dispersion. To better evaluate the BAEP recordings, taxonomic distances using clustering algorithms, and multivariate analysis of action currents distributions were applied, and a standardized method was developed using a control population (Castelo Branco et al., 1985; Marvão et al., 1985).

4.1.2. 1984–1988

- Publication of the first articles on initial findings under the team name of GIMOGMA[1]: Epilepsy (GIMOGMA, 1984a), BAEP study (GIMOGMA, 1984b), and hyper-sensibility to noise (GIMOGMA, 1984c), otherwise known as noise intolerance or annoyance. A vascular involvement began to be suspected.

Until this point, it was thought that the neurological pathology observed in this group of workers, initially termed "vibration disease", was due to excessive exposure to vibration. Neurological parameters continued to be assessed.

- Studies showed abnormal magnetic resonance imaging of the central nervous system (Cruz Maurício et al., 1988) and cognitive potentials (P300) (Moniz Botelho et al., 1988) in aircraft technicians.
- Other, non-neurological changes were identified, including damaged dental alveolar structures (Cortez-Pimentel and Castelo Branco, 1988); haemostasis and coagulation changes (Crespo et al., 1988), and abnormal retinal angiography (van Zeller et al., 1988). The latter two suggested the pathology was of a vascular nature.
- Four cases of pleural effusion developed in these workers, all of unknown aetiology. They exhibited an atypical response to standard therapeutics, and endured unusually prolonged recovery periods.

During these years, "systemic vibration disease" was the term used to identify the pathology observed in aircraft technicians (Pimenta et al., 1988). This meant that the health problems these workers were developing were not necessarily restricted to the neurological system.

- September 1987: Autopsy of an aircraft technician (Castelo Branco, 1999b). The plethora of scientific data bequeathed by this deceased patient disclosed the real extent of this pathology: 11 scars of previous silent infarct events, two previously undetected malignant tumours (kidney and brain), thickened blood vessel walls, thickened pericardium, and focal lung fibrosis.

4.1.3. 1989–1992

During this period, it was determined that the fundamental agent of disease to which aircraft technicians were exposed was ILFN (Bento Coelho et al., 1994), hence, the pathological entity was, again, renamed: "whole-body noise and vibration syndrome" (Castelo Branco, 1992).

- The thickened blood vessels and pericardium found in autopsy prompted echo-imaging studies, namely echocardiography for assessing pericardial thickening. All aircraft technicians presented abnormal pericardial and/or cardiac valve thickening (Araújo et al., 1989).
- Carotid angiodynography was used to assess carotid thickening (Albuquerque et al., 1991; Carmo et al., 1992). Simultaneously, other populations occupationally exposed to ILFN began to be studied, such as helicopter (Carmo et al., 1992) and military (Canas et al., 1993) pilots.
- Wistar rats were chosen as animal models to investigate the effects of ILFN exposure on the respiratory tract, in an attempt to explain the atypical cases of pleural effusion, of unknown aetiology.

[1]GIMOGMA—Grupo de Investigação Médica das OGMA—was the acronym for the medical reserach team that began its activity at OGMA, and that today is represented by the Center for Human Performance.

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4.1.4. 1993–1999

In 1993, during a scientific meeting sponsored by our team, the term "vibroacoustic" was proposed for this pathological entity (Castelo Branco and Rodriguez Lopez, 1999a), and "vibroacoustic syndrome" became the new name for the ailment observed in aircraft technicians and, now, also in aircraft and helicopter pilots (Castelo Branco et al., 1996).

- Animal studies showed that the respiratory tract could be considered a primary target for ILFN: abnormal amount of fibrosis/collagen was ubiquitous in trachea, lungs and pleura; damaged (sheared) tracheal and bronchial cilia; fused actin-based microvilli of tracheal and bronchial brush cells (Sousa Pereira et al., 1999a; Grande et al., 1999). The atypical cases of pleural effusion were partially explained by morphofunctional impairment of pleural microvilli (Sousa Pereira et al., 1999b), as well as of pleural phagocytic capabilities (Oliveira et al., 1999).
- Additional neurological disorders were identified in ILFN-exposed populations, such as the existence of the palmo-mental reflex, usually only seen in primates, newborns, and the elderly (Martinho Pimenta et al., 1999a); balance disturbances (Martinho Pimenta et al., 1999b), and facial dyskinesia induced by auditory stimuli (Rosado et al., 1993; Martinho Pimenta and Castelo Branco, 1999c).
- The genotoxicity of ILFN was demonstrated in both human (Silva et al., 1999, 2002a) and animal models (Silva et al., 2002b), and was confirmed by teratogenic features in mice (Castelo Branco et al., 2003 g).
- Echocardiography was deemed an unreliable diagnostic tool because there is no established procedure to assess pericardial thickening and, thus, technician subjectivity introduced a large factor of error.
- The first human pericardial fragments were studied in VAD patients who required cardiac bypass surgery for other reasons (See below): abnormal amounts of collagen as well as the neo-formation of an extra layer of tissue was shown to be the cause underlying the pericardial thickening, providing anatomical confirmation of the autopsy and echo-imaging observations (Castelo Branco et al., 1996).
- Medical files of all aircraft technicians were chronologically reviewed since their admittance to OGMA. On-the-job accidents and incidents were correlated with the existence of unmonitored ILFN exposure of the workforce (Alvarez et al., 1993), and the clinical phases of the disease were outlined (Castelo Branco and Martinho Pimenta, 1995).

In 1999, the name "VAD" was adopted, and the journal Aviation, Space & Environmental Medicine dedicated a supplemental issue to this new pathological entity (Castelo Branco et al., 1999c).

4.1.5. Since 2000

- Other ILFN-exposed professionals were studied, such as civil aviation pilots and cabin crewmembers, confirming echocardiography results of aircraft technicians and military pilots (Araújo et al., 2001).
- More neurological pathology was identified: VAD patients were found to be unable to hyperventilate when in the presence of excessive CO_2 (Reis Ferreira et al., 2003a).
- Mechanically induced cellular death was identified in the pericardia of VAD patients and it was hypothesized that this situation could be related to the large incidence of auto-immune disorders in these patients (Castelo Branco et al., 2004b).
- Further rat studies suggested that fusion of cochlear cilia (actin-based structures) may provide a biomechanical explanation for noise intolerance, or annoyance (Castelo Branco et al., 2003a).
- The first case of large-scale environmental exposure to ILFN appeared in Vieques, Puerto Rico (Torres et al., 2001). Here, ILFN was caused by military training exercises. An isolated case came from Dublin, Ireland, where buses where the source of ILFN and induced VAD in a home-maker (Monteiro et al., 2004). Another from Lisbon, where ship-to-silo and silo-to-ship loading of cereals produces ILFN in a home where both parents and 10-year-old child exhibited VAD-related signs and symptoms (Araújo et al., 2004).
- All bronchoscopic examinations of VAD patients disclosed lesions that, upon analysis, demonstrated the existence of abnormal amounts of collagen, and neo-formation of vascular beds. Disrupted collagen fibers were observed and correlated with a positive testing of anti-nuclear antibodies, providing a deeper understanding of auto-immune processes (Monteiro et al., 2004a).

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4.2. Clinical stages of VAD

In order to identify the clinical stages of VAD, as observed in aeronautical technicians, a systematic and detailed review of the medical files pertaining to the initial group of 306 aircraft technicians was undertaken in the mid 1990s. This group of 306 male individuals were all employed by OGMA for more than 10 years, and were submitted to rigorous selection criteria, as per Table 2 (Castelo Branco, 1999a).

A group of 140 technicians (average age of 42 years, SD = 10.4) remained after the application of selection criteria, i.e., 166 individuals were excluded. The medical files of these 140 technicians were comprehensively and chronologically reviewed. Simultaneously, a sociologist and a social worker interviewed family and friends to obtain additional information on the individual's behaviour outside his professional activity. The methodology to obtain a correspondence between sign/symptom and years of occupational exposure was the 50% cutoff, i.e., the sign/symptom was included in the list if it was identified in 50% ($N = 70$) of the study population. Thus, referring to Table 2, after 1–4 years of occupational exposure, at least 70 of these 140 individuals developed bronchitis, in smokers and non-smokers alike (smokers in study group: $N = 45$). Or, after 10 years of occupational activity, at least 70 exhibited headaches and nose bleeds. It should be emphasized that these signs and symptoms are not mutually exclusive, and most VAD patients suffer from more than one or two of these clinical situations, simultaneously (Castelo Branco, 1999a; Castelo Branco and Alves-Pereira, 2004a).

Table 3 refers to the signs and symptoms developed specifically by aircraft technicians working the standard 8 h/day, 5 days/week. Not all ILFN-exposed workers have this exposure schedule. For example, ship machinists can spend 3 weeks onboard ship (i.e., exposed to substantial ILFN-rich environments) and 2 weeks at home (i.e., presumably not in ILFN-rich environments) (Arnot, 2003). Other professional activities exist where the ILFN-exposure time pattern is not the standard 8-h/day exposure, such as with submarine and oil

Table 2
Conditions for study population exclusion (Castelo Branco and Rodriguez Lopez, 1999a)

Conditions	Comments
Streptococcal infections	Due to their propensity to induce extra-cellular matrix changes
Diabetes mellitus	Same as above
Pre-existing cardiovascular disease	But not labile hypertension, because it is suspected to be a measure of individual susceptibility, and because lesions are distinct from those caused by established hypertension
Tobacco abuse	Smokers of more than 20 cigarettes a day
Alcohol abuse	Drinkers with more than a liter of wine per day (10–12% alcohol content)
Drug use	Users of any recreational or psychotropic drug

Table 3
Data from a group of 140 aircraft technicians

Clinical stage	Sign/symptom
Stage I-Mild (1–4 years)	Slight mood swings, indigestion and heart-burn, mouth/throat infections, bronchitis
Stage II-Moderate (4–10 years)	Chest pain, definite mood swings, back pain, fatigue, fungal, viral and parasitic skin infections, inflammation of stomach lining, pain and blood in urine, conjunctivitis, allergies
Stage III–Severe (>10 years)	Psychiatricdisturbances, haemorrhages of nasal, digestive and conjunctive mucosa, varicose veins and haemorrhoids, duodenal ulcers, spastic colitis, decrease in visual acuity, headaches, severe joint pain, intense muscular pain, neurological disturbances

ILFN exposure time (years) refers to the amount of time it took for 70 individuals (50%) to develop the corresponding sign or symptom (Castelo Branco, 1999a).

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rig operators, astronauts, and environmental exposures in residential areas, where exposure can be continuous over long periods of time, and exists during sleeping hours. In these cases, the evolution of signs and symptoms could be greatly accelerated. For example, in the case of a Dublin homemaker, epileptic seizures consistent with VAD developed after 3 years of residence within a ILFN-infested home (Monteiro et al., 2004). If the ILFN exposure is environmental and/or leisurely, the standard 8 h/day model is also not applicable. Moreover, since different ILFN environments have unique frequency distributions, the fact that some frequency bands may be more predominant than others (i.e., concentrate more acoustical energy) can lead to the development of slightly different pathology.

4.3. Pathology associated with VAD

Other important pathologies were identified among these 140 aircraft technicians, but since they were not identified in 50% of the population, they were not included in Table 3. Nevertheless, their incidence is clinically important. Some kind of respiratory insufficiency was found in 24 of the 140 professionals, 11 were smokers. In 10 of the 24 cases, a mere light physical effort was necessary to produce symptoms. Notably, only 45 of the 140 individuals were smokers, 38 of which had over 20 years of occupational ILFN exposure.

Late-onset epilepsy was diagnosed in 22 individuals, some of whom saw their seizures subside when away from their workstation. Reflex epilepsy due to vibratory stimulus (Martinho Pimenta and Castelo Branco, 1999c) and visual stimulus was observed in two individuals. Auditory stimuli did not trigger seizures but, in some cases, triggered rage reactions and movement disorders (Martinho Pimenta and Castelo Branco, 1999d, e). Balance disturbances were also a common complaint, identified in 80 individuals, although the severity of the balance disturbance ranged from dizziness to severe vertigo (Martinho Pimenta et al., 1999f). Unique and sudden episodes of non-convulsive neurological deficit occurred in 11 individuals. These were diagnosed as cerebral ischaemic vascular accidents, which was compatible with imaging studies. EEG and multi-modal evoked potentials showed considerable power changes that were in agreement with clinical psychological and neurological evidences. Delays in multi-modal evoked potentials (including endogenous), observed in all 140 patients, are a sign of progressive neurological deterioration and early aging process, as is the appearance of the archaic palmo-mental reflex, that affects about 40% of these 140 patients.

Endocrine disorders, the most common being thyroid dysfunction, were identified in 18 cases. The overall national Portuguese rate for adult thyroid dysfunction is 0.97% vs. the 12.8% identified in our group of 140 technicians. Similarly, diabetes was seen in 16 individuals (average age 39 years, SD = 7.8) (11.4%), while the overall national rate for a similar age-group is 4.6% (Castelo Branco, 1999a). Among the 140 professionals, 28 had malignant tumours. Five of these 28 individuals exhibited simultaneous tumours of different types. All CNS tumours ($N = 5$) were malignant gliomata, and all respiratory system tumours were squamous cell carcinomas (5 in lung, 1 in larynx). To date, and to the authors' knowledge, a total of 11 VAD patients have developed respiratory tract tumours: 9 in the lung, and 2 in the glottis (3 smokers); *all* have been squamous cell carcinomas (Mendes et al., 2004; Reis Ferreira et al., 2005). Other tumours were found in the stomach ($N = 10$), colon and rectum ($N = 9$), soft tissue ($N = 1$), and bladder ($N = 1$) (Castelo Branco, 1999a). All digestive system tumours were low-differentiated adenocarcinomas. These data led to the investigation of the genotoxicity of ILFN. In both human (Silva et al., 1999, 2002a) and animal (Silva et al., 2002b) models, ILFN induced an increased frequency of sister chromatid exchanges, effectively demonstrating that ILFN is a genotoxic agent.

More recently, in 2003, a new pathological sign was identified among VAD patients: decreased respiratory drive (Reis Ferreira et al., 2003a; Castelo Branco et al., 2003b). To date, pulmonary function tests are normal in VAD patients, except the $P_{0.1}(CO_2)$ index and the metacholine reactivity test. The $P_{0.1}$ (CO_2) index is a measure of the inspiratory pressure (or suction) developed at the mouth, 0.1 s after the start of inspiration. This initial respiratory drive originates in the autonomic (or involuntary) pathway of the neural control of the respiratory function. By rebreathing CO_2, normal individuals would present a minimum six-fold increase of the $P_{0.1}(CO_2)$ index when compared to normal $P_{0.1}$. If the neural control of respiration is compromised, then a less-than six-fold increase would be expected in the $P_{0.1}(CO_2)$ index (Calverly, 1999; Cotes, 1993; Gibson, 1996). In VAD patients, all $P_{0.1}(CO_2)$ index values are below 50%, when normal values would be above 60%.

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Lastly, the issue of auto-immune diseases in ILFN-exposed individuals. In the electron microscopy studies of VAD-patient pericardial fragments, non-apoptotic cellular death was frequently observed (Castelo Branco et al., 2003c) (see below). Instead, biomechanical forces seemed to be responsible for the images of burst cells, with live organelles and no surrounding plasma membrane. Under these circumstances, the appearance of auto-immune diseases in these patients is not unreasonable. Indeed, previous studies have shown that ILFN exposure induces an accelerated onset of lupus in lupus-prone mice (Águas et al., 1999a). Lupus has also been identified in flight attendants (Araújo et al., 2001), and in entire families of islanders exposed to environmental ILFN (Torres et al., 2001). Vitiligo is another common finding, especially in the ILFN-exposed islander population. Vitiligo is associated with immune changes of CD8 and CD4 lymphocyte populations. These immune changes have also been observed in ILFN-exposed workers (Castro et al., 1999) and animal models (Águas et al., 1999b). Other authors have also corroborated the existence of auto-immune processes in noise-exposed workers (Matsumoto et al., 1989,1992; Jones et al., 1976; Soutar et al., 1974; Lippmann et al., 1973).

4.4. Some important considerations on behalf of VAD patients

Legally, the only pathology that can develop due to excessive noise exposure is hearing impairment. Therefore, occupational physicians rarely view VAD symptomatology as caused by excessive noise exposure. In fact, given the plethora of complaints associated with VAD (see Table 3), oftentimes physicians regard the patient as a malingerer or hypochondriac (Castelo Branco and Rodriguez Lopez, 1999a), especially since routine medical tests (e.g., blood chemistry analysis, EKG and EEG) do not corroborate the existence of any pathology. One of the reasons for this is that the majority of medical diagnostic tests are based on biochemical, and not biomechanical, pathways (see below). There are dire consequences for the patients, as have been candidly exposed by a Scotsman, who was employed as a motorman, and developed VAD (Arnot, 2003).

In the case of occupational exposure to ILFN, workers can develop disabilities requiring early retirement (Castelo Branco et al., 1999d). Usually ILFN-rich environments are associated with machinery that, in an ever-developing technological world, often becomes obsolete within a few years time. At present, many individuals who have developed VAD due to occupational exposures cannot prove that they have been exposed to ILFN because noise assessments do not take ILFN into account (as described above), and many of the ILFN sources have been retired.

5. VAD in light of mechanobiology

The establishment of VAD has been problematic because of several, non-typical situations that seem to defy conventional medical concepts. For example, the production of collagen, in the absence of an inflammatory process, is consistently seen in the blood and lymphatic vessel walls (Castelo Branco et al., 1999b; Reis Ferreira et al., 2003b, c; Monteiro et al., 2004a), pericardium (Castelo Branco et al., 1999b, 2003c), trachea (Reis Ferreira et al., 2003b), and lung and pleura (Reis Ferreira et al., 2003c) of VAD patients. It is also observed in the respiratory tract (Castelo Branco et al., 2003a), kidney (Castelo Branco et al., 2003d), blood and lymphatic vessels (Martins dos Santos et al., 2002, 2004, respectively) of ILFN-exposed animals.

Much of the data collected on VAD and ILFN-exposed biological tissues has been in the form of ultrastructure micrographs, obtained with scanning (SEM) and transmission (TEM) electron microscopy. The following sections will describe the anatomical findings in VAD patients' pericardia, and in the respiratory tract and cochlea of ILFN-exposed rats, based on information obtained through histological and ultrastructural studies. The implications within the context of mechanobiology will be discussed.

5.1. The pericardium

The pericardium is a fibrous sac that encases the heart, with the purpose of maintaining it in its normal position. External forces, due to respiration or changes in body posture, are absorbed by the pericardium so as to keep the heart and its cardiac rhythm intact. Consisting of three tissue layers—mesothelium, fibrosa and epipericardium—the pericardium is a highly organized mass of connective tissue, with a predominance of

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collagen fibers arranged in accordion-like bundles. Elastic fibers, much less numerous than collagen fibers, intersect the collagen bundles at right angles. This anatomical arrangement taken together with the viscoelastic properties of both collagen and elastin, provide the pericardium with the mechanical capability of protecting the integrity of the cardiac cycle. The thickness of normal parietal leaflet of the pericardium is <0.5 mm (Shabetai, 1994). The mesothelium is in direct contact with the pericardial sac, and is formed by a one-layer thick sheet of mesothelial (cuboidal) cell (MC). Anchoring junctions interconnect MC among themselves, through their cytoskeletal fibers—microtubulues—interconnected through desmosomes (see Table 1). Microtubules do not rupture when stretched, can withstand larger stresses and strains than actin filaments, and are crucial to maintain cellular integrity.

Abnormally thickened pericardia were first observed by this team in autopsy (Castelo Branco, 1999b), and later confirmed through echocardiography (Araújo et al., 1989; Marciniak et al., 1999; Torres et al., 2001). No inflammatory process was present, no cardiac dysfunction was identified and, thus, pericarditis is not an issue in VAD patients. The strange feature was that despite the extraordinary enlargement, no diastolic impairment was observed: EKGs of VAD patients were normal, as were the cardiac functional parameters assessed through echocardiography. Simultaneously, echo-imaging did not have a 1–1 correspondence with anatomical structures, thus, it became important to understand what was occurring at the anatomical level.

Since one of the consequences of ILFN exposure is thickening of blood vessel walls (Castelo Branco, 1999b; Castelo Branco et al., 1996, 1999b, 2003c; Reis Ferreira et al., 2003b, c) the recommendation for cardiac bypass surgery by other physicians is not uncommon among VAD patients. Hence, it was possible, with Hospital Ethics Committee approval and patients' fully informed consent, to study pericardial fragments of VAD patients (Castelo Branco et al., 1996, 1999b, 2004b). Pericardial thickening was confirmed anatomically, and a possible reason for the lack of diastolic dysfunction was uncovered (see Fig. 2).

When electron microscopy was used to examine these pericardial fragments, an unusual amount of cellular death was observed. This peculiar type of cellular death was related to the mechanical bursting of cells, with images of seemingly live organelles outside of the burst cytoplasmic membrane (Figs. 3 and 5). Cellular debris

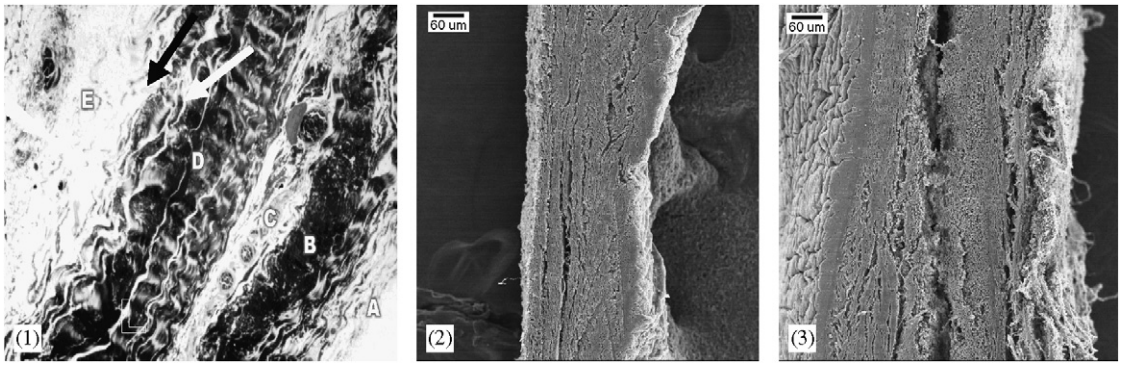


Fig. 2. (1) Light microscopy (100 ×)—VAD patient pericardium, with pericardial sac on right. Five (instead of the normal three) layers are identifiable: (A) mesothelial, (B) internal fibrosa, (C) loose tissue, (D) external fibrosa, and (E) epipericardium. The loose tissue is rich in vessels. No inflammatory cellularity was identified in any of the five layers. In both fibrous layers, wavy collagen bundles are visible, however the wave length of fibers in layer B (internal fibrosa) is smaller than that in layer D (external fibrosa). Taking together the increased amount of collagen bundles, in wavy, accordion-like arrangements, with different orientations in relation to each other, and with more than one elastic fiber accompanying the bundles at seemingly perpendicular angles (seen through electron microscopy, not shown), seems to suggest a pneumatic-like structure, designed to absorb abnormally large external forces. Similarly, this functional arrangement also explains why there is no diastolic dysfunction, despite the thickened pericardial walls. (2) SEM of non-VAD patient pericardium. Normal three layers are visible: mesothelium (white arrow), fibrosa (black arrow) and epipericardium. (3) SEM of VAD patient pericardium. Fibrosa has split into two halves (arrows) that sandwich a newly formed layer of loose tissue (L). Note that the scale in both (2) and (3) is the same. The wavy form of collagen bundles is a mechanically energy-efficient method to deal with the movement that the fibrosa must constantly undergo to follow the rhythm of the cardiac cycle. Similar to an accordion, collagen bundles will extend and contract in diastole and systole, respectively. However, during an episode of sudden and violent tachycardia (common in VAD patients), this rhythm can be greatly increased (up to 200 beats per minute, in a matter of seconds) and the mechanical stress imposed on the MC monolayer may threaten its structural integrity. One of the functions of the loose tissue layer must certainly be blood and nutrient supply to this much larger organ.

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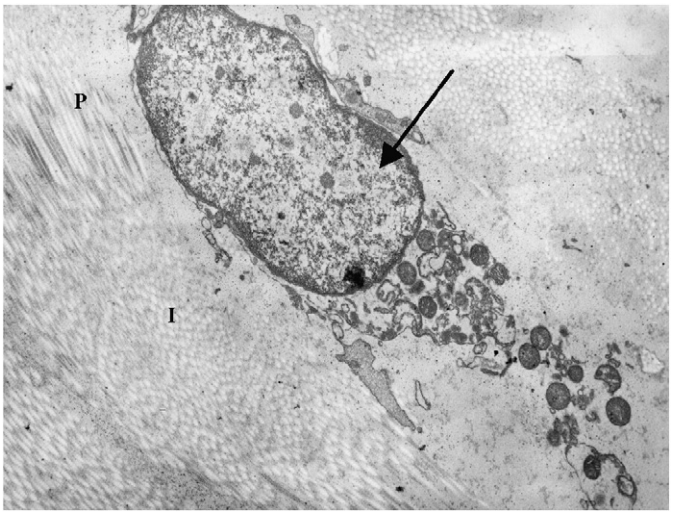


Fig. 3. (TEM) VAD patient parietal pericardium. Deep within the internal fibrosa layer: a burst myofibroblast near a small elastic fibre surrounded by abnormally abundant collagen bundles. Seemingly live organelles are outside the membrane (4000 ×).

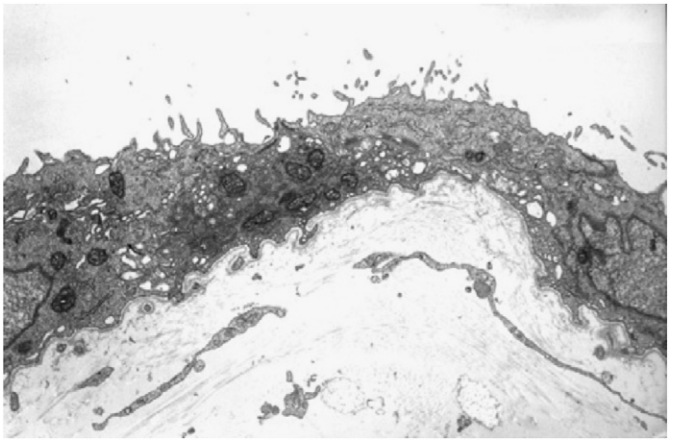


Fig. 4. (TEM) VAD patient pericardial fragment. Pericardial sac (P); Unusual number of cell–cell connections (arrow). Interdigitations (I) linking the MC layer with the subserosal basal layer, through structures reminiscent of anti-sesmic constructions (2800 ×).

was seen in all layers and in the vast majority of the images (Fig. 5). Individual, older MC were seen protruding into the pericardial sac, in a process that resembled surface extrusion of that cell (Fig. 6). Discontinuities in MC surface (in direct contact with the pericardial sac) (Fig. 5), with seemingly live organelles spewing into the pericardial sac (Fig. 6) were observed. Desmosomes, which laterally attach adjacent MC (Table 1), were more numerous than would be expected (Fig. 4). The lack of cellular anchoring junctions between MC and the subserousal basal layer (usually accomplished through hemidesmosomes, see Table 1) seems to be replaced by interdigitations whose form is reminiscent of anti-sesmic constructions (Fig. 4). MC morphology varied in accordance with the contraction wave of the cardiac rhythm (Castelo Branco et al., 1999b, 2005)

VAD patients often suffer sudden and violent tachycardia, and sudden peaks of increased arterial blood pressure (Castelo Branco, 1999a). This implies a violent and sudden kinetic changes in the cardiac (and pericardial) rhythm. During repeated, VAD-related tachycardia and hypertensive episodes, MC cells become enormously strained, and maintaining structural integrity of the MC monolayer might become an issue. Older

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MC cells may have insufficient tensile strength to undergo these violent tachycardia movements, and their extrusion (Fig. 6) from the mesothelium monolayer of cells may be an attempt to maintain structural integrity. The formation of gaps near these older MC cells (Figs. 5 and 6) seems to be an integral part of this extrusion process. The anchoring junctions between the mesothelium and its ECM sublayer seem to have been replaced with MC cytoplamsic interdigitations that dig deep into the ECM sub-layer (Fig. 4). This anchoring structure is not unlike modern anti-sesmic constructions, where building blocks fit into each other like pieces in a vertical jigsaw puzzle, providing increased plasticity, and allowing the absorption of large mechanical forces without rupturing.

The existence of such a large amount of cellular debris has been linked to a working hypothesis: The cellular debris seen in VAD patients' pericardial layers can be related to the appearance of auto-immune diseases in ILFN-exposed individuals. Seemingly live organelles that exist outside the cellular membrane envelope (Figs. 3 and 5) will not be identified by the immune system. This could trigger auto-immune disorders. No inflammatory process is tied to the removal of debris. The only visible features are an increased amount of macrophages and neovascularization, with particular relevance to the lymphatic vessels, where the drainage of debris seems to occur.



Fig. 5. (TEM) Pericardial sac (P), collagen bundles (C), cellular debris (×). Discontinuous membrane (arrow), partial loss of cytoplasm. Gaps seen near MC (circle) (4000 ×).

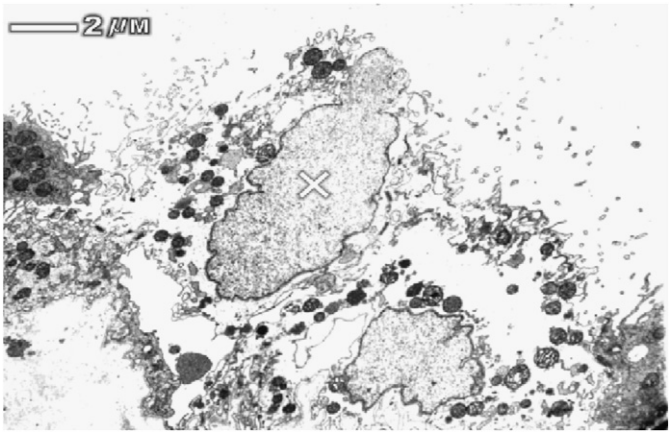


Fig. 6. (TEM) Mesothelial layer with older mesothelial cell (×) in the process of extrusion into the pericardial sac, and with large gaps on either side.

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5.2. Actin-based structures—brush cell (BC) microvilli and cochlear cilia

BC exist in the respiratory and gastrointestinal tracts. BC possess microvilli uniformly distributed over the apical surface which is known to play a role in increasing absorption surface area. The function and existence of the respiratory BC in humans is largely unknown. In the rat respiratory tract, BC are surrounded by a ring of secretory cells (SC) (Fig. 7) (Castelo Branco et al., 2003e). In ILFN-exposed rodents, microvilli clustered together and, with increasing exposure time, became fused (Fig. 8) (Castelo Branco et al., 2003a, f, g). Cochlear stereocilia also appeared fused in ILFN-exposed rats, both among themselves as well as with the upper tectorial membrane (Figs. 9 and 10) (Alves-Pereira and Castelo Branco, 2003a).

Why BC microvilli respond to prolonged ILFN stress by fusing is unknown. However, the fact that actin filaments can form both rigid (but flexible) bundles as well as gel-like networks, taken together with the fact that motor proteins connect the actin filaments core to the plasma membrane, microvilli fusion does not seem to be such a remote possibility, given the right triggering events.

If fusion of cochlea stereocilia, as a response to prolonged ILFN exposure, also occurs in humans, then this may explain the unusual auditory complaints of VAD patients. Common auditory complaints of VAD patients include "hearing too much" and "not being able to stand any type of noise, not even television or music". However, their audiograms only present hearing losses within the lower frequency bands (250 and 500 Hz), and their tympanograms are normal (Castelo Branco, 1999a). If fused among themselves and to the tectorial membrane, cilia cannot freely vibrate as is intended when the sound pressure wave is transduced within the cochlea. In fact, by becoming a rigid structure, any attempt at vibrating them might, understandably, produce discomfort. How closely related this phenomenon is to the concept of "annoyance" is still unclear, however a relationship is clearly suggested, especially since annoyance has already been specifically associated with the presence of ILFN (Persson-Waye and Rylander, 2001). In ongoing studies, fusion of actin-based structures in ILFN-exposed rodents has also been observed in the duodenum (Fonseca et al., 2005).

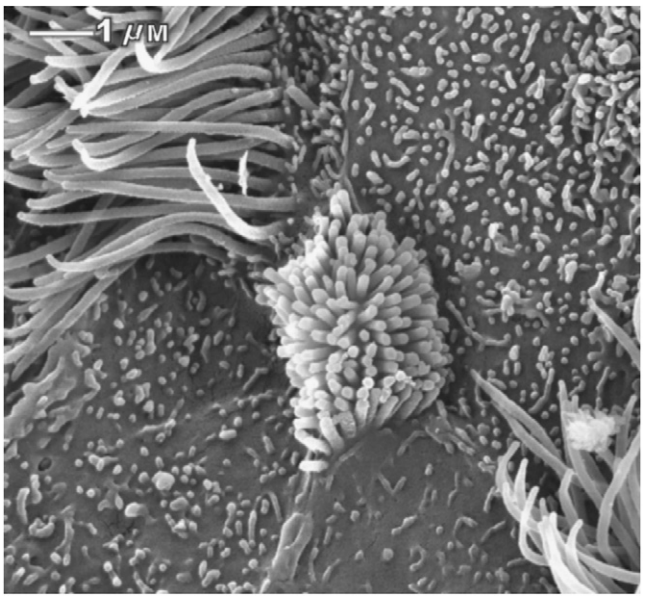


Fig. 7. (SEM) Non-exposed rat bronchial epithelium. The BC, in the center of the image, exhibits a tuft of microvilli that are individually identifiable, uniformly distributed, and sprouting upward into the airway. Surrounding the BC are SC with microvilli of different sizes. Tufts of cilia featuring vesicles are also visible. No sheared, shaggy or wilted cilia are visible. No edema is present.

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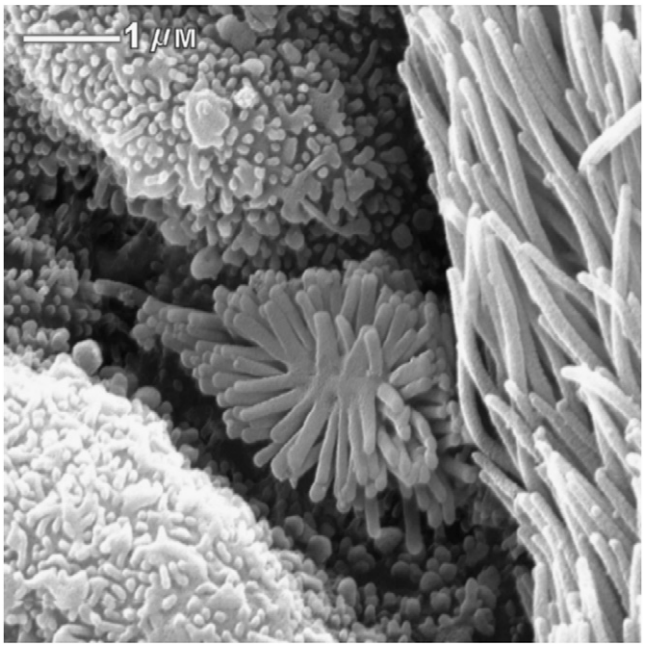


Fig. 8. (SEM) Rat bronchial epithelium exposed to 2160 h of continuous ILFN. A BC is in the centre of the image. Its microvilli are not sprouting upward and, instead, have fused, forming a central indentation that seems to be spreading outward. The prominent SC that surround the BC are swollen forming deep valleys at the intercellular junctions. SC microvilli are very irregular. Cilliary vesicles are visible.

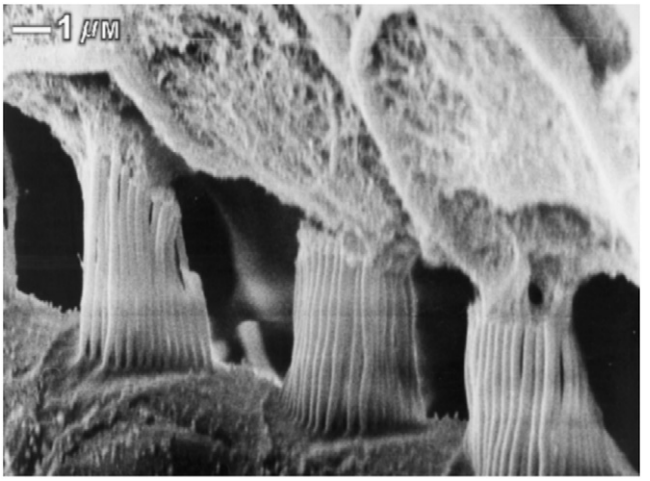


Fig. 9. (SEM) Rat cochlear stereocilia exposed to 4399 h of occupationally simulated ILFN. Cochlear stereocilia are fused amongst themselves and with the upper tectorial membrane.

5.3. Other considerations

Given the data obtained to date, the same type of analysis could be made of ILFN-exposed rat kidney glomeruli (Castelo Branco et al., 2003d; Martins dos Santos et al., 2005), or of rat tracheal epithelia (Castelo Branco et al., 2003a, f, g), or of VAD patients bronchoscopic biopsy results (Monteiro et al., 2004; Reis Ferreira et al., 2006), or of VAD patients vocal abnormalities (Mendes et al., 2005, 2006). The behaviour of respiratory tract cilliary populations is of particular interest in that they are composed of tubulin, they are anchored to the CSK actin cortex and, with ILFN exposure, they appear clipped, sheared and/or shaggy

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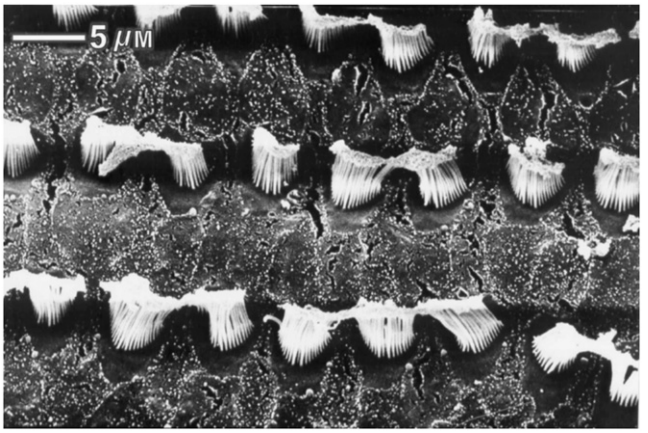


Fig. 10. (SEM) Rat cochlear stereocilia exposed to 4399 h of occupationally simulated ILFN. Cochlear stereocilia after removal of the tectorial membrane, portions of which remain fused to the stereocilia, forming bridges between adjacent cells.

(Alves-Pereira et al., 2003c). Pericardial cilia was also non-existent in VAD patients (Castelo Branco et al., 1999b). The suspicion that the tensegrity model of the cell could explain the findings in ILFN-induced biological structures was hinted at in 1999 (Alves-Pereira) and has since been the object of separate study and independent publications (Alves-Pereira and Castelo Branco, 2003a; Alves-Pereira et al., 2003b–d, 2004c).

Most medical diagnostic procedures are not based on mechanobiological features of disease. Hence, what is analysed, quantified or tested are usually parameters that depend on biochemical pathways. It cannot, therefore, be surprising that VAD present normal routine tests, such as blood chemistry analysis, EKG, and EEG, for example. With echo-imaging, where structural components can be observed, ILFN-induced pathology can be identified. With light and electron microscopy studies, morphofunctional changes can, again, be identified. However, conventional medical tests only become significantly altered in later, and irreversible, stages of VAD (see Table 3). With the tensegrity model of the cell, new avenues of research open up in the area of biochemically based tests from which biomechanical pathology can be extrapolated.

Pharmacological intervention in VAD is several years (and many Euros) away from becoming a reality. However, given that VAD is a mechanotranduction disease *par excellence* it now becomes clear where this intervention must focus: on the cellular signalling processes directly related to mechanotransduction.

6. Conclusions

ILFN is neglected as an agent of disease, and mechanotransduction is underestimated as an integral part of cellular signalling. Since VAD is caused by ILFN and explained through mechanotransduction pathways, it is not surprising that it is taking so long for the medical and scientific community to understand its existence. However, with knowledge comes responsibility, and the time has now come to take a more active position against needless suffering. The following recommendations are proposed.

6.1. Noise assessment

Hearing impairment is still a major issue within the EU and other countries, thus ceasing to perform measurements with dBA units is not a logical course of action. However, ILFN-rich environments need to be taken into account. Hence, it is proposed that *all* noise measurements be accompanied by a 1/3 octave band analysis, with no weighting (in dBLin), and down to the lowest limiting frequency permitted by the equipment at hand. If spectral analysis is not possible, then at least dBC and/or dBLin Leq measurements should be performed. In this way, not only is the acoustical environment documented for future legal and forensic purposes, but the adequate protection and prevention measures can be taken if it is known how much, and what kind, of ILFN is present in the environment.

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The same principle applies to biomedical studies, where ILFN is a possible contaminant. This is particularly true for animal studies where laboratories are kept in building basements, along with HVAC systems and elevator machinery. With human populations, it is important to obtain ILFN-exposure histories, since the effects of ILFN are cumulative (Castelo Branco et al., 1999d) and occur independent of whether the exposure was occupational, residential or leisurely. For example, an office worker may have no immediate exposure to noise on-the-job, but may live next to a bus terminal.

Control populations are especially targeted for comprehensive surveys of previous ILFN-exposure histories because, by definition, they *must not* have prior exposure to ILFN. foetal and leisurely exposures to ILFN must be included in the individual's prior history of ILFN exposure (Castelo Branco et al., 2003a, g; Araújo et al., 2004). The inadequate selection of control populations has already led to wasteful uses of resources and, of course, misleading results (ATSDR, 2001). Given the ubiquitous nature of ILFN, control populations with zero prior ILFN exposure are extremely difficult to gather. Thus, it could be feasible to undertake studies where the bio-effects of ILFN-exposed populations are compared, but only if their acoustical exposures are sufficiently well documented, in terms of exposure times and acoustical spectra.

6.2. Dosimetry

Adequate dosimetry of ILFN will be very difficult to achieve until science considers the acoustical spectrum as analogous to the electromagnetic spectrum, i.e., different frequencies have different effects on the different tissues. Thus, breaking the (lower) acoustical spectrum into infrasound versus audible frequencies is much too rudimentary.

It is proposed that the ILFN (0–500 Hz) portion of the acoustical spectrum be divided into the sub-categories listed in Table 4. Biological tissue is very sensitive to lower frequencies, below 100 Hz. Specific frequencies have been known to have a deleterious impact on specific biological tissue (e.g., Nekhoroshev and Glinchikov, 1991, 1992; Svigovyi and Glinchikov, 1987; Sidorenko et al., 1988), and a 2 Hz exposure can produce different effects than an 8 Hz exposure (Nekhoroshev and Glinchikov, 1991, 1992). Dividing the acoustical spectrum in sub-categories would eventually force bioscientists to specify the acoustical energy within each specific sub-category.

One of the most immediate problems with adequately measuring ILFN is the lack of readily available (and relatively inexpensive) instrumentation. As is well known, much of the monitoring equipment designed to assess physical agents is geared toward assessing the parameters that have been established by legislation. Thus, it is difficult (or much too expensive) to acquire the instrumentation that could adequately measure how long the acoustical energy remains at a certain dB level, for each 1/3 octave band. This would be an ideal parameter for assessing ILFN-induced pathology.

Table 4
Proposed subdivision of the lower portion (0–500 Hz) of acoustical spectrum, by 1/3 octave bands

Sub-category	1/3 octave bands (Hz)[a]	Observations
A	0–6.3	6.3 Hz is often the lower limiting frequency of standard noise measuring equipment software
B	8–12.5	Unusual behaviour in the frequencies of 8, 10 and 12.5 Hz has been detected, in residential, occupational and natural environments (unpublished results)
C	16–25	Overlapping the conventional threshold for human hearing (20 Hz)
D	31.5–63	Where many machines emit noise, includes the 50 Hz associated with high-voltage electrical distribution
E	63–160	Resonance of the thorax
F	200–500	Upper limit, with 250 Hz and 500 Hz already included in audiogram evaluations

[a]The 1/3 octave band analysis divides the acoustical spectrum into frequency bands, referred to by their central frequency. Thus, when measuring in 1/3 octave bands, values are obtained for the 1/3 octave frequency bands whose central frequency is 6.3, 8, 10, 12.5, 16, 20, 25, 31.5, 40, 50, 63, 80, 100, 125, 160, 200, 250, 315, 400 and 500 Hz (see Fig. 1). Hence, in this Table, the apparent discontinuities in the 1/3 Octave Bands column are due to the way in which science segments the acoustical spectrum.

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With animal models, some form of ILFN-exposure dosimetry has already been achieved. It is known that after a 48-h continuous exposure to ILFN, it is necessary 7 days for full recovery (Castelo Branco et al., 2003f). However, large-scale epidemiological studies are still lacking, mostly due to the difficulty of selecting adequate control populations, and funding.

6.3. Pharmacological intervention

With the integration of the tensigrity cellular model, VAD can now be viewed as a mechanotransduction disease *par excellence*. As such, new avenues of research have opened up regarding the possibility of pharmacological intervention. Since actin- and tubulin-based structures seem to be the most affected, it would make sense to focus on these biomechanical elements in order to avoid irreversible damage to individuals who *must* remain in ILFN environments for extended periods of time, such as ship and submarine workers, offshore oil and gas platforms workers, human activity onboard spacecraft, and the general population who is environmentally exposed to ILFN in the home.

6.4. Diagnosing VAD

For the purposes of an informal diagnosis, an echocardiogram to evaluate pericardial and cardiac valve thickening is essential to establish a VAD diagnosis because pericardial thickening with no diastolic dysfunction, and in the absence of an inflammatory process is a specific sign of VAD (Holt, 2000). However, given the limitations of echo-imaging procedures (discussed above) the echocardiogram is insufficient for legal and forensic purposes. Thus, if legal proof of VAD is required, then a more invasive procedure is necessary—the bronchoscopic examination (Reis Ferreira et al., 2006).

Other complementary diagnostic tests include brainstem auditory evoked potentials and cognitive evoked potentials (P300), brain magnetic resonance imaging, PCO_2 rebreathing test, blood coagulation factors and a thorough neurological examination.

Suspicion of VAD should arise if the patient exhibits one or more of the following complaints:

- "I hear too much, I'm very sensitive to noise, I can't stand any type of noise, Noise drives me crazy, Whenever there's a loud noise, all I feel like doing is screaming";
- "I wake up tired, It's not that I don't sleep enough hours, it just seems like I don't rest during my sleep";
- "Sometimes, while in a shopping mall or a restaurant, I feel like I can't breath, like I must get out of there or else";
- "I have a lot of heart palpitations, Sometimes it feels like my heart is going to leap out of my chest"; and
- "I have this cough, and I don't smoke, My throat is constantly irritated and I get hoarse for no reason, The over-the-counter medication doesn't do anything".

Or if the patient enters with one of the following diagnosis:

- Late-onset epilepsy;
- Balance disorders;
- Migraine;
- Respiratory tract tumour, especially if a non-smoker;
- Recommendation for cardiac bypass surgery; and
- Auto-immune disease, particularly systemic lupus erythematous and vitilligo.

The authors urge physicians to listen to their patients and question them about their noise exposures.

References

Águas, A.P., Esaguy, N., Castro, A.P., Grande, N.R., Castelo Branco, N.A.A., 1999a. Acceleration of lupus erythematosus-like processes by low frequency noise in the hybrid NZB/W mouse model. Aviat. Space Environ. Med. 70 (March, Suppl.), A132–A136.

009891

Águas, A.P., Esaguy, N., Castro, A.P., Grande, N.R., Castelo Branco, N.A.A., 1999b. Effect low frequency noise exposure on BALB/C mice splenic lymphocytes. Aviat. Space Environ. Med. 70 (March, Suppl.), A128–A131.

Albuquerque e Sousa, J., Dinis da Gama, A., Macedo, M.V., Cassio, I., Castelo Branco, N.A.A., 1991. Carotid angiodynographic studies in individuals occupationally exposed to noise and vibration. Aviat. Space Environ. Med. 62, 134 (Abstract).

Alenghat, F.J., Nauli, S.M., Kolb, R., Zhou, J., Ingber, D.E., 2004. Global cytoskeletal control of mechanotransduction in kidney epithelial cells. Exp. Cell. Res. 301, 23–30.

Alvarez, M., Taborda, F., Gomes, L., Marçal, C., Soutenho, A., Marmelo, J., Castelo Branco, N.A.A., 1993. Epidemiology of on-the-job accidents at OGMA. Rev. Port. Med. Mil. 4, 35–40 (in Portuguese).

Alves-Pereira, M., 1999. Extra-aural noise-induced pathology. A review and commentary. Aviat. Space Environ. Med. 70 (March, Suppl.), A7–A21.

Alves-Pereira, M., Castelo Branco, M.S.N., Motylewski, J., Pedrosa, A., Castelo Branco, N.A.A., 2001a. Airflow-induced infrasound in commercial aircraft. In: Proceedings of the Internoise 2001, The Hague, Holland, pp. 1011–1014.

Alves-Pereira, M., Castelo Branco, N.A.A., 2003a. Ciliated cells, cochlear cilia and low frequency noise. In: Proceedings of the Eighth International Congress of Noise as a Public Health Problem, Rotterdam, Holland, pp. 366–367.

Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A., 2003b. Pericardial biomechanical adaptation to low frequency noise stress. In: Proceedings of the First International Meeting on Applied Physics, Badajoz, Spain, no. 354, 6p.

Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A., 2003c. Actin and tublulin-based structures under low frequency noise stress. Proceedings First International Meeting on Applied Physics, Badajoz, Spain, no. 355, 5p.

Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A., 2003d. Low frequency noise exposure and biological tissue, reinforcement of structural integrity? In: Proceedings of the First International Meeting on Applied Physics, Badajoz, Spain, no. 356, 6p.

Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A., 2004a. Low frequency noise in trains. In: Proceedings of Internoise 2001, Prague, Czech Republic, no. 643, 5p.

Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A., 2004b. Public transportation and low frequency noise, a health hazard? In: Proceedings of the 11th International Congress on Sound and Vibration, St. Petersburg, Russia, pp. 1761–1766.

Alves-Pereira, M., Joanaz de Melo, J., Marques, M.C., Castelo Branco, N.A.A., 2004c. Vibroacoustic disease—the response of biological tissue to low frequency noise. In: Proceedings of the 11th International Meeting on Low Frequency Noise, Vibration and its Control. Maastricht, The Netherlands, 30 August–1 September, 2004, pp. 295–308.

Alves-Pereira, M., 2005. … And again low frequency noise—a possible solution. In: Proceedings of the 12th International Congress on Sound and Vibration, Lisbon, Portugal, no. 584, 8p.

Araújo, A., Ribeiro, C.S., Correia, M.J.F., Pais, F., Castelo Branco, N.A.A., 1989. Echocardiographic appearances in patients with the whole-body noise and vibration disease. MEDICEF-Direct Inform. (France) 2, 101–102.

Araújo, A., Pais, F., Lopo Tuna, J.M.C., Alves-Pereira, M., Castelo Branco, N.A.A., 2001. Echocardiography in noise-exposed flight crew. In: Proceedings of Internoise 2001, The Hague, Holland, pp. 1007–1010.

Araújo, A., Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A., 2004. Environmentally-induced vibroacoustic disease in a suburban family. In: Proceedings of the 11th International Congress on Sound and Vibration, St. Petersburg, Russia, pp. 1767–1774.

Arnot, J.W., 2003. Vibroacoustic disease I, the personal experience of a motorman. Inst. Acoustics (UK) 25 (Pt 2), 66–71.

ATSDR—Agency for Toxic Substance and Disease Registry (USA), 2001. Expert review of the Vieques heart study. Summary report for the Vieques heart study expert panel review, Contract no. 200-2000-10039. 〈www.atsdr.cdc.gov/NEWS/viequesheartreport.html〉

Bento Coelho, J.L., Ferreira, A., Serrano, J., Taborda, F., Castelo Branco, N.A.A., 1994. Noise exposure in the aeronautical industry. In: Proceedings of Internoise 1994, Yokohama, Japan, pp. 741–745.

Calverly, P.M.A., 1999. Control of breathing. In: Hughes, J.M.B., Pride, N.B. (Eds.), Lung Function Tests. Physiological Priniciples and Clinical Applications. WB Saunders, London.

Campanella, A., 2001. What you can't hear won't hurt you. The Kokomo Perspective (Indiana, USA), 17 October, pp. 1–3.

Carmo, G., Albuquerque e Sousa, J., Dinis da Gama, A., Castelo Branco, N.A.A., 1992. Carotid angiodynographic studies in helicopter pilots. Aviat. Space Environ. Med. 63, 385 (Abstract).

Canas, J., Martinho Pimenta, A.J.F., Castelo Branco, N.A.A., 1993. ERP P300 and MRI studies of the CNS in military pilots, a comparative study of early degenerative brain processes. Aviat. Space Environ. Med. 64, 451 (Abstract).

Castelo Branco, M.S.N., Castelo Branco, N.A.A., Entrudo, A., Marvão, J., 1985. A standardization method of the brainstem auditory evoked potentials. J. Soc. Ciências Med. Lisboa. 149, 214–220 (in Portuguese) *Recipient of the National Ricardo Jorge Public Health Award. 1985.

Castelo Branco, N.A.A., 1992. The whole-body noise and vibration syndrome. Rev. Port. Med. Mil. 40, 16.

Castelo Branco, N.A.A., 1999a. The clinical stages of vibroacoustic disease. Aviat. Space Environ. Med. 70 (Suppl. 3), A32–A39.

Castelo Branco, N.A.A., 1999b. A unique case of vibroacoustic disease. A tribute to an extraordinary patient. Aviat. Space Environ. Med. 70 (Suppl. 3), A27–A31.

Castelo Branco, N.A.A., Martinho Pimenta, A.J.F., 1995. The basic clinical and pathophysiological aspects of the vibroacoustic syndrome. Aviat. Space Environ. Med. 66, 504 (Abstract).

Castelo Branco, N.A.A., Águas, A.P., Sousa Pereira, A., Monteiro, E., Fragata, J.I.G., Grande, N.R., 1996. The pericardium in the vibroacoustic syndrome. Cardiovas. Diag. Prog. 13 (4), 284 (Abstract).

Castelo Branco, N.A.A., Rodriguez Lopez, E., 1999a. The vibroacoustic disease—an emerging pathology. Aviat. Space Environ. Med. 70 (March, Suppl.), A1–A6.

Castelo Branco, N.A.A., Águas, A.P., Sousa Pereira, A., Monteiro, E., Fragata, J.I.G., Tavares, F., Grande, N.R., 1999b. The human pericardium in vibroacoustic disease. Aviat. Space Environ. Med. 70 (Suppl. 3), A54–A62.

009892

Castelo Branco, N.A.A., Alves-Pereira, M. (Coords.), 1999c. Vibroacoustic disease. Aviat. Space Environ. Med. 70 (March, Suppl.), A1–A154.

Castelo Branco, N.A.A., Rodriguez Lopez, E., Alves-Pereira, M., Jones, D.R., 1999d. Vibroacoustic disease, some forensic aspects. Aviat. Space Environ. Med. 70 (Suppl. 3), A145–A151.

Castelo Branco, N.A.A., Alves-Pereira, M., Martins dos Santos, J., Monteiro, E., 2003a. SEM and TEM study of rat respiratory epithelia exposed to low frequency noise. In: Mendez-Vilas, A. (Ed.), Science and Technology Education in Microscopy. An Overview, vol. II. Formatex, Badajoz, Spain, pp. 505–533.

Castelo Branco, N.A.A., Martinho Pimenta, A.J., Reis Ferreira, J., Alves-Pereira, M., 2003b. Monitoring vibroacoustic disease. In: Proceedings of the Scuola Superiore. G. Reiss Romoli-Telecom Italia (SSGRRw), L'Aquila, Italy, no. 102, 5p. 〈http://www.ssgrr.it/en/ssgrr2003w/papers/129.pdf〉

Castelo Branco, N.A.A., Fragata, J.I., Martins, A.P., Monteiro, E., Alves-Pereira, M., 2003c. Pericardial cellular death in vibroacoustic disease. In: Proceedings of the Eighth International Congress of Noise as a Public Health Problem, Rotterdam, Holland, pp. 376–377.

Castelo Branco, N.A.A., Monteiro, E., Martins dos Santos, J., Alves-Pereira, M., 2003d. Low frequency noise and intra-cellular edema. In: Proceedings of the Eighth International Congress of Noise as a Public Health Problem, Rotterdam, Holland, pp. 378–379.

Castelo Branco, N.A.A., Monteiro, E., Costa e Silva, A., Reis Ferreira, J., Alves-Pereira, M., 2003e. Respiratory epithelia in Wistar rats. Rev. Port. Pneumol. IX (5), 381–388.

Castelo Branco, N.A.A., Gomes-Ferreira, P., Monteiro, E., Costa e Silva, A., Reis Ferreira, J., Alves-Pereira, M., 2003f. Respiratory epithelia in Wistar rats after 48 h of continuous exposure to low frequency noise. Rev. Port. Pneumol. IX (6), 473–479.

Castelo Branco, N.A.A., Monteiro, E., Costa e Silva, A., Reis Ferreira, J., Alves-Pereira, M., 2003g. Respiratory epithelia in Wistar rats born in low frequency noise plus varying amounts of additional exposure. Rev. Port. Pneumol. IX (6), 481–492.

Castelo Branco, N.A.A., Alves-Pereira, M., 2004a. Vibroacoustic disease. Noise and Health 6 (23), 3–20.

Castelo Branco, N.A.A., Fragata, J.I., Monteiro, E., Alves-Pereira, M., 2004b. Pericardial cellular death in vibroacoustic disease patients. In: Proceedings of the 11th International Congress on Sound and Vibration, St. Petersburg, Russia, pp. 1753–1760.

Castelo Branco, N.A.A., Fragata, J.I., Martins, A.P., Monteiro, E., Alves-Pereira, M., 2005. The pericardium in vibroacoustic disease I—morphological features. In: Proceedings of the 12th International Congress on Sound and Vibration, Lisbon, Portugal, 11–14 July 2005, no. 568, 9p.

Castro, A.P., Aguas, A.P., Grande, N.R., Monteiro, E., Castelo Branco, N.A.A., 1999. Increase in CD8+ and CD4+ T lymphocytes in patients with vibroacoustic disease. Aviat. Space Environ. Med. 70 (Suppl. 3), A141–A144.

Cortez-Pimentel, J.A.B., Castelo Branco, N.A.A., 1988. Systemic vibration disease VII, the dento-alveolar structure. Rev. Port. Med. Mil. 36 (1), 123–127 (in Portuguese, Abstract in English).

Cotes, J.E., 1993. Lung Function. Assessment and Applications in Medicine. Blackwell Scientific Publishing, London, p. 376.

Crespo, F., Ferreira, A.C.N., Castelo Branco, M.S.N., Castelo Branco, N.A.A., 1988. Systemic vibration disease III, coagulation and hemostasis study in patients with this disease. Rev. Port. Med. Mil. 36, 86–89 (in Portuguese, Abstract in English).

Cruz Maurício, J., Martinho Pimenta, A.J.F., Castelo Branco, N.A.A., 1988. Systemic vibration disease IV, central nervous system imaging through magnetic resonance. Rev. Port. Med. Mil. 36, 90–96 (in Portuguese, Abstract in English.).

European Communities, 2000. The Noise Policy of the European Union—Year 2. Luxembourg. 〈http://europa.eu.int/comm/environment/noise/pdf/noisebrochure.pdf〉

Fonseca, J., Mirones, J., Martins dos Santos, J., Alves-Pereira, M., Castelo Branco, N.A.A., 2005. Gastrointestinal problems in vibroacoustic disease. Proceedings 12th International Congress on Sound and Vibration, Lisbon, Portugal, 2005, no. 577, 8p.

Fuller, R.B., 1975. Synergetics. Macmillian, New York.

Gibson, G.J., 1996. Clinical tests of the respiratory function. Chapman & Hall Medicine, London, pp. 101–109.

GIMOGMA, 1984a. Epilepsy of vascular aetiology, a clinical picture of vibration disease? Rev. Port. Med. Mil. 32, 5–9 (in Portuguese).

GIMOGMA, 1984b. Evoked potential study in a population exposed to occupational vibration. Rev. Port. Med. Mil. 32, 10–16 (in Portuguese).

GIMOGMA, 1984c. Vibration and noise as the cause of acoustic hypo- and hyper-sensibility in an industrial population. Rev. Port. Med. Mil. 32, 17–20 (in Portuguese).

Grande, N., Águas, A.P., Sousa Pereira, A., Monteiro, E., Castelo Branco, N.A.A., 1999. Morphological changes in the rat lung parenchyma exposed to low frequency noise. Aviat. Space Environ. Med. 70 (Suppl. 3), A70–A77.

Holt, B.D., 2000. The pericardium. In: Furster, V., Wayne Alexander, R., Alexander (Eds.), Hurst's The Heart. McGraw-Hill Professional Publishing, New York, pp. 2061–2082.

Ingber, D.E., 2003. Mechanobiology and diseases of mechanotransduction. Ann. Med. 35, 1–14 〈http://web1.tch.harvard.edu/research/ingber/homepage.htm〉.

Ingber, D.E., 2004a. Mechanochemical basis of cell and tissue regulation. N.A.E. Bridge 34 (3), 4–10 〈http://www.nae.edu/NAE/bridgecom.nsf/weblinks/MKEZ65RHQL?OpenDocument〉.

Ingber, D.E., 2004b. In: Fliesler, N. The mechanical cell. Children's Dream Magazine. 〈http://www.childrenshospital.org/research/Site2029/mainpageS2029P23sublevel24.html〉

Jones, R.N., Turner-Warwick, M., Ziskind, M., Weill, H., 1976. High prevalence of antinuclear antibodies in sandblasters' silicosis. Am. Rev. Resp. Dis. 113, 393–395.

Lippmann, M., Eckert, H.L., Hahon, N., Morgan, W.K.C., 1973. Circulating antinuclear and rheumatoid factors in coal miners. A prevalence study in Pennsylvania and West Virginia. Ann. Int. Med. 79, 807–811.

009893

Marciniak, W., Rodriguez, E., Olsowska, K., Botvin, I., Araújo, A., Pais, F., Soares Ribeiro, C., Bordalo, A., Loureiro, J., Prazeres de Sá, E., Ferreira, D., Castelo Branco, M.S.N., Castelo Branco, N.A.A., 1999. Echocardiography in 485 aeronautical workers exposed to different noise environments. Aviat. Space Environ. Med. 70 (Suppl. 3), A46–A53.

Martinho Pimenta, A.J.F., Castelo Branco, M.S.N., Castelo Branco, N.A.A., 1999a. The palmo-mental reflex in vibroacoustic disease. Aviat. Space Environ. Med. 70 (Suppl. 3), A100–A106.

Martinho Pimenta, A.J.F., Castelo Branco, M.S.N., Castelo Branco, N.A.A., 1999b. Balance disturbances in individuals with vibroacoustic disease. Aviat. Space Environ. Med. 70 (Suppl. 3), A96–A99.

Martinho Pimenta, A.J.F., Castelo Branco, N.A.A., 1999c. Facial dyskinesia induced by auditory stimulation. A report of four cases. Aviat. Space Environ. Med. 70 (Suppl. 3), A119–A121.

Martinho Pimenta, A.J.F., Castelo Branco, N.A.A., 1999d. Epilepsy in vibroacoustic disease—a case report. Aviat. Space Environ. Med. 70 (Suppl. 3), A122–A127.

Martinho Pimenta, A.J.F., Castelo Branco, N.A.A., 1999e. Facial dyskinesia induced by auditory stimulation. A report of four cases. Aviat. Space Environ. Med. 70 (Suppl. 3), A119–A121.

Martinho Pimenta, A.J.F., Castelo Branco, M.S.N., Castelo Branco, N.A.A., 1999f. The palmo-mental reflex in vibroacoustic disease. Aviat. Space Environ. Med. 70 (Suppl. 3), A100–A106.

Martins dos Santos, J., Grande, N.R., Castelo Branco, N.A.A., Zagalo, C., Oliveira, P., 2002. Vascular lesions and vibroacosutic disease. Eur. J. Anat. 6 (1), 17–21.

Martins dos Santos, J., Grande, N.R., Castelo Branco, N.A.A., Zagalo, C., Oliveira, P., Alves-Pereira, M., 2004. Lymphatic lesions in vibroacoustic disease. Eur. J. Lymphol. 12 (40), 13–16.

Martins dos Santos, J., Albuquerque e Sousa, A., Marques, M.C., Monteiro, E., Alves-Pereira, M., Castelo Branco, N.A.A., 2005. Urinary system in vibroacoustic disease—current findings and ongoing studies. In: Proceedings of the 12th International Congress on Sound and Vibration, Lisbon, Portugal, No. 578, 8p.

Marvão, J.H., Castelo-Branco, M.S.N., Entrudo, A., Castelo Branco, N.A.A., 1985. Changes of the brainstem auditory evoked potentials induced by occupational vibration. J. Soc. Ciências Med. 149, 478–486 (in Portuguese).

Matsumoto, Y., Kawabe, M., Yasue, T., Yuguchi, M., Yoshida, I., 1989. Two cases of scleroderma associated with vibration syndrome. Jpn. J. Dermatol. 99, 155–161.

Matsumoto, Y., Yasue, T., Mizuno, N., Yoshida, I., 1992. An immunoserological study of patients with vibration syndrome. Occupat. Environ. Health 63, 357–359.

Matthews, B.D., Overby, D.R., Alenghat, F.J., Karavitis, J., Numaguchi, Y., Allen, P.G., Ingber, D.E., 2004. Mechanical properties of individual focal adhesions probed with a magnetic microneedle. Biochem. Biophys. Res. Commun. 313, 758–764.

Mendes, A., Alves-Pereira, M., Castelo Branco, N.A.A., Silva, J., Pires, F., 2005. Vibroacoustic disease and voice acoustics analysis. In: Proceedings 12th International Congress on Sound and Vibration, Lisbon, Portugal, no. 62, 9 p.

Mendes, C.P., Reis Ferreira, J., Alves-Pereira, M., Castelo Branco, N.A.A., 2004. Respiratory pathology in vibroacoustic disease I—tumours. In: Proceedings Noise and Transport 2004, St. Petersburg, Russia, no. s3–s4, 5p.

Mendes, A., Alves-Pereira, M., Castelo Branco, N.A.A., Silva, J., Pires, F., 2006. Voice acoustic patterns of patients diagnosed with vibroacoustic disease. Rev. Port. Pneumol., in press.

Moniz Botelho, J., Martinho Pimenta, A.J.F., Castelo Branco, N.A.A., 1988. Systemic vibration disease V, P300 and brain mapping. Rev. Port. Med. Mil. 36, 97–115 (in Portuguese, Abstract in English.).

Monteiro, M.B., Reis Ferreira, J., Mendes, C.P., Alves-Pereira, M., Castelo Branco, N.A.A., 2004. Vibroacoustic disease and respiratory pathology III—tracheal and bronchial lesions. In: Proceedings of the Internoise 2004, Prague, Cazech Republic, 22–25 August, no. 638, 5p.

Nekhoroshev, A.S., Glinchikov, V.V., 1991. Effect of infrasound on morphofunctional changes in myocardium exposed to infrasound. Gig. Sanit. 12, 56–58 (in Russian, Abstract in English.).

Nekhoroshev, A.S., Glinchikov, V.V., 1992. Morphological research on the liver structures of experimental animals under the action of infrasound. Aviakosm. Ekolog. Med. 26, 56–59 (in Russian, Abstract in English.).

Oliveira, M.J.R., Sousa Pereira, A., Águas, A.P., Monteiro, E., Grande, N.R., Castelo Branco, N.A.A., 1999. Effects of low frequency noise upon the reaction of pleural milky spots to mycobacterial infection. Aviat. Space Environ. Med. 70 (March, Suppl.), A137–A140.

Persson-Waye, K., Rylander, R., 2001. The prevalence of annoyance and effects after long-term exposure to low frequency noise. J. Sound Vib. 240 (3), 483–497.

Pimenta, A.J.F., Castelo-Branco, N.A.A., Manaças, J.E., Brito, J.L.R., Martins, A.S., 1988. Systemic vibration disease II, clinical elements. Rev. Port. Med. Mil. 36 (2/3), 72–85 (In Portuguese, Abstract in English).

Reis Ferreira, J., Mendes, C.P., Antunes, M., Martinho Pimenta, A.J.F., Monteiro, E., Alves-Pereira, M., Castelo Branco, N.A.A., 2003a. Diagnosis of vibroacoustic disease—preliminary report. In: Proceedings of the Eighth International Congress of Noise as a Public Health Problem, Rotterdam, Holland, pp. 112–114.

Reis Ferreira, J., Mendes, C.P., Castelo Branco, N.A.A., Monteiro, E., Alves-Pereira, M., 2003b. The human trachea in vibroacoustic disease. In: Proceedings of the Eighth International Congress of Noise as a Public Health Problem, Rotterdam, Holland, pp. 388–389.

Reis Ferreira, J., Mendes, C.P., Castelo Branco, N.A.A., Monteiro, E., Alves-Pereira, M., 2003c. The human lung and pleura in vibroacoustic disease. In: Proceedings of the Eighth International Congress of Noise as a Public Health Problem, Rotterdam, Holland, pp. 386–387.

Reis Ferreira, J., Mendes, C.P., Alves-Pereira, M., Castelo Branco, N.A.A., 2005. Respiratory pathology in vibroacoustic disease I—current findings. In: Proceedings of the 12th International Congress on Sound and Vibration, Lisbon, Portugal, no. 571, 7p.

009894

Reis Ferreira, J., Monteiro, M., Tavares, F., Serrano, I., Monteiro, E., Mendes, C.P., Alves-Pereira, M., Castelo Branco, N.A.A., 2006. Involvement of central airways in vibroacoustic disease patients. Rev. Port. Pneumol. XII (2), 93-105. *Recipeint of the 2004 Thomé Villar/Boehringer Ingelheim Award. Available: www.scielo.oces.mctes.pt/pdf/pne/v12n2/v12n2a01.pdf

Rosado, P., Martinho Pimenta, A.J.F., Castelo Branco, N.A.A., 1993. The vibroacoustic syndrome IV. Facial dyskinesia induced by auditory stimulation. Aviat. Space Environ. Med. 64, 451 (in Abstract).

Sandars, N.K., 1972. The Epic of Gilgamesh. Peguin Books, London.

Shabetai, R., 1994. Diseases of the pericardium. In: Schlant, R.C., Wayne Alexander, R. (Eds.), Hurst's The Heart, Arteries and Veins. McGraw-Hill, New York, pp. 1547–1649.

Sidorenko, I.I., Zelikman, M.K., Kaplina, A.V., 1988. Changes in eye tissues exposed to local action of infrasound. Oftalmol. Zh. 2, 109–111 (in Russian).

Silva, M.J., Carothers, A., Castelo Branco, N.A.A., Dias, A., Boavida, M.G., 1999. Sister chromatid exchanges workers exposed to noise and vibration. Aviat. Space Environ. Med. 70 (Suppl. 3), A40–A45.

Silva, M.J., Carothers, A., Castelo Branco, N.A.A., Dias, A., Boavida, M.G., 2002a. Increased levels of sister chromatid exchanges in military aircraft pilots. Mut. Res. Gene. Toxicol. Environ. Mutagen. 44 (1), 129–134.

Silva, M.J., Dias, A., Barreta, A., Nogueira, P.J., Carothers, A., Castelo Branco, N.A.A., Boavida, M.G., 2002b. Low frequency noise and whole-body vibration cause increased levels of sister chromatid exchange in splenocytes of exposed mice. Teratogen. Carcinogen. Mutagen. 22 (3), 195–203.

Sousa Pereira, A., Grande, N.R., Castelo Branco, N.A.A., 1999a. The effect of low frequency noise on rat tracheal epithelium. Aviat. Space Environ. Med. 70 (Suppl. 3), A86–A90.

Sousa Pereira, A., Grande, N.R., Castelo Branco, M.S.N., Castelo Branco, N.A.A., 1999b. Morphofunctional study of rat pleural mesothelial cells exposed to low frequency noise. Aviat. Space Environ. Med. 70 (Suppl. 3), A78–A85.

Soutar, C.A., Turner-Warwick, M., Parkes, W.R., 1974. Circulating antinuclear antibody and rheumatoid factor in coal penumoconosis. Brit. Med. J. 3, 145–147.

Svigovyi, V.I., Glinchikov, V.V., 1987. The effect of infrasound on lung structure. Gig. Truda. Prof. Zabol. 1, 34–37 (in Russian, Abstract in English.).

Torres, R., Tirado, G., Roman, A., Ramirez, R., Colon, H., Araújo, A., Pais, F., Marciniak, W., Nóbrega, J., Bordalo e Sá, A., Lopo Tuna, J.M.C., Castelo Branco, M.S.N., Alves-Pereira, M., Castelo Branco, N.A.A., 2001. Vibroacoustic disease induced by long-term exposure to sonic booms. In: Proceedings of the Internoise 2001, The Hague, Holland, pp. 1095–1098.

Van Zeller, P., Oliveira, F.C., Tavares, C., Freitas, A.M., Castelo Branco, N.A.A., 1988. Systemic vibration disease VI, retinal angiography. Rev. Port. Med. Mil. 36 (2/3), 117–125 (in Portuguese, Abstract in English).

von Gierke, H., Mohler, S., 2002. Letter to the editor, vibroacoustic disease. Aviat. Space Environ. Med. 73 (8), 828–830.

Wang, N., Butler, J.P., Ingber, D.E., 1993. Mechanotransduction across the cell surface and through the cytoskeleton. Science (Reports), New Ser. 260 (5111), 1124–1127.

Ward, W.D., 1973. Noise-induced hearing damage. In: Paparella, M.M., Shumrick, D.A. (Eds.), Otolaryngology. W.B. Saunders Co., Philadelphia, PA.

World Health Organization, 1999. Guidelines for Community Noise. Berglund, B., Lindvall, T., Schwela, D.H. (Eds.). 〈http://www.who.int/docstore/peh/noise/guidelines2.html〉

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VIBROACOUSTIC DISEASE I: THE PERSONAL EXPERIENCE OF A MOTORMAN

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1 INTRODUCTION

The aim of this paper is to provide a first-hand account of the development of vibroacoustic disease (VAD) in a motorman. I will describe the physical changes and the various costs incurred during the last five years of my professional activity, as I gradually became ill with VAD. My story is not an isolated case. In fact, VAD may be the cause of long term limiting illnesses in a large number of people who live and work in environments rich in low frequency noise (LFN) (≤ 500 Hz, including infrasound) [1].

Research indicates that the number of affected workers may be significant. Based on my personal experience, I am convinced that if protective solutions were to be implemented, an appreciable improvement would be seen in the number of workers who are off work due to sickness and incapacity. By revealing the cost of illness, I hope to emphasize the need for those exposed to LFN-rich environments to be informed of the risks involved and to be given a list of early symptoms. This would allow them to make their own choices and seek help before permanent and irreversible damage sets in.

VAD is caused by an all-pervading and insidious agent of harm - LFN. Unless the patient is aware of the early symptoms, VAD is difficult to recognize until it is too late; especially since it has not yet reached the mainstream general practitioners, nor the occupational medicine professionals. The sheer number of workers that are possibly being affected by LFN exposure will stress the importance of developing and applying solutions against this agent of disease.

2 CASE HISTORY

2.1 Employment

At the age of 17, I left school and became apprenticed to an engineering company. Eventually, after thirteen years of working as a land-based mechanic, I was hired as a motorman for a roll on roll off ferry operator sailing to the Hebridean Islands off the West Coast of Scotland. My job description consisted of performing maintenance on running medium speed diesel engines, and involved working in ship's engine rooms and machinery spaces. The usual onboard schedule was twelve hours a day, seven days per week, for periods of three weeks followed by two weeks at home in rotation.

From my Department of Transport, Seamen's Discharge Book, no. UK 092704, I worked onboard seven different vessels for nearly ten years, and was exposed to a total of 38,640 hours of LFN-rich environments. This amount of hours would be achieved in approximately 15 to 18 years of normal shore-side employment. However, crew members live onboard ships and are therefore continuously exposed to LFN-rich environments, even during their break and sleep periods.

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2.2 Personal Information

On January 15th, 2001, I was a 39 year old motorman, married and with two young children, and who had just been signed off sick from work. I have never smoked, never taken recreational drugs and I rarely drink alcohol and never to excess. I have no history of cardiovascular disease, hypertension, nor diabetes. My hobbies are local history, motor boating, swimming and do-it-yourself home improvements. I also used to enjoy cycling and hill walking. My residential environments have always been in rural parts of Britain, far from any sources of LFN.

My medical history was unremarkable. I had worked since leaving school without break, and without significant periods of sick leave, apart from a case of acute tonsillitis during which I was signed off work for a month.

2.3 From Early Warning Signs to Disabling Pathology

Within the first few years I noticed that the hearing in my right ear was much worse than in my left. For example, when I was in the engine room I could lift the ear protector off my right ear and hear virtually nothing from the roar of the main engines; if I lifted the left ear protector the noise was actually painful. The engine room was very noisy and our main concern at the time was for the pain that the main engine vibration was causing to our feet and knees. After three years the knee pain became permanent.

After approximately four years of professional activity in ships engine rooms, I began to experience what I now know to be the early stages of VAD. I began to feel what I imagine to be 'hangover' symptoms. I considered this to be particularly unfair since these symptoms appeared without my having incurred in any of the behaviors that normally cause hangovers. When off the ship, I experienced increased bowel urgency and frequency. Also at around the same time, after working very closely with the main engines for a whole three week period, I experienced extra heart beats, or flutters (tachycardia). I was sent to a Cardiologist for investigations by which time my complaint had resolved itself and no further investigations were made.

I began to have panic attacks while sleeping, and awoke abruptly, jumping up to catch my breath. I also experienced broken sleep due to burning and pins & needles sensations in my hands, later diagnosed as bilateral Carpal Tunnel Syndrome, and associated with holding vibrating equipment. Fatigue was slowly beginning to settle in. Bouts of depression began appearing, and with time became more frequent. My family noticed changes in my mood and complained that I was becoming less and less sociable. I developed a short-tempered disposition and felt more irritable.

There was an increasing pain and burning sensations in both of my knees. I lost muscle mass from around my knees. My legs became weaker and I began to have to climb stairs on all fours. At times my vision would blur, both at home and onboard ship. I noticed that I could not tolerate bright light. I used to experience a dry, tickly cough while on board the ship, and I found I was less able to fight off minor infections. All of us found that we had small nose bleeds and congested nasal passages, along with a dry irritating cough, but only while onboard ship. We thought it was caused by air dryness. Coughing often started roughly half an hour after being in the engine room. We all found this a problem and lived on cough medicine and vitamin tablets. Often the only way to stop coughing would be by thumping myself on the chest as hard as could, like an ape beating it's chest.

Areas of numbness and over-sensitivity appeared in different places on my skin. I noticed muscle twitches and spasms in my spine, and I hurt my back more easily. I was unable to control my stomach muscles as they would tighten so much by themselves that I had difficulty in breathing. When I came home from sea I couldn't bear my children running up to hug me as my stomach muscles were so painful, even before the appearance of an umbilical hernia. I developed an increased need to urinate and always felt that I had not completely emptied my bladder. Impotence developed. Noise and crowds became intolerable. Depression set in more permanently.

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After about 7 years of occupational exposure, I began to notice a perverse behaviour: I would catch myself making the wrong decision, such as opening the wrong valve or switching the wrong switch. It wasn't that I would forget to do the right thing, but I actually became convinced that I had done the correct thing. I had already witnessed this behaviour among many of the older crew-members with whom I had worked: they would draw attention to themselves by making the wrong choice, i.e., deliberately setting off fire alarms and then claiming they didn't remember, or denying they had caused the entire event.

Muscle twitches, spasms and muscle tightening became more severe and lasted for longer periods of time, often spanning whole weeks going from attack to attack. Usually, these symptoms subsided substantially during my shore leave, and two weeks at home allowed me to recovery sufficiently to return to another three weeks at sea.

In January 2001, I signed off sick with severe back pain and muscle spasms, and I have never returned to work since then. After seeing the Ferry Company doctor I was signed off unfit for duty at sea (ENG 3) with suspected spinal nerve damage. This medical condition was later excluded through the magnetic resonance imaging (MRI) tests.

Later in January 2001, I had an x-ray of the lower back showing Spondylolisthesis at L5. In February 2001, I had a private consultation with an Orthopaedic Consultant, and since nerve entrapment was suspected, a private MRI scan of my lower spine was taken in March 2001. In May 2001 I was given an ultra-sound scan of bladder at Oban hospital, showing that I was retaining urine. In August 2001 I received uro-dynamic tests at the Southern General Urology Department, which suggested that I might suffer from detrusor muscle instability, thus causing poor bladder control. Detrusitol was prescribed with limited success.

In December 2001, I underwent surgery for an umbilical hernia. It was not until after this surgery that all my health problems got worse. I suffered from increasingly broken sleep due to poor breathing and burning fingers related to carpal tunnel syndrome. My headaches became more and more severe. I noticed that I had real difficulty in concentrating for periods during the day. I suffered from daily fatigue attacks lasting three to four hours.

In October 2001, I had a consultation a Professor of Neurology, Dr. Ian Bone, from the Southern General Hospital, in Glasgow. After 16 months of tests, during which many medical conditions were excluded, such as spinal nerve damage and copper poisoning, Dr. Bone suggested that I might be suffering from VAD, as identified by Dr Nuno Castelo Branco and published on the British Medical Association's (BMA) Medline database[2]. Professor Bone arranged for a cranial MRI scan to search for possible brain lesions as seen in VAD.

2.4 The Value of a Formal Diagnosis

Desperate to find treatment for my debilitating symptoms, I found a large amount of information about VAD on the BMA Medline database. I posted a question about VAD on an Internet discussion forum and by good fortune I received an answer from one of the Portuguese experts who have been studying the effects of long-term exposure to LFN for more than twenty years, and who identified VAD. I was put in contact with the lead scientist of the VAD project, Dr. Nuno Castelo Branco.

My initial idea was to inquire about possible therapies that might be available. However, over email and without a full examination, any suggestions would be out of order. It was suggested that if I could travel to Portugal, I could be adequately examined and further advised. I traveled to Portugal and was given a wide range of physical examinations to determine whether or not I was suffering from VAD. The hallmark of VAD is pericardial thickening in the absence of an inflammatory process and with no diastolic dysfunction[3]. Echocardiography demonstrated that I had this condition. Other

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VAD tests were also positive, and the team led by Dr. Nuno Castelo Branco formally diagnosed me with VAD.

I found there was great emotional value in simply meeting and talking with medical professionals who specialized in my illness and who understood what I was experiencing. I particularly found that once given a diagnosis demonstrating the cause of my symptoms, I could reassure myself that I was not going crazy. I had often been suspected of malingering, and was informed that this was not a unique situation, and many patients had been thus accused[1]. The social implications of being considered a malingerer, even on behalf of family members employed within the health industry, were quite demoralizing.

Without a diagnosis I was left in a no-man's-land where none of the medical specialists could suggest treatment. I felt I was left to see whether my symptoms developed further into an accepted illness, or if they would just resolve themselves with the passage of time. I suggest that this is a harmful practice in itself, since the individuals who are in need of help are powerless to help themselves, and the very people who should be able to help do nothing. I also believe that leaving people to wait on incapacity benefit without a diagnosis of any sort is a dreadful waste of skills and experience.

It would be a much more efficient way to deal with workers who are incapacitated from their normal duties if, at the General Practice level, a simple decision could be taken to place the affected person onto a realistic retraining program while they await further tests and diagnosis. This would mean that the worker could find an alternative employment while waiting to find out what caused their ill health.

As my aim is to return to work and enjoy as normal a life as is possible, diagnosis is, of course, extremely valuable when approaching my doctors for treatment and therapy.

3 COST

3.1 Emotional and Social

Over the past two years, the cost to myself and my family in trying to find a diagnosis and treatment for my condition cannot be measured simply in terms of cash. Loss of self-esteem, and loss of standing in the eyes of my children and friends must also be taken into account. Social exclusion due to the lack of spending cash, and the emotional effect of the constant suggestion that I was just malingering are merely a few of the actual costs of falling ill with an occupational illness that is not yet proscribed in the UK.

I felt that doctors prejudged my case and assumed that I was either looking for a sick note to have time off work, or that I was trying to build a case to sue someone. Neither of which was anywhere near the truth, I simply could not afford to fall ill. What I truly wanted was to get treatment and get back to work as soon as possible. Today, having gone through the full circle and being in the process of returning to work as a self employed man, I feel that I have valid and pertinent questions regarding the effectiveness of the UK's system of dealing with people who are incapacitated from their usual employment.

When one marriage partner falls ill with a long term limiting illness such as that caused by VAD it naturally causes a great deal of stress between husband and wife. When, as with VAD, the illness is not proscribed, the stressors acting on the marriage relationship multiply. Stress multiplies because of things like: greatly reduced family income, prevention from claiming other benefits such as free prescriptions, doubts about the true illness of the debilitated partner i.e. whether they are as ill as they make out. These aspects, taken together with the physical debilitations described above, are a sure recipe to hinder a healthy relationship.

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The information I have gathered among researchers and colleagues alike clearly indicate that my clinical, social and financial situation is very far from being a unique case.

3.2 Pounds and Pennies

A close estimate of the financial costs incurred by my family is in the region of £50,000, due to loss of earnings, retraining, actively seeking diagnosis, and the increased expenditure associated with ill health. I have been led to believe that the number of people employed in the transport industry that sign off sick from work is twice the national average. As a Rail Maritime & Transport Union representative, I have participated in negotiations concerning pay and working conditions. I and several others present at these meetings were told that because of the high rates of sickness in the Company (a thick wad of sick notes was waved at us from across the table), we could not hope to have the rise we asked for.

Studies to date suggest that as many as 70% of people exposed for long periods to LFN may develop severe stages of VAD. Approximately 30% of the individuals studied do not develop the acute pathology associated with low frequency noise exposure[1]. Studies show that as many as 50% of men occupationally exposed to LFN for more than ten years will develop permanent debilitating illnesses, eventually leading to early disability retirement[1].

The Confederation of British Industry (CBI) and the Chartered Institute of Personnel and Development (CIPD) estimated that the direct annual cost of sickness absence ranges from £434 to £486 per employee per year[4]. Total days of certified incapacity per year for men and women have risen from £503 million in the financial year 1990/91 to £856.8 million in the year 2000/01. Days of incapacity for men of my age (81.3 million days), with a possible 20 to 25 year exposure to occupational LFN, shows a 31 fold increase to that of men under 20 years of age (2.6 million days)[5]. In 2001/02, 26% (£27.6 billion) of the UK Social Security benefit expenditure was spent on the Sick and Disabled[6].

During my stay in Lisbon, VAD researchers informed me of their success story at O.G.M.A. (an aircraft manufacturing, maintenance and rework facility which belonged to the Portuguese Air Force), where the initial studies on VAD were conducted in the early 1980's[1]. From 1987 until 1997, an echocardiography monitoring program was established among all noise-exposed workers, mostly aircraft technicians. From 1980 to 1989, there were 21 disability retirements among this group of professionals. From 1989 to 1996 there were none[1]. Similarly, during this period of time, on-the-job accidents and incidents statistically significantly reduced, and absenteeism dropped from 8.5% to 2.3%[1]. By monitoring the evolution of LFN-induced pathology, workers were removed from the noisy job before their symptoms became disabling. Given their technical qualifications, it was fairly easy to place them in other, similar jobs but where exposure to LFN was not an issue. This was only possible because the company's administration decided to allow this sort of intervention, and later reaped its benefits.

3.3 Estimate Number of People Exposed to Noise and Vibration in the UK

Noise and vibration are intimately connected. The vibration of solids can produce acoustic phenomena (noise), and noise impacting on solids can produce structural vibration. However, in the vast majority of "noisy" occupational environments, noise (especially LFN) and vibration are present simultaneously. LFN is rarely assessed, but vibration levels have been the object of interest of several research teams throughout the world. Therefore, the results obtained from a recent survey of vibration exposure levels in the UK could be used as an approximate conservative estimate of the number of people exposed to the vibroacoustic agent of disease.

Questionnaires were sent out to a random selection of 21,201 men and women from 34 general practices from across the UK, and to a further 993 men and women selected at random from the

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Armed services. During the Summer of 1997 and Winter of 1997/98, 12,907 usable responses (61% response rate) were returned. In a one-week period 7.2 million men and 1.8 million women are exposed to whole body vibration at work, if the occupational use of cars, vans and motorcycles is included. Results suggest that for all occupational sources of exposure, the personal estimated vibration dose value (eVDV) for about 374,000 men and 9000 women exceeds the action level vibration dose value of 15ms^{-1}, as defined in British Standard 6841[7].

4 FINAL COMMENTARY

Today, I have successfully retrained as a self employed CORGI registered gas installer thereby largely avoiding noise and vibration. However, my VAD symptoms still interfere with my work. Specifically, after using any type of vibration tool, I have an episode, consisting of blurred vision, severe headaches, and an overall physical and psychological indisposition. My colleagues, and many workers in the UK, continue at risk of developing on-the-job VAD. Symptoms develop over years of professional exposure to LFN and are the cause of a large number of absences due to illness. VAD can be very disabling, and can crush an individual's social and family life. Because VAD is not yet mainstream among general practitioners, nor among occupational medicine professionals, VAD goes undiagnosed in the vast majority of the cases. In fact, malingering seems to be a more frequent diagnosis than any real pathological condition. Most workers are unaware that their symptoms are directly related to their occupational environments, and are suspiciously regarded by the surrounding community. The financial toll on the UK Social Security budget is immense, as well as on company budgets due to high levels of absenteeism. I urge the appropriate authorities to implement protective measures against LFN and to inform employees who are exposed to noise and vibration, so they will be able to protect themselves.

5 REFERENCES

1. N.A.A. Castelo Branco and M. Alves-Pereira (Coord), Vibroacoustic Disease, Aviation Space & Environmental Medicine, Vol. 70 No. 3, Section II, Supplement (1999).
2. N.A.A. Castelo Branco, The Clinical Stages of Vibroacoustic Disease, Unique Identifier 10189165, Medline Identifier 99203048.
3. B.D. Holt, The pericardium. In: V. Furster , R. Wayne Alexander R, and F. Alexander (Eds). Hurst's The Heart, 10th ed, McGraw-Hill Professional Publishing, 2061-82. (2000).
4. C. Barham and J. Leonard, Trends and Sources of data on sickness absence, Labour Market Division, Office for National Statistics, www.statistics.gov.uk/cci/article.asp?id=223
5. Department for Work and Pensions, Sickness, Invalidity and Incapacity Benefit: Days of Certified Incapacity, www.statistics.gov.uk/statbase/expodata/spreadsheets/D3994.xls
6. Department for Work and Pensions, Figure 8.4, Social security benefit expenditure: by recipient group 2001/02, Great Britain, www.statistics.gov.uk/statbase/expodata/spreadsheets/D6474.xls
7. K.T. Palmer, D.N. Coggon, H.E. Bendall, and M.J. Griffin, Whole Body Vibration Occupational Exposures and their Health Effects in Great Britain. www.hse.gov.uk/research/crr_pdf/1999/CRR99233.pdf

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Vibroacoustic Disease and Respiratory Pathology III – Tracheal and Bronchial Lesions

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Abstract [638] **BACKGROUND.** Wistar rats exposed to low frequency noise (≤500 Hz, including infrasound) (LFN) have been used as experimental models for vibroacoustic disease (VAD) since 1992. The respiratory tract has been extensively studied through light microscopy, and scanning and transmission electron microscopy. LFN-induced tracheal lesions are abundant: loss of cilia, fusion of brush cell microvilli, deformation of secretory cells, and epithelial metaplasia. In VAD patients, respiratory complaints appear in the first 4 years of occupational exposure to LFN (in smokers and non-smokers alike). Thus, it became pertinent to offer a bronchoscopy examination to volunteer VAD patients, in order to investigate the type of lesions present, and whether they could be responsible for some of the respiratory pathology observed in these patients. **METHODS.** Four fully-informed and volunteer VAD patients (2 males-1 mild smoker, 2 females-1 mild smoker) received a bronchoscopy, and biopsies were removed for analysis. **RESULTS.** In all patients, small submucosal vascular-like lesions were observed in the vicinity of the spurs, consisting of increased collagen and elastin fibers. In all cases, histology disclosed cilliary abnormalities, basal membrane hyperplasia, and also thickening of vessel walls. No inflammatory process was ever identified, and no differences were seen between smokers and non-smokers. **DISCUSSION.** These data are in accordance with what has been observed in LFN-exposed animal models and also in 8 VAD patients who developed lung tumours. Moreover, this type of imaging is not frequent in the bronchoscopies given for other reasons. Taken together, it is strongly suggested that these findings could be LFN-specific.

1 INTRODUCTION

Vibroacoustic disease (VAD) is a systemic pathology caused by long-term (years) exposure to low frequency noise (<500 Hz, including infrasound) (LFN), and characterized by the abnormal growth of the extra-cellular matrices (1-3).

Respiratory complaints appear within the first 4 years of exposure, in smokers and non-smokers alike (1). Standard pulmonary function tests are usually normal in VAD patients, with the exception of focal fibrosis, as seen through high resolution CT scan (4), and a compromised neurological control of breathing, as measured by the PCO2 index obtained with the CO2-rebreathing technique (5). Non-productive cough and oropharynx infections are frequent complaints that, initially, seem reversible. In younger workers, these symptoms disappear during vacation and, in retired VAD patients, respiratory symptoms subside and often evolve to asymptomatic situations (1-3). In LFN-

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exposed rat models, 48 hours of continuous exposure showed reversibility of LFN-induced tracheal lesions (6,7). On the other hand, studies of LFN-exposed rats while *in utero* demonstrated that severe cellular damage still existed after one-year of post-birth silence (6,8).
In this report, we investigate tracheal biopsy material taken from 4 VAD patients who volunteered for a bronchoscopy, only one of whom is still being exposed to LFN.

2 METHODS

2.1 Study Population

All patients volunteered to receive a tracheal bronchoscopy. This study was approved by Hospital Scientific Ethics Committees. All 4 cases have been diagnosed with Stage III VAD. (See Table 1.)
Case 1 has been a volunteer member of the VAD (aircraft technician) study population since 1980. He discloses a very thickened pericardium (hallmark of VAD (9)) and very large delays in nerve conduction times for his age group, also common in VAD patients (10).
Case 2 has been a volunteer VAD patient since 1988, when he suffered a cerebral vascular event that left him aphasic. He also discloses the characteristic thickening of cardiac structures, as seen through echocardiography. He was a combat helicopter pilot while an officer in the Portuguese Air Force, grounded since 1979, and retired since 1991.
Case 3 is an airline flight attendant, on sick leave since 2001. She has filed for disability retirement, and contacted our team in 2002. Along with the usual cardiac structure thickening and neurophysiological abnormalities, this patient also discloses severe balance disturbances, as seen in other VAD patients (11).
Lastly, Case 4, contacted our team from abroad, and is exposed to environmental LFN in the home, in the form idling bus engines, approximately 825 hours /month. Buses are located approximately 16 meters from the windows of her home. She has been residing in these conditions since 1999. In May 2001 she suffered an epileptic seizure and, in 2003, she travelled to Portugal and was diagnosed with VAD; she discloses very thickened pericardium and thickened cardiac valves, moderate neurophysiological abnormalities, and brain MRI revealed lesions typically seen in other VAD patients (12).

Case No.	1	2	3	4
Gender	M	M	F	F
Smoker	Mild	No	No	Mild
Age	48	61	36	54
Occupation	Aircraft Technician Retired, (14 yrs)	Air Force Colonel Retired (13 yrs), Combat Pilot until 1979	Airline Crewmember, On sick leave (3 yrs)	Homemaker Still exposed to environmental LFN

Table 1: *Summary of VAD patient information.*

2.2 Bronchoscopy

The procedures were conducted under general anaesthesia, with the jet ventilation system (Sanders-Venturi valve) and through rigid bronchoscopy (Efer-Dumon, France). After identification of the lesion, it was punctured with a Wang needle which produced a slight and self limited hemorrhage. Biopsies were obtained with standard flexible forceps of the bronchofiberscope (Olympus, Japan). For safety reasons, only small volumes of biopsy material were removed.

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2.3 Light Microscopy

Specimens for light microscopy were fixed in 10% buffered formalin, sectioned and prepared for histological observation using standard methods. The sections were stained with hematoxylin-eosin, Masson trichrome solution, chronotrop aniline blue, and PAS.

3 RESULTS

In all patients, bronchoscopy imaging revealed small submucosal vascular-like lesions located distally in both tracheal and bronchial trees, uniformly distributed bilaterally near the spurs. These lesions were pink in color. Informal calculations indicated that maximal diameter of these lesions was approximately 3mm, and many were dot-like. Biopsies were performed on the abnormal mucosa (pink dots) and apparently normal mucosa (outside the pink dots). Pink areas did not bleed as would be expected from typical vascular lesions. No other abnormalities were observed. Case 1 presented the largest number of these pink lesions. Light microscopy of the non-pink areas disclosed irregular cilia distribution, with some areas containing large groupings, while others had scarce cilia. The basal membrane was thickened with abnormal amounts of collagen, and some blood vessels with thickened walls were visible.

In the abnormal (pink) areas, the number of ciliated cells and the cilia were also abnormally distributed. The basal membrane appeared even thicker than in the non-pink areas, with very large amounts of collagen. The entire basal membrane featured an abnormal neo-vascularization, disclosing very thickened blood vessel walls with scarce blood. This intense neo-vascularization was embedded in the collagen bundles that were not continuous, and exhibited ruptures. The amount of elastic fibers was also increased when compared with the non-pink areas.

No inflammatory cellularity nor processes were observed.

4 DISCUSSION

Thickening of blood vessel walls and abnormal growth of collagen was not a surprising observation since this feature has been consistently observed in LFN-exposed humans and animals:

a) in the 1987 autopsy of a VAD patient, fibrosa thickening of blood vessel intima was identified in all observed vessels. Interstitial and focal lung fibrosis was observed in both lungs (13);
b) in LFN-exposed Wistar rats, focal and interstitial fibrosis can be found along the entire respiratory epithelia. Collagen bundles appear foamy. Blood vessel intimas are very thickened with collagen. Alveoli walls are also thickened (6,8,14);
c) in electron microscopy studies of VAD-patient pericardial fragments, pericardial thickening (as seen through echocardiography), has been shown to be caused by an intense proliferation of collagen and other extra-cellular matrix components. Sandwiched within these thickened layers is a newly-formed layer of loose tissue, that includes thickened blood vessels (15-18);
d) in the lung and pleural biopsy of an aircraft technician, diagnosed with VAD (since deceased with a squamous-cell carcinoma of the lung, located in the upper right lobe), vessel walls were thickened, and focal fibrosis was a prominent feature. Thickening of the pleura, due to intense collagen proliferation, was remarkable (19); and
e) in tracheal biopsy material taken from the above patient as well as from a female military parachutist (with >2000 logged hours in military helicopters), basal membrane hyperplasia was identified (20).

This study discloses the first cases where collagen disruption was identified in humans, although foamy, degenerative foci of collagen has already been identified in LFN-exposed animal models

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(6,8). Abnormal growth of the extra-cellular matrices has been a consistent and characteristic feature of LFN-exposed humans, and is believed to be intimately related to the specific biomechanical response of living tissue to LFN-induced vibration, on the tissue and cellular structures. This is the object of independent studies (21-23).
Despite the differences in the a) time exposure patterns, and b) types of LFN-rich acoustical environments of these patients, these lesions appeared in all, although case 1 was the most severe. The fact that these pink dots never appear to exceed about 3 mm in diameter is reminiscent of the results obtained in a experiment conducted in 1969, by Ponomarkov *et al* (24). Here, dogs were exposed to 1.5-2 hours of about 120-130 dB. Under the pleura, autopsy results revealed 3mm diameter hemorrhages, which increased in number - but not in size – with increasing noise exposure time.
It is important to emphasize that even though the retired VAD patients (Cases 1-3) are no longer working in LFN-rich environments that produced the onset of their pathology, as residents of urban and suburban communities, they are still exposed to environmental LFN. LFN exposure is not restricted to the aeronautical industry and military settings. Unfortunately, as Case 4 clearly attests, excessive exposure to environmental noise can also lead to the development of VAD. Cars, public transportation, and high volume roadways are all significant sources of LFN to which Cases 1-3 are still exposed. It should be noted that for a period of time post-retirement, Case 1 took up a job as vendor, which required that he spend very large amounts of time travelling in his car, a known LFN-rich environment (25). Additionally, Case 1 has the greatest amount of cumulative exposure time to LFN. Studies in immunohistochemistry and electron microscopy are ongoing.

5 SUMMARY

The first bronchoscopies performed on 4 volunteer VAD patients demonstrate, once again, that LFN exposure induces the abnormal growth of collagen and thickening of blood vessels. These lesions may by LFN-specific.

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REFERENCES

[1] N.A.A. Castelo Branco, "The clinical stages of vibroacoustic disease", *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A32-9, (1999).
[2] N.A.A. Castelo Branco and E. Rodriguez Lopez E, "The vibroacoustic disease – An emerging pathology", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A1-6, (1999).
[3] N.A.A. Castelo Branco, E. Rodriguez Lopez, M. Alves-Pereira and D.R. Jones, "Vibroacoustic disease: some forensic aspects", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A145-51, (1999).
[4] J.M. Reis Ferreira, A.R. Couto, N. Jalles-Tavares, M.S.N. Castelo Branco and N.A.A. Castelo Branco, "Airflow limitations in patients with vibroacoustic disease", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A63-69, (1999).
[5] J. Reis Ferreira, C.P. Mendes, M. Antunes, J. Martinho Pimenta, E. Monteiro, M. Alves-Pereira and N.A.A. Castelo Branco, "Diagnosis of vibroacoustic disease - preliminary report", *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 112-14, (2003).
[6] N.A.A. Castelo Branco, M. Alves-Pereira, J. Martins dos Santos and E. Monteiro, "SEM and TEM study of rat respiratory epithelia exposed to low frequency noise". In: *Science and Technology Education in Microscopy: An Overview, Vol. II* (A. Mendez-Vilas ed.) Formatex, Badajoz, Spain, 2003, pp. 505-33.

009905

[7] N.A.A. Castelo Branco, P. Gomes-Ferreira, E. Monteiro, A. Costa e Silva, J. Reis Ferreira and M. Alves-Pereira, "Respiratory epithelia in Wistar rats after 48 hours of continuous exposure to low frequency noise," *Revista Portuguesa Pneumologia*, **IX**(6), pp. 474-9, (2003).

[8] N.A.A. Castelo Branco, E. Monteiro, A. Costa e Silva, J. Reis Ferreira and M. Alves-Pereira, "Respiratory epithelia in Wistar rats born in low frequency noise plus varying amount of additional exposure" *Revista Portuguesa Pneumologia*, **IX** (6), pp. 481-492, (2003).

[9] B.D. Holt, "The pericardium". In: *Hurst's The Heart*, (V. Furster, R. Wayne Alexander, & F. Alexander, eds.), McGraw-Hill, New York, 2001, pp. 2061-82.

[10] M.S.N. Castelo Branco, N.A.A. Castelo Branco, A. Entrudo and J. Marvão, "[A standardization method of the brainstem auditory evoked potentials]", *Revista Sociedade Ciências Médicas de Lisboa*, **149**, pp.214-220, (1985). (In Portuguese, Awarded the 1984 Ricardo Jorge National Public Health Award)

[11] A.J.F. Martinho Pimenta, M.S.N. Castelo Branco and N.A.A. Castelo Branco, "Balance disturbances in individuals with vibroacoustic disease", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A96-99, (1999).

[12] M.G. Pimenta, A.J.F. Martinho Pimenta, M.S.N. Castelo Branco and N.A.A. Castelo Branco, "ERP P300 and brain magnetic resonance imaging in patients with vibroacoustic disease", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A107-114, (1999).

[13] N.A.A. Castelo Branco, "A unique case of vibroacoustic disease. A tribute to an extraordinary patient", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A27-31, (1999).

[14] N.A.A. Castelo Branco, E. Monteiro, A. Costa e Silva, J. Martins dos Santos, J. Reis Ferreira and M. Alves-Pereira, "The lung parenchyma in low frequency noise exposed Wistar rats", *Revista Portuguesa Pneumologia*, **X**(1), pp. 77-85, (2004).

[15] N.A.A. Castelo Branco, A.P. Águas, A. Sousa Pereira, E. Monteiro, J.I.G. Fragata, F. Tavares and N.R. Grande, "The human pericardium in vibroacoustic disease", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A54-62, (1999).

[16] N.A.A Castelo Branco, A. Águas, A. Sousa Pereira, E.Monteiro, J.I.G. Fragata and N.R. Grande, "The pericardium in noise-exposed individuals", *Internoise 2001*, The Hague, Holland, pp. 1003-6, (2001).

[17] N.A.A. Castelo Branco, J.I.Fragata, A.P. Martins, E. Monteiro and M. Alves-Pereira, "Pericardial cellular death in vibroacoustic disease", *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 376-377, (2003).

[18] N.A.A. Castelo Branco, J.I. Fragata, E. Monteiro and M. Alves-Pereira, "Pericardial features in vibroacoustic disease patients", *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 380-381, (2003).

[19] J. Reis Ferreira, C.P. Mendes, N.A.A. Castelo Branco, E. Monteiro and M. Alves-Pereira, "The human lung and pleura in vibroacoustic disease", *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 386-387, (2003).

[20] J. Reis Ferreira, C.P. Mendes, N.A.A. Castelo Branco, E. Monteiro and M. Alves-Pereira, "The human trachea in vibroacoustic disease", *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 388-389, (2003).

[21] M. Alves-Pereira, J. Joanaz de Melo J and N.A.A. Castelo Branco, "Pericardial biomechanical adaptation to low frequency noise stress", *Proceedings 1st Intern. Meet. Applied Physics*, Badajoz, Spain, 2003: Abstract No. 354. (Accepted for publication, American Institute of Physics)

[22] M. Alves-Pereira, J. Joanaz de Melo J and N.A.A. Castelo Branco, "Actin and tubluin-based strucutres under low frequency noise stress. *Proceedings 1st Intern. Meet. Applied Physics*, Badajoz, Spain, 2003, Abstract No. 355. (Accepted for publication, American Institute of Physics)

[23] M. Alves-Pereira, J. Joanaz de Melo J and N.A.A. Castelo Branco, "Low frequency noise exposure and biological tissue: reinforcement of structural integrity? *Proceedings 1st Intern. Meet. Applied Physics*, Badajoz, Spain, 2003, Abstract No. 356. (Accepted for publication, American Institute of Physics)

[24] V.I. Ponomarkov, Tysik Ayu, V.I. Kudryavtseva and A.S. Barer, "Biological action of intense wide-band noise on animals", Problems of Space Biology NASA TT F-529, **7**, pp. 307-9, (1969).

[25] M. Alves-Pereira, J. Reis Ferreira, J. Joanaz de Melo, J. Motylewski, E. Kotlicka and N.A.A. Castelo Branco, "Noise and the respiratory system", *Revista Portuguesa Pnumologia*, **IX**, pp. 367-79, (2003).

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Vibroacoustic Disease in a Ten Year Old Male

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Abstract [634] Background. Restelo is a suburb of Lisbon, Portugal, located on the northern hillside margin of the River Tagus. Directly across the river is a grain cargo-ship docking site where grain in bulk is poured into merchant ships. Until 2003, this site would operate for days at a time, including at night. This team was contacted by a family residing in Restelo, who complained about the noise during the grain-site operation. Low frequency noise (LFN) was measured at the house during operation, and the entire family received volunteer medical tests because vibroacoustic disease (VAD) was suspected. This report describes the LFN-induced pathological medical findings observed in this family. **METHODS.** Mr. F., 39-year-old architect, Mrs. F., 42-year-old Forestry Engineer and P., 10-year-old son, all received an echocardiogram to determine if characteristic LFN-induced cardiac thickening was present. **RESULTS.** All exhibited some degree of pericardial thickening. Among the adult parents, the father exhibited a thicker pericardium than the mother. However, 10-year-old P. disclosed very thickened pericardium and mitral valve leaflets. No diastolic dysfunction was identified in any member of this family. **DISCUSSION.** There is no history of rheumatic fever, nor is pericarditis present, nor is asbestos an issue. As in other LFN-exposed individuals, this family exhibited thickening of cardiac structures that is characteristic of VAD patients. The severity of the cardiac pathology seen in P. is related to the fact that his exposure began *in utero*. This family has been diagnosed with environmentally-induced VAD.

1 INTRODUCTION

Mr. F. is a 39-year-old architect who lives with his family in Restelo, a suburb of Lisbon, Portugal. Restelo is located on the hillside of the northern margin of the Tagus River. Directly in front of Restelo, on the other side of the river, operates a grain cargo-ship docking site. Until 2003, this industrial complex could function at any hour of the day or night. In February 2003, Mr. F. contacted our team complaining about low frequency noise (≤500 Hz, including infrasound) (LFN) in his home.

Mr. and, Mrs. F., a 42-year-old forestry engineer, had moved into their home in August 1992. Today they have two children, P. their 10-year-old son, and an 18-month-old baby. He explained:

"Some time ago I became aware of a vibration that interfered with my concentration, although for a very long while I was unable to determine the source. I began to suspect that the vibration came from the cereal silos when I noticed that each time I felt the vibration throughout my home, a ship was docked at the site. However, no one in my home shared my perceptions of a vibration, and this left me unsure as to the external source of the disturbance. Only when the consequences of the vibration became more evident, as seen and heard in the rattling of our front door, did my wife agree that something was indeed very wrong. Whether we noticed it then, or if the rattling increased

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for some reason, is unknown. I recognized that I *felt* the disturbance more than I *heard* it."

1.1 Noise Measurement

Noise was measured at Mr. F.'s home with a real time frequency analyser (Bruel&Kjaer, 2260 Observer) equipped with a ½" microphone (Bruel&Kjaer, 4189). Sound pressure levels were documented in dBA and dBLin, and frequency distributions were obtained in 1/3 octave bands, with a lower limiting frequency of 6.3 Hz.

Measurements were made on different days, with and without the "noise" present in the home. Figures 1 and 2 show the L_{eq} for the 1/3 octave band analysis (in dB_{Lin}), as well as the overall levels, in both dBA and dB_{Lin} (Table 1). These graphs are representative of the values obtained to date.

All frequency spectra presented are compared to the cockpit of the Airbus-340. The acoustical environment within an airline cockpit is rich in LFN components (1). Both pilots and flight attendants are at risk for developing vibroacoustic disease (VAD) (2), a systemic pathology caused by excessive exposure to LFN, and characterized by the abnormal growth of the extra-cellular matrices (3). Since no standards yet exist for LFN, and specific frequency bands have not yet been linked to specific lesions, it became pertinent, among VAD-researchers, to compare other LFN-rich environments with the airline cockpit. Hence, all graphs also contain comparative information regarding this acoustical environment.

There is an increase in the acoustical energy within the home (Figures 1 and 2), that seemed to be directly related with what Mr. F. describes as the operation of the site. However, other frequency bands below 16 Hz also disclose non-trivial levels. These acoustical events were also present when the acoustical phenomena associated with the site's operation were not present (See Figure 1).

The difference in dBA vs. dBLin values is due to the predominance of LFN components. The dB-value does not provide reliable information on the lower frequency bands (<500 Hz) (4,5). The dBLin value does not reflect the differences observed in the frequency distribution of these acoustical environments (4,5).

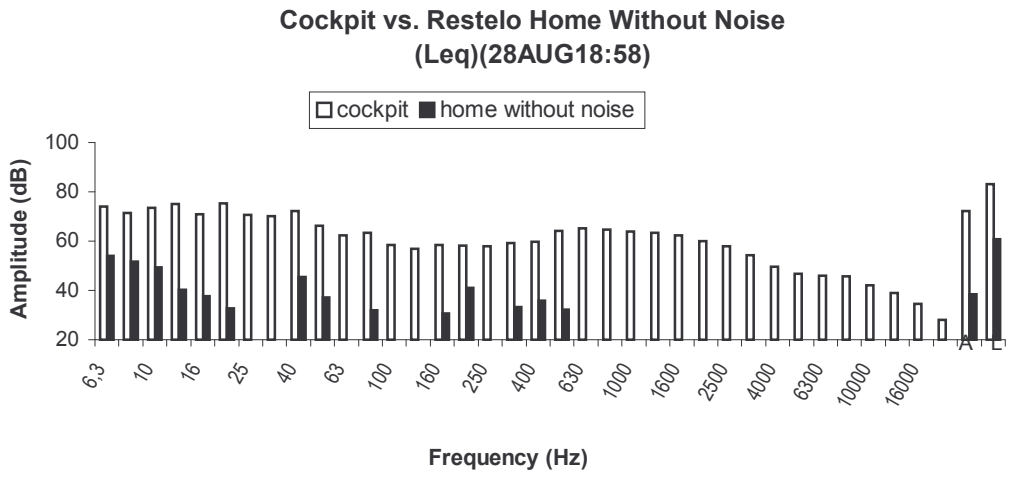


Figure 1: *Comparison of the frequency distribution obtained in the cockpit of the Airbus-340 (see text) with that obtained in the Restelo home, when the acoustical phenomena were not present (L_{eq} values). Overall dBA and dBLin levels are labelled A and L, respectively.*

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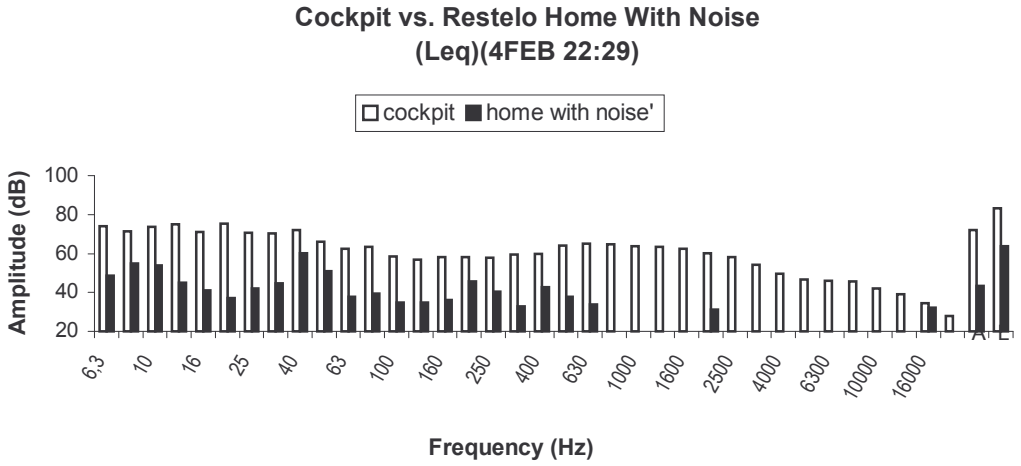


Figure 2: *Comparison of the frequency distribution obtained in the cockpit of the Airbus-340 (see text) with that obtained in the Restelo home, when the acoustical phenomena was present (L_{eq} values).Overall dBA and dBLin levels are labelled A and L, respectively.*

	Cockpit	Without Noise	With Noise
dBA	72.1	38.4	43.4
dBLin	83.2	60.7	63.7

Table1: *dB-levels values for all locations*

As a final note, even though the acoustical phenomena were very evident within the home, no confirmation was obtained from the silos authorities, attesting to the actual operation of the facility. However, each time the acoustical phenomena were present, a ship was, indeed, docked. And when no ship was seen at the facility, no similar acoustical phenomena were present in the home. The fact that other LFN components exist within the home when the silos are not in operation (Figure 1), is an indication that the silos are not the only source of LFN in this home.

1.2 Brief Medical Histories

Mr. F. is apparently asymptomatic. He complains of a lack of concentration and overall irritation, and has severe bouts of rosacea. He has always lived the suburbs of the city of Lisbon, and has been working in the center of Lisbon for the past 10 years.

Mrs. F. has been diagnosed with hepatitis A, mononucleosis and allergic rhinitis. While still a student in university, she was diagnosed with vestigial epilepsy. She complains of body aches, particularly in the right shoulder, left knee, back and neck. X-rays have not revealed any abnormalities. She has always had headaches, mostly in the back of the neck. Approximately 4 years ago, while in a shopping mall supermarket, Mrs. F. suffered a violent tachycardia, with feelings of faintness. She was taken to the hospital where a subsequent EKG did not disclose abnormalities. Mrs. F. works in governmental administrative offices, in the center of Lisbon for 16 years.

P. suffered from asthma until the age of 1 year. At the 5–8 months of age, he was medicated for reflux, and then again until he was 1 year old. At 8 months he suffered pneumonia. After the age of 1, he began to develop repeated ear infections that were not responsive to antibiotics. At age 3 he underwent ear surgery. At the age of 5, while at school, he lost his vision, and was taken to the hospital where the EKG revealed vestigial epilepsy. Nose bleeds without an apparent cause used to be frequent, but have subsided with age.

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There is no history of rheumatic fever, nor is pericarditis present, nor is asbestos an issue. Given the above acoustical and medical data, the entire family received an echocardiogram because VAD was suspected.

2 METHODS

Echocardiogram was performed on this family using HP 1500 SONOS, 2-D, M mode, color Doppler analysis and spectral Doppler. All echocardiograms were recorded on VHS video tape, focusing on pericardial, aortic and mitral valve thickening, and qualitatively evaluated using a score system from 0 to 3 points: 0 points for no thickening, and 3 points for maximum thickening (6,7).

3 RESULTS

Pericardial thickening (the hallmark of VAD (8)) was identified in all family members: Mr. F. disclosed a 2 point pericardial thickening; Mrs. F. disclosed a 1 point thickening, and P. disclosed a 3 point pericardial thickening. E > A in all cases. Mrs. F. also exhibited thickened mitral valve leaflets (1 point), as did P. (3 points).

4 DISCUSSION

This family has been diagnosed with VAD, and is currently undergoing additional testing. This cardiac pathology is common to all LFN-exposed individuals, and was first observed, through echocardiography by Prof. T. Matoba (Japan), in 1983 (9). Today it is considered VAD-specific (8). The LFN response of biological tissue is associated with the abnormal proliferation of the extra-cellular matrices. This has been observed by this team in VAD patients through autopsy (10), echocardiography (2,6,7), electron microscopy of pericardial fragments (11-14), respiratory tract biopsy material (15,16), and in LFN-exposed animal models (17,19).

Neurological damage associated with VAD were the initial observations that triggered research into LFN-induced pathology among aeronautical workers (20). Epilepsy (20,21), rage reactions (22), balance (23) and movement disorders (24) have all been observed among VAD patients. Some have been associated with brain damage, as visualized through magnetic resonance imaging and event-related potentials P300 (25), and brainstem auditory evoked potentials (26).

Immunological changes induced by LFN have been documented in LFN-exposed workers (27), as well as in LFN-exposed rodents (28). Previous studies have suggested that noise-exposed workers are more prone to develop auto-immune disease (29-33). Among VAD patients, lupus (2,7) and vitilligo (7) are common findings. In lupus-prone mice, LFN-exposure accelerated of the onset of lupus and increased mortality (34).

Respiratory pathology, in smokers and non-smokers alike, is one of the most prominent features among LFN-exposed individuals (3,35,36). Electron microscopy studies of the respiratory tract of LFN-exposed rodents has already demonstrated that LFN targets the respiratory system (17-19,37). *In utero*, LFN-exposure causes irreversible damage to the respiratory epithelium, even after the animals are kept in silence for one year post-birth (17,18). Thus it is not surprising that P. would exhibit a more severe case of VAD than his parents, especially since he continues to be exposed post-birth.

The chronological evolution of signs and symptoms of VAD, as set forth for aircraft technicians, is based on an occupational exposure, i.e., 8 hours/day, 5 days/week (3). However, when the exposure is in the home, the occupational exposure schedule is not applicable. Exposure in the home can be more severe because of the amount of hours normally spent in the home, particularly when one sleeps, and for children. In other LFN environments, such as those found in submarines, on oil rigs, and in space, the occupational-schedule is also not directly applicable.

Although VAD was initially diagnosed in LFN-exposed workers, such as aircraft technicians (3,22) military (38) and commercial (2) pilots and flight attendants, a growing number of individuals are being diagnosed with VAD due to environmental LFN exposure (7,22,39). The issue of LFN-induced pathology can no longer remain restricted to the domain of Occupational Health, but must be included in Public Health concerns.

5 SUMMARY

VAD has been diagnosed in a family whose home is exposed to environmental LFN. These are not the first documented cases of environmentally-induced VAD. The results presented herein bring the issue of LFN-induced pathology into the realm of Public Health concerns.

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REFERENCES

[1] M. Alves-Pereira, M.S.N. Castelo Branco, J. Motylewski, A. Pedrosa and N.A.A. Castelo Branco, “Airflow-induced infrasound in commercial aircraft”, *Internoise 2001*, The Hague, Holland, pp. 1011-14, (2001).

[2] A. Araujo, F. Pais, J.M.C. Lopo Tuna, M. Alves-Pereira and N.A.A. Castelo Branco, “Echocardiography in noise-exposed flight crew”, *Internoise 2001*, The Hague, Holland, pp. 1007-10, (2001).

[3] N.A.A. Castelo Branco, "The clinical stages of vibroacoustic disease", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A32-9, (1999).

[4] M. Alves-Pereira, “Extra-aural noise-induced pathology. A review and commentary. *Aviation Space Environmental Medicine*, **70** (March, Suppl), pp. A7-21, (1999).

[5] M. Alves-Pereira, J. Reis Ferreira, J. Joanaz de Melo, J. Motylewski, E. Kotlicka and N.A.A. Castelo Branco, “Noise and the respiratory system”, *Revista Portuguesa. Pnumologia*, **IX**, pp. 367-79, (2003).

[6] W. Marciniak, J. Nóbrega, A. Bordalo e Sá, J.M.C. Lopo Tuna, M.S.N. Castelo Branco, M. Alves-Pereira and N.A.A. Castelo Branco, “Vibroacoustic disease induced by long-term exposure to sonic booms”, *Internoise 2001*, The Hague, Holland, pp. 1095-98, (2001).

[7] R. Torres, G. Tirado, A. Roman, R. Ramirez, H. Colon, A. Araujo, F. Pais, W. Marciniak, J. Nóbrega, A. Bordalo e Sá, J.M.C. Lopo Tuna, M.S.N. Castelo Branco, M. Alves-Pereira and N.A.A. Castelo Branco, “Vibroacoustic disease induced by long-term exposure to sonic booms”, *Internoise 2001*, The Hague, Holland, pp. 1095-98, (2001).

[8] B.D. Holt, “The pericardium”. In: *Hurst's The Heart*, (V. Furster, R. Wayne Alexander, & F. Alexander, eds.), McGraw-Hill, New York, 2001, pp. 2061-82.

[9] T. Matoba, “Increased left ventricular function as an adaptive response in vibration disease”, *American Journal Cardiology*, **15**, pp. 1223-6, (1983).

009911

[10] N.A.A. Castelo Branco, “A unique case of vibroacoustic disease. A tribute to an extraordinary patient”, *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A27-31, (1999).
[11] N.A.A. Castelo Branco, A.P. Águas, A. Sousa Pereira, E. Monteiro, J.I.G. Fragata, F. Tavares and N.R. Grande, “The human pericardium in vibroacoustic disease”, *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A54-62, (1999).
[12] N.A.A Castelo Branco, A. Águas, A. Sousa Pereira, E.Monteiro, J.I.G. Fragata and N.R. Grande, “The pericardium in noise-exposed individuals”, *Internoise 2001*, The Hague, Holland, pp. 1003-6, (2001).
[13] N.A.A. Castelo Branco, J.I.Fragata, A.P. Martins, E. Monteiro and M. Alves-Pereira, “Pericardial cellular death in vibroacoustic disease”, *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 376-377, (2003).
[14] N.A.A. Castelo Branco, J.I. Fragata, E. Monteiro and M. Alves-Pereira, “Pericardial features in vibroacoustic disease patients”, *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 380-381, (2003).
[15] J. Reis Ferreira, C.P. Mendes, N.A.A. Castelo Branco, E. Monteiro and M. Alves-Pereira, “The human trachea in vibroacoustic disease”, *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 388-389, (2003).
[16] J. Reis Ferreira, C.P. Mendes, N.A.A. Castelo Branco, E. Monteiro and M. Alves-Pereira, “The human lung and pleura in vibroacoustic disease”, *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 386-387, (2003).
[17] N.A.A. Castelo Branco, M. Alves-Pereira, J. Martins dos Santos and E. Monteiro, “SEM and TEM study of rat respiratory epithelia exposed to low frequency noise”. In: *Science and Technology Education in Microscopy: An Overview, Vol. II* (A. Mendez-Vilas ed.) Formatex, Badajoz, Spain, 2003, pp. 505-33.
[18] N.A.A. Castelo Branco, E. Monteiro, A. Costa e Silva, J. Reis Ferreira and M. Alves-Pereira, “Respiratory epithelia in Wistar rats born in low frequency noise plus varying amount of additional exposure” *Revista Portuguesa Pneumologia*, **IX** (6), pp. 481-492, (2003).
[19] N.A.A. Castelo Branco, E. Monteiro, A. Costa e Silva, J. Martins dos Santos, J. Reis Ferreira and M. Alves-Pereira, “The lung parenchyma in low frequency noise exposed Wistar rats”, *Revista Portuguesa Pneumologia*, **X**(1), pp. 77-85, (2004).
[20] N.A.A. Castelo Branco and E. Rodriguez Lopez E, "The vibroacoustic disease – An emerging pathology", *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A1-6, (1999).
[21] GIMOGMA. [Epilepsy of vascular etiology, a clinical picture of vibration disease?] *Revista Portuguesa Medicina Militar*, **32**, pp.5-9, (1984). (In Portuguese)
[22] N.A.A. Castelo Branco, E. Rodriguez Lopez, M. Alves-Pereira and D.R. Jones, "Vibroacoustic disease: some forensic aspects", *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A145-51, (1999).
[23] A.J.F. Martinho Pimenta, M.S.N. Castelo Branco and N.A.A. Castelo Branco, “Balance disturbances in individuals with vibroacoustic disease”, *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A96-99, (1999).
[24] A.J.F. Martinho Pimenta and N.A.A. Castelo Branco,”Facial dyskinesia induced by auditory stimulation. A report of four cases”, *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A119-121, (1999).
[25] M.G. Pimenta, A.J.F. Martinho Pimenta, M.S.N. Castelo Branco and N.A.A. Castelo Branco, “ERP P300 and brain magnetic resonance imaging in patients with vibroacoustic disease”, *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A107-114, (1999).
[26] M.S.N. Castelo Branco, N.A.A. Castelo Branco, A. Entrudo and J. Marvão, “[A standardization method of the brainstem auditory evoked potentials]”, *Revista Sociedade*

009912

Ciências Médicas de Lisboa, **149**, pp.214-220, (1985). (In Portuguese, Awarded the 1984 Ricardo Jorge National Public Health Award)

[27] A.P. Castro, A.P. Aguas, N.R. Grande, E. Monteiro and N.A.A. Castelo Branco, "Increase in CD8+ and CD4+ T lymphocytes in patients with vibroacoustic disease", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A141-4, (1999).

[28] A.P. Águas, N. Esaguy, A.P. Castro, N.R. Grande and N.A.A. Castelo Branco, "Effect low frequency noise exposure on BALB/C mice splenic lymphocytes", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A128-31, (1999).

[29] Y. Matsumoto, T. Yasue, N. Mizuno and I. Yoshida, "An immunoserological study of patients with vibration syndrome", *Occupational & Environmental Health*, **63**, pp. 357-9, (1992).

[30] Y. Matsumoto, M. Kawabe, T. Yasue, M. Yuguchi and I. Yoshida, "Two cases of scleroderma associated with vibration syndrome", *Japanese Journal Dermatology*, **99**, pp. 155-61, (1989).

[31] R.N. Jones, M. Turner-Warwick, M. Ziskind and H. Weill, "High prevalence of antinuclear antibodies in sandblasters' silicosis", *American Review Respiratory Diseases*, **113**, pp. 393-95, (1976).

[32] C.A. Soutar, M. Turner-Warwick and W.R. Parkes, "Circulating antinuclear antibody and rheumatoid factor in coal penumoconosis", British Medical Journal, **3**, pp. 145-7, (1974).

[33] M. Lippmann, H.L. Eckert, N. Hahon and W.K.C. Morgan, "Circulating antinuclear and rheumatoid factors in coal miners. A prevalence study in Pennsylvania and West Virginia", *Annals Internal Medicine*, **79**, pp. 807-11, (1973).

[34] A.P. Águas, N. Esaguy, A.P. Castro, N.R. Grande and N.A.A. Castelo Branco, "Acceleration of lupus erythematosus-like processes by low frequency noise in the hybrid NZB/W mouse model", *Aviation Space Environmental Medicine*, **70** (3, Suppl), pp. A132-6, (1999).

[35] J. Reis Ferreira, C.P. Mendes, M. Antunes, J. Martinho Pimenta, E. Monteiro, M. Alves-Pereira and N.A.A. Castelo Branco, "Diagnosis of vibroacoustic disease - preliminary report", *Proceedings 8th Intern. Conf. Noise as Public Health Problem (ICBEN)*, Rotterdam, Holland, pp. 112-14, (2003).

[36] J.M. Reis Ferreira, A.R. Couto, N. Jalles-Tavares, M.S.N. Castelo Branco and N.A.A. Castelo Branco, "Airflow limitations in patients with vibroacoustic disease", *Aviation Space Environmental Medicine* **70** (3, Suppl), pp. A63-69, (1999).

[37] N.A.A. Castelo Branco, P. Gomes-Ferreira, E. Monteiro, A. Costa e Silva, J. Reis Ferreira and M. Alves-Pereira, "Respiratory epithelia in Wistar rats after 48 hours of continuous exposure to low frequency noise," *Revista Portuguesa Pneumologia*, **IX**(6), pp. 474-9, (2003).

[38] N.A.A. Castelo Branco, E. Monteiro, M. Alves-Pereira, A.P. Águas, A. Sousa Pereira and N.R. Grande, "Morphological changes in the pericardia of military helicopter pilots", Proc. Microscopy Barcelona 2001, pp. 318-19 (2001).

[39] N.A.A. Castelo Branco, J. Reis Ferreira, M.C. Marques and M. Alves-Pereira, "Vibroacoustic disease: the impact of low frequency noise on quality of life", *ECQual-Journal European Commission on Quality of Life*, 2004 (In Press).

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Google Maps



EXHIBIT
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Decibel Readings taken in 2016 and 2017 by Vicki May Lynch, Nebraska



12/19/16 - Wind Towers - Pointed - To S-W -
Tower Noise - Loud - Roaring Wind
That Never get here - Wind Speed 10 mph
Annoying - At Best -

12/20/16 West / S-W Wind Tower "Loud" Roaring Wind
Can Alissia - No Return
* Call Josh - & he met with us To
discuss Noise Issue - Said he would
Try to help. - Willing Listener -

12-22-16 S-Wind - 6mph - Sound Loud - Roaring
Wind Sound - Can hear In house if
TV is off - NOT Pleasant !

12-23-16 S-Wind @ 19 Sound Worse than Yesterday
Very Disturbing To Say The
Least. Loud Roaring Sound
Not Natural -

12-24-16 S-W @ 15-20 MPH Sond Loud - Can Hear
in House - Not Pleasant -

12-25-16 South / S East - Very Loud Roar -
Can hear in house - Very
Disturbing Did Not Work

12-27-16 S-SE @ South Sound Roaring - Can
64-82 Hear in House - NOT Pleasant
Dec. Vibrates in Wife can feel - The
Sound - Very disturbing -

12-31-16 Source 10 - Roaring Sound - Could
Hear in House -
NOT Pleasant.

1-1-16 EAST / South / ~~SE~~ 8-10 - Roaring - Sound
out Side. + Can Hear Thump
62-81 Sound in House - Worse
Decebells when this direction - Very Disturbing
~~Thump~~. Ringing in Ears inside

1-2-17 N-Wind 15-20 Strong Roar in
House. Foggy + Damp Air
Decebells 67-82 O/S 58-64 I/side
Worst Night yet! -

1-8-17 S-SE wind Strong 15-20 mph - Wind Noise
Stronger. Tonal Noise. Very Annoying
in + out of House -

1-9-17

8:00 AM wind. S-SE 12 mph -
71-89 - 91 Rear Decibels
Very Loud Roar - Even in House
Could Hear -
7:04 PM - 73-88 Dbs. Outside
57-63 ✓ Inside
No or Little wind on Place.
SSE - 12 MPH.

1-10-17

E-SE @ 10 mph. 75-88 - Decibles. Loud
Banging - Hear in House -
Vibrating in Victim Ear -
Very Annoying -

1-13-16

8:08 AM SE - 8 mph 31-33 A
Sound like Thunder. 51-53 C
+ Smaller Roar -
1:19 PM - Roar Sound 37-39 A
S-SE 11 - Visible Hears Thump F/S 53-57 C
S-6 mph. 7:11 P.M. 29-32 A
49-52 C
9:30 PM S-SE @ 8 mph 31-33 A
41-48 C

1-15-17

9:20 AM S-9 mph. 31-34 A
Roar Sound - Little wind 51-54 C
on Place -

009918

1-18-17 Roar S-e8 mph. A - 31-36
Noise - Thumping in House C - 51-56
NOT Pleasant -
705 PM S e8 mp. C 56-58
Loud Roar A 36-38

1-3-17 7:25 PM Roar-Sound A - 32-38
S-19 Can hear C - 51-55
when TV is off -

1-5-17 8:20 PM S-SE e11 A 31-36
Roar - Sound - C 56-67

~~1-6-17~~ 1-19-17 9:34 AM S-15 Front A - 41-45
C - 59-69

Thunder Sound -
Rumble. Here Back A - 41-44
in House - C ~~89~~ - 59-67
7:00 PM S-SE e15
A - 45-54
C 71-83

1-20-17 S-~~19~~ 15 - 5:50 AM
Rower Thumping A - ~~39~~ - 47
Heard In House C 59-74

3-18-17	SE-11	A 37-43
	Loud Roar	C - 57-63
	Rumble - Very Disturbing -	
4-1-17	SE - 9 6:30	A 38-45
	Roar - Rumble.	C 58-65
	Not Good -	
		A 31-43
	8:40 S-SE-9	C 61-50
	Very Disturbing	
4-11-17	8:14 P.m SSE-9	A 44-54
	Roar - Dryer Sound -	C 71-84
	Loud - Not Natural -	
4-13-17	7:53 SE-9	A 30-36
	Low Roar -	C 46-52
4-14-17	5:35 SE-9	A 31-36
	Loud Roar - Rumble.	C 51-58
4-17-17	8:00 P.m Loud Roar	A 36-47
	SSE Jet Engine Running	C 66-73
	12	

April 23 | 8:30 Am SSW 13 MPH | A 34-41.8
Loud Roar - Thumping | C 52-64
Disturbing

8:20 pm SW at 14 MPH | A-38-48
loud roar w/thumping! | C 58-67

5-7-17 SSE @ 10 | A 37-44
Annoying thumping | C 52-64

5-12-17 | 7:00 Am. S-SW 10 | A 32-45
Roar - an - Natural | C 49-52

5-13- | 6:45 | Loud Roar & Thunder | A-39-51
5-16 | Very Disturbing - | C-51-56
5-12 | Heard over TV In house | A-38-44
Thumping - Very distrubing | C-57-67

5-14- | 8:00 Am S-6 - | A 37-48
C - 49-60
12:00 - } | A 39-48
Loud Roar - | C - 52-60
Thumping - Not Natural.

5-24 | 9:00 P.M SE-@ 10 mph | A 39-45
Have Thumbing | C ~~58-64~~
+ Roar

5-25-17 | 5:00 Am. | S-SE @ 18 mph | A 44-55 @C-59-77

Very Disturbing Here in House —

Time 2 6:00 Am | S-SE @ 9 | A 39-45 C 56-68

Loud Roar — Not Natural

6:8 6:32 Am | SSW @ 8 | 38-54 a 51-59 c

Roaring jet sound

July 20 - 10:00 PM 33-36
53-63

July 24 - 6:15 AM 35-38
SE 5 mph
Loud - Banging -

July 25 S-SE all
8:30 PM Loud in House

7/30/17 S-SE 12 mph A 35-40
7:53 PM Here in house. C 57-65

8-4-17 SE @ 11
7:45 P.M Thumping - See it in Chat

8-4-17 - Louder
9:20 PM than before

8-5-17 SE 15
5:24 AM Thumping - Loud

8-5-17 10:00 AM
48-56 A SE
58-83 C Thumping. Can easily hear indoors!

8-8-17 7:30 PM SE 12 37-44 A
Thumping - Heard IDRs 52-60 C

			Wind
8-9-17	5:57 am	38-49 a	SSE 8 MPH
		Thumping Sound	
		54-62 c	
	6:53 am	38-48.9 a Roaring	W
		56-68 c	8-11 MPH
8-15-17	7:40 pm	44-53 a	
	ESE W@10 MPH	66-80 c	
		Thumping, roaring sound	
8-17-17	9:27 PM	44-45 a	W SSE 6
		56-61 c	
		Roaring Sound	
8-18-17	8:28 PM	38-45 a	SSE 11
		66-80 c	
		Roaring	
8-23-17	5:33 AM		SSE 7
		37 a	
		55 c	
	6:14 AM	Loud wooshing	SSE 7
		42 a	
		59 c	
		Starting to Roar + thump	

8-23-17	7:54 AM	41.5 a	SOUTH 8MPH
BACK PORCH		59 c	
FP	8:35 pm	44-45 a	SSE 8 MPH
		63-72 c	
8-24-17	6:55 AM	35-36 a	ESE 8 MPH
		57-68 c	
	Loud Woosking w thumping		
	9:15 AM	33.7-43	S @ 9 mph
		55-66.8 c	
	Woosking + thumping		
	10:00 PM	44-45 a	ESE 4 MPH
		55 c	
8-25-17	6:11 AM	44-51 a	~~E~~ SSE 10 MPH
		68-77 c	
	Roaring + thumping		
	11:49 AM	39-55 a	S 15
		55-78 c	
	Roaring		

8-26-17 6:10 AM SSE 12 MPH

44-53.6 a

63-78 C

Roaring + thumping

7:56 AM 40-44 a SSE 8 MPH

53-58 c

8-30-17 3:22 am 44.3-49.4 a SSE 6 MPH

Roaring 55.6-58.1 c

6:21 am 46.1-51.1 a S7

53.7-62.8 C

Roaring Jet

9:49 38.7-45.8 S8

55.2-58.9

Roaring

8-31 9:51 42-44- A S7

55-62 C

Roaring

9-1 5:26 AM 44.7-61.1 a SSE 13

55.3-81 c

Roaring + thumping

8:08 AM 41.9-53.1 a AT HOUSE S12

49.8-60.1 a AT BARN

55.7-80.1 c AT HOUSE

Roaring + thumping

9/1 1:46 P.M. 38-46 A SW MPH
58-67 C
Roar -

6:57 PM 48.1 - 53.4 A SSE 11
61.9 - 84.2 C
Roaring with thumping

9-3-17 7:26 AM 37.1 - 42 a 9 MPH
54.9 - 60 C
Roaring + thumping

9-7-17 6:29 AM S-SW 6 36-38 - A
43-46 - C

SSW 8 MPH

F Porch 35.8 - 45.4
Back Porch 36.4 - 44.3 a
57.5 - 68.3 C

9-8 10:14 PM SSE 8 42-45 A
Loud Roar 53-57 C

9-9-17 6:28 AM 42.8 - 51.1 a SSE 6
55.3 - 64.7 C
Roaring + thumping

9:33 AM 37.2 - 48.5 a SSE 7
60.3 - 76.5 C

9-9-17 3:49 pm

44.7 - 53.9 a
68.1 - 84.5 c
SE 16

5:30 pm 48 - 57 A
67 - 81 C
SE @ 20

9-10-17 4:42 am 68.1 - 83.4 c
48.7 - 61 a
S-17 mph
Thumping!!!

7:16 33 - 41 A
63 - 70 C
Roaring
S-14 mph

9-11-17 5:28 AM 42 - 44.7 a
53.4 - 59.7 c
Roaring
S-11 mph

9-12-17 6:46 AM 40.3 - 43.1 a
50.7 - 58.6 c
Roaring + thumping
SES-8 mph

9-12-17 6:57 34 - 38 A
54 - 63 C
Roaring - thumping
S-10

8:20 pm 40.6 - 51.5 A
58.7 - 69 C
Roaring + thumping
SSE-8

EXPLAIN SOUND DECIBELS!

9-18-17	5:37 AM SSE 10	Loud - Rom -	46-53 A 73-80 C
	6:22pm	35.5-44.4 a 49-65.5 c	S @ 11 mph
9-19-	5:44 AM	44-56 A 78-83 C Loud Roar - In Home Noise.	SSE @ 10
9/19	7:54 P.m	39-45 A 57-64 C Loud Roar	SSE @ 9
9-21	6:40 AM	38-45 A 64-73 C	SSE @ 8
9-29-17	8:49pm	38.3-41.5 a 53.1-66 C Roar	E 9 MPH
9-30-17		38.6-45.3 a 61.3-70 C Roaring & thumping	SE 10

10-7-17	7:55 pm	33.4 - 36.1 a 53.1 - 56.6 c Roaring jet sound	SSE 7
10-11-17	6:01 AM	35.4-43.3 a 53.8 - 56.6 c Starting to Roar	SE 7
10-11-17	7:25 P.M	38 - 41 A 57-63 C Roar -	SSE 14
	8:01 P.M	43-52 A 63-72 C Roaring & thumping	SSE 11
10-12-17	5:10 AM	38.8 - 54.4 a 60.1 - 80.6 c	S 14
10-16-17	9:51 AM	40.1 - 53.3 a 58.2 - 75.3 c Roaring	S 11
10/17/17	7:53 PM	33-37 A 49-56 C	SSE 8

10/17/ - 8:40 PM 34-36 A SSE 9
55-61 C
Roaring —

10/19/17 7:13 AM 33-35 A SSW 5
51-54 C
Roaring -

10/19/17 9:49 pm 41.5 - 57.7 - A Back Porch SSE 10
- C
Roaring & thumping
Can hear inside

10/20/17 5:35 AM 41.3 - 44.4 A S16
59.3 - 67 C
Roaring - thumping
Can hear inside

8:09 AM 59.9 - 84.4 C S17

8:04 PM. 48 - 54 A S - 18
72 - 88 C
Loud Roar / thump. very

10-25-17 9:00 Pm 31.6 - 41.7 a S10
54.4 - 57.1 c
Wooshing starting to thump

11/1/17 — 36-44 — A
5:40 Am — 58-61 — C — 5.019
ROA - Def Rlsm.

11/1/17 — 8:27 Am — 36--41 — A
61-74 — C
Roaring —

11/5/17 — Very Few Turbines Turning 20%-25%
C — Wnd S/S South - 8-15 mph.

11/6/17 — Wnd SSE @ 10 - Not many Turbines Turning —
Not Normal —

11/7/17 — 7:00 AM Turbines turning really slow. NNW 6 mph
No turbines Running at 3:00 pm

11/8/17 — Large blocks of turbines not running - Windy out

11/9/17 — Turbines barely turning 7:30 Am — W N 11 mph
" " " 12:18 PM — Wind N 10 mph
Large Blocks Not turning " " " 2:45 Pm — NNE 7 mph

11/10/17 8:45 AM Large Blocks of turbine not running Wind SSE 25

@ 11/10 6:27 PM 39-43 C SE 12 MPH
57-63 A
No - Bird, Cricket - Traffic
Very little Ground Noise -
Can Hear in House.

11/12/17 33-38 C
7:30 PM 64-68 A SSE 9
Roar -

11/13/17 8:56 PM 34-36 C
54-56 A Se 8
Roar

11/14/17 7:23 38-43 C Se 16
68-73 A
Roar - Constent

11/27/2017 3:00 PM 32-40 C SWSE 16
54-60 A

11-29/2017 6:29 PM 33-36 A S e 8
56-62

12/3/16 7:10 PM 34-38 A SE 14
58-63 C
Rain - Rain

1-6-18 6:11 PM 34-38 A SE 8
54-56 C

1-12-18 Turbine closest to road south has something hanging from nacelle. Several turbines not turning
1-13-18 "

1-25-18 6:30 PM SSE 14

2-2-18 8:20 PM. SSE 12 39-43 A
57-63 C

3-3-18 5:07 AM SSE ~~14~~ 35-37 A
58-61 C

8:10 AM ~~S~~SE 10 mph 40.9-54 A
60.2-84 C

3-10-18 SE 9 mi peak
thumping & rumbling

9

3-23	6:28 AM		38-43 A 63-68 C RAIN	E-17

6-1-18	6:26 AM	SSE W @ 7	Loud - Roaring
6-4-18	7:00 AM	S @ 12	Roaring - Can hear in closed up house!
6-5-18	5:45 AM	S @ 12	
	9:54 PM	SSE 8 MPH	Roaring, Thundering Awful! Can't sleep in bedroom!
6-8-18	5:11 AM	ESE 6 MPH	Roaring, Can hear indoors with house closed!
6-10-18	5:54 AM	ESE 14 MPH	ROARING, THUNDERING, FREIGHT TRAIN SOUND!

6-14-18

6-15-18

8-2-18 6:09 AM Thumping, rumbling W- 35 mph
noise

EXHIBIT I-6

See attached flash drive for videos 1-6a through I-6e

EXHIBIT I-6a

See attached flash drive for videos 1-6a- May Video 1

EXHIBIT I-6b

See attached flash drive for videos 1-6b May Video 2

EXHIBIT I-6c

See attached flash drive for videos 1-6c May Video 3

EXHIBIT I-6d

See attached flash drive for videos 1-6d May Video 4

EXHIBIT I-6e

See attached flash drive for videos 1-6e May Video 5

EXHIBIT I-7

See flash drive for audio clip for ExhibitI-7 - Audio Clip Recording

Taken By Vicki May on 7/28/2018

1341 Yew Ave



Imagery ©2018 Google, Map data ©2018 Google 500 ft

EXHIBIT
Int. I-8

EXHIBIT I-9

See flash drive for video clip for Exhibit I-9 Video of Turbines Located Around Scott Rueter's Farmhouse (Previous Residence) Taken on November 22, 2016

EXHIBIT I-10

See flash drive for video clip for Exhibit I-10 - Video of Malfunctioning Turbine on Scott Rueter's Property (Section 21, Fairview Township, Osceola County, Iowa) Taken on June 5, 2017

Google Maps 40263 293rd St



Imagery ©2018 Google, Map data ©2018 Google 1000 ft

EXHIBIT
Int. I-11

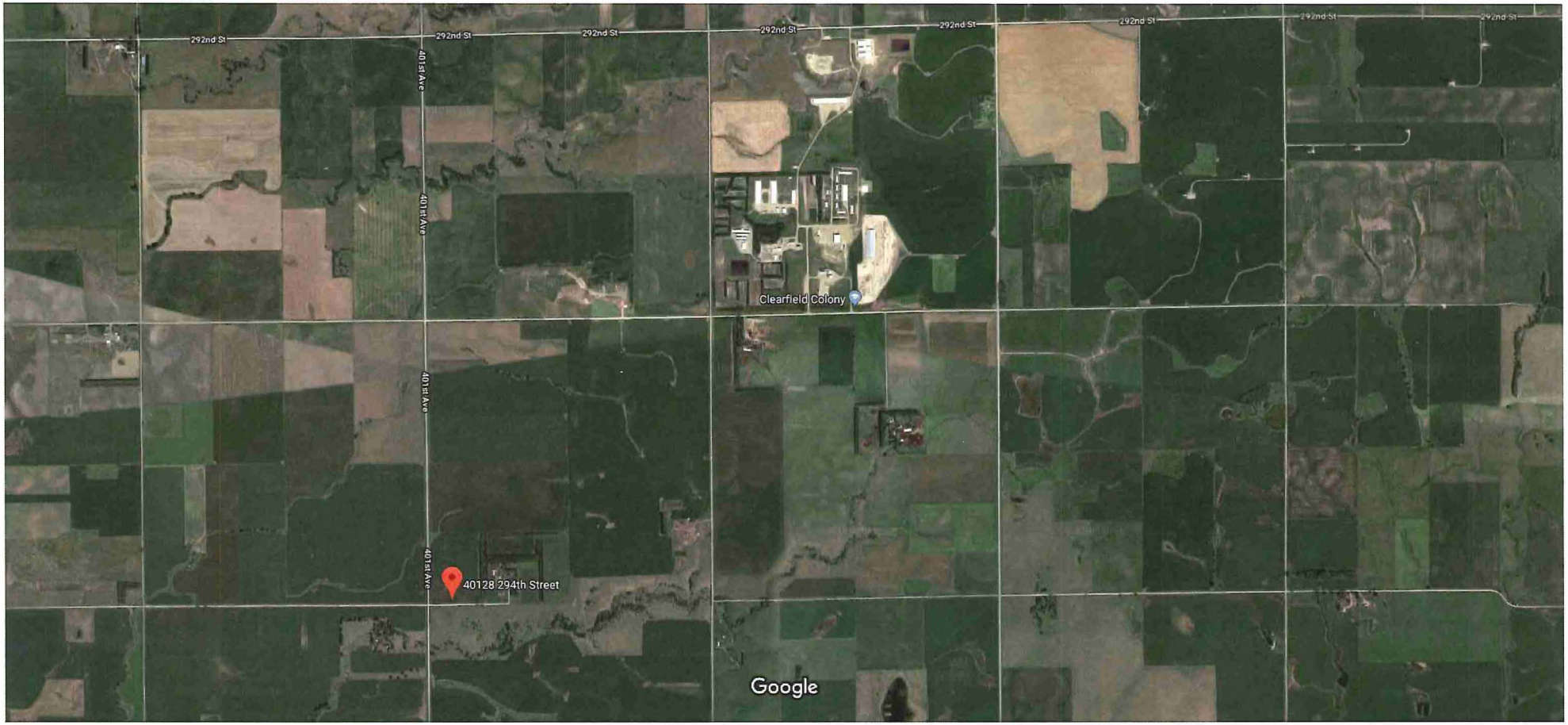


EXHIBIT

Int. I-12

009948

40128 294th St



Imagery ©2018 Google, Map data ©2018 Google 1000 ft

EXHIBIT
Int. I-13

009949

DEFINITIONS

Definitions

For the purpose of this Ordinance, unless otherwise stated, words used in the present tense include the future; the singular number includes the plural and the plural the singular; the word shall means mandatory, not discretionary; the word may is permissive; the word person includes a firm, association, organization, partnership, trust, company or corporation, as well as, an individual; the word lot includes the word plat or parcel; and the words used or occupied include the words intended, designed, or arranged to be used or occupied.

Terms

For the purpose of this Ordinance, certain terms or words used herein shall be interpreted as follows:

Accessory Use or Structure - A use or structure on the same lot with, and of a nature customarily incidental and subordinate to, the principal use or structure.

Actual Construction - Actual construction is hereby defined to include the placing of construction materials in permanent position and fastened in a permanent manner. Where excavation or demolition or removal of an existing building has been substantially commenced, preparatory to rebuilding, such excavation or demolition or removal shall be deemed to be actual construction, provided that work shall be carried on diligently.

Agriculture - The planting, cultivating, harvesting and storage of grains, hay or plants, fruits, or vineyards along with the raising and/or feeding of less than one thousand (1,000) animal units of livestock and/or poultry in an animal feeding operation as defined by this ordinance.

An animal feeding operation as defined by this ordinance is not considered an agricultural use. The processing and/or storage of raw agricultural products, including facilities such as grain elevators and ethanol plants, shall not be considered an agricultural use if such use constitutes the main or principal use on a lot or parcel.

Agriculture Product Processing Facility - A business activity customarily designed to process raw agricultural products into value added products. Agricultural processing facilities include, but are not limited to; feed mills, ethanol plants, and soy bean processing facilities.

All Weather Road - A roadway in which emergency vehicles and local traffic may pass at all times not to include severe weather events such as snow drifting and surface flooding. ***(Amended 11/3/15)***

Animal Feeding Operation - A facility where more than one thousand (1,000) animal units are stabled, confined, fed, or maintained in either an open or housed lots for a total of 45 days or more in any 12-month period and the open lots do not sustain crops, vegetation, forage growth, or post-harvest residues in the normal growing season. Two (2) or more facilities under common ownership are a single animal feeding operation if they adjoin each other (within one (1) mile), or if they use a common area or system for the disposal of manure. For the purpose of this ordinance animal units of differing species shall not be totaled to constitute an animal feeding operation as defined herein.

EXHIBIT
Int. I-14

009950

Animal Units - A unit of measure for livestock. One (1) animal unit is equivalent to:

1	feeder or slaughter beef animal;
.5	horse;
.7	mature dairy cattle;
.27	farrow-to-finish sows;
2.13	swine in a production unit (breeding, gestating and farrowing);
10	nursery swine less than 55 pounds;
2.5	finisher swine over 55 pounds;
10	sheep or lambs;
30	laying hens or broilers;
5	ducks; and
55	turkeys.

Animal Unit Conversion Table
A conversion table designed to integrate the definition of an animal feeding operation with the animal unit definition.

Animal Species	1,000 Animal Units
Feeder or Slaughter Beef Animal	1,000
Horses	500
Mature Dairy Cattle	700
Farrow to Finish Sows	270
Swine in a Production Unit	2,130
Nursery Swine Less than 55 Pounds	10,000
Finisher Swine Over 55 Pounds	2,500
Sheep	10,000
Laying Hens or Broilers	30,000
Ducks	5,000
Turkeys	55,000

Animal Waste Facility - A structure designed and constructed to store and/or process animal waste. Animal waste facilities include but are not limited to; holding basins, lagoons, pits and slurry stores.

Automobile-Machinery Service Station - Building and premises where motor fuel, oil, grease, batteries, tires, and parts may be supplied and dispensed at retail, and where, in addition, customary repair services may be rendered.

Board – The County Commission, Planning Commission, Board of Adjustment or other governmental body governing the district this ordinance refers to. ***(Amended 11/3/15)***

Buildable Area - The portion of a lot remaining after required yards have been provided.

Building - Any structure for the support, shelter and enclosure of persons, animals, chattels, or moveable property of any kind.

009951

Campground - Any premises where two (2) or more camping units are parked or placed for camping purposes, or any premises used or set apart for supplying to the public camping space for two (2) or more camping units for camping purposes, which include any buildings, structures, vehicles or enclosures, used or intended for use or intended wholly, or in part, for the accommodation of transient campers for monetary gain.

Camping Unit - Any vehicle, tent, trailer or portable shelter used for camping purposes.

Commercial Trucking Terminal - A building or structure where seven (7) or more commercially licensed trucks or tractors are rented, leased, kept for hire, or stored or parked for compensation, or from which trucks or tractors, stored or parked on the property, are dispatched for hire as common carriers, and which may include warehouse space.

Conditional Use - A conditional use is a use that would not be appropriate, generally or without restriction, throughout the zoning district, but which, if controlled as to number, area, location or relation to the neighborhood, would promote the public health, safety, welfare, morals, order, convenience, appearance, prosperity or general welfare.

Construction – Any clearing of land, excavation, or other action that would adversely affect the natural environment of the site or route but does not include changes needed for temporary use of sites or routes for non-utility purposes, or uses in securing survey or geological data, including necessary borings to ascertain foundation conditions. ***(Amended 11/3/15)***

Domesticated Large Animals - Any animal that through long association with man, has been bred to a degree which has resulted in genetic changes affecting the temperament, color, conformation or other attributes of the species to an extent that makes it unique and different from wild individuals of its kind. For the purpose of this ordinance the definition shall include, but is not limited to, animals commonly raised on farms and ranches, such as cattle, horses, hogs, and mules.

Dwelling Unit - One (1) room, or rooms connected together, constituting a separate, independent housekeeping establishment for owner occupancy, or rental or lease on a weekly, monthly, or longer basis and physically separated from any other rooms or dwelling units which may be in the same structure and containing independent cooking and sleeping facilities.

Dwelling, Multiple Family - A residential building designed for, or occupied by, two (2) or more families, with the number of families in residence not exceeding the number of dwelling units provided.

Dwelling, Single Family - A detached residential dwelling unit other than a manufactured home designed for or occupied by one (1) family only.

Family - Any number of individuals living together as a single housekeeping unit, in which not more than four (4) individuals are unrelated by blood, marriage or adoption. This definition shall not include foster families as regulated by the State of South Dakota.

Farm, Ranch, Orchard - An area of twenty (20) acres or more which is used for growing usual farm products, vegetables, fruits, trees, and grain, and for the raising thereon of the usual farm poultry and farm animals such as horses, cattle, and sheep, and including the necessary accessory uses for raising, treating, and storing products raised on the premises; but excluding an Animal Feeding Operation.

The processing and storage of raw agricultural products, such as grain elevators and ethanol plants, shall not be considered a farm, ranch or orchard if such constitutes the main or principal use on the lot or parcel

Farm Building - All buildings and structures needed in agricultural operation, including dwellings for owners, operators, farm laborers employed on the farm, and other family members.

Farm Occupation - A business activity customarily carried out on a farm by a member of the occupant's family without structural alterations in the building or any of its rooms, without the installation or outside storage of any machinery, equipment or material other than that customary to normal farm operations, without the employment of persons not residing in the home, which does not cause the generation of additional traffic in the area. Farm occupations include, but are not limited to, seed sales and custom combining support facilities.

Farmstead - The area within or adjacent to the shelterbelt protecting the house and main buildings, including, the driveway and the land lying between the farmstead and the road.

Farm Unit - All buildings and structures needed in an agricultural operation, including dwellings for owners, operators, and other family members.

Fishery - As defined by South Dakota Administrative Rules, Sections 74:03:03:02 and 74:03:03:03 (August 8, 1994). Bon Homme County as described in Section 74:03:03:07. Lakes Bucholz, Clear, Cosby, Hieb, Kloucek, Schaefer and Tyndall Kids Pond (Section 74:03:03:03(6)) are warm water marginal fish life propagation waters and Lake Henry (Section 74:03:03:03(5)) is defined as warm water permanent fish life propagation waters. The Missouri River (Section 74:03:04:04 (1,4,7,8,11) domestic water supply, warm water permanent fish life propagation waters, immersion recreation waters, limited-contact recreation waters, commerce and industry waters. Choteau Creek from Lewis and Clark Lake to S34, T96N, R63W as described in 74:03:04:04 (5,8) and Emanuel Creek from Lewis and Clark Lake to S20 T94N R60W are warm water semi-permanent fish life propagation waters and warm water marginal fish life propagation waters. Dry Choteau Creek as described in 74:03:04:04 (6,8) from Choteau Creek to S.D. Highway 50 is warm water marginal fish life propagation waters and limited-contact recreation waters.

Flammable or Combustible Liquids, or Hazardous Material - Flammable material is any material that will readily ignite from common sources of heat, or that will ignite at a temperature of 600°F or less. Flammable liquid is any liquid having a flash point below 100°F and having vapor pressure not exceeding forty (40) pounds per square inch (absolute) at 100°F. Combustible liquid is any liquid having a flash point at or above 100°F. Hazardous material includes any flammable solids, corrosive liquids, radioactive materials, oxidizing materials, highly toxic materials, poisonous gases, reactive materials, unstable materials, hypergolic materials, pyrophoric materials, and any substance or mixture of substances which is an irritant, a strong sensitizer or which generates pressure through exposure to heat, decomposition or other means.

Game Farm - An area of five (5) acres or more which is used for producing hatchery raised game and nondomestic animals for sale to private shooting preserves.

Game Lodge - A building or group of two (2) or more detached, or semi-detached, or attached buildings occupied or used as a temporary abiding place of sportsmen, hunters and fishermen, who are lodged, with or without meals, and in which there are more than two (2) sleeping rooms.

Grain Elevator - Grain storage facilities, which are the principal and primary use of the lot. Said facilities are generally equipped with devices for housing and discharging significant quantities of grain. This definition does not include normal farm product storage and warehousing facilities such as grain bins and where such storage is an accessory use to the parcel.

Habitable Residential Dwelling – A structure designed and constructed for residential purposes to which utilities such as water and electrical are either active or readily accessible upon the property. Structures currently not occupied shall be able to facilitate human occupation with minor repairs or renovations as determined by the Zoning Administrator. ***(Amended 11/3/15)***

High Voltage Transmission Line – A conductor of electric energy with a minimum voltage of 69 kilovolts and associated facilities. ***(Amended 11/3/15)***

Hobby Farm - An activity carried out in rural residential areas which include the planting, cultivating, harvesting and storage of grains, hay or plants, fruits, or vineyards.

The raising and feeding of livestock and poultry shall be considered as part of a hobby farm if the area, in which the livestock or poultry is kept, is two (2) acres or more in area for every two (2) domesticated large animals, and if such livestock does not exceed ten (10) animals; or the raising of livestock and poultry is incidental or supplemental to the residential use and is not primarily for the growing of crops or raising of livestock.

Home Occupation - A business activity customarily carried on in the home by a member of the occupant's family without structural alterations in the building or any of its rooms, without the installation or outside storage of any machinery, equipment or material other than that customary to normal household operations, without the employment of persons not residing in the home, which does not cause the generation of additional traffic in the street.

Horticulture - The science or art of cultivating fruits, vegetables, flowers, and plants.

Junkyard - A place where non recyclable waste, having no economic value, or waste which is recyclable, but has no chance of being recycled is deposited.

Kennel - Any place where dogs, cats, or other domesticated animals are housed, groomed, bred, boarded, trained, harbored, kept or sold for commercial purposes.

Large Wind Energy System or LWES – All WES facilities excluding Small Wind Energy Systems. ***(Amended 11/3/15)***

Lagoon - Any pond, basin, or other impoundment made by excavation or earthfill for storage or treatment of animal waste.

Lot - Land occupied or to be occupied by a building and its accessory building(s) having its principal frontage upon a public street or officially approved place.

Lot Depth - The average horizontal distance between the front and rear lot lines.

Lot Frontage - The portion of the lot nearest the street. For the purpose of determining yard requirements on corner lots and through lots, all sides of a lot adjacent to streets shall be considered frontage, and yards shall be provided as indicated under Yards in this article.

Lot Line - The legally defined limits of any lot.

Lot Width - The mean horizontal distance between the side lot lines of a lot measured at right angles to the depth or the same distance measured at the front building line.

Manufactured Home - A moveable or portable dwelling which is eight (8) feet or more in width and thirty-two (32) feet or more in length, constructed on a chassis, and which is designed to be towed, designed for year-round occupancy, primarily to be used without a permanent foundation, but which may sit on a permanent foundation, and designed to be connected to utilities. It may consist of one (1) or more units, separately transportable, but designed to be joined together into one (1) integral unit.

The following shall not be included in this definition:

a. Travel trailers, pickup coaches, motor homes, camping trailers, or other recreational vehicles.

b. Manufactured modular housing which is designed to be set on a permanent foundation, and which uses standard sheathing, roofing, siding, and electrical, plumbing, and heating systems.

Mobile Home - See Manufactured Home

Modular Home - A structure or building module that is manufactured at a location other than the site upon which it is installed and used as a residence; transportable in one or more sections on a temporary chassis or other conveyance device; and to be used as a permanent dwelling when installed and placed upon a permanent foundation system. This term includes the plumbing, heating, air conditioning, and electrical systems contained within the structure.

Navigable Waters - A body of water presently being used or is suitable for use for transportation and commerce, or if it has been so used or was suitable for such use in the past**.**

Open Sales Area - Any open land or area used or occupied for the purpose of displaying for sale new or secondhand merchandise, including but not limited to, passenger cars or trucks, farm machinery, construction machinery, motor scooters or motorcycles, boats, trailers, aircraft, and monuments.

Ownership Line - A line defining ownership of property under one owner of record

Person – An individual, partnership, joint venture, private or public corporation, association, firm, public service company, cooperative, political subdivision, Municipal Corporation, government agency, public utility district, consumer's power district, or any other entity, public or private, however organized. ***(Amended 11/3/15)***

Private Shooting Preserves - An acreage of at least one hundred and sixty (160) acres and not exceeding one thousand two hundred and eighty (1,280) acres either privately owned or leased on which hatchery raised game is released for the purpose of hunting, for a fee, over an extended season.

Property Line - The division between two (2) parcels of land, or between a parcel of land and the road.

Route – The location of a High Voltage Transmission Line between two end points. The route may have a variable width of up to 1.25 miles. ***(Amended 11/3/15)***

Salvage Yard - The use of more than one (1) acre of open storage on any lot, portion of lot, or tract of land for the sale, storage, keeping, or for the abandonment, dismantling, or wrecking of automobiles or other vehicles, machines, or parts thereof.

Semi-Portable Agricultural Structures - Anything which requires placement on the ground for agriculture related purposes. Semi-portable agricultural structures include, but are not limited to, feed bunks, calving, lambing, or farrowing sheds, and temporary grain storage facilities.

Shelterbelt - A barrier consisting of trees and shrubs that reduces erosion and protects against the effects of wind and storms. For the purposes of this ordinance a shelterbelt shall include ten (10) or more trees planted in a line, separated by a distance of forty (40) feet or less.

Shelterbelt Restoration - The removal and replacement of two (2) or more rows of trees or of trees totaling one-half (1/2) acre or more, whichever is greater, in an existing shelterbelt.

Small Wind Energy System or SWES – A WES facility with a single Tower Height of less than seventy-five (75) feet used primarily for on-site consumption of power. ***(Amended 11/3/15)***

Street Line - The right-of-way line of a street or road.

Structure - Anything constructed or erected which requires location on the ground, or attached to something having a fixed location on the ground. Among other things, structures include, but are not limited to, buildings and manufactured homes. This definition does not include semi-portable agricultural structures.

Swine, Farrow-to-Finish - An animal husbandry operation including all elements of an animal's life cycle and generally includes a single site operation with breeding, gestating, farrowing, nursery, feeder, and finisher stages of swine. The operation is viewed as a complete operation and is different from multi-site production methods

Swine, Feeder - A swine of an intermediate stage of growth; removed from a nursery facility at an approximate weight of fifty-five (55) pounds then sold and/or moved to a finishing unit.
Swine, Finish - A swine weighing between an approximate weight of fifty-five (55) pounds and the standard slaughter weight for the specific genetic makeup of the animal. This term shall also include replacement stock raised to an adult stage for the purposes of reproduction or show. A swine in a finishing unit may be part of either a single or multi-site production system.

Swine, Nursery - A young swine weaned from a sow and placed in a unit for the purpose of growth to an approximate weight of fifty-five (55) pounds. A swine in a nursery unit may be part of either a single or multi-site production system.

Swine, Production Unit - A swine unit primarily focused on the breeding, gestating, and farrowing of swine. This unit may include newly farrowed swine not yet weaned from the sow. A swine production unit may be part of either a single or multi-site production system.

System Height – The height above grade of the tallest point of the WES, including the rotor radius. ***(Amended 11/3/15)***

Tower Height – The height above grade of the fixed portion of the tower, excluding the wind turbine itself. ***(Amended 11/3/15)***

Turbine – The parts of the WES including the blades, generator and tail. ***(Amended 11/3/15)***

Used Vehicles Dealer - Any person who, for commission or with intent to make a profit or gain sells, exchanges, rents with option to purchase, offers or attempts to negotiate a sale or exchange of used vehicles or who is engaged in the business of selling used vehicles; or any person who sells five (5) or more used vehicles or offers for sale five (5) or more used vehicles at the same address or telephone number in any one calendar year.

Utility – Any person engaged in the generation, transmission or distribution of electric energy in this state including, but not limited to, a private investor owned utility, a cooperatively owned utility, a consumers power district and a public or municipal utility. ***(Amended 11/3/15)***

Variance - A variance is a relaxation of the terms of the zoning ordinance where such variance will not be contrary to the public interest and where, owing to conditions peculiar to the property and not the result of the actions of the applicant, a literal enforcement of the ordinance would result in unnecessary and undue hardship. As used in this ordinance, a variance is authorized only for area and size of structure or size of yards and open spaces; establishment or expansion of a use otherwise prohibited shall not be allowed by variance, nor shall a variance be granted because of the presence of nonconformities in the zoning district or uses in an adjoining district or because of conditions created by the landowner. All required setbacks are eligible for variances within the provisions of this Ordinance.

Vehicle - Any new or used automobile, truck, truck tractor, motorcycle, motor home, trailer, semi trailer, or travel trailer of the type and kind required to be titled and registered under Chapters 32-3 and 32-5 of SDCL, except manufactured homes, mobile homes, mopeds or snowmobiles.

Vehicle Dealer - Any person who, for commission or with intent to make a profit or gain, sells, exchanges, rents with the option to purchase, offers or attempts to negotiate a sale or exchange new, or new and used vehicles, or who is engaged wholly or in part in the business of selling new, or new and used vehicles.

Wind Energy System or WES – A commonly owned and/or managed integrated system that converts wind movement into electricity. All of the following are encompassed in this definition of system:

a) Tower or multiple towers, including foundations;
b) Generator(s);
c) Blades;
d) Power collection systems, including pad mount transformers;
e) Access roads, meteorological towers, on-site electric substation, control building and other ancillary equipment and facilities. ***(Amended 11/3/15)***

Yard - An open space at grade, other than a court or plaza, between a structure and the adjacent lot lines, unoccupied and unobstructed by any portion of a structure from the ground upward. All yards shall be measured from the property line or road right-of-way where applicable.

Yard, Front - An open, unoccupied space on a lot facing a street and extending across the front of the lot between the side lot lines. Measured from the road right-of-way to the structure.

Yard, Side - An open, unoccupied space on the same lot with a building situated between the building and sideline of the lot and extending through from the front yard to the required rear yard. Any lot line not the rear line or a front line shall be deemed a sideline.

Yard, Rear - An open, unoccupied space extending across the rear of a lot from one side lot line to the other side lot line.

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ARTICLE 1

JURISDICTION

Section 101 General

This Ordinance shall be known and shall be cited and referred to as "The Zoning Ordinance of Bon Homme County, South Dakota", to the same effect as if the full title were stated.

Section 103 Jurisdiction

The provisions of this Ordinance shall apply within the unincorporated areas of Bon Homme County, South Dakota, excluding the incorporated communities of Avon, Scotland, Springfield, Tabor, and Tyndall, as established on the map entitled "The Official Zoning Map of Bon Homme County, South Dakota".

Section 105 Provisions of this Ordinance Declared to the Minimum Requirements

In their interpretation and application, the provisions of this Ordinance shall be held to be minimum requirements, adopted for the promotion of the public health, safety, morals, or general welfare. Whenever the provisions of this Ordinance require a greater width or size of yards, courts or other spaces, or require a greater percentage of lot to be left unoccupied, or impose other higher standards than are required, in any other Ordinance, the provisions of this Ordinance shall govern. Wherever the provisions of any other ordinance require a greater width or size of yards, courts, or other open spaces, or require a greater percentage of lot to be left unoccupied, or impose other higher standards than are required by the provisions of this Ordinance, the provisions of such Ordinance shall govern.

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ARTICLE 2

APPLICATION OF DISTRICT REGULATIONS

Section 201 General

The regulations, set forth by this Ordinance within each district, shall be minimum regulations and shall apply uniformly to each class or kind of structure or land, except as hereinafter provided.

Section 203 Zoning Affects Every Building and Use

No building, structure, or land shall hereafter be used or occupied, and no building or structure or part thereof shall hereafter be erected, constructed, reconstructed, moved, or structurally altered except in conformity with all of the regulations herein specified for the district in which it is located.

Section 205 Performance Standards

No building or other structure shall hereafter be erected or altered, without obtaining a permit, to:

1. accommodate or house a greater number of families;
2. occupy a greater area of the lot; or
3. have narrower or smaller rear yards, front yards, side yards, or other open spaces.

Section 207 Yard and Lot Reduction Prohibited

No yard or lot existing at the time of passage of this Ordinance shall be reduced in dimensions or area below the minimum requirements set forth herein. Yards or lots created after the effective date of this Ordinance shall meet at least the minimum requirements established by this Ordinance.

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ARTICLE 3

ESTABLISHMENT OF DISTRICTS

Section 301 Districts Created

For the purpose of this Ordinance, there are hereby created four (4) types of districts by which the jurisdictional area defined in Section 103 shall be divided.

AG - Agricultural

RR - Rural Residential

PTR - Platted Town Site Residential

RC - Rural Commercial

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ARTICLE 4

OFFICIAL ZONING MAP AND BOUNDARY INTERPRETATION

Section 401 General

The County is hereby divided into zones, or districts, as shown on the Official Zoning Map, which, together with all explanatory matter thereon, is hereby adopted by reference and declared to be a part of this Ordinance. The Official Zoning Map shall be identified by the signature of the Chairman of the County Commissioners, attested by the Auditor, and bearing the seal of the County, under the following words: "This is to certify that this is the Official Zoning Map referred to in Section 401 of Ordinance No. 99-1 of Bon Homme County, South Dakota, as amended" together with the date of the adoption of this Ordinance.

Section 403 Zoning Map Changes

If, in accordance with the provisions of this Ordinance, changes are made in the district boundaries or other matter portrayed on the Official Zoning Map, such changes shall be entered on the Official Zoning Map promptly after the amendment has been approved by the County Commissioners, with an entry on the Official Zoning Map as follows: "on [date], by official action of the Bon Homme County Commission, the following [change] changes were made in the Official Zoning Map: [brief description of nature of change]," which entry shall be signed by the Chairman of the Commission and attested by the Auditor. No amendment to this Ordinance which involves matters portrayed on the Official Zoning Map shall become effective until after such change and entry has been made on said map.

No changes of any nature shall be made in the Official Zoning Map or matters shown thereon except in conformity with the procedures set forth in this Ordinance.

Any unauthorized change of whatever kind by any person or persons shall be considered a violation of this Ordinance and punishable as provided under Section 1503.

Regardless of the existence of purported copies of the Official Zoning Map which may, from time to time, be made or published, the Official Zoning Map which shall be located in the office of the Zoning Administrator shall be the final authority as to the current zoning status of land and water areas, buildings, and other structures in the County.

Section 405 Zoning Map Replacement

In the event that the Official Zoning Map becomes damaged, destroyed, lost or difficult to interpret because of the nature or number of changes and additions, the Bon Homme County Commission may, by resolution, adopt a new Official Zoning Map, which shall supersede the prior Official Zoning Map. The new Official Zoning Map may correct drafting or other errors or omissions in the prior Official Zoning Map, but no such correction shall have the effect of amending the original Official Zoning Map or any subsequent amendment thereof.

The new Official Zoning Map shall be identified by the signature of the Chairman of the County Commission, attested by the Auditor, and bearing the seal of the County, under the following words:

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"This is to certify that this Official Zoning Map supersedes and replaces the Official Zoning Map adopted [date of adoption of map being replaced] as part of Ordinance No. 99-1 as amended of Bon Homme County, South Dakota."

Unless the prior Official Zoning Map has been lost, or has been totally destroyed, the prior map or any significant parts thereof remaining, shall be preserved, together with all available records pertaining to its adoption or amendment.

Section 407 Rules for Interpretation of District Boundaries

Where uncertainty exists as to the boundaries of districts as shown on the Official Zoning Map, the following rules shall apply:

1. Boundaries indicated as approximately following the center lines of streets, highways, or alleys shall be construed to follow such center lines;

2. Boundaries indicated as approximately following platted lot lines shall be construed as following such lot lines;

3. Boundaries indicated as approximately following city limits shall be construed as following such city limits;

4. Boundaries indicated as following railroad lines shall be construed to be midway between the main tracks;

5. Boundaries indicated as following shore lines shall be construed to follow such shore lines, and in the event of change in the shore line shall be construed as moving with the actual shore line; boundaries indicated as approximately following the center line of streams, rivers, canals, lakes, or other bodies of water shall be construed to follow such center lines;

6. Boundaries indicated as parallel to or extensions of features indicated in subsections 1 through 5 above shall be so construed. The scale of the map shall determine distances not specifically indicated on the Official Zoning Map; and

7. Where physical or cultural features existing on the ground are at variance with those shown on the Official Zoning Map, or in other circumstances not covered by subsections 1 through 6 above, the Planning Commission shall interpret the district boundaries.

ARTICLE 5

AGRICULTURAL DISTRICT (AG)

Section 501 Intent

The intent of Agricultural Districts (AG) is to protect agricultural lands and lands consisting of natural growth from incompatible land uses in order to preserve land best suited to agricultural uses and land in which the natural environment should be continued and to limit residential, commercial, and industrial development to those areas where they are best suited for reasons of practicality and service delivery.

Section 503 Permitted Principal Uses and Structures

The following principal uses and structures shall be permitted in an Agricultural District (AG):

1. agriculture;
2. farm;
3. ranch;
4. orchard;
5. farm occupations;
6. public parks and public recreational areas;
7. farm buildings;
8. farm drainage and irrigation systems, flood control and watershed structures and erosion control devices meeting all county, state, and soil conservation district regulations;
9. manufactured homes;
10. historic sites;
11. veterinary services and kennels;
12. off-site and on-site signs;
13. cemeteries;
14. schools public and private;
15. campgrounds;
16. single-family dwellings;
17. additional farm dwellings;
18. churches;
19. rodeo grounds and arenas;
20. shelterbelts; and
21. stock dams.

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Section 505 Permitted Accessory Uses and Structures

The following accessory uses and structures shall be permitted in an Agricultural District (AG):

1. roadside stands for sales of farm products, fish bait, and other approved products;
2. home occupations;
3. professional offices; and
4. accessory uses, not specifically regulated by ordinance and structures customarily incidental to permitted uses and structures when established within the space limits of this district.

Section 507 Conditional Uses

After the provisions of this Ordinance relating to conditional uses have been fulfilled, the Board of Adjustment may permit as conditional uses in an Agricultural District (AG):

1. utility substations, television, radio, and telephone relay stations;
2. airports;
3. automobile and equipment sales;
4. fairgrounds, race tracks, and amusement parks;
5. golf courses, country clubs, and golf driving ranges;
6. amphitheaters, stadiums, arenas, and fieldhouses;
7. go-cart tracks, riding stables, playfields, athletic fields, bowling alleys, swimming pools, permanent automobile parking;
8. agricultural product processing facilities;
9. grain elevators;
10. municipal sewage disposal and/or treatment sites, animal feeding operation lagoons and holding facilities;
11. commercial trucking terminals;
12. sales and auction yards and barns;
13. private or commercial outdoor recreation areas;
14. sanitary landfills and similar facilities;
15. wildlife and game production areas;
16. fireworks stands;
17. animal feeding operations;
18. bed and breakfast commercial operations;
19. game farms;
20. private shooting preserves;
21. game lodges;

22. extraction of sand, gravel, or minerals provided such uses meet requirements for conducting surface mining activities of SDCL 45-6B;
23. salvage yards/junk yards; and
24. any facility engaged in the manufacture, wholesale distribution, retail sale or storage of flammable or combustible liquids, or hazardous material.

Section 509 Classification of Unlisted Uses

In order to insure that the zoning ordinance will permit all similar uses in each district, the Board of Adjustment, upon its own initiative or upon written application, shall determine whether a use not specifically listed as a permitted, accessory or conditional use in a District shall be deemed a permitted, accessory or conditional use in one or more districts on the basis of similarity to uses specifically listed.

Section 511 Prohibited Uses and Structures

All uses and structures which are not specifically permitted as principal, accessory or conditional uses or approved as such within the provisions of Section 509 shall be prohibited from an Agricultural District (AG).

Section 513 Minimum Lot Requirements

1. The minimum lot area per single-family dwelling unit, manufactured or modular homes shall be five (5) acres.

2. The minimum lot frontage shall be two hundred and fifty (250) feet.

3. An additional dwelling unit may be allowed if they are to be occupied by other members of the family farm unit, the Board of Adjustment may reduce the required area following the procedures of a variance.

4. The Zoning Administrator may allow construction of single and multi-family dwelling units not in conformance with this provision only on those lands organized as a 501(d), non-profit religious and apostolic associations as described in the United States Tax Code. Prior to issuance of a building permit or permission to proceed said entity shall file the Articles of Incorporation and other requested documentation with the Zoning Administrator. Construction activities carried on under this provision shall be in conformance with all other provisions of this ordinance.

Section 515 Minimum Yard Requirements for Dwellings, Manufactured or Modular Homes

All yards must meet the following criteria as measured from the lot lines. This Section shall apply to all buildings and structures, including but not limited to decks, patios, and garages:

1. There shall be a front yard of not less than a depth of seventy-five (75) feet.

2. There shall be a rear yard of not less than a depth of twenty (25) feet.

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3. There shall be two (2) side yards, each of which shall not be less than twenty-five (25) feet.

4. The Zoning Administrator may allow construction of single and multi-family dwelling units not in conformance with this provision only on those lands organized as a 501(d); non-profit religious and apostolic associations as described in the United States Tax Code. Prior to issuance of a building permit or permission to proceed said entity shall file the Articles of Incorporation and other requested documentation with the Zoning Administrator. Construction activities carried on under this provision shall be in conformance with all other provisions of this ordinance.

Section 517 Prohibition of View Obstruction

1. There shall be no obstruction, such as buildings, structures, grain bins, baled or stacked agricultural products, large rocks or rock piles, dead plant material, volunteer trees, and shelter belts that may cause view obstruction, snow build-up or safety hazards within seventy five (75) feet of the road right-of-way between the dates of November 1 and April 1.

2. The purpose of this Section is to keep the right-of-ways free and clear of snow build-up and, further, to promote traffic safety along road rights-of-way and at intersections.

Section 519 Animal Feeding Operations Performance Standards

1. Animal Feeding Operations shall submit animal waste management system plans and specifications for review and approval prior to construction, and a Notice of Completion for a Certificate of Compliance, after construction, to the South Dakota Department of Environment and Natural Resources.

2. Prior to construction, such facilities shall obtain a storm water permit for construction activities from the South Dakota Department of Environment and Natural Resources. The storm water plan required by the permit must be developed and implemented upon the start of construction.

3. All animal feeding operation's confinement and waste facilities shall comply with the following setbacks;

Public Wells	1,000 feet
Private Wells	250 feet
Operators Well	150 feet
Property Lines delineating a change in ownership	300 feet
Road Right-of-Ways	300 feet
Lakes, Rivers, Streams Classified as Fisheries	500 feet

4. Applicants must present a nutrient management plan, with the initial application documents, which will assure offensive odors, and runoff will be kept to a minimum.

   Examples of such management shall include at least:

   a. Proposed maintenance of holding ponds.

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b. Nutrient transportation equipment free of leaks or spillage hazards.

c. Land application process and/or methods

d. Legal description and map of area to be utilized for nutrient application.

5. Animal waste facilities shall be located no closer than one (1) mile from any incorporated municipality or rural residential district.

6. Animal waste facilities shall be located no closer than one (1) mile from any residential dwelling, one dwelling unit is allowed on the facility site. The owner of a residential dwelling may request the Board of Adjustment to review the facility and the Board may, by variance, waive or decrease the required separation distance. An easement, approved by the States Attorney must then be recorded with the County Register of Deeds in order that any future owners can be informed.

7. Animal waste shall be transported no further than five (5) miles from the point of origination for land application.

8. Animal Feeding Operations shall have a minimum lot size of five (5) acres.

9. The Zoning Administrator will automatically transfer a conditional use permit for all land approved as a conditional use for the purpose of operating an Animal Feeding Operation if:

   a. The current owner notifies the Zoning Administrator and Secretary of the Department of Environment and Natural Resources at least thirty (30) days in advance of the proposed transfer date;

   b. The notice includes a written agreement between the existing and new owners containing a specific date for transfer of permit responsibility, coverage and liability between them; and

   c. The new owner or operator submits a Certification of Applicant Form to the County and DENR.

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ARTICLE 6

RURAL RESIDENTIAL DISTRICT (RR)

Section 601 Intent

The intent of Rural Residential Districts (RR) is to provide for residential uses of varying types and other compatible uses in a pleasant and stable environment.

Section 603 Permitted Principal Uses and Structures

The following principal uses and structures shall be permitted in a Rural Residential District (RR):

1. single-family dwellings;
2. multi-family dwellings;
3. manufactured homes;
4. modular homes;
5. horticulture;
6. churches, synagogues, and temples;
7. nursery, primary, intermediate, secondary schools and day care facilities;
8. public recreational and park facilities;
9. cemeteries;
10. utility substations;
11. long term care facilities;
12. medical and other health facilities; and
13. governmental services.

Section 605 Permitted Accessory Uses and Structures

1. home occupations and professional offices; and
2. accessory uses and structures normally appurtenant to the permitted uses and structures when established within space limits of this district.

Section 607 Conditional Uses

After the provisions of this Ordinance, relating to conditional uses have been fulfilled, the Board of Adjustment may permit as conditional uses in a Rural Residential District (RR):

1. convenience stores;
2. colleges and universities;

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3. golf courses, fairgrounds, rodeo grounds, and country clubs;
4. campgrounds;
5. hobby farms;
6. retail sales;
7. automobile service stations; and
8. game lodges.

Section 609 Classification of Unlisted Uses

In order to insure that the zoning ordinance will permit all similar uses in each district, the Board of Adjustment, upon its own initiative or upon written application, shall determine whether a use not specifically listed as a permitted, accessory or conditional use in a District shall be deemed a permitted, accessory or conditional use in one or more districts on the basis of similarity to uses specifically listed.

Section 611 Prohibited Uses and Structures

All uses and structures which are not specifically permitted as principal, accessory or conditional uses or approved as such within the provisions of Section 609 shall be prohibited from Rural Residential Districts (RR).

Section 613 Minimum Lot Requirements

1. The minimum lot area shall be one acre for a single-family dwelling unit, manufactured or modular homes;

2. The minimum lot area for a multi-family dwelling unit shall be ten thousand (10,000) square feet per unit; and

3. The minimum lot width shall be one hundred fifty (150) feet.

Section 615 Minimum Yard Requirements

All yards must meet the following criteria as measured from the lot lines. This Section shall apply to all buildings and structures, including but not limited to decks, patios, and garages:

1. There shall be a front yard of not less than a depth of seventy-five (75) feet;

2. There shall be a rear yard of not less than a depth of twenty-five (25) feet; and

3. Each side yard shall not be less than twenty-five (25) feet

ARTICLE 7

PLATTED TOWN SITE RESIDENTIAL (PTR)

Section 701 Intent

The intent of Platted Town Site Residential Districts (PTR) is to provide for residential uses of all currently platted property within unincorporated town sites, such as the Apple Tree, Bon Homme, Dempster's Cove, and Running Water, and other compatible uses in a pleasant and stable environment.

Section 703 Permitted Principal Uses and Structures

The following principal uses and structures shall be permitted in a Platted Town Site Residential District (PTR):

1. single-family dwellings;
2. multi-family dwellings;
3. manufactured homes;
4. modular homes;
5. horticulture;
6. churches, synagogues, and temples;
7. nursery, primary, intermediate, secondary schools and day care facilities;
8. public recreational and park facilities;
9. cemeteries;
10. utility substations;
11. convalescent, nursing, and rest homes;
12. medical and other health facilities;
13. governmental services; and
14. game lodges.

Section 705 Permitted Accessory Uses and Structures

1. home occupations and professional offices; and
2. accessory uses and structures normally appurtenant to the permitted uses and structures when established within space limits of this district.

Section 707 Conditional Uses

After the provisions of this Ordinance, relating to exceptions have been fulfilled, the Planning Commission may permit as exceptions in Platted Town Site Residential Districts (PTR):

1. convenience stores;

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2. colleges and universities;
3. golf courses, fairgrounds, rodeo grounds, and country clubs;
4. campgrounds;
5. hobby farms;
6. retail sales;
7. automobile service stations; and
8. grain elevators.

Section 709 Classification of Unlisted Uses

In order to insure that the zoning ordinance will permit all similar uses in each district, the Board of Adjustment, upon its own initiative or upon written application, shall determine whether a use not specifically listed as a permitted, accessory or conditional use in a District shall be deemed a permitted, accessory or conditional use in one or more districts on the basis of similarity to uses specifically listed.

Section 711 Prohibited Uses and Structures

All uses and structures which are not specifically permitted as principal, accessory or conditional uses or approved as such within the provisions of Section 709 shall be prohibited from Platted Town Site Residential Districts (PTR).

Section 713 Minimum Lot Requirements

1. The minimum lot area shall be two thousand five hundred (2,500) square feet for single family dwelling, manufactured or modular homes;

2. The minimum lot width shall be twenty-five (25) feet; and

3. The minimum lot depth shall be one hundred (100) feet.

Section 715 Minimum Yard Requirements

All yards must meet the following criteria as measured from the lot lines. This Section shall apply to all buildings and structures, including but not limited to decks, patios, and garages:

1. There shall be a front yard of not less than a depth of twenty-five (25) feet;

2. There shall be a rear yard of not less than a depth of five (5) feet; and

3. Each side yard shall not be less than five (5) feet.

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ARTICLE 8

RURAL COMMERCIAL DISTRICT (RC)

Section 801 Intent

The intent of the Rural Commercial Districts (RC) is to provide commercial areas for those establishments which can function most satisfactorily in an area directly related to a major vehicular circulation route due to the nature of the merchandise handled and the display space required, particularly items requiring expansive display area such as motor vehicles, trailers, and farm implements; the method of transport required of the purchaser for the merchandise handled, particularly goods customarily traded in bulk such as lumber or feed requiring access for the customer to the sales area; primary dependence upon vehicular, as opposed to pedestrian, access such as drive-in facilities and all types of automotive and farm implement services; or the clientele toward which the establishments are primarily oriented.

Section 803 Permitted Principal Uses and Structures

The following principal uses and structures shall be permitted in a Rural Commercial District (RC):

1. retail sales;
2. wholesale sales;
3. funeral and crematory services and supplies;
4. agriculture;
5. farm products warehousing and storage;
6. refrigerated warehousing;
7. household goods warehousing and storage;
8. general warehousing and storage;
9. automobile and machinery sales, repair and services;
10. veterinary services;
11. contract construction services;
12. bus garaging and equipment maintenance;
13. motor freight terminals, garaging, maintenance;
14. libraries; museums, art galleries; planetaria; aquariums; historic and monument sites; auditoriums; exhibition halls; and arcades;
15. miniature golf, gymnasiums and athletic clubs, swimming pools, tennis courts, ice skating, roller skating;
16. parks;
17. theaters; stadiums; drive-in movies; arenas and field houses; race tracks; fairgrounds; amusement parks, golf driving ranges; go-cart tracks; golf courses and country clubs; riding stables; playfields and athletic fields; bowling; and swimming pools;

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18. communication and utility uses;
19. automobile-machinery service stations;
20. motels; and
21. off-site and on-site signs.

Section 805 Permitted Accessory Uses and Structures

The following accessory uses and structures shall be permitted in Rural Commercial Districts (RC):

Accessory uses normally appurtenant to the permitted principal uses and structures when established in conformance within the space limits of this district.

Section 808 Conditional Uses

After the provisions of this resolution relating to conditional uses have been fulfilled, the Board of Adjustment may permit as conditional uses in the Rural Commercial Districts (RC):

1. other trade and service uses which are similar to the permitted principal uses and which are in harmony with the intent of this district;
2. campgrounds;
3. any facility engaged in the manufacture, wholesale distribution, retail sale or storage of flammable or combustible liquids, or hazardous material; and
4. grain elevators.

Section 809 Classification of Unlisted Uses

In order to insure that the zoning ordinance will permit all similar uses in each district, the Board of Adjustment, upon its own initiative or upon written application, shall determine whether a use not specifically listed as a permitted, accessory or conditional use in a District shall be deemed a permitted, accessory or conditional use in one or more districts on the basis of similarity to uses specifically listed.

Section 811 Prohibited Uses and Structures

All uses and structures which are not specifically permitted as principal, accessory or conditional uses or approved as such within the provisions of Section 809 shall be prohibited from Rural Commercial Districts (RC).

Section 813 Minimum Lot Requirements

1. The minimum lot area shall be one (1) acre.

2. The minimum lot width shall be one hundred and fifty (150) feet.

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Section 815 Minimum Yard Requirements

1. There shall be a front yard of not less than a depth of seventy-five (75) feet;
2. There shall be a rear yard of not less than a depth of twenty-five (25) feet; and
3. Each side yard shall be not less than twenty-five (25) feet.

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ARTICLE 9

SUPPLEMENTARY DISTRICT REGULATIONS

Section 901 Accessory Buildings

No accessory building shall be erected in any required yard area and no separate accessory building shall be erected within five (5) feet of any other building.

Section 903 Erection of More than One Principal Structure on a Lot

In any district, more than one structure, housing a permitted or permissible principal use, may be erected on a single lot, provided, that yard and other requirements of this Ordinance shall be met for each structure as though it were on an individual lot.

Section 905 Shelterbelts

All shelterbelts shall be seventy-five (75) feet from the road right-of-way to the first row of trees.

Section 907 Recording of Conditions

All zoning agreements including conditions prescribed by the Board of Adjustment must be recorded at the Register of Deeds Office prior to issuance of a building permit.

Section 909 Signs

All land lying within one (1) mile of an incorporated municipality and adjoining a designated primary roadway or lying on the same side of the road and within one (1) mile of a currently established business located within the rural areas shall be designated rural commercial for the sole purpose of the construction and placement of signs, displays, and devices. Placement of said signs shall comply with SDCL 31-29 and ARSD 70:04:03.

Section 911 Right-of Way Obstructions

It shall be unlawful for any person to place any obstruction in a road right-of-way without prior written permission from the Bon Homme County Highway Superintendent. Said obstacles may include but are not limited to signs, fences, and trees. Temporary obstacles may be placed within six (6) feet of the traveled surface upon written authorization of the Highway Superintendent.

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ARTICLE 10

ADMINISTRATIVE PROCEDURE AND ENFORCEMENT

Section 1001 Bon Homme County Zoning Administrator

An administrative official who shall be known as the Zoning Administrator and who shall be designated by the Bon Homme County Commission shall administer and enforce this ordinance. They may be provided with the assistance of such other persons as the County Commission may direct.

If the Zoning Administrator shall find that any of the provisions of this Ordinance are being violated, they shall notify in writing the person responsible for such violations, indicating the nature of the violation and ordering the action necessary to correct it. They shall order discontinuance of illegal use of land, buildings or structures; removal of illegal buildings or structures or of illegal additions, alterations, or structural changes; discontinuance of any illegal work being done; or shall take any other action authorized by the Ordinance to insure compliance with or to prevent violation to its provisions.

Section 1003 Right of Entry

Whenever necessary to make an inspection to enforce any of the provisions of this ordinance, or whenever the Zoning Administrator or an authorized representative has reasonable cause to believe that there exists in any building or upon any premises an ordinance violation, the Zoning Administrator or an authorized representative may enter such building or premises at all reasonable times to inspect the same or to perform any duty imposed upon the Zoning Administrator by this ordinance, provided that if such building or premises be occupied, they shall first present proper credentials and request entry; and if such building or premises be unoccupied, they shall first make an reasonable effort to locate the owner or other persons having charge or control of the building or premises and request entry. If such entry is refused, the Zoning Administrator or an authorized representative shall have recourse to every remedy provided by law to secure entry.

When the Zoning Administrator or an authorized representative shall have first obtained a proper inspection warrant or other remedy provided by law to secure entry, no owner or occupant or any other persons having charge, care or control of any building or premises shall fail or neglect, after proper request is made as herein provided, to promptly permit entry therein by the Zoning Administrator or an authorized representative for the purpose of inspection and examination pursuant to this ordinance.

Section 1005 Bon Homme County Planning Commission

The Bon Homme County Commission shall appoint a Planning Commission of five (5) members; the total membership of which shall be an uneven number and at least one (1) member shall be a county commissioner. The term of each of the appointed members of the Planning Commission shall be for three to five years. When the Planning Commission is first appointed the lengths of the terms shall be varied so that no more than one-third (1/3) of the terms shall expire in the same year. Meetings shall be scheduled and held at the call of the Chairman, at such other times as the Planning Commission may determine. The Chairman, or in their absence, the Acting Chairman, may administer oaths and compel the attendance of witnesses. All meetings shall be open to the public.

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Upon notification of a proposed revision, modification, change or amendment to the zoning ordinance or any part thereof the Planning Commission shall schedule a public hearing. Said public hearing shall not be less than ten (10) days after notice has been published in the County's legal newspapers. Any person may appear and request or protest the proposed change.

The Planning Commission shall keep a record of all proceedings, including minutes, showing the vote of each member upon each question, or if absent or failure to vote indicating such fact, and shall keep records of its examinations and other official actions, all of which shall be a public record and be immediately filed with the Zoning Administrator. The Planning Commission shall adopt from time to time, subject to the approval of the County Commission, rules and regulations, as it may deem necessary for the conduct of its affairs and to carry the appropriate provisions of this Ordinance into effect.

Section 1007 Bon Homme County Board of Adjustment

The Bon Homme County Planning Commission shall serve as the Board of Adjustment. The Board of Adjustment is hereby designated to hear all requests for variances, conditional uses and zoning appeals. The Board of Adjustment may, in specific cases to avoid unwarranted hardship which constitutes an unreasonable deprivation of use as distinguished from the mere grant of a privilege, make upon an affirmative vote of two-thirds (2/3) of the full membership of the Board of Adjustment, conditional uses or grant variances to the terms of the regulations or controls, subject to appropriate conditions or safeguards being adopted by the Board of County Commissioners.

The Zoning Administrator shall act as secretary to the Board of Adjustment when acting in zoning cases, but shall take no part in the deliberations. Meetings of the Board of Adjustment acting in zoning cases shall be held at the call of the Chairperson and at such other times, as the Board shall determine. Such Chairperson, or in his/her absence, the Acting Chairperson, may administer oaths and compel the attendance of witnesses.

All meetings of the Board of Adjustment shall be open to the public. The Board, acting in zoning appeal cases, shall keep minutes of its proceedings, showing the vote of each member upon each question, or if absent or failing to vote, indicating such fact, and shall keep records of its examinations and other official actions, all of which shall be immediately filed in the office of the Zoning Administrator and shall be a public record. The Board of Adjustment, acting in zoning appeals cases, shall adopt from time to time, subject to the approval of the County Commission, such rules and regulations as it may deem necessary to carry the appropriate provisions of this Ordinance into effect.

Section 1009 Bon Homme County Commission

The Bon Homme County Commission may amend, supplement, change, modify, or repeal any regulation, restriction, boundary, or enforcement provision established in the comprehensive plan or adjuncts thereto. The County Commission shall forward a copy of the proposed changes to the Planning Commission for public review. Upon receipt of the comments from the Planning Commission the County Commission shall publish a notice of public hearing no less than ten (10) days in advance in the County's legal newspapers. The Board of County Commissioners shall thereafter either adopt or reject such amendment, supplement, change, modification, or repeal. If adopted the Board of County Commissioners shall direct the Planning Commission to prepare a summary of the action. Upon completion of the summary the States Attorney shall review the same and direct the County Auditor to have said summary published once in the legal newspapers.

Section 1011 Building Permits Required

No building or other structure shall be erected, moved, added to, removed, demolished, burned, or use changed without a permit issued by the Zoning Administrator. No building permit shall be issued by the Zoning Administrator except in conformity with the provisions of this Ordinance unless they received a written order from the Board of Adjustment in the form of an administrative review, conditional use, or variance as provided by this Ordinance.

Concrete flatwork of less than five hundred (500) square feet shall be exempt from obtaining a building permit provided such work is done at or immediately above grade.

Section 1013 Applications for Building Permits, Conditional Uses and Variances

All applications for building permits, conditional uses and variances must be signed or approved in writing by the owner of record. In the event the owner of record has a binding purchase agreement contingent on the approval of the application, the potential purchaser may submit and sign all documents required for application. All building permit, conditional use and variance applications shall be accompanied by a site plan including but not limited to the following items; drawn to scale, including a north arrow, showing the property lines, actual dimensions and shape of the lot to be built upon, the exact sizes and locations on the lot of buildings already existing, if any; and the location and dimensions of the proposed building or alteration. Refer to document entitled Site Plan Requirements for a detailed example of site plan requirements.

The application shall include such other information as may be lawfully required by the Zoning Administrator, including: existing or proposed building or alterations; existing or proposed uses of the building and land; the number of families, housekeeping units, rental units, or animal units the building is designed to accommodate; conditions existing on the lot; and such other matters as may be necessary to determine conformance with, and provide for the enforcement of, this Ordinance.

One copy of the plans shall be returned to the applicant by the Zoning Administrator after they shall have marked such copy either as approved or disapproved and attested to the same by their signature on such copy. If a building permit is refused, the Zoning Administrator shall state the reasons for such refusal in writing. The Zoning Administrator shall retain the original and one copy of the plans, similarly marked. The issuance of a building permit, shall, in no case, be construed as waiving any provisions of this Ordinance.

Section 1015 Expiration of Building Permit, Conditional Uses and Variances

If the work described in any building permit, conditional use or variance application has not begun within one hundred and eighty (180) days or has not been substantially completed within two (2) years of the date of issuance thereof, said permit shall expire; it shall be canceled by the Zoning Administrator and written notice thereof shall be given to the persons affected. The notice shall state that further work as described in the canceled permit or application shall not proceed unless, and until, a new building permit, conditional use or variance application has been approved and all required fees have been paid.

Section 1017 Construction and Use to be as Provided in Application, Plans, Permits, and Application for Zoning Compliance

Building permits issued on the basis of plans and applications approved by the Zoning Administrator authorize only the use, arrangement, and construction set forth in such approved plans and applications,

and no other use, arrangement or construction. Use arrangement, or construction at variance with that authorized shall be deemed a violation of this Ordinance, and punishable as provided by Section 1503 of this ordinance.

Section 1019 Schedule of Fees, Charges, and Expenses

The Bon Homme County Commission shall establish a schedule of fees, charges, and expenses and a collection procedure for variances, conditional uses amendments, appeals and other matters pertaining to this Ordinance. The schedule of fees shall be posted in the office of the Zoning Administrator and may be altered or amended only by the Bon Homme County Commission. Until all application fees, charges, and expenses have been paid in full, no action shall be taken on any application or appeal.

Section 1021 Building Permit in a Conspicuous Place

All building permits issued by the Zoning Administrator must be placed in a conspicuous location on the building site for the duration of the construction of work described.

Section 1023 Bad Actor Legislation

The Bon Homme County Commission may reject an application for any permit filed for a variance, conditional use or otherwise for the reasons and on the grounds set forth in SDCL 1-40-27, as revised and amended. Such rejection shall be based upon a specific finding by the Commission that the applicant has engaged in the activity identified in the aforesaid statute. The burden on the Commission to make the specific finding provided for herein shall be by a preponderance of the evidence.

ARTICLE 11

BOARD OF ADJUSTMENT
APPEALS, VARIANCES AND CONDITIONAL USES

Section 1101 Members, Terms, Meetings, Rules

The Bon Homme County Planning Commission shall serve as the Board of Adjustment. The Board of Adjustment is hereby designated to hear all requests for variances, conditional uses, and zoning appeals. The Zoning Administrator shall act as secretary to the Board of Adjustment when acting in zoning cases, but shall take no part in the deliberations. Meetings of the Board of Adjustment shall be held at the call of the Chairperson and at such other times as the Board shall determine. Such Chairperson, or in his/her absence, the Acting Chairperson, may administer oaths and compel the attendance of witnesses.

All meetings of the Board of Adjustment shall be open to the public. The Board shall keep minutes of its proceedings, showing the vote of each member upon each question, or if absent or failing to vote, indicating such fact, and shall keep records of its examinations and other official actions, all of which shall be immediately filed in the office of the Zoning Administrator and shall be a public record. The Board of Adjustment shall adopt from time to time, subject to the approval of the County Commission, such rules and regulations, as it may deem necessary to carry the appropriate provisions of this Ordinance into effect.

Section 1103 Appeals to Board of Adjustment, Record of Appeals, Hearing, and Stays

Any decision rendered by the Zoning Administrator may be appealed to the Board of Adjustment. An appeal stays all proceedings in furtherance of the action appealed from, unless the officer from whom the appeal is taken certifies to the Board of Adjustment after the notice of appeal shall have been filed with them, that by reason of facts stated in the certificate a stay would, in their opinion, cause imminent peril to life or property. In such case, proceedings shall not be stayed otherwise than by a restraining order which may be granted by the Board of Adjustment or by a court of record on application or notice to the officer for whom the appeal is taken and on due cause shown.

Section 1105 Board of Adjustment Hearings and Notice

Each session of the Board of Adjustment at which a hearing is held shall be a public meeting with notice of hearing to be published at least ten (10) days prior to the date of the hearing in the legal newspapers of the County.

Section 1107 Powers and Duties

The Board of Adjustment shall have the following powers and duties:

APPEALS:

A. The Board of Adjustment shall have the power to hear and decide appeals where it is alleged there is error in any order, requirement, decision or determination made by an administrative official or agency based on or made in the enforcement of any zoning

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regulation or any regulation relating to the location or soundness of structures or to interpret any map. An appeal will not be heard until:

1. The applicant or any other person aggrieved by the decision of an administrative official or agency shall file a written appeal with the Zoning Administrator within five working days of the decision.

2. The administrative official or agency from whom the appeal is taken shall forthwith transmit to the Board of Adjustment all the papers constituting the record upon which the action appealed was taken.

3. Written notice shall be given to the appellant seven days prior to meeting.

4. The appellant or an authorized agent shall be present at the meeting. Failure to provide a representative may constitute grounds for a denial.

5. The administrative official or agency shall present their decision to the Board of Adjustment for review.

6. The Board of Adjustment shall either uphold, overrule or amend the decision of the Zoning Administrator.

CONDITIONAL USES

B. The Board of Adjustment shall have the power to hear and decide, in accordance with the provisions of this ordinance, requests for conditional uses or for decisions upon other special questions upon which the Board of Adjustment is authorized by this ordinance to pass; to decide such questions as are involved in determining whether conditional uses should be granted; and to grant conditional uses with such conditions and safeguards as are appropriate under this ordinance, or to deny conditional uses when not in harmony with the purpose and intent of this ordinance. A conditional use shall not be granted by the Board unless and until:

1. A written application for a conditional use is submitted, indicating the section of this ordinance under which the conditional use is sought and stating the grounds on which it is requested.

2. Notice of public hearing shall be given at least ten (10) days in advance by publication in the legal newspapers of the County. The owner of the property for which conditional use is sought or his agent shall be notified by mail.

3. A notice shall be posted in a conspicuous place on or near the property upon which action is pending. Such notice shall be not less than seventeen (17) inches in height and eleven (11) inches in width with a white background and black letters not less than one (1) inch in height. Such posted notice shall be so placed upon such premises that it is easily visible from the road and shall be so posted at least seven (7) days before the date of such hearing. It shall be unlawful for any person to remove, mutilate, destroy or change such posted notice prior to such hearings.

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4. The public hearing shall be held. Any party may appear in person, or by agent or attorney.

5. The applicant or an authorized agent shall be present at the hearing. Failure to provide a representative may constitute grounds for a denial.

6. The Board of Adjustment shall make a finding that it is empowered under the section of this ordinance described in the application to grant the conditional use, grant with conditions, or deny the conditional use, and that the granting of the conditional use will not adversely affect the public interest.

7. Before any conditional use is granted, the Board of Adjustment shall make written findings certifying compliance with the specific rules governing individual conditional uses and that satisfactory provision and arrangement has been made concerning the following, where applicable:

   a. ingress and egress to property and proposed structures thereon with particular reference to automotive and pedestrian safety and convenience, traffic flow and control, and access in case of fire or catastrophe;

   b. off-street parking and loading areas where required; with particular attention to the items in (a) above and the economic, noise, glare, odor or other effects of the conditional use on adjoining properties and properties generally in the district;

   c. refuse, waste and service areas, with particular reference to the items in (a) and (b) above;

   d. utilities, with reference to locations, availability, and compatibility;

   e. screening and buffering with reference to type, dimensions, and character;

   f. signs, if any, and proposed exterior lighting with reference to glare, traffic safety, economic effect and compatibility and harmony with properties in the district;

   g. required yards and other open spaces; and

   h. general compatibility with adjacent properties and other property in the district;

VARIANCES

C. The Board of Adjustment shall have the power to hear requests for variances from this Ordinance in instances where strict enforcement would cause unnecessary hardship, and to grant such variances only when the following provisions apply:

   1. No such variance shall be authorized by the Board of Adjustment unless it finds that the strict application of the ordinance would produce undue hardship; such hardship is not shared generally by other properties in the same zoning district

and the same vicinity; the authorization of such variance will not be of substantial detriment to adjacent property and the character of the district will not be changed by the grant of the variance; and the granting of such variance is based upon reasons of demonstrable and exceptional hardship as distinguished from variations for purposes of convenience, profit, and caprice.

2. No variance shall be authorized unless the Board of Adjustment finds that the condition or situation of the property concerning or the intended use of the property concerned, or the intended use of the property is not of so general or recurring a nature as to make reasonably practicable the formulation of a general regulation to be adopted as an amendment of this ordinance.

3. A variance from the terms of this ordinance shall not be granted by the Board of Adjustment unless and until a written application for a variance is submitted demonstrating that special conditions and circumstances exist which are peculiar to the land, structure, or building involved and which are not applicable to other lands, structures, or buildings, in the same district; that literal interpretation of the provisions of this ordinance would deprive the applicant of rights commonly enjoyed by other properties in the same district under the terms of this ordinance; that the special conditions and circumstances do not result from the actions of the applicant; and that granting the variance requested will not confer on the applicant any special privilege that is denied by this ordinance to other lands, structure, or buildings in the same district.

4. No non-conforming use of neighboring lands, structures, or buildings in the same district, and no permitted or non-conforming use of lands, structures or buildings in other districts shall be considered grounds for the issuance of a variance.

5. Notice of public hearing shall be given, as in Section 1107 (B)(2), (B)(3) above; the public hearing shall be held. Any party may appear in person or by agent or by attorney; the Board of Adjustment shall make findings that the requirements of this Section have been met by the applicant for a variance; the Board shall further make a finding that the reasons set forth in the application justify the granting of the variance, and the variance is the minimum variance that will make possible the reasonable use of the land, building, or structure; the Board of Adjustment shall further make a finding that the granting of the variance will be in harmony with the general purpose and intent of this ordinance, and will not be injurious to the neighborhood, or otherwise detrimental to the public welfare.

6. The applicant or an authorized agent shall be present at the hearing. Failure to provide a representative may constitute grounds for a denial.

7. In granting any variance, the Board of Adjustment may prescribe appropriate conditions and safeguards in conformity with this ordinance. Violation of such conditions and safeguards, when made a part of the terms under which the variance is granted, shall be deemed a violation of this ordinance and punishable under Section 1503 of this ordinance.

8. Under no circumstances shall the Board of Adjustment grant a variance to allow a use not permissible under the terms of this ordinance in the district involved, or

any use expressly or by implication prohibited by the terms of this ordinance in said district.

D. The Board of Adjustment has the powers of a Zoning Administrator on Appeals and Reversing Decision of the Zoning Administrator.

In exercising the above-mentioned powers, the Board of Adjustment may reverse or affirm, wholly or partly, or may modify the order, requirement, decision or determination appealed from, and may make such order, requirement, decision, or determination as ought to be made, and to that end shall have all the powers of the officer from whom the appeal is taken.

The concurring vote of two-thirds (2/3) of the full membership of the Board of Adjustment shall be necessary to reverse any order, requirement, decision or determination of any such officer, or to decide in favor of the applicant on any matter upon which it is required to pass under this Ordinance or to effect any variation in this Ordinance.

E. Any persons, jointly or severally aggrieved by a decision of the Board of Adjustment, or any taxpayer, or any officer, department, board, or bureau of the County, may appeal to the Board of County Commissioners and by a majority vote reverse any decision of the Board of Adjustment. The applicant or any other person aggrieved by the decision of the Board of Adjustment shall file a written appeal with the County Auditor within five (5) working days of the Board of Adjustment decision. The County Auditor shall present the Board of Adjustment's decision to the Board of County Commissioners for review. Notice of the meeting shall be given as required by Section 1107 B(2) B(3). Review may be sought by a court of record of such decision, in a manner provided by the laws of the State of South Dakota.

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ARTICLE 12

DUTIES OF THE ZONING ADMINISTRATOR, BOARD OF COUNTY COMMISSIONERS AND COURTS ON MATTERS OF APPEAL

Section 1201 Duties of Zoning Administrator, Board of Adjustment, County Commission and Courts on Matters of Appeal

It is the intent of this Ordinance that all questions of interpretation and enforcement shall be first presented to the Zoning Administrator, and that such questions shall be presented to the Board of Adjustment only on appeal from the decision of the Zoning Administrator, and that such questions shall be presented to the County Commission only on appeal from the decision of the Board of Adjustment and that recourse from the decisions of the County Commission shall be to the courts as provided by law.

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ARTICLE 13

AMENDMENTS

Section 1301 Regulations

The regulations, restrictions, and boundaries set forth in this Ordinance may from time to time be amended, supplemented, changed, or repealed, provided that such modification or repeal in each instance be proposed in an Ordinance presented to the governing body for adoption in the same manner and upon the same notice as required for the adoption of the original Ordinance.

Prior to consideration of amending, supplementing, changing, modifying or repealing this Ordinance by the governing body, notice of public hearings shall be provided as follows:

1. If the proposed changes affect a particular piece of property a notice shall be posted in a conspicuous place on or near the property upon which action is pending. Such notice shall be not less than seventeen (17) inches in height and eleven (11) inches in width with a white background and black letters not less than one and one-half (1.5) inches in height. Such posted notice shall be so placed upon such premises that it is easily visible from the road and shall be so posted at least seven (7) days before the date of such hearing. It shall be unlawful for any person to remove, mutilate, destroy or change such posted notice prior to such hearings.

2. At least ten (10) days before the date of the Planning Commission hearing, the County shall have published in the County's legal newspapers a notice of the time, place, and subject matter of such hearing.

3. The Planning Commission shall hold the Public Hearing, review the proposed amendment(s) and make recommendations to the County Commission.

4. The applicant or an authorized agent shall be present at the meeting. Failure to provide a representative may constitute grounds for a denial.

5. At least ten (10) days before the date of the County Commission hearing, the County shall have published a notice of the time, place, and subject matter of such hearing in the County's legal newspapers.

6. The County Commission shall hold the Public Hearing, review the proposed amendment(s) and by Ordinance deny or pass the recommendations.

7. The applicant or an authorized agent shall be present at the meeting. Failure to provide a representative may constitute grounds for a denial.

8. If the changes are adopted the Planning Commission shall prepare a summary of the changes.

9. Once the summary is prepared the States Attorney shall review the changes and forward the changes to the County Auditor for publishing.
10. The summary of changes must be published once in the in the County's legal newspapers. The changes will take effect twenty (20) days after publication.

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ARTICLE 14

NON-CONFORMANCE

Section 1401 General

Within the districts established by this Ordinance or amendments that may later be adopted, there exists (a) lots, (b) structures, (c) uses of land and structures, and (d) characteristics of use which were lawful before this Ordinance was passed or amended, but which would be prohibited, regulated, or restricted under the terms of this Ordinance or future amendment; it is the intent to permit these nonconformities to continue until they are removed, but not to encourage their survival. It is further the intent that nonconformities shall not be enlarged upon, expanded, or extended, nor be used as grounds for adding other structures or uses prohibited elsewhere in the same district.

Nonconforming uses are declared to be incompatible with permitted uses in the districts involved. A nonconforming use of a structure, a nonconforming use of land, or a nonconforming use of structure and land in combination shall not be extended or enlarged after passage of this revised Ordinance by attachment on a building or premises of additional signs intended to be seen from off the premises, or by the addition of other uses, of a nature which would be prohibited generally in the district involved.

To avoid undue hardship, nothing in this Ordinance shall be deemed to require a change in the plans, construction, or designated use of any building on which actual construction was lawfully begun prior to the effective date of adoption or amendment of this Ordinance and upon which actual building construction has been carried on diligently. Actual construction is hereby defined to include the placing of construction materials in permanent position and fastened in a permanent manner. Where excavation or demolition or removal of an existing building has been substantially begun preparatory to rebuilding, such excavation or demolition or removal shall be deemed to be actual construction, provided that work shall be carried on diligently.

Section 1403 Nonconforming Lots of Record

In any district in which single-family dwellings are permitted, single-family dwelling and customary accessory buildings may be erected on any single lot of record at the effective date of adoption or amendment of this Ordinance, not withstanding limitations imposed by other provisions of this Ordinance. Such lots must be in separate ownership and not of continuous frontage with other lots in the same ownership. This provision shall apply even though such lots fail to meet requirements for area or width, or both, that are generally applicable in the district, provided that yard dimensions and requirements other than those applying to area or width, or both, of the lot shall conform to the regulations for the district in which such lot is located.

Variance of other yard requirements shall be obtained only through action of the Board of Adjustment.

Section 1405 Nonconforming Uses of Land or Land with Minor Structures Only

Where at the time of passage of this revised Ordinance lawful use of land exists, which would not be permitted by the regulations imposed by this Ordinance, and where such use involves no individual structure with a replacement cost exceeding one thousand (1,000) dollars, the use may be continued so long as it remains otherwise lawful, provided:

1. No such nonconforming use shall be enlarged or increased, nor extended to occupy a greater area of land than was occupied at the effective date of adoption or amendment of this Ordinance;

2. No such nonconforming use shall be moved, in whole or in part, to any portion of the lot or parcel other than that occupied by such use at the effective date of adoption or amendment of this Ordinance;

3. If any such nonconforming use of land ceases, for any reason, for a period of more than one (1) year, any subsequent use of such land shall conform to the regulations specified by this Ordinance for the district in which such land is located; and

4. No additional structure, not conforming to the requirement of this Ordinance, shall be erected in connection with such nonconforming use of land.

Section 1407 Nonconforming Structures

Where a lawful structure exists at the effective date of adoption or amendment of this Ordinance, that could not be built under the terms of this Ordinance by reason of restrictions on area, lot coverage, height, yards, its location on the lot, or other requirements concerning the structure, such structure may be continued so long as it remains otherwise lawful, subject to the following provisions:

1. No such nonconforming structure may be enlarged or altered in any way, which increases its nonconformity, but any structure or portion thereof, may be altered to decrease its nonconformity;

2. Should such nonconforming structure, or nonconforming portion of structure, be destroyed by any means, to an extent of more than seventy-five (75) percent of its replacement cost at the time of destruction, it shall not be reconstructed except in conformity with the provisions of this Ordinance; and

3. Should such structure be moved for any reason for any distance whatever, it shall thereafter conform to the regulations for the district in which it is located after it is moved.

Section 1409 Nonconforming Uses of Structures or of Structures and Premises in Combination

If the nonconforming use involving individual structures with a replacement cost of one thousand (1,000) dollars or more, or of structure and premises in combination, exists at the effective date of adoption or amendment of this Ordinance that would not be allowed in the district under the terms of this Ordinance, the nonconforming use may be continued so long as it remains otherwise lawful, subject to the following provisions:

1. No existing structure devoted to a use not permitted by this Ordinance in the district in which it is located shall be enlarged, extended, constructed, reconstructed, moved or structurally altered except in changing the use of the structure to a use permitted in the district in which it is located;

2. Any nonconforming use may be extended throughout any part of a building which was manifestly arranged or designed for such use at the time of adoption or amendment of this Ordinance, but no such use shall be extended to occupy any land outside such building;

3. If no structural alterations are made, any nonconforming use of a structure or structure and premises may, as a conditional use, be changed to another nonconforming use provided that the Board of Adjustment, either by general rule or by making findings in the specific case, shall find that the proposed use is equally appropriate or more appropriate to the district than the existing nonconforming use. In permitting such change, the Board of Adjustment may require appropriate conditions and safeguards in accordance with the provisions of this Ordinance;

4. Any structure, or structure and land in combination, in or on which a nonconforming use is superseded by a permitted use, shall thereafter conform to the regulations for the district, and the nonconforming use may not thereafter be resumed;

5. When a nonconforming use of a structure, or structure and premises in combination, is discontinued or abandoned for a period of more than one (1) year (except when government action impedes access to the premises), the structure, or structure and premises in combination, shall not thereafter be used except in conformity with the regulations of the district in which it is located; and

6. Where nonconforming use status applies to a structure and premises in combination, removal or destruction of the structure shall eliminate the nonconforming status of the land.

Section 1411 Uses Under Conditional Use Provisions are Conforming Uses

Any use, which is permitted as a conditional use in a district, under the terms of this Ordinance, shall be deemed a conforming use in such district without further action. A nonconforming use can never be allowed in a defined district without a change in the district definition or boundaries.

Permitted Principal Uses	Conditional Uses	Nonconforming
Allowed within defined district.	Allowed within defined district AFTER Board grants permission.	Never allowed within defined district without change in district definitions or boundaries.

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ARTICLE 15

VIOLATIONS, COMPLAINTS, PENALTIES, AND REMEDIES

Section 1501 Complaints Regarding Violations

Whenever a violation of this Ordinance occurs, or is alleged to have occurred, any person may file a complaint. Such complaint stating fully the causes and basis thereof shall be filed with the Zoning Administrator. The Zoning Administrator shall record properly such complaint with the Board of Adjustment and investigate and take action thereon as provided by this Ordinance.

If the Zoning Administrator shall find that any of the provisions of this Ordinance are being violated, they shall notify, in writing by certified mail with return receipt, the person responsible for such violations, indicating the nature of the violation and ordering the action necessary to correct it. The party responsible for the violation shall respond within seven (7) working days from receipt of the letter; otherwise, they will be considered in violation and punishable under Section 1503.

Section 1503 Penalties for Violations

The owner or agent of a building or premises in or upon which a violation of any provisions of this Ordinance has been committed or shall exist, or lessee or tenant of an entire building or entire premises in or upon which such violation shall exist, shall be guilty of a Class II misdemeanor and shall be punished by a fine not to exceed two hundred (200) dollars or imprisonment for not more than thirty (30) days in the County jail, or both, and in addition shall pay all costs and expenses involved in the case. Each day such violation continues shall be a separate offense.

Any architect, engineer, builder, contractor, agent or other person who commits, participates in, assists in or maintains such violation may each be found guilty of a separate offense and suffer the penalties herein provided.

In case any building or structure is erected, constructed, reconstructed, altered, repaired, converted, or maintained, or any building, structure or land is used in violation of this Ordinance, appropriate authorities of the county may institute any appropriate action or proceedings to prevent such unlawful erection, construction, reconstruction, alteration, repair, conversion, maintenance, or use; to restrain, correct or abate such violation; to prevent the occupancy of said building, structure or land; or to prevent any illegal act, conduct, business or use in or about such premises.

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ARTICLE 16

LEGAL STATUS PROVISIONS

Section 1601 Separability

Should any article, section, or provisions of this Ordinance be declared by the courts to be unconstitutional or invalid, such decision shall not affect the validity of this Ordinance as a whole, or any part thereof other than the part so declared to be unconstitutional or invalid.

Section 1603 Purpose of Sub-Titles

The sub-titles appearing in connection with the foregoing sections are inserted simply for convenience, to serve the purpose of any index and they shall be wholly disregarded by any person, officer, court or other tribunal in construing the terms and provisions of this Ordinance.

Section 1605 Repeal of Conflicting Ordinances

All ordinances or parts of resolutions in conflict with this Ordinance, or inconsistent with the provisions of this Ordinance, are hereby repealed to the extent necessary to give this Ordinance full force and effect.

Section 1607 Effective Date

This Ordinance shall take effect and be in force from and after its adoption

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ARTICLE 17

WIND ENERGY SYSTEMS (WES)

(Amended 11/3/15)

Section 1701 Intent

The intent of this ordinance is to ensure that the placement, construction and modification of a Wind Energy System (WES) facility is consistent with the Bon Homme County's land use policies, to minimize the impact of WES facilities, to establish a fair and efficient process for review and approval of applications, to assure a comprehensive review of environmental impacts of such facilities, and to protect the health, safety and welfare of the County's citizens.

Section 1703 Authority and Jurisdiction

South Dakota Codified Law 11-2-2 delegates the responsibility to the Board of County Commissioners of each county to adopt and enforce regulations designed for the purpose of promoting health, safety, and general welfare of the county.

Section 1705 Federal and State Requirements

All WES facilities shall meet or exceed standards and regulations of the Federal Aviation Administration and South Dakota State Statutes and any other agency of federal or state government with the authority to regulate WES facilities.

Section 1707 Requirements for Siting Small Wind Energy Systems

A Small Wind Energy System shall be a permitted use in all zoning districts subject to the following requirements:

a) Setbacks. The minimum setback distance between each wind turbine tower and all surrounding property lines, overhead utility or transmission lines, other wind turbine towers, electrical substations, public roads and **h*abitable residential dwellings*** shall be equal to no less than one point one (1.1) times the system height, unless written permission is granted by each affected person.

b) Access. All ground mounted electrical and control equipment shall be labeled or secured to prevent unauthorized access, and the tower shall be designed and installed so as to not provide step bolts or a ladder readily accessible to the public for a minimum height of eight (8) feet above the ground.

c) Lighting. A SWES shall not be artificially lighted unless such lighting is required by the Federal Aviation Administration.

d) Noise. SWES facilities shall not exceed forty-five (45) dBA, as measured at the closest neighboring **h*abitable residential dwelling***. The level, however, may be exceeded during short-term events such as utility outages or wind storms.

e) Shadow Flicker. When determined appropriate by the County, a Shadow Flicker Control System shall be installed upon all turbines which will cause a perceived shadow effect upon a habitable residential dwelling. Such system shall limit blade rotation at those times when shadow flicker exceeds thirty (30) minutes per day or thirty (30) hours per year at perceivable shadow flicker intensity as confirmed by the Zoning Administrator are probable.

The permittees shall submit a report of predicted shadow flicker levels at habitable residential dwellings within one and one-half miles of proposed tower locations to the Board no less than forty five (45) days prior to commencing construction.

f) Appearance, Color, Finish. The SWES shall remain painted or finished the color or finish that was originally applied by the manufacturer, unless approved in the building permit.

g) Signs. All signs, other than the manufacturer's or installer's identification, appropriate warning signs, or owner identification on a wind generator, tower, building, or other structure associated with a SWES visible from any public road shall be prohibited.

h) Code Compliance. A SWES shall comply with all applicable state construction and electrical codes, and the National Electrical Code.

i) Utility Notification. No SWES shall be installed until evidence has been given that the utility company has been informed of the customer's intent to install an interconnected customer-owned generator. Off-grid systems shall be exempt from this requirement.

Section 1709 Permit Requirements

a) A building permit shall be required for the installation of a SWES.

b) The building permit shall be accompanied by a plot plan which includes the following:

1. Property lines and physical dimensions of the property;
2. Location, dimensions, and types of existing major structures on the property;
3. Location of the proposed SWES;
4. The right-of-way of any public road that is contiguous with the property;

5. Any overhead utility lines;
6. Wind system specifications, including manufacturer and model, rotor diameter, tower height, and tower type (monopole, lattice, guyed);
7. Tower foundation blueprints or drawings;
8. Tower blueprint or drawing;
9. Proof of notification to the utility in the service territory in which the SWES is to be erected, consistent with the provisions of 5(3)(h) herein; and
10. The status of all necessary interconnection agreements or studies.

c) Expiration. A permit issued pursuant to this ordinance shall expire if:

1. The SWES is not installed and functioning within twenty-four (24) months from the date the permit is issued; or
2. The SWES is out of service or otherwise unused for a continuous 12-month period.

Section 1711 Abandonment

A SWES that is out-of-service for a continuous 12-month period will be deemed to have been abandoned. The Board may issue a Notice of Abandonment to the owner of a SWES that is deemed to have been abandoned. The owner shall have the right to respond to the Notice of Abandonment within thirty (30) days from Notice receipt date. The Board shall withdraw the Notice of Abandonment and notify the owner that the Notice has been withdrawn if the owner provides information that demonstrates the SWES has not been abandoned.

If the SWES is determined to be abandoned, the owner of the SWES shall remove the wind generator from the tower at the Owner's sole expense within three (3) months of receipt of Notice of Abandonment. If the owner fails to remove the wind generator from the tower, the Board may pursue legal action to have the wind generator removed at the owner's expense.

Section 1713 Building Permit Procedure

a) An owner shall submit an application to the Board for a building permit for a SWES. The application must be on a form approved by the Board and must be accompanied by two (2) copies of the plot plan identified.

b) The Board shall issue a permit or deny the application within one month of the date on which the application is received.

c) The Board shall issue a building permit for a SWES if the application materials show that the proposed SWES meets the requirements of this ordinance.

d) If the application is approved, the Board will return one signed copy of the application with the

permit and retain the other copy.

e) If the application is rejected, the Board will notify the applicant in writing and provide a written statement of the reason why the application was rejected. The applicant may reapply if the deficiencies specified by the Board are resolved.

f) The owner shall conspicuously post the building permit on the premises so as to be visible to the public at all times until construction or installation of the SWES is complete.

Section 1715 Violations

It is unlawful for any person to construct, install, or operate a SWES that is not in compliance with this ordinance or with any condition contained in a building permit issued pursuant to this ordinance. SWES facilities installed prior to the adoption of this ordinance are exempt.

Section 1717 Severability

The provisions of this ordinance are severable, and the invalidity of any section, subdivision, paragraph, or other part of this ordinance shall not affect the validity or effectiveness of the remainder of the ordinance.

Section 1719 Requirements for Siting Large Wind Energy Systems

A Large Wind Energy System as defined herein shall be a permitted use in all zoning districts subject to the standards identified within the following sections.

Section 1721 Mitigation Measures

a) Site Clearance. The permittees shall disturb or clear the site only to the extent necessary to assure suitable access for construction, safe operation and maintenance of the LWES.

b) Topsoil Protection. The permittees shall implement measures to protect and segregate topsoil from subsoil in cultivated lands unless otherwise negotiated with the affected landowner.

c) Compaction. The permittees shall implement measures to minimize compaction of all lands during all phases of the project's life and shall confine compaction to as small an area as practicable.

d) Livestock Protection. The permittees shall take precautions to protect livestock on the LWES site from project operations during all phases of the project's life.

e) Fences. The permittees shall promptly replace or repair all fences and gates removed or damaged by project operations during all phases of the project's life unless otherwise negotiated with the fence owner.

f) Roads

1. Public Roads. Prior to commencement of construction, the permittees shall identify all state, county or township "haul roads" that will be used for the WES project and shall notify the state, county or township governing body having jurisdiction over the roads to determine if the haul roads identified are acceptable. The governmental body shall be given adequate time to inspect the haul roads prior to use of these haul roads. Where practicable, existing roadways shall be used for all activities associated with the WES. Where practicable, all-weather roads shall be used to deliver concrete, turbines, towers, assemble nacelles and all other heavy components to and from the turbine sites.

2. The permittees shall, prior to the use of approved haul roads, make satisfactory arrangements with the appropriate state, county or township governmental body having jurisdiction over approved haul roads for construction of the WES for the maintenance and repair of the haul roads that will be subject to extra wear and tear due to transportation of equipment and WES components. The permittees shall notify the County Zoning Office of such arrangements.

3. Turbine Access Roads. Construction of turbine access roads shall be minimized. Access roads shall be low profile roads so that farming equipment can cross them and shall be covered with Class 5 gravel or similar material. Access roads shall avoid crossing streams and drainage ways wherever possible. If access roads must be constructed across streams and drainage ways, the access roads shall be designed in a manner so runoff from the upper portions of the watershed can readily flow to the lower portion of the watershed.

4. Private Roads. The permittees shall promptly repair private roads or lanes damaged when moving equipment or when obtaining access to the site, unless otherwise negotiated with the affected landowner.

5. Control of Dust. The permittees shall utilize all reasonable measures and practices of

construction to control dust during construction.

(g) Soil Erosion and Sediment Control Plan. The permittees shall develop a Soil Erosion and Sediment Control Plan prior to construction and submit the plan to the County Zoning Office no less than forty five (45) days prior to commencing construction. The Soil Erosion and Sediment Control Plan shall address the erosion control measures for each project phase, and shall at a minimum identify plans for grading, construction and drainage of roads and turbine pads; necessary soil information; detailed design features to maintain downstream water quality; a comprehensive re-vegetation plan that uses native plant species to maintain and ensure adequate erosion control and slope stability and to restore the site after temporary project activities; and measures to minimize the area of surface disturbance. Other practices shall include containing excavated material, protecting exposed soil, stabilizing restored material and removal of silt fences or barriers when the area is stabilized. The plan shall identify methods for disposal or storage of excavated material.

Section 1723 Setbacks

a) Distance from currently occupied off-site residences, business and public buildings shall be not less than one thousand (1,000) feet. Distance from the residence of the landowner on whose property the tower(s) are erected shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater. For the purposes of this section only, the term "business" does not include agricultural uses.

b) Distance from right-of-way (ROW) of public roads shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater.

c) Distance from any property line shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater, unless appropriate easement has been obtained from adjoining property owner.

Section 1725 Electromagnetic Interference

The permittees shall not operate the LWES so as to cause microwave, television, radio, or navigation interference contrary to Federal Communications Commission (FCC) regulations or other law. In the event such interference is caused by the LWES or its operation, the permittees shall take the measures necessary to correct the problem.

Section 1727 Lighting

Towers shall be marked as required by the Federal Aviation Administration (FAA). There shall be no lights on the towers other than what is required by the FAA.

This restriction shall not apply to infrared heating devices used to protect the monitoring equipment.

Section 1729 Turbine Spacing

The turbines shall be spaced no closer than is allowed by the turbine manufacturer in its approval of the turbine array for warranty purposes.

Section 1731 Footprint Minimization

The permittees shall design and construct the WES so as to minimize the amount of land that is impacted by the WES. Associated facilities in the vicinity of turbines such as electrical/electronic boxes, transformers and monitoring systems shall to the extent practicable be mounted on the foundations used for turbine towers or inside the towers unless otherwise allowed by the landowner on whose property the LWES is constructed.

Section 1733 Electrical Cables

The permittees shall place electrical lines, known as collectors, and communication cables underground when located on private property except when total distance of collectors from the substation require an overhead installation due to line loss of current from an underground installation. This paragraph does not apply to feeder lines.

Section 1735 Feeder Lines

The permittees shall place overhead electric lines, known as feeders, on public rights-of-way if a public right-of-way exists or immediately adjacent to the public right-of-way on private property. Changes in routes may be made as long as feeders remain on public rights-of-way or immediately adjacent to the public right-of-way on private property and approval has been obtained from the governmental unit responsible for the affected right-of-way. If no public right-of-way exists, the permittees may place feeders on private property. When placing feeders on private property, the permittees shall place the feeder in accordance with the easement(s) negotiated. The permittees shall submit the site plan and engineering drawings for the feeder lines to the Board no less than forty five (45) days prior to commencing construction.

Section 1737 Height from Ground Surface

The minimum height of blade tips at their lowest possible point shall be twenty-five (25) feet above grade.

Section 1739 Towers

a) Color and finish shall be as required by State and Federal regulations to include those of the Federal Aviation Administration

b) All towers shall be singular tubular design, unless approved by the Board.

Section 1741 Noise and Shadow Flicker

Noise level produced by the LWES shall not exceed forty five (45) dBA, average A-weighted sound pressure at the perimeter of occupied residences existing at the time the permit application is filed, unless a signed waiver or easement is obtained from the owner of the residence.

The permittees shall submit a report of predicted noise levels at habitable residential dwellings within one mile of proposed tower locations to the Board no less than forty five (45) days prior to commencing construction.

When determined appropriate by the County a Shadow Flicker Control System shall be installed upon all turbines which will cause a perceived shadow effect upon a habitable residential dwelling. Such system shall limit blade rotation at those times when shadow flicker exceeds thirty (30) minutes per day or thirty (30) hours per year at perceivable shadow flicker intensity as confirmed by the Zoning Administrator are probable.

The permittees shall submit a report of predicted shadow flicker levels at habitable residential dwellings within one and one-half miles of proposed tower locations to the Board no less than forty five (45) days prior to commencing construction.

Section 1743 Permit Expiration

The permit shall become void if no substantial construction has been completed within three (3) years of issuance.

Section 1745 Required Information for Permit Application

a) Boundaries of the site proposed for LWES and associated facilities on United States Geological Survey Map or another map as appropriate.

b) Map of easements for LWES.

c) Map of occupied residential structures, business and public buildings within one half mile of the proposed LWES site boundaries.

d) Preliminary map of sites for LWES, access roads and utility lines. Location of other LWES within five (5) miles of the proposed LWES site.

e) Project-specific environmental and cultural concerns (e.g. native habitat, rare species, and migratory routes). This information shall be obtained by consulting with the following agencies with evidence of such consultation included within the application

1. South Dakota Department of Game, Fish and Parks;
2. U.S. Fish and Wildlife Service; and
3. South Dakota State Historical Society

f) Project schedule

g) Mitigation measures

h) Status of interconnection studies/agreements.

Section 1747 Decommissioning

a) Cost Responsibility. The owner or operator of a LWES is responsible for decommissioning that facility and for all costs associated with decommissioning that facility and associated facilities. The decommissioning plan shall clearly identify the responsible party.

b) Useful Life. A LWES is presumed to be at the end of its useful life if the facility generates no electricity for a continuous period of twelve (12) months. The presumption may be rebutted by submitting to the Board for approval of a plan outlining the steps and schedule for returning the LWES to service within twelve (12) months of the submission.

c) Decommissioning Period. The facility owner or operator shall begin decommissioning a LWES facility within eight (8) months after the time the facility or turbine reaches the end of its useful life, as determined in 14(b). Decommissioning must be completed with eighteen (18) months after the facility or turbine reaches the end of its useful life.

d) Decommissioning Requirements. Decommissioning and site restoration includes dismantling and removal of all towers, turbine generators, transformers, overhead and underground cables, foundations, buildings and ancillary equipment to a depth of forty-two (42) inches; and removal of surface road material and restoration of the roads and turbine sites to substantially the same physical condition that existed immediately before construction of the LWES. To the extent possible, the site must be restored and reclaimed to the topography and topsoil quality that existed just prior to the beginning of the construction of the commercial wind energy conversion facility or wind turbine. Disturbed earth must be graded and reseeded, unless the landowner requests in writing that the access roads or other land surface areas be retained.

e) Decommissioning Plan. Prior to commencement of operation of a LWES facility, the facility owner or operator shall file with the Board the estimated decommissioning cost per turbine, in current dollars at the time of the application, for the proposed facility and a decommissioning plan that describes how the facility owner will ensure that resources are available to pay for decommissioning the facility at the appropriate time. The Board shall review a plan filed under this section and shall approve or disapprove the plan within six (6) months after the decommissioning plan was filed. The Board may at any time require the owner or operator of a LWES to file a report describing how the LWES owner or operator is fulfilling this obligation.

f) Financial Assurance. After the tenth (10th) year of operation of a LWES facility, the Board may require a performance bond, surety bond, letter of credit, corporate guarantee or other form of financial assurance that is acceptable to the Board to cover the anticipated costs of decommissioning the LWES facility.

g) Failure to Decommission. If the LWES facility owner or operator does not complete decommissioning, the Board may take such action as may be necessary to complete decommissioning, including requiring forfeiture of the bond. The entry into a participating landowner agreement shall constitute agreement and consent of the parties to the agreement, their respective heirs, successors, and assigns, that the Board may take such action as may be necessary to decommission a LWES facility and seek additional expenditures necessary to do so from the facility owner.

Section 1749 Pre-construction Filing

At least forty-five (45) days prior to commencement of construction, the applicant/permittee shall submit reports of predicted noise levels, predicted shadow flicker levels, soil erosion and control plan, final maps depicting the approximate location of the proposed wind turbines, access roads and collector and feeder lines. Upon completion, the applicant shall also supply an "as-built" ALTA survey indicating that the proposed facilities are in compliance with the setbacks in the permit.

GE
Energy

Ice Shedding and Ice Throw – Risk and Mitigation

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Greenville, SC



Ice Shedding and Ice Throw – Risk and Mitigation

Introduction

As with any structure, wind turbines can accumulate ice under certain atmospheric conditions, such as ambient temperatures near freezing (0°C) combined with high relative humidity, freezing rain, or sleet. Since weather conditions may then cause this ice to be shed, there are safety concerns that must be considered during project development and operation. The intent of this paper is to share knowledge and recommendations in order to mitigate risk.

The Risk

The accumulation of ice is highly dependent on local weather conditions and the turbine's operational state.[2,4] Any ice that is accumulated may be shed from the turbine due to both gravity and the mechanical forces of the rotating blades. An increase in ambient temperature, wind, or solar radiation may cause sheets or fragments of ice to loosen and fall, making the area directly under the rotor subject to the greatest risks[1]. In addition, rotating turbine blades may propel ice fragments some distance from the turbine—up to several hundred meters if conditions are right.[1,2,3] Falling ice may cause damage to structures and vehicles, and injury to site personnel and the general public, unless adequate measures are put in place for protection.

Risk Mitigation

The risk of ice throw must be taken into account during both project planning and wind farm operation. GE suggests that the following actions, which are based on recognized industry practices, be considered when siting turbines to mitigate risk for ice-prone project locations:

- **Turbine Siting:** Locating turbines a safe distance from any occupied structure, road, or public use area. Some consultant groups have the capability to provide risk assessment based on site-specific conditions that will lead to suggestions for turbine locations. In the absence of such an assessment, other guidelines may be used. Wind Energy Production in Cold Climate[6] provides the following formula for calculating a safe distance:

  1.5 * (hub height + rotor diameter)

  While this guideline is recommended by the certifying agency Germanischer Lloyd as well as the Deutsches Windenergie-Institut (DEWI), it should be noted that the actual distance is dependant upon turbine dimensions, rotational speed and many other potential factors. Please refer to the *References* for more resources.

- **Physical and Visual Warnings:** Placing fences and warning signs as appropriate for the protection of site personnel and the public.[4]

- **Turbine Deactivation:** Remotely switching off the turbine when site personnel detect ice accumulation. Additionally there are several scenarios which could lead to an automatic shutdown of the turbine:

  - Detection of ice by a nacelle-mounted ice sensor which is available for some models (with current sensor technology, ice detection is not highly reliable)
  - Detection of rotor imbalance caused by blade ice formation by a shaft vibration sensor; note, however, that it is possible for ice to build in a symmetric manner on all blades and not trigger the sensor[2]
  - Anemometer icing that leads to a measured wind speed below cut-in

- **Operator Safety:** Restricting access to turbines by site personnel while ice remains on the turbine structure. If site personnel absolutely must access the turbine while iced, safety precautions may include remotely shutting down the turbine, yawing to place the rotor on the opposite side of the tower door, parking vehicles at a distance of at least 100 m from the tower, and restarting the turbine remotely when work is complete. As always, standard protective gear should be worn.

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References

The following are informative papers that address the topic of wind turbine icing and safety. These papers are created and maintained by other public and private organizations. GE does not control or guarantee the accuracy, relevance, timeliness, or completeness of this outside information. Further, the order of the references is not intended to reflect their importance, nor is it intended to endorse any views expressed or products or services offered by the authors of the references.

[1] *Wind Turbine Icing and Public Safety – a Quantifiable Risk?:* Colin Morgan and Ervin Bossanyi of Garrad Hassan, 1996.

[2] *Assessment of Safety Risks Arising From Wind Turbine Icing:* Colin Morgan and Ervin Bossanyi of Garrad Hassan, and Henry Seifert of DEWI, 1998.

[3] *Risk Analysis of Ice Throw From Wind Turbines:* Henry Seifert, Annette Westerhellweg, and Jürgen Kröning of DEWI, 2003.

[4] *State-of-the-Art of Wind Energy in Cold Climates:* produced by the International Energy Agency, IEA, 2003.

[5] *On-Site Cold Climate Problems:* Michael Durstewitz, Institut fur Solare Energieversorgungstechnik e.V. (ISET), 2003.

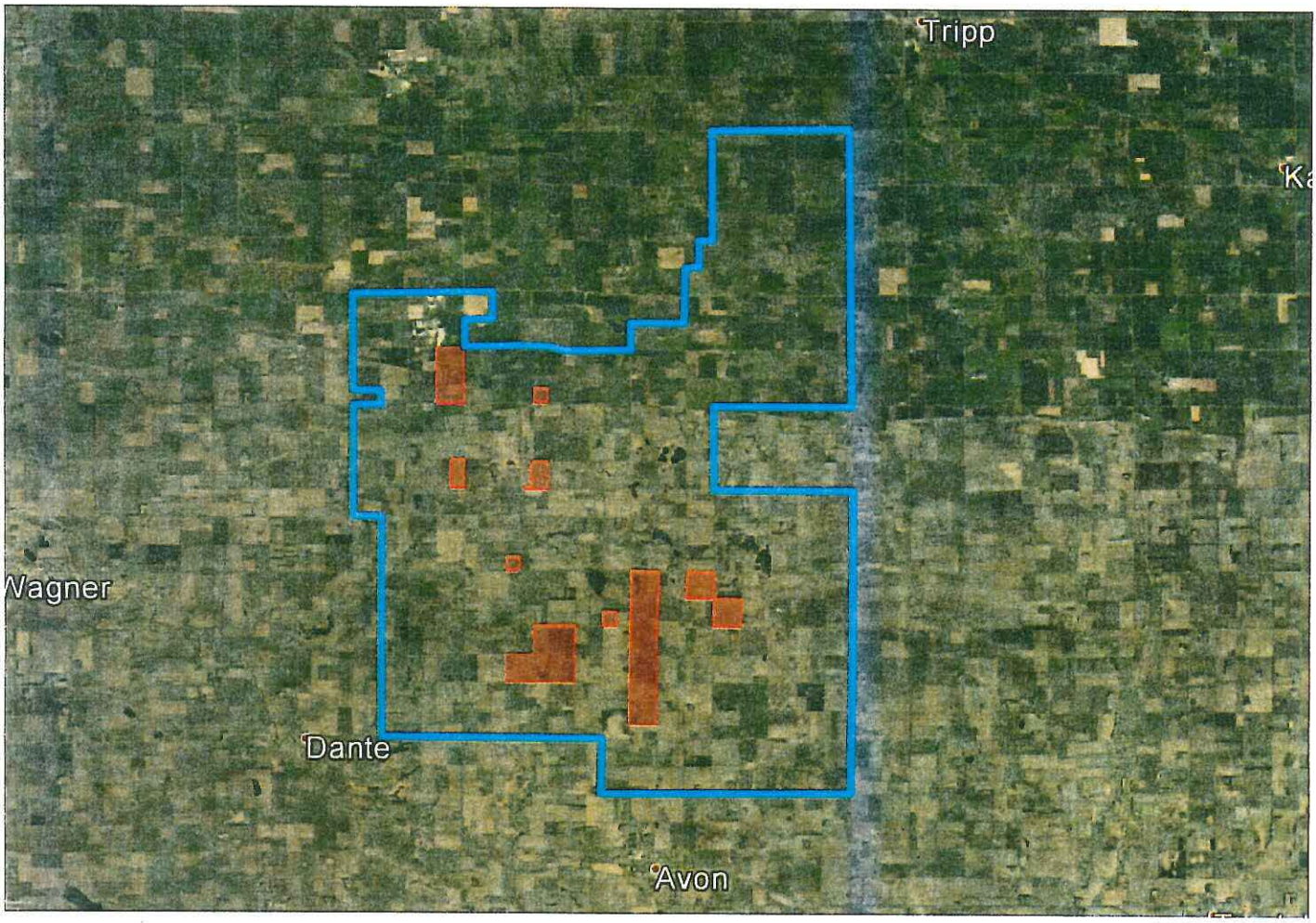
[6] *Wind Energy Production in Cold Climate:* Tammelin, Cavaliere, Holttinen, Hannele, Morgan, Seifert, and Säntti, 1997.

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GER-4262 (04/06)

WELCOME!



Prevailing Wind Park

220 MW
Power Generation

Q4 2018
Start of Construction

Q4 2019
Commercial Operations

S·POWER
An AES and AIMCo Company

EXHIBIT
Int. I-18

S·POWER
An AES and AIMCo Company

Project Summary

- **Project Name:** Prevailing Wind Park, LLC
- **Project Location:** Rural Tripp and Avon, SD
- **Amount of MW Developed:** 220 MW
- **Length of PPA:** 30 years
- **Project Footprint:** 32,000 acres



About sPower

sPower, an AES and AIMCo company, is a leading renewable energy Independent Power Producer (IPP). On February 28, 2018, sPower announced a 30-year Power Purchase Agreement (PPA) for the Prevailing Wind Park Project in South Dakota.

150+ Projects Across the United States	1.1 GW Operating Assets	13.0 GW Operating | Construction | Pipeline	4 BILLION kWh and Counting

About Prevailing Winds

d-working South Dakotans saw opportunity to further economic development in South Dakota by building another wind project. Prevailing Winds, LLC was created and expanded to include **150 local investors, land owners and community members** from South Dakota.

Prevailing Winds, LLC's Mission: Bring the benefits of wind power to the Avon-Tripp area families, landowners, government and communities as well as other South Dakota citizens and businesses.

Project Timeline

2007 | 2008 | 2009 | 2010 | 2011 | 2012 | 2013 | 2014 | 2015 | 2016 | 2017 | 2018 | 2019

Q1 2007
Wind power development started with public meetings in the area towns, which lead to the building of the Beethoven project.

Dec 2014
A group of South Dakotans dedicated to growing renewable energy in the State forms Prevailing Winds, LLC and development begins.

Oct 2017
sPower purchases Prevailing Winds, LLC and forms Prevailing Wind Park, LLC.

Feb 2018
sPower secures Power Purchase Agreement (PPA).

Q4 2018
Construction commences.

Commercial Operations Begin – **Q4 2019**

Electricity Generated

Enough electricity to meet the consumption of **90,000 South Dakota Homes.**

Equivalent to avoiding **486,000 Metric Tons of CO_2 Emissions** Annually.

Annual greenhouse gas emissions over the course of one year from **103,000 Passenger Vehicles.**

Project Benefits

Economic Benefits

- New Income Source for Landowners
- Tax Revenue Generated: Over $56,000,000 Over the Life of the Project
- Shared Among: Schools | Counties | Townships | State

Local Jobs

- 200 Jobs During Peak Construction
- Approximately 8 to 10 Long-Term Operations & Management Positions

Bon Homme Wind Ordinance Explained

When the Beethoven Wind Farm was constructed, Bon Homme County required a conditional use permit and building permits to build the wind turbines. At that time there was no wind energy ordinance in Bon Homme County. So the County looked to the SDPUC, there they found the draft model ordinance that was created as a starting point to help counties develop their own ordinance. In that draft model ordinance, they recommended 55dB as the maximum sound level, a minimum 1000 foot setback from residences, 500 foot or 1.1 times the height setback from right-of-ways of public roads and property lines of non-participants. Also included was a group of standards to assure a review of environmental impacts of wind turbines and other standards to protect the health, safety and welfare of the County's residents. The County used these recommendations to permit the Beethoven Wind Farm.

In 2015, at the suggestion of some Bon Homme County residents, the Bon Homme County Zoning Board undertook the task of writing a wind ordinance. The Zoning Board held a number of meetings with these residents, were the residents requested a two mile setback from residences. The Zoning Board also consulted with Brian McGinnis of District III as to the structure of the potential new ordinance. Mr. McGinnis advises the county on zoning procedures. A key issue for the zoning commission was balancing the rights of county residents to use their land for their own economic benefit, while at the same time protecting residents who do not want to participate in a wind facility from potential impacts.

The Zoning Board found three key areas of potential impact that should be of concern to residents. The first consideration was physical safety. The towers need to be far enough away from residences so that even if a wind turbine would fail from some unforeseen catastrophe event, there would be no impact to a nearby residence. For this, Bon Homme County adopted the state standard of a 1000 foot setback, for safety reasons.

The next consideration was sound. The sound generated by a turbine is dependent on turbine model, and the amount of sound heard at a residence is also dependent on elevation differences between the tower and the residence, and the orientation of the house and number of wind turbines in proximity to the residence. So a distance-based setback for sound would be inappropriate. The Zoning Board settled on a hard limit of 45dB sound level as the maximum sound a turbine is allowed to project to the outside of the residences. For a point of reference, 40dB is the sound level of a quiet library, 50dB is the sound level of the average home and 60dB is the sound level of conversation. According to Ron Hornstra, president of Prevailing Winds, if a wind tower in Bon Homme County makes more than 45dB of noise at your house, you would be able to get that turbine shut off.

The final consideration for the Zoning Board was shadow flicker, which is the shadow cast on a residence when the turbine blade passes between a residence and the sun. If a turbine is located east of a residence, there may be some shadow flicker in the morning at certain times of the year when the sun and turbine line up with your residence. If the tower is west of the residence, the shadow flicker will be in the evening. If the tower is north of the residence, there will be very little or no shadow flicker ever. The county ordinance states that a turbine may cast no more than 30 hours of shadow flicker onto a residence in a year, and no more than 30 minutes during any single day.

All wind turbine locations must meet the most stringent of all three of these requirements and more.

Roland Jurgens, project manager for Prevailing Winds, states that, for most residences, the shadow flicker ordinance will govern the setback. If the tower is east or west of a residence, the shadow flicker will generally require the wind turbines to be 1500 feet to 2800 feet or more from a residence, depending on the wind turbine used. And he states that if the tower is north of a residence, the noise requirement could require the turbine to be at around 1400 feet away, again depending on the wind turbine used. Jurgens also said that the 1000 foot setback did not come into play in the Beethoven wind farm, and won't for the Prevailing Winds wind farm. There is really no way to site a turbine 1000 feet from a residence and be in compliance with Bon Homme County ordinance.

Ron Hornstra commented that Prevailing Winds feels that the Bon Homme County wind ordinance is well thought out and protects both the landowners who want to participate in the project and those that do not equally.

Submitted by Erik Johnson, Avon

9-8-17

EXHIBIT Int. I-20

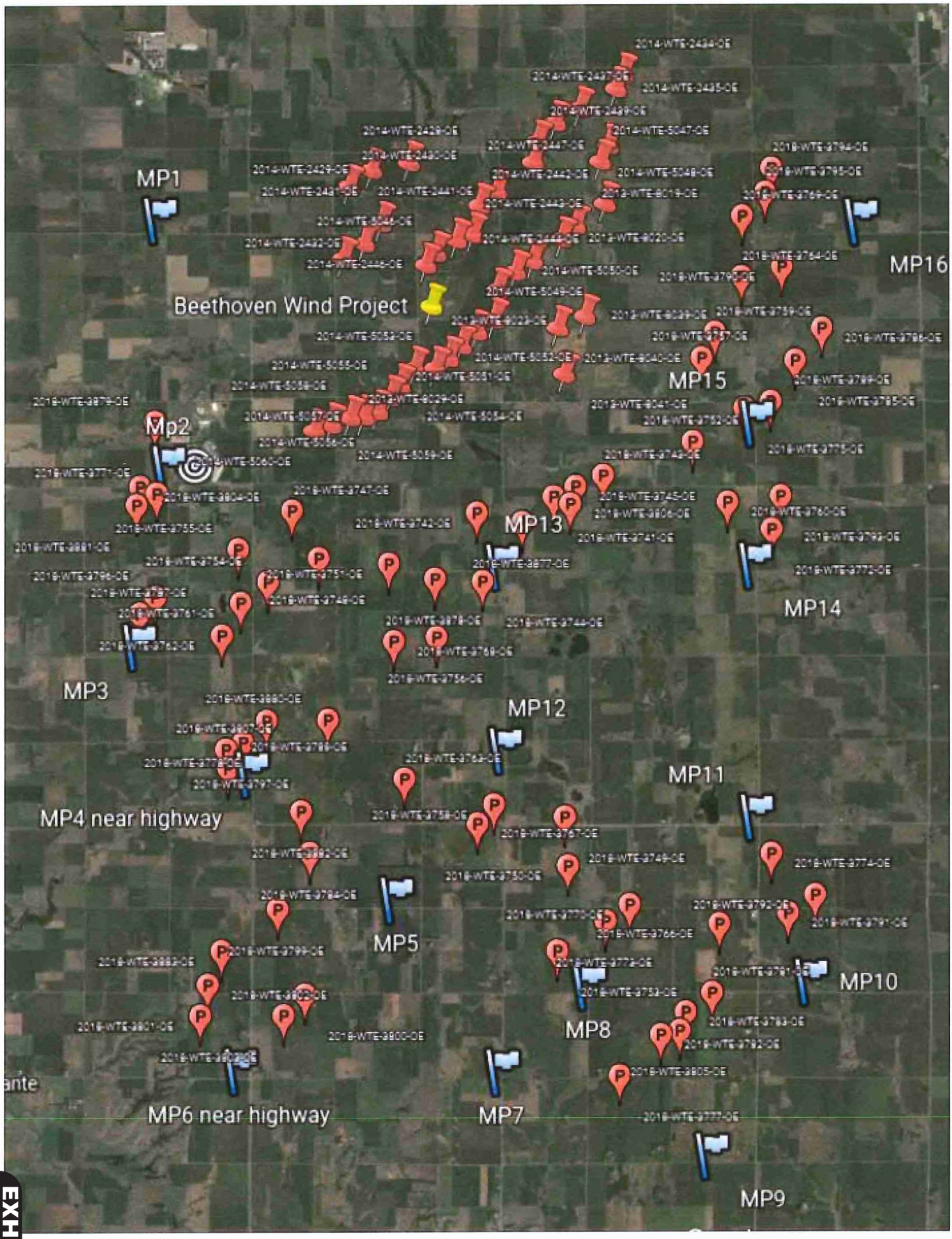


EXHIBIT
Int. I-21

CHARLES MIX COUNTY
STATES ATTORNEY
PO BOX 370
LAKE ANDES, SOUTH DAKOTA 57356
605-487-7441

August 22, 2018

South Dakota Public Utilities Commission
500 East Capital Ave.
Pierre, SD 57501

RE: APPLICATION BY PREVAILING WIN PARK, LLC FOR A PERMIT OF WIND ENERTY FACILITY IN BON HOMME COUNTY, CHARLES MIX COUNTY AND HUTCHINSON COUNTY, SOUTH DAKOTA FOR THE PREVAILING WIND EL 18-026

Dear SDPUC Commission:

This letter is to follow up the phone conversation we had concerning Charles Mix County, SD, and Keith Mushitz's notice of intervening party and the STATE'S FIRST SET OF DATA REQUESTS TO CHARLES MIX COUNTY

As I stated during that phone conversation, Charles Mix County by and through its Commission Chairman, Keith Mushitz, sought to be an intervening party in the above entitled action. In hind sight, I am not sure that was necessary. Applicant has met with the Charles Mix County Commission concerning its project and the concerns of that board Charles Mix County is presently not zoned. In these meetings, the Applicant listened to the county's concerns about parameters of the project. In the end, Applicant agreed to build the project in Charles Mix County in a manner that reflects the Commission's wishes, i.e., Tower Setbacks, Tower Noise (DB level), Shadow Flickering, etc.. The Applicant signed an Affidavit and provided the Commission with that document which outlines these commitments. A copy of that Affidavit is attached hereto.

Given that, the County's request to intervene was only to provide the SDPUC with notice this agreement, to provide the SDPUC with the parameters of the agreement and to request that the SDPUC consider implementing Charles Mix County parameters in the final permit, if given, to the Applicant.

Thus, Charles Mix County does not plan to take depositions, testify or present witnesses during the application process of Applicant. In fact, Charles Mix County has no intention of attending any of the hearings unless called upon.

EXHIBIT
Int. I-22

Thank you very much for your office's guidance in helping Charles Mix County better understand the Application process.

Sincerely,

Scott J. Podhradsky
Deputy State's Attorney
Charles Mix County

In the Matter of the Prevailing Wind Park Project in Charles Mix County, South Dakota

State of South Dakota)
) SS.
County of Charles Mix)

Affidavit of Peter Pawlowski

Peter C. Pawlowski, Vice President, Wind, Sustainable Power Group, LLC ("sPower") of the City of Salt Lake City, County of Salt Lake, State of Utah, being duly sworn on oath, deposes and states that the proposed Prevailing Wind Park will comply with the following requirements in Charles Mix County, South Dakota ("County"):

1. Prevailing Wind Park, LLC ("Prevailing Wind Park") is proposing to construct a wind energy system and associated facilities in Bon Homme, Charles Mix, Hutchinson and Yankton counties, South Dakota. As noted on its website, Basin Electric Power Cooperative has contracted to purchase the 200 megawatts of energy to be generated by the Project.[1] Up to 23 of the proposed turbines and associated facilities ("Project") would be located in Charles Mix County.

2. Prevailing Wind Park has been working cooperatively with the County to address questions regarding the Project.

3. Prevailing Wind Park is a wholly-owned subsidiary of sPower. In my position as Vice President, Wind, sPower, I am authorized to make commitments on behalf of Prevailing Wind Park.

4. Prevailing Wind Park hereby commits to the County Board of Commissioners that the Project will adhere to the following requirements:

   Setbacks.

   (a) Turbine tower distance from currently inhabited rural residence of a nonparticipating landowner shall be not less than three and a half (3.5) times the system height or two thousand feet (2,000) feet, whichever is greater. Turbine tower distance from the residence of the landowner on whose property the tower(s) are erected shall be not less than one thousand (1,000) feet.

   (b) Turbine tower distance from right-of-way of public roads shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater.

[1] https://www.basinelectric.com/About-Us/Organization/At-a-Glance/.

(c) Turbine tower distance from any property line shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater, unless a waiver has been obtained from adjoining property owner.

Noise. Noise from the wind turbines will not exceed 43 dBA at any existing nonparticipating residences and 45 dBA at existing participating residences, unless a signed waiver is obtained from the owner of the residence.

Shadow Flicker. Shadow flicker produced by the wind turbines will not exceed 30 hours per year and/or 30 minutes per day at currently inhabited residences of non-participants.

Lighting. The towers shall be lit using an Aircraft Detection Lighting System ("ADLS"), pending Federal Aviation Administration approval. The ADLS is designed to mitigate the impact of nighttime lights by deploying a radar-based system around a windfarm, turning lights on only when low-flying aircraft are detected nearby. The ADLS sends a signal to keep the light off until a plane is detected, then it stops sending the signal and the lights operate normally until the plane leaves the area and the off signal resumes.

Ice Detection. Prevailing Wind Park will use two methods to detect icing conditions on turbine blades: (1) sensors that will detect when blades become imbalanced or create vibration due to ice accumulation; and (2) meteorological data from on-site permanent meteorological towers, on-site anemometers, and other relevant meteorological sources that will be used to determine if ice accumulation is occurring. These control systems will either automatically shut down the turbine(s) in icing conditions (per the sensors) or Prevailing Wind Park will manually shut down turbine(s) if icing conditions are identified (using meteorological data). Turbines will not return to normal operation until the control systems no longer detect an imbalance or when weather conditions either remove icing on the blades or indicate icing is no longer a concern. Prevailing Wind Park will pay for any documented damage caused by ice thrown from a turbine

5. Prevailing Wind Park further commits to submitting this affidavit in the proceeding currently pending at the South Dakota Public Utilities Commission, *In the Matter of the Application by Prevailing Wind Park, LLC for a permit of a Wind Energy Facility in Bon Homme, Hutchinson and Charles Mix Counties*, Docket EL18-026.

Peter C. Pawlowski

Subscribed and sworn to before me
this 9th day of August, 2018

Notary Public
Expires 4-19-2023

SEAL
SARA CLAYTON
Notary Public
SOUTH DAKOTA

From PUC Site 2008

DRAFT MODEL ORDINANCE FOR SITING OF WIND ENERGY SYSTEMS (WES)

1. **Purpose**
2. **Authority and Jurisdiction**
3. **Federal and State Requirements**
4. **Definitions**
5. **Requirements for Siting Small Wind Energy Systems**
6. **Requirements for Siting Large Wind Energy Systems**

1. Purpose

The purpose of this ordinance is to ensure that the placement, construction and modification of a Wind Energy System (WES) facility is consistent with the County's land use policies, to minimize the impact of WES facilities, to establish a fair and efficient process for review and approval of applications, to assure a comprehensive review of environmental impacts of such facilities, and to protect the health, safety and welfare of the County's citizens.

2. Authority and Jurisdiction

South Dakota Codified Law 11-2-2 delegates the responsibility to the Board of County Commissioners of each county to adopt and enforce regulations designed for the purpose of promoting health, safety, and general welfare of the county.

3. Federal and State Requirements

All WES facilities shall meet or exceed standards and regulations of the Federal Aviation Administration and South Dakota State Statutes and any other agency of federal or state government with the authority to regulate WES facilities.

4. Definitions

1. **Board** – The County Commission, City Commission, or other governmental body governing the district this ordinance refers to.
2. **Construction** – Any clearing of land, excavation, or other action that would adversely affect the natural environment of the site or route but does not include changes needed for temporary use of sites or routes for non-utility purposes, or uses in securing survey or geological data, including necessary borings to ascertain foundation conditions.
3. **High Voltage Transmission Line** – A conductor of electric energy and associated facilities.
4. **Large Wind Energy System** or **LWES** – All WES facilities excluding Small Wind Energy Systems.
5. **Person** – An individual, partnership, joint venture, private or public corporation, association, firm, public service company, cooperative, political subdivision, municipal corporation, government agency, public utility district, consumers power district, or any other entity, public or private, however organized.

EXHIBIT
Int. I-23

6. **Route** – The location of a High Voltage Transmission Line between two end points. The route may have a variable width of up to 1.25 miles.
7. **Small Wind Energy System** or **SWES** – A WES facility with a single Tower Height of less than seventy-five (75) feet used primarily for on-site consumption of power.
8. **Tower Height** – The height above grade of the fixed portion of the tower, excluding the wind turbine itself
9. **System Height** – The height above grade of the tallest point of the WES, including the rotor radius.
10. **Turbine** – The parts of the WES including the blades, generator and tail.
11. **Utility** – Any person engaged in the generation, transmission or distribution of electric energy in this state including, but not limited to, a private investor owned utility, a cooperatively owned utility, a consumers power district and a public or municipal utility.
12. **Wind Energy System** or **WES** – A commonly owned and/or managed integrated system that converts wind movement into electricity. All of the following are encompassed in this definition of system:
    (a) Tower or multiple towers, including foundations;
    (b) Generator(s);
    (c) Blades;
    (d) Power collection systems, including padmount transformers;
    (e) Access roads, meteorological towers, on-site electric substation, control building, and other ancillary equipment and facilities; and
    (f) Electric interconnection systems or portion thereof dedicated to the WES.

5. Requirements for Siting Small Wind Energy Systems

1. Standards

   A Small Wind Energy System shall be a permitted use in all zoning districts subject to the following requirements:

   (a) Setbacks. The minimum setback distance between each wind turbine tower and all surrounding property lines, overhead utility or transmission lines, other wind turbine towers, electrical substations, public roads and dwellings shall be equal to no less than one point one (1.1) times the system height, unless written permission is granted by each affected person.

   (b) Access. All ground mounted electrical and control equipment shall be labeled or secured to prevent unauthorized access, and the tower shall be designed and installed so as to not provide step bolts or a ladder readily accessible to the public for a minimum height of eight (8) feet above the ground.

   (c) Lighting. A SWES shall not be artificially lighted unless such lighting is required by the Federal Aviation Administration.

(d) Noise. SWES facilities shall not exceed fifty-five (55) dBA, as measured at the closest neighboring inhabited dwelling. The level, however, may be exceeded during short-term events such as utility outages or wind storms.

(e) Appearance, Color, Finish. The SWES shall remain painted or finished the color or finish that was originally applied by the manufacturer, unless approved in the building permit.

(f) Signs. All signs, other than the manufacturer's or installer's identification, appropriate warning signs, or owner identification on a wind generator, tower, building, or other structure associated with a SWES visible from any public road shall be prohibited.

(g) Code Compliance. A SWES shall comply with all applicable state construction and electrical codes, and the National Electrical Code.

(h) Utility Notification. No SWES shall be installed until evidence has been given that the utility company has been informed of the customer's intent to install an interconnected customer-owned generator. Off-grid systems shall be exempt from this requirement.

2. Permit Requirements

(a) Building Permit. A building permit shall be required for the installation of a SWES.

(b) The building permit shall be accompanied by a plot plan which includes the following:

(1) Property lines and physical dimensions of the property;

(2) Location, dimensions, and types of existing major structures on the property;

(3) Location of the proposed SWES;

(4) The right-of-way of any public road that is contiguous with the property;

(5) Any overhead utility lines;

(6) Wind system specifications, including manufacturer and model, rotor diameter, tower height, and tower type (monopole, lattice, guyed);

(7) Tower foundation blueprints or drawings;

(8) Tower blueprint or drawing;

(9) Proof of notification to the utility in the service territory in which the SWES is to be erected, consistent with the provisions of 5(3)(h) herein; and

(10) The status of all necessary interconnection agreements or studies.

(c) Expiration. A permit issued pursuant to this ordinance shall expire if:

(1) The SWES is not installed and functioning within twenty-four (24) months from the date the permit is issued; or

(2) The SWES is out of service or otherwise unused for a continuous 12-month period.

3. Abandonment

   (a) A SWES that is out-of-service for a continuous 12-month period will be deemed to have been abandoned. The Board may issue a Notice of Abandonment to the owner of a SWES that is deemed to have been abandoned. The owner shall have the right to respond to the Notice of Abandonment within thirty (30) days from Notice receipt date. The Board shall withdraw the Notice of Abandonment and notify the owner that the Notice has been withdrawn if the owner provides information that demonstrates the SWES has not been abandoned.

   (b) If the SWES is determined to be abandoned, the owner of the SWES shall remove the wind generator from the tower at the Owner's sole expense within three (3) months of receipt of Notice of Abandonment. If the owner fails to remove the wind generator from the tower, the Board may pursue legal action to have the wind generator removed at the owner's expense.

4. Building Permit Procedure

   (a) An owner shall submit an application to the Board for a building permit for a SWES. The application must be on a form approved by the Board and must be accompanied by two (2) copies of the plot plan identified.

   (b) The Board shall issue a permit or deny the application within one month of the date on which the application is received.

   (c) The Board shall issue a building permit for a SWES if the application materials show that the proposed SWES meets the requirements of this ordinance.

   (d) If the application is approved, the Board will return one signed copy of the application with the permit and retain the other copy.

   (e) If the application is rejected, the Board will notify the applicant in writing and provide a written statement of the reason why the application was rejected. The applicant may reapply if the deficiencies specified by the Board are resolved.

   (f) The owner shall conspicuously post the building permit on the premises so as to be visible to the public at all times until construction or installation of the SWES is complete.

5. Violations. It is unlawful for any person to construct, install, or operate a SWES that is not in compliance with this ordinance or with any condition contained in a building permit issued pursuant to this ordinance. SWES facilities installed prior to the adoption of this ordinance are exempt.

6. Severability. The provisions of this ordinance are severable, and the invalidity of any section, subdivision, paragraph, or other part of this ordinance shall not affect the validity or effectiveness of the remainder of the ordinance.

6. Requirements for Siting Large Wind Energy Systems

1. Mitigation Measures

   (a) Site Clearance. The permittees shall disturb or clear the site only to the extent necessary to assure suitable access for construction, safe operation and maintenance of the LWES.

   (b) Topsoil Protection. The permittees shall implement measures to protect and segregate topsoil from subsoil in cultivated lands unless otherwise negotiated with the affected landowner.

   (c) Compaction. The permittees shall implement measures to minimize compaction of all lands during all phases of the project's life and shall confine compaction to as small an area as practicable.

   (d) Livestock Protection. The permittees shall take precautions to protect livestock on the LWES site from project operations during all phases of the project's life.

   (e) Fences. The permittees shall promptly replace or repair all fences and gates removed or damaged by project operations during all phases of the project's life unless otherwise negotiated with the fence owner.

   (f) Roads

      (1) Public Roads. Prior to commencement of construction, the permittees shall identify all state, county or township "haul roads" that will be used for the WES project and shall notify the state, county or township governing body having jurisdiction over the roads to determine if the haul roads identified are acceptable. The governmental body shall be given adequate time to inspect the haul roads prior to use of these haul roads. Where practicable, existing roadways shall be used for all activities associated with the WES. Where practicable, all-weather roads shall be used to deliver concrete, turbines, towers, assemble nacelles and all other heavy components to and from the turbine sites.

      The permittees shall, prior to the use of approved haul roads, make satisfactory arrangements with the appropriate state, county or township governmental body having jurisdiction over approved haul roads for construction of the WES for the maintenance and repair of the haul roads that will be subject to extra wear and tear due to transportation of equipment and WES components. The permittees shall notify the County Zoning Office of such arrangements.

      (2) Turbine Access Roads. Construction of turbine access roads shall be minimized. Access roads shall be low profile roads so that farming equipment can cross them and shall be covered with Class 5 gravel or similar material. Access roads shall avoid crossing streams and drainage ways wherever possible. If access roads must be constructed across streams and drainage ways, the access roads shall be designed in a manner so runoff from the upper

portions of the watershed can readily flow to the lower portion of the watershed.

(3) Private Roads. The permittees shall promptly repair private roads or lanes damaged when moving equipment or when obtaining access to the site, unless otherwise negotiated with the affected landowner.

(4) Control of Dust. The permittees shall utilize all reasonable measures and practices of construction to control dust during construction.

(g) Soil Erosion and Sediment Control Plan. The permittees shall develop a Soil Erosion and Sediment Control Plan prior to construction and submit the plan to the County Zoning Office. The Soil Erosion and Sediment Control Plan shall address the erosion control measures for each project phase, and shall at a minimum identify plans for grading, construction and drainage of roads and turbine pads; necessary soil information; detailed design features to maintain downstream water quality; a comprehensive re-vegetation plan that uses native plant species to maintain and ensure adequate erosion control and slope stability and to restore the site after temporary project activities; and measures to minimize the area of surface disturbance. Other practices shall include containing excavated material, protecting exposed soil, stabilizing restored material and removal of silt fences or barriers when the area is stabilized. The plan shall identify methods for disposal or storage of excavated material.

2. Setbacks

LWES shall meet the following minimum spacing requirements.

(a) Distance from currently occupied off-site residences, business and public buildings shall be not less than one thousand (1,000) feet. Distance from the residence of the landowner on whose property the tower(s) are erected shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater. For the purposes of this section only, the term "business" does not include agricultural uses.

(b) Distance from right-of-way (ROW) of public roads shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater.

(c) Distance from any property line shall be not less than five hundred (500) feet or one point one (1.1) times the system height, whichever is greater, unless appropriate easement has been obtained from adjoining property owner.

3. Electromagnetic Interference. The permittees shall not operate the LWES so as to cause microwave, television, radio, or navigation interference contrary to Federal Communications Commission (FCC) regulations or other law. In the event such interference is caused by the LWES or its operation, the permittees shall take the measures necessary to correct the problem.

4. Lighting. Towers shall be marked as required by the Federal Aviation Administration (FAA). There shall be no lights on the towers other than what is required by the FAA.

This restriction shall not apply to infrared heating devices used to protect the monitoring equipment.

5. Turbine Spacing. The turbines shall be spaced no closer than is allowed by the turbine manufacturer in its approval of the turbine array for warranty purposes.

6. Footprint Minimization. The permittees shall design and construct the WES so as to minimize the amount of land that is impacted by the WES. Associated facilities in the vicinity of turbines such as electrical/electronic boxes, transformers and monitoring systems shall to the extent practicable be mounted on the foundations used for turbine towers or inside the towers unless otherwise allowed by the landowner on whose property the LWES is constructed.

7. Electrical Cables. The permittees shall place electrical lines, known as collectors, and communication cables underground when located on private property except when total distance of collectors from the substation require an overhead installation due to line loss of current from an underground installation. This paragraph does not apply to feeder lines.

8. Feeder Lines. The permittees shall place overhead electric lines, known as feeders, on public rights-of-way if a public right-of-way exists or immediately adjacent to the public right-of-way on private property. Changes in routes may be made as long as feeders remain on public rights-of-way or immediately adjacent to the public right-of-way on private property and approval has been obtained from the governmental unit responsible for the affected right-of-way. If no public right-of-way exists, the permittees may place feeders on private property. When placing feeders on private property, the permittees shall place the feeder in accordance with the easement(s) negotiated. The permittees shall submit the site plan and engineering drawings for the feeder lines to the Board before commencing construction.

9. Height from Ground Surface. The minimum height of blade tips at their lowest possible point shall be twenty-five (25) feet above grade.

10. Towers

    (a) Color and Finish. The finish of the exterior surface shall be non-reflective or matte.

    (b) All towers shall be singular tubular design, unless approved by the Board.

11. Noise. Noise level produced by the LWES shall not exceed 55 dBA, average A-weighted sound pressure at the perimeter of occupied residences existing at the time the permit application is filed, unless a signed waiver or easement is obtained from the owner of the residence.

12. Permit Expiration. The permit shall become void if no substantial construction has been completed within three (3) years of issuance.

13. Required Information for Permit Application.

    (a) Boundaries of the site proposed for LWES and associated facilities on United States Geological Survey Map or other map as appropriate.

    (b) Map of easements for LWES.

(c) Map of occupied residential structures, business and public buildings within one half mile of the proposed LWES site boundaries.

(d) Preliminary map of sites for LWES, access roads and utility lines. Location of other LWES within five (5) miles of the proposed LWES site.

(e) Project-specific environmental and cultural concerns (e.g. native habitat, rare species, and migratory routes). This information shall be obtained by consulting with the following agencies:

(1) South Dakota Department of Game, Fish and Parks;

(2) U.S. Fish and Wildlife Service; and

(3) South Dakota State Historical Society

Evidence of such consultation shall be included in the application.

(f) Project schedule.

(g) Mitigation measures.

(h) Status of interconnection studies/agreements.

14. Decommissioning

(a) Cost Responsibility. The owner or operator of a LWES is responsible for decommissioning that facility and for all costs associated with decommissioning that facility and associated facilities. The decommissioning plan shall clearly identify the responsible party.

(b) Useful Life. A LWES is presumed to be at the end of its useful life if the facility generates no electricity for a continuous period of twelve (12) months. The presumption may be rebutted by submitting to the Board for approval of a plan outlining the steps and schedule for returning the LWES to service within twelve (12) months of the submission.

(c) Decommissioning Period. The facility owner or operator shall begin decommissioning a LWES facility within eight (8) months after the time the facility or turbine reaches the end of its useful life, as determined in 14(b). Decommissioning must be completed with eighteen (18) months after the facility or turbine reaches the end of its useful life.

(d) Decommissioning Requirements. Decommissioning and site restoration includes dismantling and removal of all towers, turbine generators, transformers, overhead and underground cables, foundations, buildings and ancillary equipment to a depth of forty-two (42) inches; and removal of surface road material and restoration of the roads and turbine sites to substantially the same physical condition that existed immediately before construction of the LWES. To the extent possible, the site must be restored and reclaimed to the topography and topsoil quality that existed just prior to the beginning of the construction of the commercial wind energy conversion facility or wind turbine. Disturbed earth must be graded and reseeded, unless the landowner requests in writing that the access roads or other land surface areas be retained.

(e) Decommissioning Plan. Prior to commencement of operation of a LWES facility, the facility owner or operator shall file with the Board the estimated decommissioning cost per turbine, in current dollars at the time of the application, for the proposed facility and a decommissioning plan that describes how the facility owner will ensure that resources are available to pay for decommissioning the facility at the appropriate time. The Board shall review a plan filed under this section and shall approve or disapprove the plan within six (6) months after the decommissioning plan was filed. The Board may at any time require the owner or operator of a LWES to file a report describing how the LWES owner or operator is fulfilling this obligation.

(f) Financial Assurance. After the tenth (10th) year of operation of a LWES facility, the Board may require a performance bond, surety bond, letter of credit, corporate guarantee or other form of financial assurance that is acceptable to the Board to cover the anticipated costs of decommissioning the LWES facility.

(g) Failure to Decommission. If the LWES facility owner or operator does not complete decommissioning, the Board may take such action as may be necessary to complete decommissioning, including requiring forfeiture of the bond. The entry into a participating landowner agreement shall constitute agreement and consent of the parties to the agreement, their respective heirs, successors, and assigns, that the Board may take such action as may be necessary to decommission a LWES facility and seek additional expenditures necessary to do so from the facility owner.

15. Pre-construction Filing. At least forty-five (45) days prior to commencement of construction, the applicant/permittee shall submit final maps depicting the approximate location of the proposed wind turbines, access roads and collector and feeder lines. Upon completion, the applicant shall also supply an "as-built" ALTA survey indicating that the proposed facilities are in compliance with the setbacks in the permit.

Reece M. Almond

From: Roland Jurgens <rjurgens@thorstadcompanies.com>
Sent: Wednesday, October 28, 2015 2:38 PM
To: Eric Elsberry
Subject: RE: A few questions

Hi Eric, In general the setbacks proposed in Lincoln County really do nothing to increase safety or further protect the public, they just make the wind farm more expensive and unnecessarily spread out across a larger area, potentially effecting more people.

For example the State requires 500 feet or 1.1 time total height from a non-participants property lines, so even if a tower could tip straight over the tower would land within the project. A 2 times the commercial height from any exterior property line does not increase tip over safety or access to aerial crop spraying or change the visual effect of the turbine. 2 times the commercial height from any exterior property line does increase the length of the access roads and negatively affects a farmers operation when working his field for no reason. It also makes a project more expensive to build, decreasing the chances that a project will be built.

The 1.25 times the total height from the right-of-way line of any public road or highway again does nothing to increase safety over the State's 1.1 time total height from the right-of-way line. It just increases the length of the access road and negatively affects a farmers operations again.

The 1,500 feet from any habitable structure is fine but if you are really worried about safety, I would use the ice throw formula plus 10% instead; Max Throw = 1.5(2R + H). Because it is possible that 1500 feet is not enough in the future if turbine blades get larger as they have been. It also needs to be inhabited structure not any habitable structure.

The 45dB participant, 35dB non-participant noise restraint is absolutely the best way to protect non-participants. It will move turbines away from the people that don't like them. The reality is when the other setbacks are increased it forces us to put the turbines closer to people's houses and farm places because you paint us into the proverbial corner and unnecessarily limit where we can put the turbines.

The limits on Shadow flicker does the same thing, it will move turbines away from the people that don't like them. Which is good.

Setbacks like Lincoln County are proposing do more harm than good, sadly I have seen it happen more than once.

Hope this helps, I think you guys have it right if you just add the 35dB non-participant noise restraint.
Roland

P.S. I also understand that wind project supporter are asking Lincoln County why there ordinance is not like Bon Homme County and every other County in state that has wind farms and wind farm zoning. I am sure their ordinance is going to change too.

From: Eric Elsberry [mailto:bhcem@hcinet.net]
Sent: Wednesday, October 28, 2015 1:10 PM
To: Roland Jurgens
Subject: A few questions

Good Morning,

EXHIBIT
Ibt. I-24

Mr. Hubner was in my office Friday and directed me to look at the site we-caresd.org. I went and looked at the site and saw that Lincoln Co. was working on their ordinance. I got in touch with the Lincoln Co. Zoning and they directed me to the site licolncountysd.org and their wind energy article is 13.02. While looking at that article, I was wondering how part of it would affect how you design the site plan. I am looking at their setbacks and they are 2 times the commercial height from any exterior property line, 1.25 times the total height from the right-of-way line of any public road or highway, and 1,500 feet from any habitable structure. If we used these setbacks, and implemented a 45dB participant, 35dB non-participant noise restraint, would there be a big impact on how the site plan would be altered?
Thanks,
Eric

Second International Meeting
on
Wind Turbine Noise
Lyon France September 20 – 21 2007

In-Home Wind Turbine Noise Is Conducive to Vibroacoustic Disease

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Abstract
Introduction. This team has systematically studied the effects of infrasound and low frequency noise (ILFN, <500 Hz) in human and animal models since 1980. Recently, yet another source of ILFN has appeared: wind turbines (WT). Like many other ILFN-generating devices, WT can greatly benefit humankind if, *and only if*, responsible measures are taken for their implementation. Vibroacoustic disease (VAD) is the pathology that is acquired with repeated exposures to ILFN environments (occupational, residential *or* recreational). This has been demonstrated in numerous scientific articles published in peer-reviewed academic journals over the past 27 years. **Goal.** To evaluate if ILFN levels obtained in a home near WT are conducive to VAD. **Methods**. Case 1: documented in 2004, in-home ILFN levels generated by a port grain terminal (GT), 2 adults and a 10-year-old child diagnosed with VAD. Case 2: isolated farm in agricultural area, four 2MW WT that began operation in Nov 2006, located between 300 m and 700 m from the residential building, 3 adults and 2 children (8 and 12-years-old). ILFN levels of Case 2 were compared to those in Case 1. In both, ILFN was assessed in 1/3 octave bands, without A-weighting, (i.e. in dB Linear). In Case 1, the lower limiting frequency was 6.3 Hz, while in Case 2, it was 1 Hz. **Results.** ILFN levels within the 6.3-31.5 Hz range in the home of Case 2 were higher than those obtained in the home of Case 1. Above, 31.5 Hz, levels varied but were comparable. **Discussion.** ILFN levels contaminating the home of Case 2 are sufficient to cause VAD. This family has already received standard diagnostic tests to monitor clinical evolution of VAD. Safe distances between WT and residences have not yet been scientifically established, despite statements by other authors claiming to possess this knowledge. Acceptance, as fact, of statements or assertions not supported by valid scientific data, defeats all principles on which true scientific endeavor is founded. Widespread statements claiming no harm is caused by in-home ILFN produced by WT rotating blades are fallacies that cannot, in good conscience, continue to be perpetuated. In-home ILFN generated by WT blades can lead to severe health problems, specifically, VAD. Real and efficient zoning for WT must be *scientifically* determined, and quickly adopted, in order to competently and responsibly protect Public Health.

EXHIBIT
Int. I-25(a)

010040

Initial Disclaimer

The authors and the research team they represent would like to clarify that:

a) *No member of this team is party to anti-technology sentiments;*
b) *Large industrial plants, such as grain terminals, as well as alternative forms of renewable energy, such as wind turbines, are considered welcome additions to modern technological society by all members of this team;*
c) *The data reported herein have been scrutinized under one, and only one, agenda - that of pure scientific inquiry;*
d) *In no way can or should this report be construed as a document arguing against the implementation of wind turbines and/or grain terminals;*
e) *No member of this research team is employed by the firm that conducted the acoustical measurements reported in this article, nor are there any commercial, financial or professional agreements (contractual or otherwise) between the aforementioned accredited firm and any member of this team;*
f) *The consulting activities provided by these authors to Family R are of a purely academic and scientific nature and hence are* pro bono.

Introduction

In March of 2007, this team was contacted by an attorney-at-law representing the R. Family, in a case involving the placement of 4, 2 MW wind turbines (WT) near family R.'s property. Located between 321.8m and 642.0m from the residential building (Figs. 1, 2), the 4 WT became operational in November 2006. Two days later the R. family contacted a lawyer to begin court proceedings in order to have the WT removed.

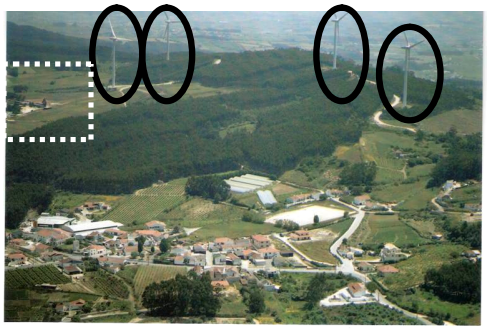


Figure 1. Aerial view of the WT home of Family R., isolated on upper left (dashed square) with the four wind turbines nearby (ovals).



Figure 2. WT home with the two of the turbines (arrows) at approximately 322m and 642m from the home.

In order for acoustical assessments to be accepted as legal documents, they must be performed by an accredited firm. In February 2007, the R. Family hired such a firm – dBLab (1) - to conduct continuous, 12-day, acoustical, wind speed, and vibration measurements. Although Portuguese noise legislation (D.L. 9/2007, January 17th) does not require acoustical evaluations of frequencies below 50Hz, nor dB Linear (dBL) measurements (without the A-weighting network), the accredited firm was additionally asked to obtain data for the entire frequency spectra, down to the lowest limiting frequency of the equipment in use, in 1/3 octave bands and in dBL. Data

were made available to this team, within legal terms and with written consent on behalf of Family R., as well as the accredited firm.

This report documents the levels of infrasound and low frequency noise (ILFN, 6.3Hz-500Hz) encountered in this particular home, due to the operation of 4, 2-Megwatt WT.

Methods

dBLab used two appropriately calibrated and certified 01dB Symphonie sound level meters, equipped with ½" microphone (GRAS, model 23606). Measurements were obtained in periods of 30-min, continuously for 12 days, between Apr 5th-16th, 2007. The lower limiting frequency was 1 Hz. Simultaneous and synchronized accelerometer and wind speed data were also acquired. Measurements were taken within the Master bedroom of Family R., in accordance with the procedures stipulated by Portuguese (NP 1730, Pt 1&2, 1996 and DL 9/2007, January 17th) and International Law (ISO1996, 2003). Measurements conducted outside of the residential building and accelerometer data have not yet been fully analyzed and will not be considered in this report.

Results

Noise Analysis As Per Current Legislation

In accordance with the noise study conducted by dBLab, legally stipulated annoyance levels were exceeded during day (7am-8pm), evening (8-11pm) and night (11pm-7am) hours. dBA noise levels were also exceeded for a sensitive zone during night hours, but were within legal limits for a mixed zone. The local Municipal Authorities where the Family R.'s property is located have not yet classified the area as sensitive *or* mixed.

Wind Speed varied between 0 and 12.6 Km/h. According to the Portuguese Institute of Meteorology (Table 1), the average wind speed during the month of April 2007 was well below the average values of the previous years.

	2004	2005	2006	2007
Jan	6,6	5	6,4	4,6
Feb	7,4	8,8	8,6	6,8
Mar	11,3	12,3	10,4	11
Apr	11,4	11,5	10,7	8,9
May	11,5	13,1	10	10,7
Jun	12,6	11,9	11,7	12,8
Jul	13,1	14,3	12,2	
Aug	13,4	11,6	12,3	
Sep	9,1	10,9	10,9	
Oct	9,2	10	10,9	
Nov	5,6	6,7	6,1	
Dec	6,5	7,8	5,3	

Table 1. Monthly average wind speeds (in Km/h), from 2004 onwards, obtained at the relevant meteorological station near the property of family R, as provided and certified by the Portuguese Institute of Meteorology, on July 27th, 2007.

Acoustical Analysis of Infrasound and Low Frequency Noise

As per the request of this team, spectral analysis of 1/3 octave bands ranging from 1-500 Hz, in dBL were also obtained. Figure 3 compares the residual (no WT blade movement) and environmental (with rotating WT blades) measurements taken within the Master bedroom.

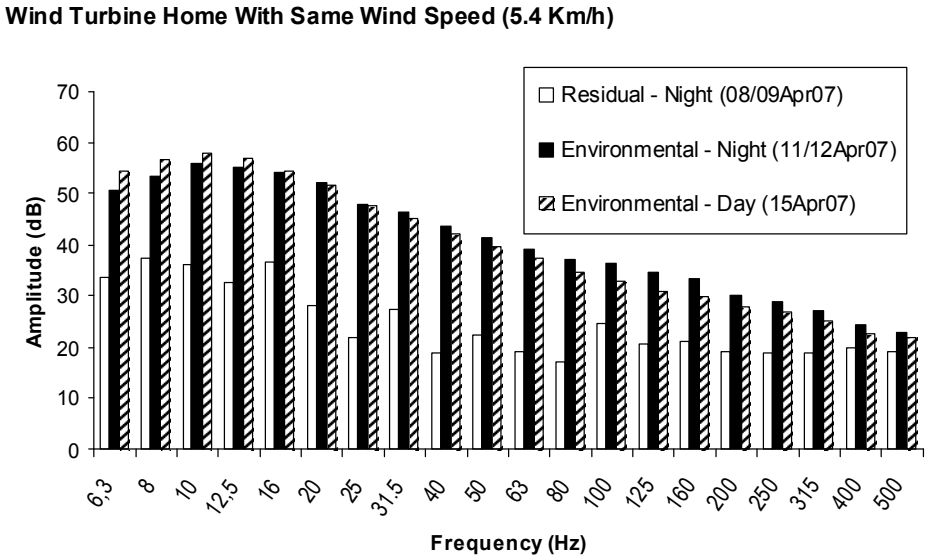


Figure 3. Comparison of 1/3 octave ILFN levels, in dBL, of the *Residual* (no WT blade movement) measurement and the *Environmental* (with rotating WT blades) measurement in the WT home Master Bedroom, with the same recorded wind speed.

Since ILFN is not yet recognized as an agent of disease, it is not covered by legislation. Therefore, permissible exposure levels for ILFN have not yet been determined, and dose-response relationships are unknown. Hence, no adequate standard exists to appropriately compare the ILFN levels in Figure 3 within the context of human health effects.

Analysis Within the Context of Vibroacoustic Disease

Although no generally accepted standard exists for linking the ILFN levels shown in Figure 3 to human health effects, the authors propose that a standard can be established by comparing WT-produced ILFN levels to ILFN levels that have been shown to be conducive to vibroacoustic disease (VAD).

Since 1980, this team has been systematically studying the effects of ILFN on human and animal models. As a result, an illness termed VAD (2-4) has been identified and can be readily diagnosed through echocardiography (5-8) and bronchoscopy (9-13) examinations.

On March 8th, 2007, the first time, the Portuguese Ministry of Labour, through its National Center for Occupational Diseases, granted 100% professional disability to a 40-year-old flight attendant, for having developed VAD during her professional activity. She was diagnosed with VAD in 2001, at the age of 34.

Within the context of VAD studies, the compilation of data on ILFN-rich environments has been ongoing since 2003. Hence, this team is uniquely positioned to provide pertinent data with which to compare the ILFN levels obtained in the bedroom of Mr. and Mrs. R.

Although VAD has been mostly studied within occupational settings (3), in 2004 this team documented (14) its first Portuguese case of environmental VAD in Family F. Residential ILFN was produced by a port Grain Terminal (GT) within line of sight of the home (Figs. 4, 5). From 1982 until 2003, this GT was allowed to operate at any time of the day or night. Operating hours were only restricted in 2003 when new legislation mandated that noisy industrial activities must cease at 11 p.m.

Acoustical measurements at the Family F. home were conducted with a Bruel & Kjaer 2260 sound level meter, equipped with a ½" microphone (B&K, model 4189). Measurements were obtained in periods of 15-min, for 3 hours, starting at 9 p.m. (evening period) on Feb 4th, 2004. The lower limiting frequency was 6.3 Hz (14).



Figure 4. Trafaria Deep Water Grain Terminal (TDWGT).



Figure 5. View from the GT home of Family F., located in Lisbon. Across the Tagus River is the TDWGT.

Figure 6 compares the ILFN levels obtained in the WT home of Family R., with those obtained in the GT home of Family F. Below 31.5 Hz, all 1/3 octave bands have higher dB Linear readings in the WT home than in the GT home. The two peaks detected within the GT home, at 40Hz and 50Hz, are specifically related to unidentified GT operations. Above 200 Hz, the GT has higher dB levels than the WT home. The remaining 1/3 octave bands showed similar levels in both homes.

Manifestation of Vibroacoustic Disease in Family F. – GT Home

"Mr. F. is apparently asymptomatic. He complains of a lack of concentration and overall irritation, and has severe bouts of rosacea. He has always lived in the suburbs of the city of Lisbon, and has been working in the centre of Lisbon for the past 10 years. Mrs. F. has been diagnosed with hepatitis A, mononucleosis and allergic rhinitis. While still a student in university, she was once diagnosed with a late-onset epileptic seizure, for which she is currently unmedicated. She complains of body aches, particularly in the right shoulder, left knee, back and neck. X-rays have not revealed any abnormalities. She has always had headaches, mostly radiating along the back of the neck. Approximately 4 or 5 years ago, while in a shopping mall supermarket, Mrs. F. suffered a violent tachycardia, with feelings of faintness. She

was taken to the hospital where a subsequent EKG did not disclose abnormalities. Mrs. F. has worked in governmental administrative offices, in the centre of Lisbon, for the past 16 years. Ten-year-old P. suffered from asthma until the age of 1 year. At 5–8 months of age, he was medicated for reflux, and then again until he was 1 year old. At 8 months he suffered pneumonia. After the age of 1, he began to develop repeated ear infections that were not responsive to antibiotics. At age 3 he underwent ear surgery. At the age of 5, at school, he suddenly lost his vision and was taken to the hospital, where the EEG revealed an epileptic seizure. Nose bleeds without an apparent cause used to be frequent, but have subsided with age. There is no history of rheumatic fever, radiation or asbestos exposure" (14).

Through echocardiography, all three members of this family showed characteristic thickening of cardiovascular structures normally seen in VAD patients (14), namely the pericardium and mitral valve (2, 3, 5-8). The most severe cardiovascular condition was observed in 10-year-old P., most probably because the mother spent the pregnancy gestation months in that same ILFN-rich home. For a more detailed description of this case, see (14).

Late-onset epilepsy, nose bleeds, tachycardia, muscular and joint pain with no imaging corroboration despite sustained patient complaints, are common in VAD patients (2, 3). Respiratory pathology has already been closely linked to ILFN exposure, both by this team (2-4, 9-13, for example) and by other authors (15-17, for example). This family continues to be followed by this team, and has chosen to remain in the ILFN-rich home, but they have relocated their bedrooms to the back of the house.

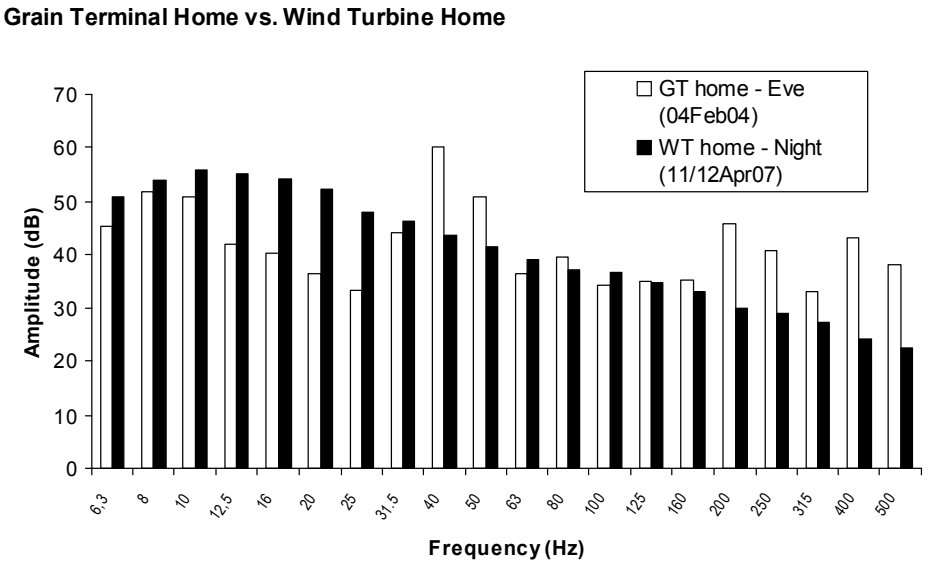


Figure 6. Comparison of the frequency spectra obtained in the GT home of Family F., in 2004, with those obtained in the WT home of Family R., in 2007. The 40Hz peak in the GT home is specifically associated with GT operations. Within the range of 6.3Hz to 31.5Hz, 1/3 octave bands disclose higher dBL levels in the WT home than in the GT home.

Manifestation of Vibroacoustic Disease in Family R. – WT Home

Mr. R has deep concerns about his memory loss, increased irritability and progressive intolerance toward audible noise, all of which he complained about at the very first meeting with this team, in March 2007. Both Mr. and Mrs. R. have developed great difficulty in sleeping continuously throughout the night, as well as non-specific body pain. Upon visiting a general physician at the local State Health Center, Mr. R was prescribed 2 analgesics (anti-inflammatory and spasmolytic) and 2 tranquilizers (diazepam-based and short-term sleep-inducer).

Echocardiograms (routine, non-invasive VAD diagnostic test) of Mr. and Mrs. R. disclosed slight to moderate pericardial thickening (between 1.7mm and 2.0mm, normal for the equipment in use: <1.2mm). Respiratory drive was below normalized values in both adults (46%-53%, normal: >60%), suggesting the existence of brain lesions in the areas responsible for the neurological control of breathing (3).

In mid-March, Mr. and Mrs. R received a letter from their 12-year-old son's school, expressing concern for the growing difficulties of an otherwise outstanding student, "*particularly in English, Humanities and Physical Education. He progressed in Mathematics, which is a field that naturally attracts his type of intelligence. However, in the above mentioned coursework, it seems that [the child] has lost interest, makes a lesser effort, as if he were permanently tired. In Physical Education, an abnormal amount of tiredness is also observed. Is [the child] leading a healthy life? Does he sleep sufficient hours during the night?*"

Given the above school information, and since cognitive and memory disturbances are common and well documented in VAD patients (2, 3), the child received neurophysiological evaluation. Brainstem auditory evoked potentials disclosed asymmetries in the right and left nerve conduction times, and the right I-V interlatency value was at the threshold of normal values (4.44ms). The endogenous evoked potential P300 recording occurred at 352ms (normal: 300ms). This measure reflects the time it takes to recognize and memorize infrequent stimuli. *"Although this result is in accordance with the child's school report indicating that cognitive processes are affected, it is not possible to state that this situation is irreversible. Moreover, in children, P300 recordings often disclose variations that are difficult to interpret. Nevertheless, initial clinical signs of Stage-I VAD are characterized by this type of cognitive impairment and, as such, may be reversible at this clinical stage provided ILFN exposure is suspended"* (18).

The R. Family income is provided by breeding bulls and raising and training horses for bullfights. Horses have exhibited an abnormal behaviour, lying down and sleeping during the day. Tissue fragments have been removed from the farm animals that have been scheduled for slaughter, and will be submitted to the light and electron microscopy analyses that this team usually conducts on ILFN-exposed tissue fragments (3,4). These procedures will be repeated every 6 months, and follow-up reports will ensue. Mr. R. has resident employees who are also receiving all medical tests.

DISCUSSION

ILFN Levels

ILFN levels within the 1/3 octave bands ranging from 6.3Hz to 31.Hz are larger in the WT home than in the GT home. Other bands have a similar dBL level with the exception of the 40Hz and 50Hz bands, and those equal or above 200 Hz. Previous studies strongly suggest that infrasound (≤ 20 Hz) exposure is specifically associated with pericardial thickening (3, 7, 19). Since the family living in the GT home has developed VAD due to in-home ILFN exposure, it is reasonable to assume that the WT family will also progressively develop VAD.

In a perfect world, designed for the most efficient and accurate scientific studies, all noise assessments ought to be conducted with the same equipment and with the same procedures. This is not feasible. So, despite on-site and factory calibrations, a legitimate question will always remain: can the differences between the ILFN levels in the homes of Family F. and Family R. be due to differences in the noise measuring equipment and procedures alone? Despite this legitimate question, these data are sufficient to warrant precautionary measures.

If and when moderate and severe VAD-related symptoms are documented in Family R., this question can be put to rest. This course of action, however, lacks any ethical basis, and could potentially result in a lawsuit, due to negligence.

Occupational vs. Residential Exposures

Occupational exposures to ILFN occur at larger dBL levels (See Fig. 7). However, residential exposure occurs over longer periods of time, affecting all family members (particularly children), and is present during sleep time. In this team's experience, residential ILFN exposures lead to accelerated progression of VAD (9).

Drama or Challenge

Neither the authors nor the team they represent are oblivious to the implications that this study brings to light. However, dramatization of a problem rarely leads to any sort of solution.

Instead of attempting to appease those who are vehement about the notion that WT are inconsequential to human health, and to avoid the useless acrimonious debate that usually ensues after these type data is presented, a challenge is offered up: *zoning laws*. And this immediately leads to the issue of safe distances from the aerodynamic pressure waves produced by rotating WT blades.

Safe Distances…

Scientists have not yet established safe distances between residential buildings and WT. Many scientists have not yet recognized that ILFN is an agent of disease. Hence, the fact that rotating WT blades (analogous to aircraft propellers, or rather, helicopter rotor blades) produce acoustic pressure waves consistent with ILFN phenomena is, to many, simply irrelevant – because ILFN is (erroneously) assumed to be harmless.

…And the Scientific Method

Following a logical rationale: If ILFN is not suspected as an agent of disease, then it does not need to be assessed or investigated as to potential health hazards. Most scientists and physicians do not, therefore, possess information regarding any aspect of ILFN. It follows that until scientific data exist on the subject, no credible claims can be made regarding safe distances between rotating WT blades and residential homes.

Some team could develop an equation that would determine, on a case-specific basis, the safe distance between a home and WT, since the amount of ILFN will depend simultaneously on several factors, including: distance to the building, blade size, wind speed and direction, geology, type of terrain, building resonance properties of both ILFN and solid-to-solid ground vibration transmission, type of vegetation as well as its quantity and distribution.

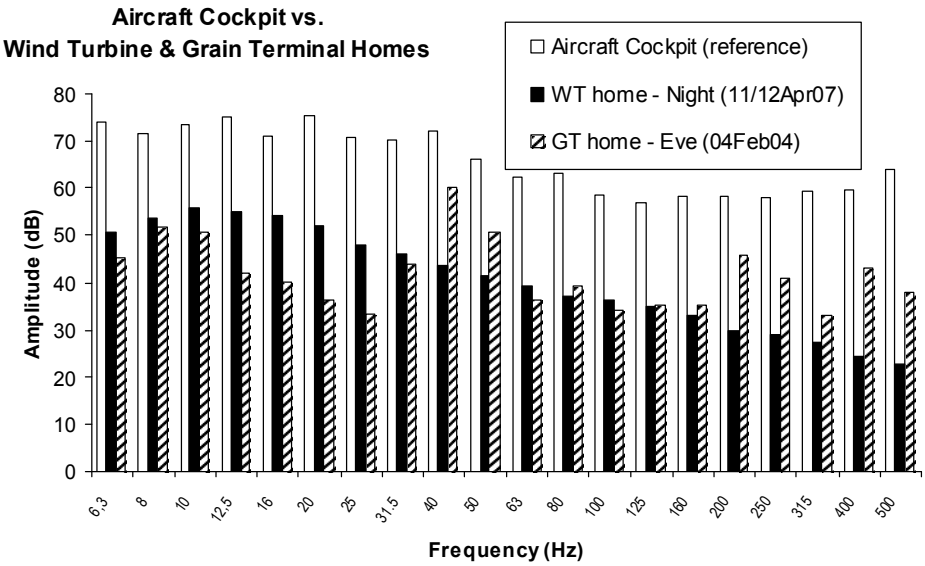


Figure 7. Comparison of 1/3 octave ILFN levels, in dBL, of the WT home, the GT home, and the reference ILFN levels used for occupational exposures by this team (Aircraft Cockpit (19)).

Prospects of New Data

As certified by the Portuguese Institute of Meteorology (Table 1), the average monthly wind speeds in August are generally higher than in April. Hence, dBLab will repeat all measurements during the month of August. WT-generated ILFN is strongly related to the acoustic pressure waves aerodynamically generated by rotating blades. This is somewhat analogous to the infrasound (≤ 20 Hz) levels in airplane cockpits, which are larger with increasing aircraft speed, due to the impact of the aerodynamic airflow on the nose of the aircraft (19).

The neurophysiology evaluation of the child will be repeated in late September, after he has spent 2 months away from the ILFN-contaminated home, and in December, non-invasive VAD diagnostic tests will be repeated on all family members and

resident employees. Electron microscopy studies of animal tissue fragments are ongoing.

CONCLUSIONS

ILFN levels within the range of 6.3Hz to 31.5Hz obtained at the WT home are higher than those obtained at the GT home. Family members residing in the GT home have been diagnosed with ILFN-induced pathology, i.e., VAD. With time, it is highly probable that the family residing at the WT home will also develop severe VAD, since they are already exhibiting symptoms consistent with early VAD.

Precautionary measures regarding the placement of WT near inhabited buildings are justified. Safe distances between WT rotating blades and inhabited buildings have not yet been determined by the scientific community.

REFERENCES

1. dBLab – Laboratório de acústica e vibrações, Lda. www.absorsor.pt. (dblab@absorsor.pt).
2. Castelo Branco NAA (1999). The clinical stages of vibroacoustic disease. *Aviation, Space & Environmental Medicine*, 70 (3, Suppl): A32-9.
3. Castelo Branco NAA & Alves-Pereira M (2004). Vibroacoustic disease. *Noise & Health*, 6(23): 3-20.
4. Alves-Pereira M & Castelo Branco NAA (2007). Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signalling. *Progress Biophysics & Molecular Biology*, 93:256-279.
5. Marciniak W, Rodriguez E, Olsowska K, Botvin I, Araujo A, Pais F, Soares Ribeiro C, Bordalo A, Loureiro J, Prazeres de Sá E, Ferreira D, Castelo Branco MSNAA, Castelo Branco NAA (1999). Echocardiography in 485 aeronautical workers exposed to different noise environments. *Aviation, Space & Environmental Medicine*, 70 (3, Suppl): A46-53.
6. Torres R, Tirado G, Roman A, Ramirez R, Colon H, Araujo A, Pais F, Marciniak W, Nóbrega J, Bordalo e Sá A, Lopo Tuna JMC, Castelo Branco MSNAA, Alves-Pereira M, Castelo Branco NAA (2001). Vibroacoustic disease induced by long-term exposure to sonic booms. *Proceedings Internoise 2001*, The Hague, Holland: 1095-98.
7. Araujo A, Pais F, Lopo Tuna JMC, Alves-Pereira M, Castelo Branco NAA (2001). Echocardiography in noise-exposed flight crew. *Proceedings Internoise 2001*, The Hague, Holland 2001: 1007-10.
8. Araujo A, Carranca J, Alves-Pereira M, Castelo Branco NAA (2005). Echocardiography in vibroacoustic disease. *Proceedings 12th International Congress on Sound & Vibration*, Lisbon, Portugal, No. 567 (9 pages).
9. Bento Monteiro M, Reis Ferreira J, Mendes CP, Alves-Pereira M, Castelo Branco NAA (2004). Vibroacoustic disease and respiratory pathology III – Tracheal and bronchial lesions. *Proceedings Internoise 2004*, Prague, Czech Republic, No. 638 (5 pages).
10. Monteiro M, Reis Ferreira J, Mendes CP, Serrano I, Tavares F, Alves-Pereira M, Castelo Branco NAA (2005). espiratory pathology in vibroacoustic disease II – Specific morphological changes. *Proceedings 12th International Congress on Sound & Vibration*, Lisbon, Portugal, No. 572 (9 pages).

11. Reis Ferreira J, Monteiro M, Tavares F, Monteiro E, Mendes CP, Alves-Pereira M, Castelo Branco NAA (2006). Involvement of central airways in vibroacoustic disease patients. *Revista Portuguesa Pneumologia* XII(2), 93-105. (In English.)
Granted the 2004 Thomé Villar/Boehringer Ingelheim award, attributed by the Portuguese Lung Society
Available:www.sppneumologia.pt/publicacoes/?imc=50n&publicacao=22&edicao=1862&fmo=pa
12. Castelo Branco NAA, Reis Ferreira J, Alves-Pereira M (2007). Respiratory pathology in vibroacoustic disease: 25 years of research. *Revista Portuguesa Pneumologia*, XIII(1), 129-35. (In English.)
Available:www.sppneumologia.pt/publicacoes/?imc=50n&publicacao=22&edicao=1893&fmo=pa
13. Monteiro M, Reis Ferreira J, Alves-Pereira M, Castelo Branco NAA (2007). A medical test for noise-induced respiratory tract lesions. *Proceedings Internoise 2007*, Istanbul, Turkey. (In Press.)
Awarded the 2006 "More Prevention, Better Living" Prize for Scientific Research, attributed by the Portuguese National Institute for Safety and Health in the Workplace.
14. Castelo Branco NAA, Araujo A, Joanaz de Melo J, Alves-Pereira M (2004). Vibroacoustic disease in a 10-year-old male. *Proceedings Internoise 2004*, Prague, Czech Republic, No. 634 (7 pages).
15. Mohr GC, Cole JN, Guild E von Gierke HE (1965). Effects of low-frequency and infrasonic noise on man. *Aerospace Medicine*, 36, 817-24.
16. Ponomarkov VA, Tysik A, Kudryavtseva VI, Barer AS (1969). Biological action of intense wide-band noise on animals. *Problems of Space Biology - NASA TT F-529*, 7(May), 307-9.
17. Svigovyi VI, Glinchikov VV (1987). The effect of infrasound on lung structure. *Gigiena Truda Profissional Zabol*, 1, 34-7. (In Russian.)
18. Castelo Branco NAA (2007). Medical report on Family R. Documentation pertaining to legal proceeding in this case.
19. Alves-Pereira M, Castelo Branco MSNA, Motylewski J, Pedrosa A, Castelo Branco NAA (2001). Airflow-induced infrasound in commercial aircraft. *Proceedings Internoise 2001*, The Hague, Holland, 1011-14.

14th International Meeting
on
Low Frequency Noise and Vibration and its Control
Aalborg, Denmark 9 – 11 June 2010

Family with wind turbines in close proximity to home: follow-up of the case presented in 2007

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EXHIBIT
Int. I-25(b)
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Summary

In 2007, at the 2nd International Conference on Wind Turbine (WT) Noise, held in Lyon, France, low frequency noise (<500 Hz, LFN)-induced pathology, consistent with vibroacoustic disease (VAD), was shown to be emerging in the R. Family, exposed to residential LFN generated by 4 WT installed in close proximity (300-700 m) to their home. Herein, a follow-up is provided.

The wife and 2 children no longer reside within that home. Mr. R., however, must remain to care for the thoroughbred Lusitanian horses and bulls that he trains and breeds for bullfights. In addition to the continued deterioration of Mr. R's health and well-being, his financial situation is aggravated by the condition now appearing in his horses during the first year of life. Between 2000 and 2006, 13 healthy thoroughbred Lusitanian horses were born and raised on Mr. R's property. All horses (N=4) born or raised after 2007 developed asymmetric flexural limb deformities. WT began operations in November 2006. No other changes (constructions, industries, etc) were introduced into the area during this time.

Tissue analyses of the defected tendons were performed and revealed the classical features of LFN-induced biological responses: thickening of blood vessel walls due to proliferation of collagen in the absence of an inflammatory process.

1. Initial Disclaimer

The authors and the research team they represent would like to clarify that:
a) No member of this team is party to anti-technology sentiments;
b) Wind turbines, are considered welcome additions to modern technological society by all members of this team;
c) The data reported herein have been scrutinized under one, and only one, agenda - that of pure scientific inquiry;
d) In no way can or should this report be construed as a document arguing against the implementation of any and all wind turbines;
e) There are no commercial, financial or professional agreements (contractual or otherwise) between the accredited firms involved in noise measurements and any member of this team;
f) The consulting activities provided by these authors to Family R are of a purely academic and scientific nature and hence are pro bono*.*

2. Introduction

This report is a follow-up of a previous paper presented at the 2nd Wind Turbine Noise Conference, held in Lyon France in 2007. Herein, an update is given on the case of family R.

2.1 Background

In March of 2007, this team was contacted by an attorney-at-law representing the R. Family, in a case involving the placement of 4, 2 MW wind turbines (WT) near family R.'s property. Located between 321.8m and 642.0m from the residential building (Figs. 1, 2), the 4 WT became operational in November 2006. Two days later the R. family sought legal counsel to begin court proceedings in order to have the WT removed.

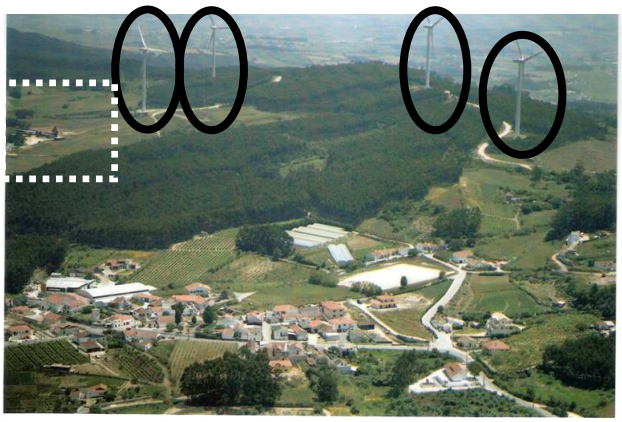


Figure 1. Aerial view of the WT home of Family R., isolated on upper left (dashed square) with the four wind turbines nearby (ovals).



Figure 2. WT home with the two of the turbines (arrows) at approximately 322m and 642m from the home.

Complaints of health effects reported in 2007:
"Mr. R has deep concerns about his memory loss, increased irritability and progressive intolerance toward audible noise, all of which he complained about at the very first meeting with this team, in March 2007. Both Mr. and Mrs. R have developed great difficulty in sleeping continuously throughout the night, as well as non-specific body pain. Upon visiting a general physician at the local State Health Center, Mr. R was prescribed 2 analgesics (anti-inflammatory and spasmolytic) and 2 tranquilizers (diazepam-based and short-term sleep-inducer) (...) In mid-March, Mr. and Mrs. R received a letter from their 12-year-old son's school, expressing concern for the growing difficulties of an otherwise outstanding student, 'particularly in English, Humanities and Physical Education. He progressed in Mathematics, which is a field that naturally attracts his type of intelligence. However, in the above mentioned coursework, it seems that the child has lost interest, makes a lesser effort, as if he were permanently tired. In Physical Education, an abnormal amount of tiredness is also observed. Is the child leading a healthy life? Does he sleep sufficient hours during the night?'" (Alves-Pereira & Castelo Branco 2007).

Noise assessments were subsequently conducted by an accredited firm hired by the R Family. Measurements were obtained in periods of 30-min, continuously for 12 days, between Apr 5th-16th, 2007. The lower limiting frequency was 1 Hz. Simultaneous and synchronized accelerometer and wind speed data were also acquired. Measurements were taken within the Master bedroom of Family R., in accordance with the procedures stipulated by Portuguese (NP 1730, Pt 1&2, 1996 and DL 9/2007, January 17th) and International Standards (ISO1996, 2003).

Figure 3 shows an example of the low frequency noise (LFN) measured within the R family home (Alves-Pereira & Castelo Branco 2007).

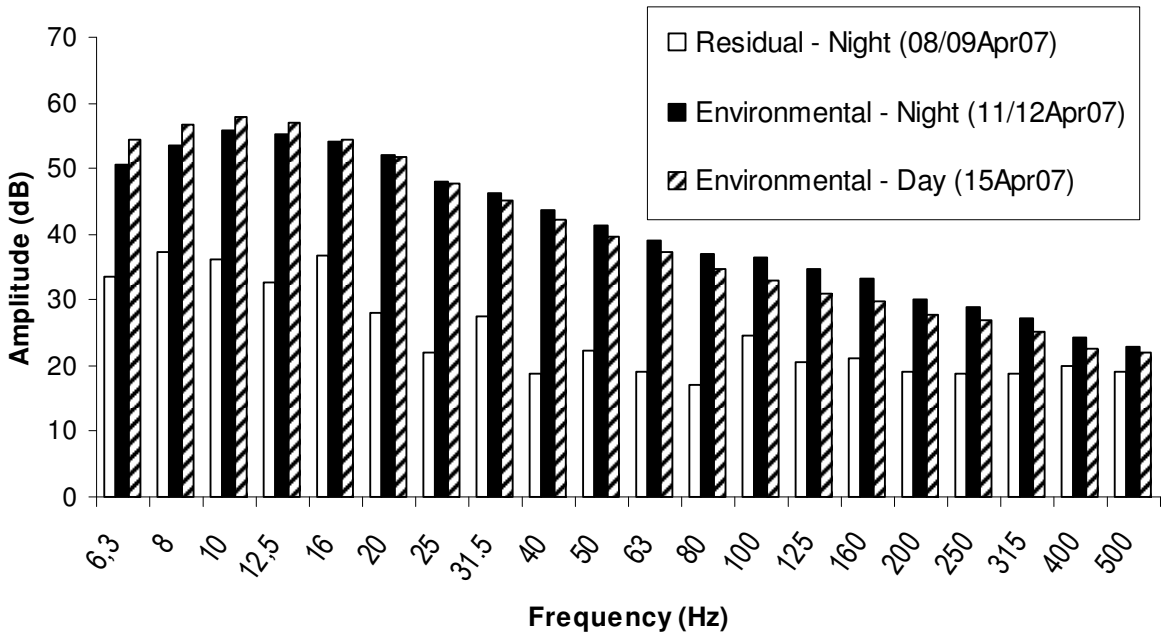


Figure 3. Comparison of 1/3 octave ILFN levels, in dBLinear, of the *Residual* (no WT blade movement) measurement and the *Environmental* (with rotating WT blades) measurement in the WT home Master Bedroom, with the same recorded wind speed.

The R. Family income is provided by breeding bulls and raising and training thoroughbred Lusitanian horses for bullfights. In 2007, Mr. R reported that his horses were exhibiting abnormal behaviours, such as lying down and sleeping during the day.

2.2 LFN-induced pathology

The biological effects of LFN exposure have been under study by this team for the past 3 decades. As a result, vibroacoustic disease (VAD) was defined as a LFN-induced extra-auditory pathology, characterized by the abnormal growth of collagen and elastin fibers in the absence of an inflammatory process (Castelo Branco 1999, Holt 2001, Castelo Branco & Alves-Pereira 2004, Alves-Pereira & Castelo Branco 2007b). Objective diagnostic tests to evaluate the severity of LFN-induced lesions have already been identified and were offered to the R family.

Echocardiograms of Mr. and Mrs. R. disclosed slight to moderate pericardial thickening (between 1.7 mm and 2.0 mm, normal for the equipment in use: <1.2mm). Respiratory drive was below normalized values in both adults (46%-53%, normal: >60%), suggesting the existence of brain lesions in the areas responsible for the neurological control of breathing.

Given the aforementioned school information, and since cognitive and memory disturbances are common and well documented in VAD patients (Castelo Branco 1999, Castelo Branco & Alves-Pereira 2004), the child received a neurophysiological evaluation.

Brainstem auditory evoked potentials disclosed asymmetries in the right and left nerve conduction times, and the right I-V interlatency value was at the threshold of normal values (4.44 ms). The endogenous evoked potential P300 recording occurred at 352 ms (normal: 300 ms). This measure reflects the time it takes to recognize and memorize infrequent stimuli. “Although this result is in accordance with the child’s

school report indicating that cognitive processes are affected, it is not possible to state that this situation is irreversible. Moreover, in children, P300 recordings often disclose variations that are difficult to interpret. Nevertheless, initial clinical signs of Stage-I VAD are characterized by this type of cognitive impairment and, as such, may be reversible at this clinical stage provided low frequency noise exposure is suspended" (Castelo Branco 2007).

3. Follow-up of Legal Proceedings

Legal proceedings are still ongoing. Nevertheless, given the gravity of the situation, the court has ordered that WT be shut down during evening and night hours (8 pm - 7 am), and that the WT closest to the home (322 m, Fig. 2) be permanently shut down. It was subsequently removed by its owners (See Figure 4A). Despite the court's decision, more WT continue to be installed in the immediate vicinity surrounding the R family home (See Figure 4B).



A B

Figure 4A and 4B. Aerial view of WT near Mr. R's farm in 2010. The long arrow (white) pointing upward indicates the site from where 1 of the 4 original WT was removed by its owners after a court order decreed that it be permanently shut down. (See Figure 1.) The circles indicate the remaining 3 WT of the original 4 and which, by court order, are forbidden to operate between 8 pm and 7 am. The smaller arrows (red) on the left pointing downward indicate WT installed after legal proceedings were initiated in 2007. The home is outlined by the dashed square.

4. Follow-up of Family Health

In September of 2007, after summer vacation during which the 12-year-old child was away from his home for 2 months, the neurophysiology results improved: endogenous evoked potential P300 recording occurred at 302 ms (normal: 300 ms). Mrs. R and the 2 children no longer reside within that home, having moved to a residential suburban area near Lisbon. Today, this child is again an outstanding student, at the top of his class.

Mr. R, however, must remain to care for the thoroughbred Lusitanian horses and bulls that he trains and breeds for bullfights. His health situation is continuously and visibly deteriorating. Intolerance to noise has become more severe. Situations compatible with an unregulated sympathetic nervous system have become frequent. Mr. R is currently undergoing a battery of neurophysiological tests which include multimodal evoked potentials, brain-mapping under different stimuli, and sleep studies. Mr. R's cognitive impairment has become more pronounced.

5. Equine Flexural Limb Deformities

Equine flexural limb deformities (EFLD) are clinical conditions that can occur in horse limbs causing the inability to place the hoof flatly on the ground. EFLD is often referred to as “tip-toeing”, because the hoof seems to be tilted off the ground, as if the horse were tip-toeing. Boxy foot or club foot are other common names for this condition.

EFLD can be caused by a variety of factors, such as congenital, nutritional, infectious and traumatic (Auer and Stick 2007). In adult horses, EFLD are mainly caused by infections or traumatic events. In foals, EFLD usually develop within 6 weeks to 8 months of age, and are most often a consequence of the inability of the deep digital flexor tendon to follow bone growth during the rapid growth period that occurs during the first year of life.

Treatment of EFLD does not have a bad prognosis when applied in a timely fashion. If at a sufficiently early age, splints complemented by medication may be used to help the foal straighten the limb, thus promoting the extension of the deep digital flexor tendon while accompanying bone growth. More severe situations may require a surgical procedure (desmotomy) to sever the accessory ligament of the deep digital flexor tendon (check ligament), thus releasing the flexor tendon and allowing it to take its position. In the most severe cases, the deep digital flexor tendon must also be severed. EFLD are highly undesirable situations, particularly for thoroughbreds, most often leading to permanent disability of the adult horse.

5.1. Horses born and raised on Mr. R’s farm

Between 2000 and 2006, 13 healthy thoroughbred Lusitanian horses were born and raised on Mr. R’s property.

All 4 horses born or raised after 2007 developed asymmetric EFLD. Figures 5 and 6 illustrate this condition.



Figure 5. *Espartaco* (Born 02May09, father: Zircão, mother: Vassoura.) Thoroughbred Lusitanian born and raised on Mr. R’s farm, exhibiting a normal right hoof and pronounced EFLD in the left limb.



Figure 6. *Engenheiro* (Born 17May09, father: Zircão, mother: Zizi.) Thoroughbred Lusitanian born and raised on Mr. R’s farm, exhibiting a normal left hoof and pronounced EFLD in the right limb.

All horses before and after 2007 had the same quantity and quality of diet, experienced the same housing conditions, and were submitted to the same daily exercise routines.

6. Tissue Analysis

Given the prior knowledge this team has acquired regarding expected outcomes of LFN exposure, and in an attempt to understand the remarkable frequency of EFLD in Mr. R's farm, biopsies were obtained from five different foals, as described in Table 1.

Table 1. Description of horses from which biopsies were obtained.

Case No.	Name & Birth Date	Origin	Procedure
1 Corrective Surgery	Canela 26FFEB07	Father: Operário Mother: Juvita *Born on Mr. R's farm*	EFLD corrective surgery with desmotomy of the check ligament in MAR08. After surgery, was allowed to spend time outdoors. Biposy obtained in MAR10.
2 Corrective Surgery	Desplante 02APR08	Father: Importante, Mother: Vassoura, *Acquired by Mr. R from Herdade das Silveiras in JUN09. At the same time, Vassoura was also acquired by Mr. R from the same breeders.*	Came to Mr. R's farm at age of 15 days. EFLD corrective surgery with desmotomy of the check ligament in JUN09. Biopsy obtained at the time of surgery in JUN09. After surgery, maintained in enclosed area.
3 (Control)	Dondoca 04ABR08	Father: Urânio Mother: Scalabitana. *Acquired by Mr. R from Olívio Pedrosa Marques in JUN09.*	Came to Mr. R's farm at the age of 14 months. Served as a control sample. Biopsy of left limb check ligament in JUN09.
4 Biopsy only	Espartaco 02May09 (See Fig. 5)	Father: Zircão, Mother: Vassoura. *Born on Mr. R's farm*	Biopsy of check ligament obtained in MAR10. Due to ongoing legal proceedings, no corrective surgery was performed.
5 Biopsy only	Engenheiro 17May09 (See Fig. 4)	Father: Zircão, Mother: Zizi. *Born on Mr. R's farm*	Biopsy of check ligament obtained in MAR10. Due to ongoing legal proceedings, no corrective surgery was performed.

Case 1 was born on Mr. R's farm in February 2007, developed EFLD and received corrective surgery in March 2008. After surgery, she was allowed to exercise in the outdoors. Today, the untreated limb has also developed asymmetrical EFLD and the treated limb seems to be recuperating (Figure 7). Check ligament biopsy was obtained in March 2010.

Case 2 (and his mother) was purchased by Mr. R in April 2008 and arrived at Mr. R's farm at the age of 15 days. EFLD developed over several months (Figure 9) and, in June 2009, corrective surgery was performed (Figure 10). After surgery, and unlike Case 1, this horse was not allowed to remain outdoors and was kept in the enclosed area. Today, no signs of EFLD are visible in either limb (Figure 8).



Figure 7. Case No. 1 (*Canela*) in March 2010. After surgery, she was allowed to go outdoors. EFLD is visible in both limbs.



Figure 8. Case No. 2 (*Desplante*) in March 2010. After surgery, he was maintained in enclosed area. No sign of EFLD is visible.



Figure 9. Case No. 2 (*Desplante*) before surgery. EFLD is visible in both forelimbs. Surgery was performed on left limb due to the larger severity in that limb.



Figure 10. Case No. 2 (*Desplante*). Desmotomy and biopsy of the check ligament of the left limb by author Costa Pereira (June 2009).

By June 2009, it had already been decided that biopsies would be taken for study. Within this scope, Mr. R purchased Case 3, from an entirely different breeder and age-matched with Case 2, in order to serve as control sample. In June 2009, check ligament biopsy was obtained from this horse.

Cases 4 and 5 (Figs. 5, 6) were born on Mr. R's farm and developed asymmetrical EFLD. Biopsies were obtained March 2010. No corrective surgery was performed on these two equines. Based on studies conducted by Auer and Stick (2006), no foal surgically treated for EFLD after 8 months of age had a favorable outcome.

Tissue fragments were prepared for light microscopy using standard gomori e trichromic masson stainings.

Check ligament fragments had normal appearances in all samples. In the three foals born and raised on Mr. R's farm (Cases 1, 4, 5), blood vessel walls were abnormally thickened due to the presence of collagen. Case 2 also disclosed collagen-thickened blood vessel walls. No such images were visible in fragments from Case 3. Cells normally associated with inflammatory processes were absent from all images.

7. Discussion

There are certainly a variety of pathways that can lead to incorrect positioning of the deep distal flexor tendon in equine forelimbs. Of the 13 foals born on Mr R's property before 2007, none developed EFLD. Since 2007, the 3 foals born and raised on this farm, all have developed EFLD. The foal purchased by Mr. R from Herdade das Silveiras, a prestigious breeder of Lusitanian thoroughbreds, arrived at Mr. R's farm at the age of 15 days and began showing signs of EFLD after several months (Case 2). This suggests that the agents responsible for EFLD were not present on Mr. R's farm until 2007, after which they seem to have started to influence the onset of EFLD. Three new foals more were born in 2010 but are, as yet, too young to manifest any visible signs of EFLD. Three mares are currently pregnant.

After surgery, Case 1 was allowed to remain in the outdoor pastures, while Case 2 was not. Mr. R's farm possesses a closed riding arena, adjacent to the stable area. Case 2 was exercised exclusively in this enclosed area. Case 1 has subsequently developed EFLD in the untreated limb while Case 2 has not. It would seem that the remaining within enclosed spaces might influence the onset of EFLD.

The asymmetrical development of EFLD in the forelimbs is commonly observed in most forms of EFLD, and is most probably related to the slightly asymmetrical load the equine places on one limb relative to the other when experiencing some degree of pain or discomfort. The asymmetry is associated with the degree of severity since, in most acquired EFLD, the onset is usually bilateral (Auer and Stick 2006). Other diseases affecting equine forelimbs can also develop first on one side and then on the opposite limb (navicular disease, for example).

Tissue analysis revealed:

- thickened blood vessel walls in the 3 foals born and raised on Mr. R's farm (Cases 1, 4, 5)
- thickened blood vessel walls in the foal who arrived on the farm at 15 days of age (Case 2)
- absence of thickened blood vessel walls in the equine age-matched with Case 2 and purchased from a different breeder.

These morphological changes in the absence of inflammatory processes are consistent with observations made over the past 30 years in autopsy, biopsy material from LFN-exposed individuals, and tissue fragments of LFN-exposed laboratory animals (Alves-Pereira & Castelo Branco 2007, for example). This particular type of tissue reorganization is the hallmark of LFN-induced response.

Since the year 2000 until today, the only significant change in the area of Mr. R's farm has been the installation of WT. No gas pipelines, large industrial plants, highways, railways, transportation terminals, or mining activities have been introduced in the vicinity within the last decade. The area is officially classified as rural. Under these circumstances, it is sensible to suspect that LFN is playing a role in the pathways leading to the onset of this form of EFLD.

8. Conclusions

This is a follow-up to the 2007 case of family R, exposed to LFN generated by WT, and who had begun to develop signs of LFN-induced pathology consistent with vibroacoustic disease. Although Mrs. R and both children no longer reside in this home and have been regaining their health, Mr. R must remain to care for thoroughbred Lusitanian horses and bull that he breeds for bullfighting. As a result,

his health has deteriorated rapidly, and now includes a more pronounced cognitive impairment and noise intolerance.

The pattern of onset of equine flexural deformities before and after the installation of WT in the farm's vicinity, led to check ligament biopsies of 4 foals raised on Mr. R's farm, and of 1 foal purchased by Mr. R from another breeder. It was shown that all 3 foals raised on Mr. R's farm disclosed the classical signs of LFN-induced responses: thickening of blood vessel walls due to proliferation of collagen in the absence of inflammatory processes. The purchased yearling foal did not exhibit these morphological features.

The results presented herein strongly suggest that the presence of LFN-generating WT in the vicinity of this horse breeding farm can play a significant role in the triggering and onset of equine flexural limb deformities.

References

M Alves-Pereira, NAA Castelo Branco. "On the impact of infrasound and low frequency noise on public health - two cases of residential exposure", RLCTS, 4(2), 186-200, 2007a.
http://revistas.ulusofona.pt/index.php/revistasaude/article/viewFile/670/564

M Alves-Pereira, NAA Castelo Branco NAA. "Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signalling", Progress Biophysics & Molecular Biology, 93:256-279, 2007b.

JA Auer, JA Stick (Eds): "Equine Surgery" (3rd Ed.). St. Louis: Saunders Elsevier, pp 1150-1165, 2006.

NAA Castelo Branco. "The clinical stages of vibroacoustic disease", Aviation, Space & Environmental Medicine, 70 (3, Suppl): A32-9, 1999.

NAA Castelo Branco, M Alves-Pereira. "Vibroacoustic disease", Noise & Health, 6(23): 3-20, 2004.

NAA Castelo Branco. "Medical report on Family R". Documentation pertaining to legal proceedings in this case, 2007.

BD Holt. "The pericardium". In: V Furster , R Wayne Alexander, F Alexander, editors. Hurst's The Heart. New York: McGraw-Hill Professional Publishing, pp2061-82, 2001.

Low Frequency Noise-Induced Pathology: Contributions Provided by the Portuguese Wind Turbine Case

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Summary
In November 2006, 4 Industrial Wind Turbines (IWT) were installed in the vicinity of a residential dwelling in Portugal. In March 2007, this team was contacted by the family requesting assistance in dealing with their Infrasound & Low Frequency Noise (ILFN) problem that they claimed was being generated by the IWT. The family began legal proceedings for the removal of the IWT, and in September 2007, this team's first report was presented at the 2nd International Meeting on Wind Turbine Noise. In June 2010, a follow-up report of this case was presented at the 14th International Meeting on Low Frequency Noise and Vibration and its Control, wherein ILFN-induced pathology was confirmed through histology in this family's thoroughbred horses. The goal of this report is to provide second follow-up to this case, five years later.

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1. Introduction

(Please see disclaimer notice provided at the end of this report.) Over the past 35 years, this team of scientists has studied the biological response to Infrasound & Low Frequency Noise (ILFN) exposure in both human populations [1-4] and laboratory animal models [5-7]. Initially prompted by the signs and symptoms observed among a population of aeronautical technicians [3], by the mid-1990's, study populations included commercial airline pilots and flight attendants [8,9]. In 2001, the team began receiving complaints from families regarding ILFN in their homes [10], and subsequently the inquiry was widened to include residential exposures [11]. By 2007, this team had already accumulated a vast body of knowledge regarding the biological effects of ILFN exposure, including a clinical protocol for establishing the extent of ILFN-induced pathology [1,2,12]. It is within this context that private citizens worldwide have requested our assistance with their ILFN-related issues.

2. First Contact in March 2007

In November 2006, 4 2-MW Industrial Wind Turbines (IWT) were installed in the vicinity of the farm of Family R. This rural dwelling also housed the family's business: breeding thoroughbred Lusitanian horses and bulls for bullfighting events. The IWT were installed at a distance of 321-642 m from the residential home. Complaints of sleep disturbances were first reported in December 2006. In mid-March, Mr. and Mrs. R received a letter from their 12-year-old son's schoolteacher, expressing concern for the growing difficulties in an otherwise outstanding student, "*particularly in English, Humanities and Physical Education. He progressed in Mathematics, which is a field that naturally attracts his type of intelligence. However, in the above mentioned coursework, it seems that [the child] has lost interest, makes a lesser effort, as if he were permanently tired. In Physical Education,*

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an abnormal amount of tiredness is also observed. Is [the child] leading a healthy life? Does he sleep sufficient hours during the night?" This immediately prompted the parents to begin legal proceedings and seek medical assistance, and thus, this team's first contact with Family R. During the month of April 2007, Family R hired an accredited acoustical firm to conduct continuous acoustical monitoring of ILFN both inside and outside their home, for a period of 2 weeks, and that included real time wind speed data. Legally stipulated annoyance levels were exceeded during the day (7am-8pm), evening (8-11pm) and night (11pm-7am) hours. dBA noise levels were also exceeded for a sensitive zone during night hours, but were within legal limits for a mixed zone. Municipal Authorities where Family R.'s property is located had not yet classified the area as sensitive *or* mixed. Numerical data regarding acoustical and wind speed information, independently collected by the accredited firm, was provided to this team for analysis. Figure 1 provides a visual comparison of ILFN levels inside the master bedroom under different conditions.

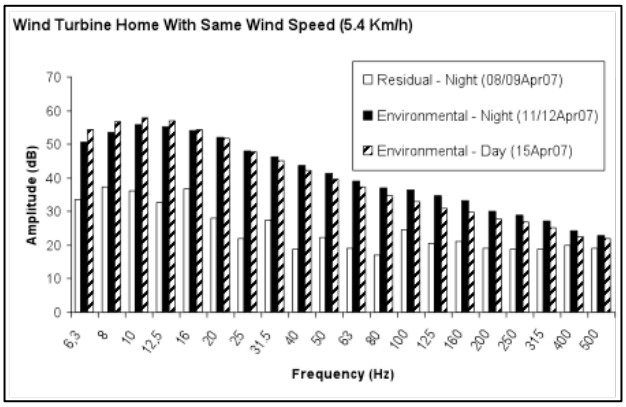


Figure 1. Comparison of ILFN levels in 1/3 octave bands, in dBL, *at the same windspeed*, and recorded inside the master bedroom. Residual (no IWT blade movement); Environmental (with rotating IWT blades) [13].

These ILFN levels were compared to those obtained in other ILFN-rich residences and occupational environments [13], and were deemed to be problematic for the health of this family. In order to obtain relevant clinical data, specific medical examinations were provided. For the rationale justifying the medical examinations reported herein, please see [1-3, 12]. The 12-year-old child received a neurological test assessing cortical nerve conduction times: P300 Event Related Evoked Potentials (ERP). P300 ERP disclosed nerve conduction time to be 352 ms, when expected value should be closer to 300 ms. Brainstem Auditory Evoked Potentials (BAEP) disclosed asymmetries in the right and left nerve conduction times, and the right I-V interval interlatency value was at the threshold of normal (4.44 ms). Mr. and Mrs. R. disclosed slight to moderate pericardial thickening: between 1.7 mm and 2.0 mm (normal for the equipment in use: <1.2mm) [12]. Respiratory drive was below normalized values in both adults (46%-53%, normal: >60%), suggesting the existence of brain lesions in the areas responsible for the neurological control of breathing [14]. Observations made by the family included animal behavioural changes: Horses were seen to lie down and sleep during the day; Dogs were lethargic, and no longer jumped up requesting attention from their owners; Ants simply disappeared. All data was presented at the 2nd International Meeting on Wind Turbine Noise, held in Lyon, France in September 2007, where this team concluded that precautionary measures regarding the placement of IWT near residential dwellings were justified, and that safe distances between IWT rotating blades and inhabited buildings had not yet been determined by the scientific community [13].

3. 2010 Update and Equine Pathology

By 2010, Mrs. R and the children no longer resided in their rural home, having moved to a suburban apartment. For Mr. R, however, this was not an option given the care required by thoroughbred Lusitanian horses and prize bulls that, as his only source of income, he bred and trained for bullfights. After the summer vacation in 2007, spent away from the farm, the 12-year-old child had again received the P300 ERP examination that, this time, disclosed nerve conduction times much closer to normal: 302 ms. In 2010, this child was again an outstanding student, top of his class. Over these 3 years, Mr. R's health and wellbeing had continuously and visibly deteriorated: intolerance to (any) noise had become more severe; situations compatible with an unregulated sympathetic nervous system increased in frequency; and cognitive impairment became more pronounced. As a result of the first phase of the (still ongoing) legal proceedings, IWT were

ordered to be shut down during evening and night hours (8 pm - 7 am), and the IWT closest to the home (322 m) was ordered to be removed. Meanwhile, however, more IWT continued to be installed in the immediate vicinity surrounding the R family home. Legal proceedings underway refer only to the first 4 IWT. The R family was now being confronted with yet another problem that would severely aggravate their already delicate financial situation: a condition was now developing in the horses during the first year of life that was substantially reducing the family's equine income. Between 2000 and 2006, 13 healthy thoroughbred Lusitanian horses were born and raised on Mr. R's property. All horses (N=4) born after 2007 on his farm developed asymmetric flexural limb deformities. Besides the IWT installed in November 2006, no other changes (constructions, industries, etc) were introduced into the area during this time. Detailed results from the tissue study carried out among these equines and their corresponding controls were presented at the 14th International Meeting on Low Frequency Noise and Vibration and its Control, held in Aalborg, Denmark, in June 2010 [15]. One of this team's most consistent findings over the past 30 years of histological and ultrastructural analyses of human and animal ILFN-exposed tissues has been the organized proliferation of collagen and elastin fibers in the absence of an inflammatory process [1,2,5,6]. This same morphological feature appeared in the tissue fragments of the horses that were born, *or raised*, on Mr. R's farm after 2007 [15]. Equine flexural limb deformities developed by Mr. R's horses was the object of a Master's Thesis defended at the School of Veterinary Medicine, Technical University of Lisbon in 2012 [16].

4. 2015 Update

In terms of legal proceedings, in May 2013 the Supreme Court of Justice of Portugal decided that the remaining 3 IWT had to be removed from the vicinity of Mr. R's property [17]. The developer is reportedly appealing to the European Courts. Given the installation of other IWT in the immediate vicinity, the R Family property will still potentially be impacted by ILFN, although new studies to that effect would be required. Mr. R continues to live away from Mrs. R and the children, and his health has further deteriorated. The respiratory drive value that in 2007 was 46% (normal: >60%) is now at 28%. The development of balance disturbances associated with loss of consciousness has apparently caused several falls, requiring medical treatment for facial and rib fractures. This situation is still under clinical study, as late-onset epilepsy is one of the most severe outcomes of excessive ILFN exposure. However, diagnosis of this neurological disorder requires time to be properly established within a clinical context. While disconcerting, this is unsurprising given the already documented cases of balance disturbances and late-onset epilepsy among ILFN-exposed persons [18-20]. To date, the only remarkable element in the severe degradation of Mr. R's health is its rapidity, when compared to the evolution of this pathology seen in workers exposed to occupational ILFN. Ultimately, though, this observation is unsurprising, given the distinct time-exposure patterns of residential vs. occupational exposures.

5. The Scientific Approach

This IWT case is but one of many residential ILFN cases with which this team has been confronted, i.e., IWT are but one in a long list of ILFN-generating industries capable of disturbing citizens' right to a healthy physical environment in their homes. The approach applied to this IWT case is consistent with this team's long-standing methodology: confirmation of the presence of the agent of disease (ILFN), and clinical assessment of the parameters deemed relevant by 30 years of past research. Other agencies and institutions have studied health impacts caused by the proximity of IWT to residential dwellings [21,22 for example]. Apparently, however, all have chosen subjective and clinically unreliable measures to characterize "health impacts," unsurprisingly leading to many inconclusive results. Hypersensitivity to noise or intolerance toward sounds, commonly called *annoyance*, is frequently considered a measure of health. Usually there are large variations in the levels of annoyance assessed among family members living in the same ILFN-rich home, and among families and neighbours living in clusters of homes under the same ILFN exposure conditions. Since no prior noise exposure histories are generally included in these "health impact" studies, the fact that annoyance levels are directly related to prior and overall noise exposure

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histories remains, therefore, unaccounted for. The same lack of information occurs for concurrent occupational or recreational exposures – no information of this type is generally collected among these populations. But even if annoyance levels were correctly assessed, their relationship to a proper "health impact" remains a mystery, with the exception of the relatively vague notion of "quality of life". Medical scholars trained in the scientific method know that the measure of annoyance *is not* a clinically objective parameter. Hence, any statement regarding "health impacts" merely based on (incomplete) assessments of annoyance levels is, at best, perplexing. Medical scholars trained in epidemiology will also recognize that transverse studies are quite insufficient to establish causality (or lack thereof) between an agent of disease and a specific pathology. This is particularly true when prior exposures to that same agent of disease under study are not taken in account. Having put forth why annoyance is an improper measure, both in terms of its extraordinary subjectivity and of its clinical uselessness, it is now pertinent to share this team's (long) experience with the medical concept of annoyance, which was termed in 1984 as hypersensitivity to noise or intolerance to sound [23]. Normal Wistar rats, independent of age, react to the sound of a blown kiss by becoming tense and discretely irritated. In rats exposed for extended periods of time to ILFN, the sound of a blown kiss produces a dramatic startle response, some of them losing balance, and falling backwards [24]. Fusion of actin-based, cochlear stereocilia, both amongst themselves as well as with the upper tectorial membrane, was observed in ILFN-exposed rat cochlea. These results were presented at the 8th International Meeting of the Commission on the Biological Effects of Noise, held in Rotterdam, The Netherlands, in 2003, and wherein it was postulated that the fusion of these actin-based structures could be an organic basis for the hypersensitivity to noise and intolerance to sound observed in ILFN-exposed populations, i.e., an organic-based hypothesis for annoyance [24,25]. If so, it would follow that annoyance is the product of cumulative exposures to ILFN, and not necessarily a hereditary trait related to an oversensitive auditory function. If this were indeed the case, however, *the needs of the many outweigh the needs of the few* would justify the exclusion of this "sensitive" segment of the population from the decision-making process.

6. Looking Ahead

Mr. R is not alone when it comes to life disruption due to the installation of IWT in the proximity of a residential home. This problem has become a societal issue compelling an intelligent approach, rather than a crystallized view based on antiquated notions and concepts. It is clear that the great motivation for marshalling energy and effort toward industrial wind farms is legitimately based on a worldwide shortage of electrical energy. It would seem, however, that under the guise of *the needs of the many outweigh the needs of the few*, humans are collectively forgetting their own history that has unequivocally shown the benefits of dealing with adverse health effects outright, rather than later. The dichotomy stipulating that either one accepts the development of adverse health effects among certain segments of the population, *or*, one does not have sufficient electrical energy for all the modern gadgets (including cars), is archaic and fictitious. An effort toward developing and implementing appropriate construction techniques that would minimize the deleterious effects of in-home ILFN could be, perhaps, an excellent beginning. The hindrance to this apparently viable beginning is the *sine qua non* prior recognition that ILFN is, *de facto*, a physical agent of disease.

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References

[1] M. Alves-Pereira, N.A.A. Castelo Branco: Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling. Progress Biophysics & Molecular Biology 93 (2007) 256-279. www.academia.edu/10671962/Vibroacoustic_disease_Biological_effects_of_infrasound_and_low-frequency_noise_explained_by_mechanotransduction_cellular_signalling.

[2] N.A.A. Castelo Branco, M. Alves-Pereira: Vibroacoustic disease. Noise & Health 6 (2004) 3-20.

[3] N.A.A Castelo Branco: The clinical stages of vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A32-9.

[4] N.A.A Castelo Branco: Vibroacoustic disease – An emerging pathology. Aviation, Space & Environmental Medicine 70 (1999) A1-6.

[5] N.A.A. Castelo Branco, J. Reis Ferreira, M. Alves-Pereira: Respiratory pathology in vibroacoustic disease-25 years of research. Revista Portuguesa de

Pneumologia XIII (2007) 129-135. www.scielo.oces.mctes.pt/pdf/pne/v13n1/v13n1a08

[6] N.A.A. Castelo Branco, E. Monteiro, A. Costa e Silva, J. Reis Ferreira, M. Alves-Pereira. Respiratory epithelia in Wistar rats born in low frequency noise plus varying amounts if additional exposure. Revista Portuguesa de Pneumlogia IX (2003) 481-492.

[7] M.J.R. Oliveira, A. Sousa Pereira, A.P. Águas, E. Monteiro, N.R. Grande, N.A.A. Castelo Branco. Effects of low frequency noise upon the reaction of pleural milky spots to mycobacterial infection. Aviation, Space & Environmental Medicine 70 (1999) A137-40.

[8] A. Araujo, F. Pais, J.M.C. Lopo Tuna, M. Alves-Pereira, N.A.A. Castelo Branco: Echocardiography in noise-exposed flight crew. Proc. Internoise 2001, 1007-10 (ISBN: 9080655422).

[9] M. Alves-Pereira, M.S.N. Castelo Branco, J. Motylewski, E. Kotlicka, A. Pedrosa, N.A.A Castelo Branco: Airflow-induced infrasound in commercial aircraft. Proc. Internoise 2001, 1011-14 (ISBN: 9080655422).

[10] R. Torres, G. Tirado, A. Roman, R. Ramirez, H. Colon, A. Araujo, F. Pais, J.M.C. Lopo Tuna, M.S.N. Castelo Branco, M. Alves-Pereira, N.A.A Castelo Branco: Vibroacoustic disease induced by long-term exposure to sonic booms. Proc. Internoise 2001, 1095-98 (ISBN: 9080655422).

[11] A. Araujo, M. Alves-Pereira, J. Joanaz de Melo, N.A.A. Castelo Branco: Vibroacoustic disease in a ten-year-old male. Proc. Internoise 2004, No. 634 (7 pages) (ISBN: 8001030555).

[12] N.A.A. Castelo Branco, M. Alves-Pereira, A.J.F. Martinho Pimenta, J. Reis Ferreira: Clinical protocol for evaluating pathology induced by low frequency noise exposure. Euronoise 2015, paper no. 601.

[13] M. Alves-Pereira, N.A.A. Castelo Branco: In-home wind turbine noise is conducive to vibroacoustic disease. Proc. 2nd Intl Meet Wind Turbine Noise, 2007, Paper No. 3 (11 pages).

[14] J. Reis Ferreira, J. Albuquerque e Sousa, P. Foreid, M. Antunes, S. Cardoso, M. Alves-Pereira, N.A.A. Castelo Branco: Abnormal respiratory drive in vibroacoustic disease. Revista Portuguesa de Pneumologia XII (2006) 369-74. www.scielo.oces.mctes.pt/pdf/pne/v12n4/v12n4a03

[15] N.A.A. Castelo Branco, T. Costa e Curto, L. Mendes Jorge, J. Cavaco Faísca, L. Amaral Dias, P. Oliveira, J. Martins dos Santos, M. Alves-Pereira: Family with wind turbines in close proximity to home: follow-up of case presented in 2007. Proc. 14th Intl Meet Low Frequency Noise, Vibration and its Control 2010, 31-40.

[16] T. Costa e Curto: [Acquired flexural deformity of the distal interphalangic joint in foals]. Masters Thesis. School of Veterinary Medicine, Technical University of Lisbon (2012) (In Portuguese). www.repository.utl.pt/bitstream/10400.5/4847/1/Deformaçao%20flexural%20adquirida%20da%20articulaçao%20interfalangica%20distal%20em%20poldros.pdf

[17] Supreme Court of Justice of Portugal. Decision No. 2209/08.oTBTVD.L1.S1, 30 May 2013 (In Portuguese). www.dgsi.pt/jstj.nsf/954f0ce6ad9dd8b980256b5f003fa814/4559d6d733d1589780257b7b004d464b?OpenDocument

[18] A.J.F. Martinho Pimenta, M.S.N. Castelo Branco, N.A.A. Castelo Branco: Balance disturbances in individuals with vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A100-6.

[19] A.J.F. Martinho Pimenta, N.A.A. Castelo Branco: Epilepsy in vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A119-21.

[20] GIMOGMA*: [Epilepsy of vascular etiology, a clinical picture of vibration disease? Revista Portuguesa de Medicina Militar 32 (1984) 5-9.

[21] Executive Office of Energy and Environmental Affairs of the State of Massachusetts, USA: Wind turbine health impact study: Report of an independent expert panel. January 2012. www.mass.gov/eea/docs/dep/energy/wind/turbine-impact-study.pdf

[22] Health Canada: Wind turbine noise and health study – Summary of results. Department of the Government of Canada. 2014. www.hc-sc.gc.ca/ewh-semt/noise-bruit/turbine-eoliennes/summary-resume-eng.php

[23] GIMOGMA*: [Noise and vibration at the origin of hypo- and hyper acoustic sensitivities in an industrial population]. Revista Portuguesa de Medicina Militar 32 (1984) 17-20 (In Portuguese).

[24] N. Lousã, E. Monteiro, M. Alves-Pereira, N.A.A. Castelo Branco: Rat cochlea exposed to low frequency noise. Proc. 8th ICBEN, 2003, 43-5 (ISBN: 9080799017).

[25] M. Alves-Pereira, J. Joanaz de Melo, N.A.A. Castelo Branco: Actin- and tubulin-based structures under low frequency noise stress. In: A. Méndez-Vilas (ed.). Recent Advances in Multidisciplinary Applied Physics. Elesevier, London, 2005, pp. 955-79 (ISBN: 9780080446486).

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Disclaimer

The authors and the research team they represent would like to clarify that:

No member of this team is party to anti-technology sentiments;

Alternative forms of renewable energy, such as industrial wind turbines, are considered welcome additions to our modern technological society, by all members of this team;

The data reported herein have been scrutinized under one, and only one, agenda - that of pure scientific inquiry;

In no way can or should this report be construed as a document arguing for or against the implementation of industrial wind turbines;

No member of this research team is employed by the firm that conducted the acoustical measurements reported in this article, nor are there any commercial, financial or professional agreements (contractual or otherwise) between the aforementioned accredited firm and any member of this team;

The consulting activities provided by these authors to Family R are of a purely academic and scientific nature and hence are *pro bono*.

Clinical Protocol for Evaluating Pathology Induced by Low Frequency Noise Exposure

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Summary
Segments of the general population who complain about infrasound & low frequency noise (ILFN) in their homes or in their workplaces continue to increase. These individuals often complain about similar sets of concurrent symptoms, and frequently attribute their ailments directly to ILFN exposure. Oftentimes, however, routine clinical evaluations of these individuals reveal no apparent dysfunction, and patients with persistent complaints are subsequently referred to psychology or psychiatry health professionals. The goal herein is to present an objective clinical protocol that scientifically evaluates these complaints, leading to the elimination of malingerers, and to the proper medical assistance of those developing ILFN-induced lesions.

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Introduction

Since 1980, this team of scientists has studied the biological response to Infrasound & Low Frequency Noise (ILFN) exposure in both human populations [1-3] and laboratory animal models [4,5]. Over the decades, a valid clinical protocol has been established in order to ascertain a differential diagnosis for ILFN-exposed persons. The goal of this report is to list the medical diagnostic complementary tests relevant for evaluating ILFN-induced pathology. The rationale justifying each diagnostic test included in this ILFN clinical protocol will be but briefly described; extensive details can be found in the references provided.

2. Vibroacoustic Disease

The nosological entity triggered by excessive exposure to ILFN has been termed vibroacoustic disease (VAD) [1-3]. Despite the overwhelming antagonism with this nomenclature [6, for example], this time these authors have opted to use this specific name rather than "ILFN-induced pathology." VAD is a whole-body pathology characterized by proliferation of extra-cellular matrices (collagen and elastin) in the absence of an inflammatory process. VAD is a consequence of the disruption and restructuring of tissue and cellular components in order to maintain functional structural integrity in the presence of a mechanical agent of disease [further refs in 1]. The onset of VAD is insidious and frequently misdiagnosed. Individual ILFN exposure patterns dictate the time-evolution and ultimate severity of lesions, i.e., exposure times vs. recovery periods is of crucial importance, both for a prognostic view and for an appropriately detailed clinical history of ILFN exposure. Generally, ILFN exposure can be occupational or residential. With occupational exposures, recovery periods are usually certain, given the scheduled end of the workshift and the existence of weekends. Residential exposures, however, do not enjoy this type of recovery periods. The onset of VAD among individuals who are exposed to ILFN in their homes has been observed to be more rapid than those who are exposed to occupational ILFN. Over the past decades, there has been much controversy regarding several issues related to human health

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and noise exposure, such as, a) the role and range of the human auditory system in ILFN-rich environments; b) the physiological pathways responsible for inadequate quality of sleep, for hypersensibility and intolerance to sound, and for cardiovascular disease; and c) the parameters used for measuring ILFN, and for assessing health impacts on ILFN-exposed populations. These issues have been extensively addressed by this team of researchers, and are currently beyond the scope of this report. The clinical protocol herein defined will provide family physicians and epidemiologists with objective, useful and clinically valid data that could ensure appropriate medical assistance to potential VAD patients.

3. Clinical Protocol for ILFN Exposures

3.1. Fundamentals

Auditory effects of ILFN exposure differ from the classical picture of hearing loss due to acoustic trauma. In VAD, patients report of *hearing too much*, and commonly lower the volume of audio devices (as opposed to increasing the volume to hear better). Whether in occupational or environmental ILFN exposures, early symptoms frequently include mood disorders and sleep disturbances. As overall exposure time accumulates, physicians are often confronted with a myriad of complaints referring to a wide variety of organs and systems. If unfamiliar with VAD, extensive complementary diagnostic tests will be prescribed to confirm or exclude a clinical suspicion. Most of these medical examinations will disclose negative or borderline values in ILFN-exposed patients, i.e. useless for a diagnosis. Generally, at this point, the clinician refers the patient to psychiatric care, with the likely suspicion of malingering or hypochondria. If, however, the physician suspects that ILFN may be playing a role in the patient's condition, the following complementary diagnostic tests are recommended for confirmation or exclusion of that hypothesis.

3.2. Echocardiogram

Thickening of the parietal pericardium was first observed in autopsy [7], then studied through echocardiography [8-10], and then confirmed through surgical pathology [11,12]. Pericardial thickening in the absence of diastolic dysfunction, and in the absence of an inflammatory process, is a hallmark response to ILFN exposure in humans. In VAD, pericardial echogenicity is visible with a GAIN setting of <45. See Fig. 1.

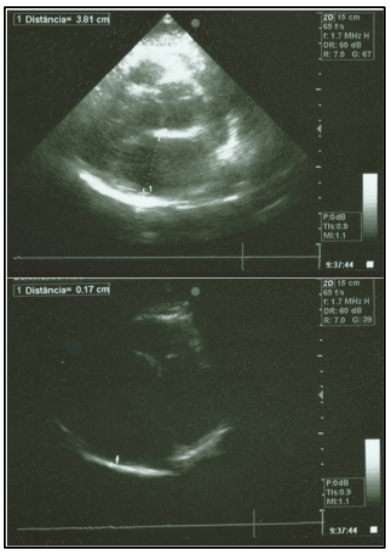


Figure 1 shows echocardiograms of Mr. R [13-15]. *Top image*: Gain set at 67. *Bottom image*: Gain set at 39.

Valve pathology may also be present, particularly in the more severe cases [8-10]. When thickened pericardia are observed through echocardiography, and no accompanying diastolic dysfunction exists, this indicates that significant ILFN exposure has taken place. Echo-imaging alone, however, is insufficient to establish a full VAD diagnosis. Firstly, because this organic manifestation is a *sign* of ILFN exposure, and not a nosological entity in itself. Secondly, no standardized methodology exists to numerically evaluate pericardial thickness. Hence, an undesirable element of subjectivity is introduced with this diagnostic test, making it susceptible to erroneous interpretations [16, for example] and/or deliberate manipulations. With the introduction of GAIN considerations, however, the degree of subjectivity of this imaging technique can be drastically reduced. Echocardiography is, therefore, the diagnostic method of choice for exposure confirmation and patient selection.

3.3. Brain MRI

This imaging test includes the evaluation of structural changes in brain tissues. Albeit non-specific to VAD, brain tissue changes in ILFN-exposed patients include: a) hyperintense foci in T2 of the subcortical and periventricular white

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matter, basal ganglia and brain stem, and b) cerebral atrophy and dilation of the perivascular Virchow-Robin spaces. The most frequent locations for these appearances are the sublenticular and periatrial areas, and semioval centers [17]. While these situations may be expected in older populations, they are not desirable among working populations within the 35-55 year age range. VAD patients often exhibit low tolerance to this examination, and frequently remain bed-ridden for the rest of the day.

3.4. P300 Event Related Evoked Potentials

This non-invasive and objective neurological test evaluates nerve conduction times of processes that occur in the cerebral cortex. The P3 and N2 components of Event Related Potentials (ERP) are related to decision processing and stimuli classification, respectively, and increased latencies in these components have been associated with cognitive deterioration [18]. Longer latencies and lower amplitudes of ERP are observed in ILFN-exposed individuals [17]. VAD patients who present with the above described brain MRI lesions, also exhibit significant latencies of the endogenous N2 component, and significantly decreased amplitudes of the P3 component [17]. The presence of cortical lesions, confirmed through brain MRI, is associated with the changes seen in the P300 ERP values. Frontal topography and multipeaked appearances, as observed in these ILFN-exposed individuals, are similar to those found in the elderly and in patients with degenerative processes of the brain.

3.5. Brainstem Auditory Evoked Potentials

This is another non-invasive and objective neurological test. Here, nerve conduction times of processes occurring within the brainstem are evaluated. These can be altered due to demyelinization foci or expansive lesions. In ILFN-exposed individuals, brainstem auditory evoked potentials (BAEP) exhibit increased latencies in wave intervals III, IV and V [19]. Increased BAEP wave V interval latencies were correlated with hyperintense foci in T2 observed in the brainstem through MRI. VAD patients often develop balance disturbances, and these were associated with the existence of asymmetric values for the BAEP wave V interval latency, in both ears [20]. BAEP results suggest that some central dysfunction is occurring at the level of the brainstem. This is further corroborated by the results obtained in ILFN-exposed individuals through the next diagnostic test in this discussion.

3.6. PCO_2 respiratory drive evaluation

This evaluation is performed within the context of lung function tests. The neurological centers of the control of breathing are located in the brainstem. This control system modulates respiratory rate and respiratory (either inspiratory or expiratory) pressure depending on CO_2 concentration: as CO_2 concentration increases, so does the neurologically-controlled respiratory rate and respiratory pressure drive. In VAD patients, this increased respiratory rate and drive in the presence of increased CO_2 is only mildly observed [21]. The significance of this impaired partially autonomic reflex among ILFN-exposed individuals is not yet clearly understood, and raises many (more) questions regarding the pathophysiological mechanisms of this agent of disease. Nevertheless, it further corroborates the existence of brainstem lesions in ILFN-exposed humans. In respiratory functional tests, changes of metacholine sensitivity is common in VAD patients, probably related to the cellular changes in epithelial bronchial cells which present cellular cholinergic degranulation processes [1,4,22].

3.7. Bronchoscopy

This is a highly invasive examination, and is only recommended for forensic purposes within the context of legal proceedings. In VAD patients, vascular-like lesions are observed in both tracheal and bronchial trees, uniformly distributed bilaterally near the spurs [23]. Biopsies of these lesions were taken and studied with light and electron microscopy, revealing the same features observed before in ILFN-exposed human and animal samples, namely, organized proliferation of collagen and elastin in the absence of an inflammatory process [1,2]. Respiratory diseases (specifically, asthma-like conditions, and squamous cell carcinomas, particularly of the right lung) that develop among noise-exposed populations should be carefully considered in light of the morphological impact that ILFN has on respiratory tract structures [22].

3.8. Voice Acoustic Analysis

In a more recent area of study for VAD researchers, this non-invasive test evaluates changes in voice production as a consequence of

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physiological changes of the laryngeal anatomical structure system. Morphological changes of the respiratory tract structures, such as those seen in ILFN-exposed human and animal models, can alter several parameters associated with voice acoustics. The fundamental frequency among three vowels significantly increases with increasing ILFN exposure time [24,25]. Changes in other voice acoustic parameters, such as jitter and shimmer perturbation measures, harmonic-to-noise ratio, and maximal phonational frequency range, also exhibited changes but these are not yet fully understood. Bronchoscopy imaging of the vocal folds revealed the same, above-mentioned, vascular-like lesions as observed in the tracheal and bronchial trees [23]. This non-invasive, voice acoustic evaluation that seems to reflect a dose-response pattern for VAD patients is poised to become an invaluable complementary diagnostic tool for ILFN-induced pathology

3.9. Hemostasis and Coagulation Parameters

In extreme stress environments, states of elevated hypercoagulability have been documented [26,27]. In ILFN-exposed individuals, spontaneous platelet aggregation was observed in the most severe cases while platelet aggregation values are abnormally high in all VAD patients. In ILFN-exposed persons the plasminogen activator inhibitor-1 (PAI-1) is significantly increased, even after several days post-ILFN exposure [27]. Increased PAI-1 is an indicator of fibronolysis inhibition and activation of coagulation, leading to situations of hypercoagulability.

3.10. Immunological Parameters

Autoimmune disorders, particularly collagenous diseases, are common among the more extensively ILFN-exposed individuals [1,9]. Lupus-prone animal models exposed to ILFN saw an earlier onset and higher mortality rate than the non-noise-exposed control group [28]. ILFN-exposed Wistar rats saw the pleural immune mechanisms highly impaired, when compared to non-exposed controls [5]. In ILFN-exposed animals, splenic $CD4^+$, $CD8^+$, and IgM^+B lymphocyte populations were decreased when compared to non-exposed controls [29]. In VAD patients, the number of circulating $CD8^+$ and $CD4^+$ T lymphocytes was significantly increased when compared to non-noise-exposed controls [30]. Pericardial fragments of VAD patients observed through electron microscopy revealed remarkable amounts of cellular debris due to non-programmed (non-apoptotic) cellular death [1,11,12]. It is, therefore, unsurprising that VAD patients often develop lupus, vitiligo, and other autoimmune disorders [1]. Hence, complementary examinations should include assays for antinuclear and anti-mitochondrial antibodies, C-reactive protein, and systemic lupus erythematous.

3.11. Exposure Histories

To assess the probability of a patient's symptomatology being associated with excessive ILFN, a comprehensive noise exposure history must be taken. Not only will this aid diagnosis, but it will also provide valuable prognostic information. Noise exposure histories begin with fetal exposure that will depend on the mother's profession and residential conditions. Residential exposures during childhood are of fundamental importance given the cellular processes that occur only during this time of human physical and emotional growth. The relative position of bedrooms in relation to noisy streets, potentially concurrent with occupational exposures, and a variety of different types of recreational noise exposures, must all be taken into account in order to obtain an accurate clinical picture of the patient. The development of ailments in the individual's history should be viewed in light of the chronological exposure to noise. Each type of individual noise source also provides key information for ascertaining the extent of the risk factors for VAD to which the individual has been exposed. Documentation of the time spent away from the ILFN-rich environments (i.e., recovery periods) is crucial for characterizing the evolution of VAD.

4. Health Impact of ILFN Exposure

Over the past decade, the health impact of ILFN exposure has taken on a new life due to the worldwide energy crisis and the urgent implementation of numerous industrial wind turbines (IWT), as decreed by many governments. Since little regard was initially given to the ILFN generated by these devices, it was not long after their installation that families living in their vicinity began having complaints. Recognizing the problematic situation, several agencies and institutions worldwide began conducting studies and publishing papers with titles suggesting that health impacts were being evaluated among these residential populations. However, upon a closer examination, significant design flaws rendered

most of these studies useless [31, for example]. While this may not have been a concerted effort, it is however a consequence of the perpetuation of an historical fact: acousticians, usually with no medical background, are generally greatly involved in noise-impact study designs. Therefore, information on the "health impacts," as rudimentarily obtained through questionnaires or interviews, is often deemed sufficient in order to establish whether or not ILFN is, *de facto*, having an impact on the health of human populations. Objective clinical evaluations of families and animals living in the vicinity of IWT are not commonly performed. ILFN is a physical agent of disease, and infrasound (<20 Hz) is internationally classified as *non-ionizing radiation*. The type of toxicological effect and the evolution and onset of disease caused by ILFN exposure is partially analogous to that caused by radiation. As with radiation exposure, a) the onset of ILFN-induced disease greatly depends on time-exposure patterns; b) different wavelengths of the physical agent affect different organs and tissues; c) the individual need not perceive the agent of disease for a pathological effect to occur within the body; and d) prior exposure histories are a determining factor of key importance. These facts *must* condition the design of studies purporting to evaluate the health impacts of residential ILFN exposure; but currently, they do not. It is recognized that there is an added cost to conducting real clinical studies among ILFN-exposed populations. The next obvious question, though, is: what is the added cost associated with continuing with the *status quo*. While this issue is way beyond the scope of this paper, it is nevertheless pertinent to recall episodes in humanity's recent history, having to do with asbestos, smoking and pesticides.

5. Looking Forward

In the interest of becoming a responsible and mature human society, activities that are deleterious to human health and wellbeing should not be hidden, obfuscated, or otherwise camouflaged. History unequivocally demonstrates the benefits of dealing outright with any potential human-health issue. It is recognized that the production of electrical energy has become the warp and woof of modern societies, and hence the urgency to procure new ways of harvesting energy that can be rapidly (and inexpensively) transformed into electrical energy. There need not be, however, such antagonistic, acrimonious and spiteful endeavors between ILFN disturbed families and *industry*, be it in of the energy, transportation, military, entertainment or manufacturing sectors of society. Having contextualized the issue, it is therefore of paramount importance that the clinical parameters used to evaluate the specific type of pathology caused by excessive ILFN exposure be the most appropriate for the job. Audiometry, electrocardiography, cortisol levels, and number of nighttime arousals are remarkably imprecise measures to clinically evaluate the adverse health impacts of noise exposure. Whether transverse or longitudinal epidemiological studies are planned, the clinical parameters required to ascertain the extent of ILFN-induced pathology have herein been outlined. It is hoped that this report will contribute to a symbiotic relationship between the ILFN-generating industries and world citizens.

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References

[1] M. Alves-Pereira, N.A.A. Castelo Branco: Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling. Progress Biophysics & Molecular Biology 93 (2007) 256-279.

[2] N.A.A. Castelo Branco, M. Alves-Pereira: Vibroacoustic disease. Noise & Health 6 (2004) 3-20.

[3] N.A.A Castelo Branco: The clinical stages of vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A32-9.

[4] N.A.A. Castelo Branco, E. Monteiro, A. Costa e Silva, J. Reis Ferreira, M. Alves-Pereira. Respiratory epithelia in Wistar rats born in low frequency noise plus varying amounts of additional exposure. Revista Portuguesa de Pneumlogia IX (2003) 481-492. europepmc.org/abstract/MED/15190433

[5] M.J.R. Oliveira, A. Sousa Pereira, A.P. Águas, E. Monteiro, N.R. Grande, N.A.A. Castelo Branco. Effects of low frequency noise upon the reaction of pleural milky spots to mycobacterial infection. Aviation, Space & Environmental Medicine 70 (1999) A137-40.

[6] S. Chapman, A. St. George: How the factoid of wind turbines causing 'vibroacoustic disease' came to be 'irrefutably demonstrated'. Australian & New Zealand Journal of Public Health 37 (2013) 244-49.

[7] N.A.A. Castelo Branco: A unique case of vibroacoustic disease. A tribute to an extraordinary patient. Aviation, Space & Environmental Medicine 70 (1999) A27-31.

[8] W. Marciniak, E. Rodriguez, K Olsowska, I. Botvin, A. Arauj, F. Pais, C. Socares Ribeiro, A. Bordalo, J. Loureiro, E. Prazeres de Sá, D. Ferreira, M.S.N. Castelo Branco, N.A.A. Castelo Branco: Echocardiography in 485 aeronautical workers exposed to different noise environments. Aviation, Space & Environmental Medicine 70 (1999) A46-53.

[9] R. Torres, G. Tirado, A. Roman, R. Ramirez, H. Colon, A. Araujo, F. Pais, J.M.C. Lopo Tuna, M.S.N. Castelo Branco, M. Alves-Pereira, N.A.A Castelo Branco: Vibroacoustic disease induced by long-term exposure to sonic booms. Proc. Internoise 2001, 1095-98 (ISBN: 9080655422).

[10] A. Araujo, J. Carranca, M. Alves-Pereira, N.A.A. Castelo Branco: Echocardiography in vibroacoustic disease. Proc. 12th ICSV 2005, No.567 (9 pages).

[11] N.A.A. Castelo Branco, A.P. Águas, A. Sousa Pereira, E. Monteiro, J.I.G Fraggata, F. Tavares, N.R. Grande: The human pericardium un vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A54-62.

[12] N.A.A. Castelo Branco, J.I. Fragata, A.P.Martins, E. Monteiro, M. Alves-Pereira: The pericardium in vibroacoustic disease I – morphological features. Proc. 12th ICSV 2005, No.568 (9 pages).

[13] M. Alves-Pereira, N.A.A. Castelo Branco: In-home wind turbine noise is conducive to vibroacoustic disease. Proc. 2nd Intl Meet Wind Turbine Noise, 2007, Paper No. 3 (11 pages).

[14] N.A.A. Castelo Branco, T. Costa e Curto, L. Mendes Jorge, J. Cavaco Faísca, L. Amaral Dias, P. Oliveira, J. Martins dos Santos, M. Alves-Pereira: Family with wind turbines in close proximity to home: follow-up of case presented in 2007. Proc. 14th Intl Meet Low Frequency Noise, Vibration and its Control, 2010, 31-40.

[15] N.A.A. Castelo Branco, M. Alves-Pereira, A.J.F. Martinho Pimenta, J. Reis Ferreira: Low frequency noise-induced pathology: Contributions provided by the Portuguese wind turbine case. EuroNoise 2015, paper no. 602.

[16] ATSDR – Agency for Toxic Substance and Disease Registry: Expert review of the Vieques heart study. Summary report for the Vieques heart study expert panel review. (2001) Contract No. 200-2000-10039. www.atsdr.cdc.gov/sites/vieques/heart_study_summary.html.

[17] M.G. Pimenta, A.J.F. Martinho Pimenta, M.S.N. Castelo Branco, N.A.A. Castelo Branco: ERP P300 and brain magnetic resonance imaging in patients with vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A107-14.

[18] L. Gomes, A.J.F. Martinho Pimenta, N.A.A. Castelo Branco: Effects of occupational exposure to low frequency noise on cognition. Aviation, Space & Environmental Medicine 70 (1999) A115-18.

[19] J. H. Marvão, M.S.N. Castelo Branco, A. Entrudo, N.A.A. Castelo Branco: [Changes of the brainstem auditory evoked potentials induced by occupational vibration]. Jornal da Sociedade das Ciências Médicas 149 (1985) 478-486 (In Portuguese).

[20] A.J.F. Martinho Pimenta, M.S.N. Castelo Branco, N.A.A. Castelo Branco: Balance disturbances in individuals with vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A100-6.

[21] J. Reis Ferreira, J. Albuquerque e Sousa, P. Foreid, M. Antunes, S. Cardoso, M. Alves-Pereira, N.A.A. Castelo Branco: Abnormal respiratory drive in vibroacoustic disease. Revista Portuguesa de Pneumologia XII (2006) 369-74. www.scielo.oces.mctes.pt/pdf/pne/v12n4/v12n4a03

[22] N.A.A. Castelo Branco, J. Reis Ferreira, M. Alves-Pereira: Respiratory pathology in vibroacoustic disease-25 years of research. Revista Portuguesa de Pneumologia XIII (2007) 129-135. www.scielo.oces.mctes.pt/pdf/pne/v13n1/v13n1a08

[23] J. Reis Ferreira, M. B. Monteiro, F. Tavares, I. Serrano, E. Monteiro, C. P. Mendes, M. Alves-Pereira, N.A.A. Castelo Branco: Involvement of central airways in vibroacoustic disease. Revista Portuguesa de Pneumologia XII (2006) 93-105. www.scielo.oces.mctes.pt/pdf/pne/v12n2/v12n2a01

[24] A. P. Mendes, I. Bonança, A. Jorge, M. Alves-Pereira, N.A.A. Castelo Branco, M. Caetano, N. Oliveira, A. Graça, C. Santos, R. Ferraria: Voice acoustic profile of males exposed to occupational infrasound and low frequency noise. Laryngology & Voice. 4 (2014) 12-20. www.laryngologyandvoice.org/temp/JLaryngolVoice4112-3980982_110329.pdf

[25] A. P. Mendes, A. Graça, A. Jorge, M. Alves-Pereira, N.A.A. Castelo Branco, A. Freitas, M. Laranjeira, I. Bonaça: The effects of ILFN-exposure on voice acoustic parameters of commercial cabin crewmembers. Laryngology & Voice. 2 (2012) 70-80. www.laryngologyandvoice.org/temp/JLaryngolVoice2270-459212_124521.pdf

[26] G. Biondi, S. Farrace, G. Mameli, F. Marongiu: Is there a hypocoagulable state in military fighter pilots Aviation, Space & Environmental Medicine 67 (1996) 568-71.

[27] L. Cunha Ribeiro, F. F.Crespo, I. Freira, H. Afonso, M.S.N. Castelo Branco, M.C. Marques, M. Alves-Pereira, N.A.A. Castelo Branco: Hemostasis and coagulation changes in vibroacoustic disease. Proc. 12th ICSV 2005, No.564 (8 pages).

[28] A.P. Águas, N. Esaguy, A.P. Castro, N.R. Grande, N.A.A. Castelo Branco: Acceleration of lupus erythematosus-like processes by low frequency noise in the hybrid NZB/W mouse model. Aviation, Space & Environmental Medicine 70 (1999) A132-6.

[29] A.P. Águas, N. Esaguy, A.P. Castro, N.R. Grande, N.A.A. Castelo Branco: Effect of low frequency noise exposure on BALB/C mice splenic lymphocytes. Aviation, Space & Environmental Medicine 70 (1999) A128-31.

[30] A.P. Castro, A.P. Águas, N.R. Grande, E. Monteiro, N.A.A. Castelo Branco: Increase in CD8+ and CD4+ T lymphocytes in patients with vibroacoustic disease. Aviation, Space & Environmental Medicine 70 (1999) A141-4.

[31] Executive Office of Energy and Environmental Affairs of the State of Massachusetts, USA: Wind turbine health impact study: Report of an independent expert panel. January 2012. www.mass.gov/eea/docs/dep/energy/wind/turbine-impact-study.pdf

12th ICBEN Congress on
Noise as a Public Health Problem

Case Report: Cross-Sensitisation to infrasound and low frequency noise

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ABSTRACT

This Case Report describes an episode experienced by two noise-sensitised individuals during a field trip. Exposed to residential infrasound and low frequency noise due coal mining activities, the subjects reacted suddenly, strongly and unexpectedly to pressure pulses generated by a wind farm located at a different town, approximately 160 km by road from their residence. Simultaneous physiological data obtained in one subject and subjective sensations occurring during the episode are reported. Acoustical evaluations of the location of the episode are also reported. The possibility of a nocebo effect as an etiological factor for their bodily reactions is cogently eliminated. The degree of cross-sensitisation to acoustical phenomenon depends on prior exposure histories, and on the temporal characteristics of the acoustical phenomenon.

BACKGROUND

Many families residing in the vicinity of coal mining operations in New South Wales, Australia, report adverse health effects in response to infrasound and low frequency noise (ILFN). Mr & Mrs T and their children moved to this area in December 2009. Subsequent to significant health deterioration in all members of the family, noise measurement equipment was installed in the T home starting in 2015, and infrasound and low frequency components were monitored on a 24-hr basis for several months. Medical histories of the adult family members were recorded. Within the scope of CSI-ACHE (Citizen Science Initiative – Acoustical Characterization of Human Environments [1]), the family volunteered to wear physiological monitoring devices while at home, so that specific acoustical environments could be correlated with specific physiological responses. CSI-ACHE also includes families whose residential exposure to ILFN is caused by industrial wind turbines (IWT), and who have been reporting similar symptoms as the T family. The goal of this report is to document the reactions endured by Mr and Mrs T, as observed by 3 of the 4 authors and as registered on

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electronic devices, when in the presence of infrasound generated by a nearby wind farm. Informal acoustical evaluations are also presented.

Female Subject – Mrs. T.

Caucasian, age 44, homemaker, non-smoker, no alcoholic habits. Mother has history of heart disease, and worked at processed food factories and as a sewing machinist while pregnant with Mrs T. Father died of asbestos-related mesothelioma. As a child, Mrs T had her tonsils and appendix removed and, as a teenager, developed mononucleosis and herpes simplex 1. Subject began living in urban environments at age 16, in the suburbs of Sydney, Australia. Mrs T had a hairdresser apprenticeship at age 17, but developed severe dermatitis with associated chemicals. Began administrative office work. At this time, she lived next to railway lines and power lines "that hummed," but moved out one year after the birth of her first child in 1993. Two years later, the family moved to another home, where mould was a significant issue and finally led to their departure. In 2002, Mrs T had her second child, and began shift-work in the food industry. Later, she became a homemaker and domestically raised poultry for eggs. In December 2009, the T family moved from suburban Sydney to the Central Tablelands, in New South Wales. No significant exposure to radiation or asbestos was identified.

Male Subject – Mr. T.

Caucasian, age 40, public employee, non-smoker, no alcoholic habits. Mother has heart disease and worked as a sewing machine operator until she discovered she was pregnant. Father has diabetes. Three of the subject's four grandparents died of smoking-related lung cancer. Until the age of 4, subject lived in busy western suburb of Sydney; then moved to a more rural suburb where he lived until age 18. No childhood or adolescent medical conditions of note. Started working on motorcycles at age 10. At 18 he worked in a refrigerated warehouse for approximately 3 years. Subsequently, he worked in the automobile restoration business for 10 yrs, during which protective equipment was always used. Periodic testing subsequent to chemical exposures during automotive restoration work always returned as "cleared." Changed to an administrative job in the insurance sector and then obtained a teacher's certificate for automotive restoration, painting and rectification work. After moving to the Central Tablelands in December 2009, he obtained a position as a public servant that he maintains to this day. Mr T has never worked the 'graveyard' shift. No significant exposure to radiation or asbestos was identified.

HEALTH COMPLAINTS SINCE 2009

After moving to the new home, Mr and Mrs T noticed that their house often vibrated, audibly rattling windows and the shower screen. Within a year, Mr and Mrs T began awakening in the middle of the night, sensing vibration through their bed and audibly hearing home structures rattling. By the end of 2010, Mrs T reported feeling vibration through her feet. Mr T's systematic awakening in the night was attributed to his snoring, which was also blamed as the cause of Mrs T's interrupted sleep. By 2011, Mrs T developed numbness in her face and fingers and had difficulty swallowing. Multiple sclerosis was suspected but, after medical testing, was eliminated as a cause of her symptoms. Sleep continued to be disrupted, but was mainly attributed to Mr T's loud snoring. In 2012, Mrs T took a cleaning job at a local resort hotel. Although she wore gloves and no dermatitis developed, she felt sick and thought it was the chemicals, so she quit. Also in 2012, prior symptoms persisted, and new medical conditions developed, particularly in Mrs T: digestive problems, unexplained joint pain, shortness of breath and nausea. A colonoscopy revealed secil polyps; an inflammatory process in the stomach was identified (no *Helicobacter pylori* detected); it was suggested that

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the shortness of breath was related to anxiety. Mrs T developed a “pulsating head pressure” which impeded her from lying down to sleep, and was forced to sleep sitting up. She also began developing balance disorders. In 2013, all these symptoms collectively persisted. In this same year, the T family took a family trip to another part of the country, and realized that their symptoms greatly subsided. This led them to first suspect their home environment as the cause of their health complaints. Since Mr T’s snoring was still the main contender for the couple’s sleep disruption, in late 2013, Mr T had his tonsils removed. While the snoring did indeed cease, awakenings in the middle of the night did not. Starting in 2014, Mrs T began developing the feeling of intense coldness. Today, Mrs T reports nausea, shortness of breath and difficulty swallowing that occur in wave-like episodes, and simultaneously with other members of the family; increased emotional instability and no stamina or will to complete tasks. Mr T reports pulsating head pressure, nausea, and shortness of breath; about his symptoms he says: “I am about one year behind my wife.” The T family is currently undergoing diagnostic testing for vibroacoustic disease [2-5].

RESIDENTIAL ILFN EXPOSURE

Acoustical monitoring conducted on the T family property (Nov 2015-Feb 2016, and then again Sept-Oct 2016) was performed in accordance with the requirements of Australian Standard AS1055-1997. Two sets of instrumentation were used: one set external to the dwelling, in free-field, and the other inside the master bedroom.

Instrumentation used for the measurements included a SVAN 979 sound level meter with a GRAS 40AZ microphone, set to record and sample measurements 10 times per second for statistical purposes and, simultaneously, to record 24-bit 10-minute wave files for subsequent analyses if required. The sound level meter was field calibrated using a sound level calibrator (B&K 4230), before and after measurements, and did not exhibit any deviation. A secondary system using an INFILTEC micro-barometer was used to record pressure variations in the infrasound region. The output of the INFILTEC unit was connected to a RAND ACOUSTICS serial data logger type SDL2. The logger was set to record data 50 times per second, and store data files in one-hour blocks. The micro-barometer and logger were calibrated using a GRAS 42AE calibrator. All instrumentation used for these sound level measurements hold current calibration to manufacturers’ specifications, as required by Australian Standard AS1259.

Results obtained in the T home have not yet been submitted for publication. Figure 1 is an informal representative sonogram from inside the master bedroom, captured shortly after midnight. This sonogram is the result of passing the signal from a SVAN 979 through a bank of 1/24-octave, narrow-band filters compliant with ANSI® S1.11-2004 standard, class 0. These are uncalibrated, relative levels of acoustical energy within the 10-100 Hz range.

The large number of horizontal lines indicates the presence of strong tonal components within the soundscape. The strongest is at 50 Hz, which would be assumed to come from the house’s electrical mains, as would any harmonics. Several of the other tones, however, appear to be harmonics of a fundamental frequency slightly over 10 Hz, which did not arise from any source within the house.

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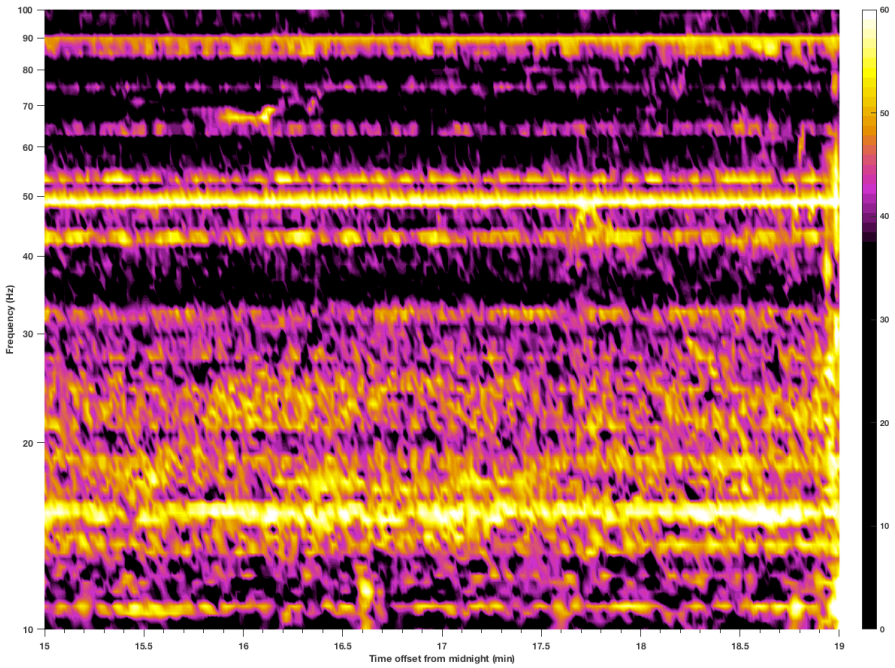


Figure 1: Sonogram showing the relative levels of acoustical energy within the 10-100 Hz range, as frequently encountered in the Master Bedroom of the T family home (around midnight). The predominance of horizontal lines indicates acoustic sources with distinctive tonal characteristics.

THE EXPERIMENT

Background

In 2015, the T family hosted a team of researchers who were taking measurements in parallel with those already in place and described above. One of these used a prototype micro-barometer to measure infrasound in addition to 'Biopatch' physiological monitors (Medtronic Zephyr™ [6]) to record heart rate, heart rate variability, respiration rate, skin temperature, energy usage, movement and orientation.

Mr & Mrs T reported becoming acutely and severely distressed whilst driving through the township of Taralga (Southern Highlands, New South Wales) earlier that year. This location has an IWT development in its proximity (Fig 2). Mr T reported developing pressure bolts, headaches and nausea while Mrs T had developed a sudden, severe headache and unexplained anxiety. This report presented itself to CSI-ACHE as an opportunity to further explore the correlation between physiological response and specific acoustic components in the soundscape. On December 4th a field trip was made to several locations near IWT developments while Mr & Mrs T were each wearing a Biopatch.

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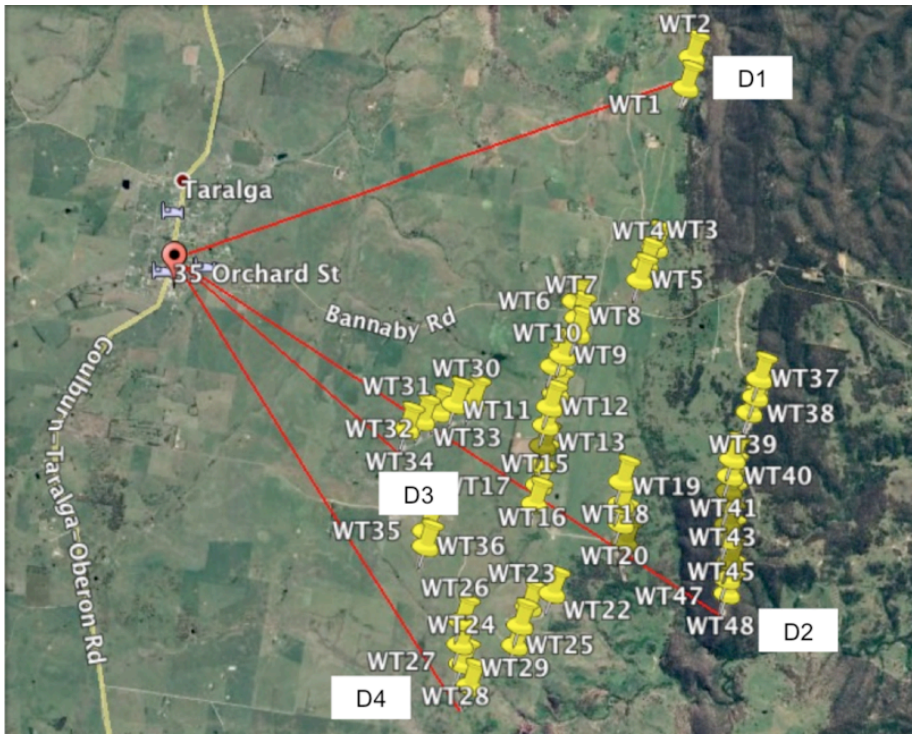


Figure 2: Reconstruction using an image obtained with Google Earth showing the relative positions of the Township of Taralga and the IWT developments. D1 ≈ 6030 m (WT2), D2 ≈ 7288 m (WT48), D3 ≈ 3342 m (WT34), D4 ≈ 5964 m (WT28).

The Episode

On December 4th, 2015, the recorded incident happened at the very end of a long day that had been disappointing scientifically in that very few of the IWTs in the visited areas were operating. No acute responses had been recorded up to that point. On the way home, a stop was made in Taralga to make use of the public toilets. These were located behind the Taralga Memorial Hall on Orchard Street. An aerial plan view from Google Earth has been annotated to show the relative placement of the facilities and the vehicle (Fig 3).

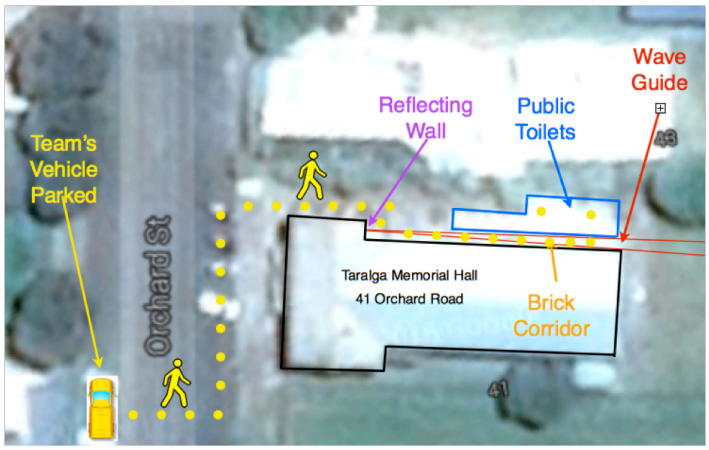


Figure 3: Annotated plan view of the public facilities behind the Taralga Memorial Hall.

The hall is of solid brick construction with the facilities at the left side of the building towards the rear where there is a short corridor (Fig 4).

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Figure 4: Path to public toilets (**A**), brick corridor (**B**) and the reflecting wall (**C**).

Mrs T and a member of the research team left the parked vehicle and walked to the toilets behind the hall. On turning right down the side of the hall and passing the reflecting wall, Mrs T experienced a sudden reaction of nausea and a feeling that she was swaying, “like being on a ship at sea,” requiring her to grasp the handrail to maintain her balance. She described “waves of pressure” sweeping over her, nausea, and a feeling of extreme anxiety. No obvious sources of ILFN were present on the day in question. Trees obscured the IWTs, and neither person was aware of their proximity.

Physiological Response During the Episode

Figure 5 shows the physiological responses of Mrs T between 5:00 pm to 6:00 pm, as obtained with the Biopatch (Medtronics, Zephyr™ [6]), during the episode described above.

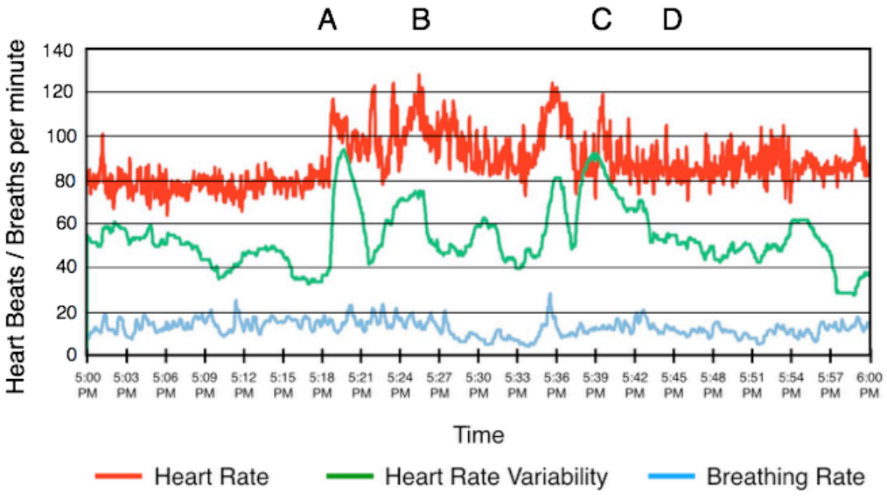


Figure 5: Mrs T’s physiological responses between 5 and 6 pm, as recorded with the Zephyr™ Biopatch.

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At approximately 5:18 pm, Mrs T exited the vehicle and walked toward the public toilet entrance. At the precise moment she passed the reflecting wall (Fig 4C) she immediately experienced sudden tachycardia accompanied by feelings of vertigo and anxiety. Her heart rate variability spiked from 40 to 90 beats per minute (Fig 5A). After steadying herself using the handrail she managed to walk to the toilets and return to the vehicle a few minutes later. At this point a researcher retrieved the SAM infrasound unit [7] and returned to the start of the guardrail (Fig 4A), recording for about 12 minutes (until approx 5:32) in Mrs T's presence. Her heart rate again spiked, as did her heart rate variability (Fig 5B). The team returned to the vehicle and drove off at around 5:33 pm, at which time Mrs T's heart rate and heart rate variability returning to baseline values (Fig 5).

Mr T was driving. Having driven maybe 1 km, he looked to the right where a break in the houses allowed the public toilet entrance to become visible. He reacted violently and instantly. He stopped the vehicle, jumped out and, in the middle of the road, doubled up and underwent dry retching for some time. In the same time (5:36, Fig 5-C), Mrs T's heart rate again spiked to 120 beats per minute (bpm) and her heart rate variability hit 90 bpm. The entire team was shocked at this physical reaction, as the onset was so rapid and so physiologically violent. Unfortunately, Mr T's reaction was so severe that it dislodged the Biopatch sensor, so no usable data could be obtained for him from the incident. After a few minutes, Mr T returned to the vehicle, extremely embarrassed for his sudden, uncontrollable reaction. Team members offered to drive but he stated that he was better in control of the vehicle as he was feeling some vertigo. A second attempt to drive off only managed to move the car forward a few metres before Mr T again brought the vehicle to an emergency stop, this time running into the grassy side, where he continued to dry retch uncontrollably for about 5 minutes. At around 5:39 pm, Mr T had recovered sufficiently to attempt to drive off again, reporting that the symptoms did not finally subside until about one hour later. Mrs T's heart rate returned to baseline values after the team had driven off, although she reported inexplicable anxiety and pressure for the next half hour. (NB: The Biopatch real-time clock was later found to be approximately 7 minutes slow.)

Acoustical Analysis

The results of the informal recordings with a prototype infrasound recorder unit [7] are shown here. Data was captured continuously during the journey with approximately 12 minutes being recorded at the toilet location. The resultant sonogram is shown in Fig 6 from the time the digital filters settled until the instrument was returned to the car. It represents non-calibrated, relative levels of acoustical energy.

The sonogram shows a large proportion of energy in near 1 Hz and below. Between about 1 Hz and 3 Hz vertical striping can be seen within the frequency bands. The striping occurs at frequencies below the band in which they appear, e.g., in the 1–1.5 Hz bands the striping occurs at about 3–4 second intervals. This indicates the presence of amplitude modulation.

Figure 7 shows a 5-minute sonogram obtained on the outskirts of Taralga during the time when Mr T was suffering his episode. The car motor was running during this time. The same features indicating amplitude modulation can be seen in the 1–3 Hz region.

In view of the discovery of this amplitude modulation, the time signals were isolated from Figure 7 for the 1 and 1.25 Hz 1/24-octave bands (Figure 8).

It can be seen that the infrasound levels at these two frequencies were being modulated at a frequency of about 0.25 Hz, or a pulse every four seconds. The modulation depth is of the order of 5 dB. This depth of modulation would be readily noticeable as a change in perceived loudness had the carrier frequencies been in the audible range instead of the infrasound

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range. It can also be seen that the sound level of the 1.25 Hz band dropped by 20 dB and returned despite the 1 Hz band remaining relatively unchanged. The infrasound unit did not record the audible frequency range and so it is not possible to state whether amplitude modulation also occurred in that region.

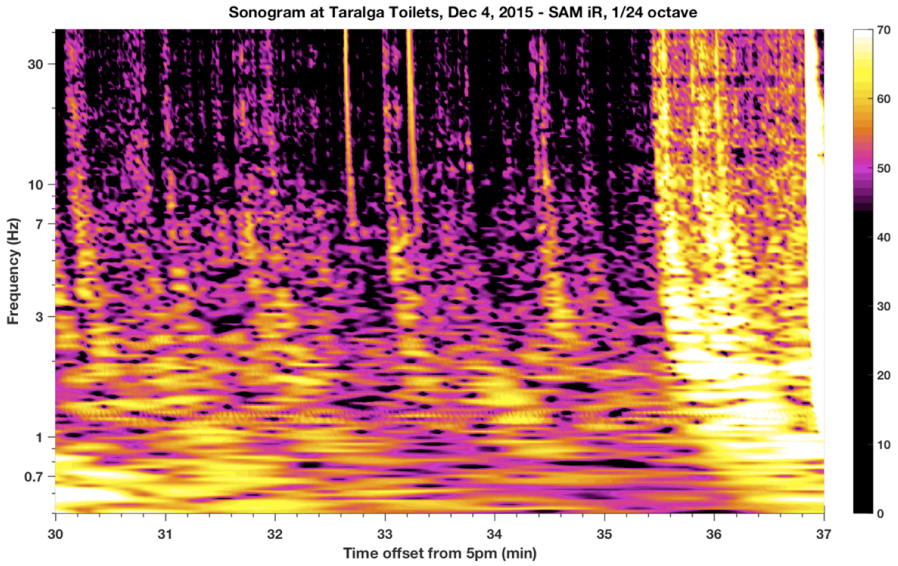


Figure 6: Sonogram at Taralga public toilets, showing the relative levels of acoustical energy within the 0.5–41 Hz frequency range.

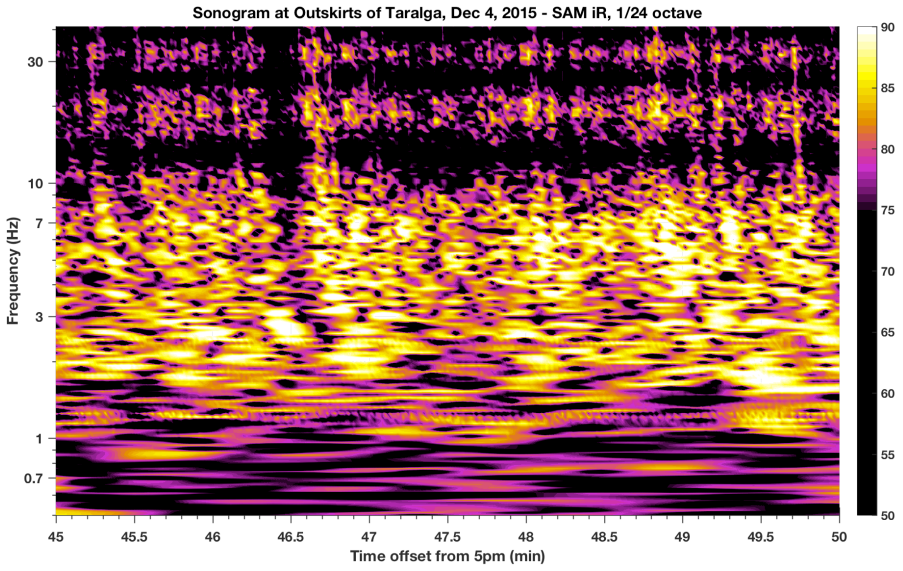


Figure 7: 5-minute sonogram from the outskirts of Taralga showing the relative levels of acoustical energy within the 0.5-41 Hz frequency range.

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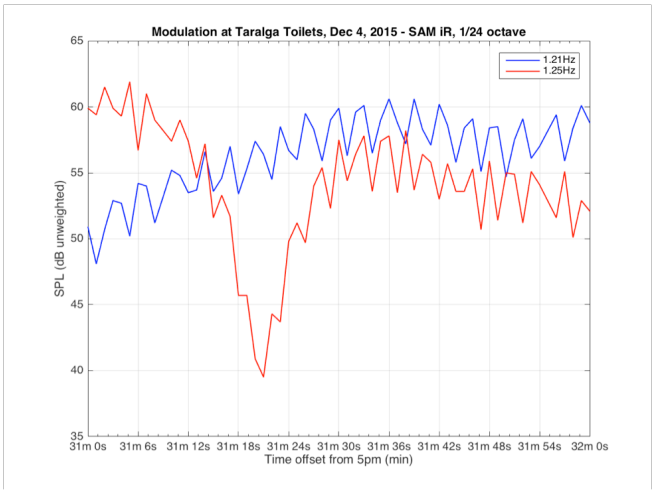


Figure 8: 60 seconds of modulation taken from the Taralga toilets dataset. (Sound levels in this sonogram have not been calibrated and only the relative sound levels should be considered.)

DISCUSSION AND CONCLUSION

At present, Mr & Mrs T have been exposed to excessive levels of ILFN in their residence for seven years. This experiment documents how sensitised they had become to this acoustical agent of disease at the end of five years. The immediacy of the onset of extreme bodily symptoms speaks to the nature of the agent of disease, and to the importance of prior exposure histories when current reactions are examined.

Sensitisation to airborne pressure waves rich in low frequency components is a phenomenon well-known to acousticians working in the field of low-frequency noise and vibration. It was described in 1985 in the seminal work of Dr. Neil Kelley. [8] A recent animal study [9] suggests that the degree of impulsiveness of the sound, or sound character, is as important as the frequency and peak amplitude. In 2011 Gotz & Janik [9] showed that wild seals exposed to impulsive sound became progressively sensitised to the sound and distressed by it. In contrast, a control group of wild seals exposed to the same sound level, but without the impulsivity, habituated to the sound. Another example of animal sensitisation can be found in laboratory experiments with Wistar rats exposed to ILFN. Here, non-exposed animals react to the sound of a blown kiss by becoming tense and nervously looking around for its source. After exposure, the same sound of a blown kiss induces tremors, rising on the hind legs and falling backwards [10].

The concept of ‘noise annoyance’ is a form of noise sensitisation. Classical hearing loss does not necessarily imply the existence of ‘noise annoyance.’ Indeed, annoyance to noise (any noise) is often the first symptom associated with excessive ILFN exposure [3,4], as opposed to having hearing difficulties or decreased speech intelligibility, i.e., the typical initial symptoms of classical hearing impairment. Many authors [for example, 11] still defend that ‘annoyance’ occurs as the consequence of an indirect pathway associated with noise exposure. Normally, those authors place annoyance on par with a “cognitive and emotional response” [11]. As suggested in 2003 [10], the organic lesions seen in the actin-based, cochlear cilia of ILFN-exposed laboratory rats could provide a physical, anatomical aetiology for the phenomenon of noise annoyance [12].

The notion that a nocebo effect may have been the aetiological factor for Mr and Mrs T’s episode in Taralga is only here entertained due to suggestions made by other authors [13]. (This issue is discussed in more detail by these authors in [14].) In the episode described herein, no hint of a nocebo effect is possible, since it was already “on-the-way-home,” *after* the experiment had been officially terminated; there was no reason to believe that there would

be any adverse elements in the acoustic soundscape at the toilet stop. The Gotz & Janik experiment with seals [9] provides further scientific evidence that a nocebo effect cannot be used to dismiss such reactions as psychosomatic in origin, as does the laboratory experiment with Wistar rats [10]. Studies that have provided objective, clinical diagnostic tests to ILFN-exposed individuals have also negated the possibility of a nocebo effect [2-5]. Emesis, or involuntary vomiting, is controlled by the autonomic nervous system, as is the neurological control of breathing. It is therefore interesting to note that ILFN-exposed individuals who have undergone lung functional tests including the pCO_2 respiratory drive, reveal an impaired neurological response to the presence of excessive CO_2 (i,e, no hyperventilation is triggered) [15]. As an ending note on the nocebo issue, Figure 9A shows the domestic poultry eggs collected by Mrs T at her home in 2015. The teratogenic effect in Mrs T's chick (Fig 9B) is similar in nature to that observed in ILFN-exposed rats (Fig 9C) [16]. Increased frequency of sister chromatid exchanges, as seen in ILFN exposed workers and animals [17-20], corroborate the genotoxicity of ILFN.



A B C

Figure 9: Photographs taken by Mrs T (Dec 2015) of the domestic poultry gestated and raised on their property. **A.** Abnormal hatching of turkey eggs. **B.** Teratogenesis in a chick gestated and born on the T family property. **C.** Teratogenesis in a Wistar rat gestated and born in ILFN [16].

The evolution of symptoms in Mr T occurred more slowly than in Mrs T. This is unsurprising since Mrs T spends more time in the home than Mr T. Prior noise exposure histories of Mrs T suggest that she had already lived in a home where ILFN was an issue, and may have had non-trivial *in utero* exposure. The evolution of signs and symptoms (clinical stages) associated with occupational exposures to ILFN has already been identified [2]. Residential exposures to ILFN however, have been observed to have an earlier onset than occupational exposures and an accelerated evolution [21-24]. It is postulated that exposure to ILFN during sleep-time may be playing an important role in this matter. Mrs T seems to have a predisposition for immune system disorders, as gleaned from her anamenesis summarised above (i.e., medical and familial histories self-reported to clinically-trained professionals [25]), and that can be a concomitant situation when living in ILFN-contaminated homes. ILFN exposure has already been shown to modulate the immune system in both human [26, for example] and animal models [for example, 27,28]. Balance disorders and other neurological impairments have also been previously documented in ILFN-exposed workers [29,30].

As is so often the case with wind turbine emissions, the blade-pass frequency manifests as a modulation of the overall sound emitted. This can be likened to listening to a radio where the volume control is being slowly turned up and down at low infrasound frequencies [31]. Wind turbines are not thought of as emitting these low frequencies as separate tones, rather they are simply artefacts of the modulation of the total sound envelope. It is likely that when residents complain about infrasound or pulsing noise, it is the total sound envelope changing in volume in a periodic fashion that is being perceived. How the human body responds to modulated infrasound is still a matter of debate and further research is urgently needed to determine the exact biological mechanism of action.

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Acknowledgements

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Disclaimer

The authors 1) Do not harbour anti-technology sentiments; 2) Consider industrial activities to be important to modern technological societies; 3) Have presented this report under one and only on agenda—pure scientific inquiry; 4) Are not producing a report arguing against industrial developments.

REFERENCES

[1] Bakker, H. H. C., Alves-Pereira, M., Summers, S. R. (2017) Citizen Science Initiative: Acoustical Characterisation of Human Environments. Paper presented at the ICBEN 2017, Zurich, Switzerland (Paper No. 3653).

[2] Castelo Branco, N.A.A. (1999) Clinical stages of vibroacoustic disease. *Aviation, Space & environmental Medicine*, 70 (March Suppl), A32-9.

[3] Castelo Branco, N.A.A., Alves-Pereira, M. (2004) Vibroacoustic disease. Noise & Health 6(239, 3-30.

[4] Alves-Pereira, M, Castelo Branco N.A.A. (2007) Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling. Progress Biophysics & Molecular Biology, vol 93: 256-279.

[5] Castelo Branco, N.A.A., Alves-Pereira, M., Martinho Pimenta, A., Reis Fereira, J. (2015) Clinical protocol for evaluating pathology induced by low frequency noise exposure. Paper presented at the Euronoise 2015, Maastricht, The Netherlands (Paper No.601)

[6] Medtronic (2017) Zephyr™ Biopatch, Annapolis, MD, USA. https://www.zephyranywhere.com

[7] SAM infrasound unit was the prototype recording system for CSI-ACHE. The unit now in use by CSI-ache is the SAM Scribe. See: Bakker, H., Rapley, B., Summers, R., Alves-Pereira, M., Dickinson, P. (2017) An affordable recording instrument for the acoustical characterisation of human environments. Paper presented at the ICBEN 2017, Zurich, Switzerland (Paper No. 3654)

[8] Kelley, N.D., McKenna, H.E., Hemphill, R.R., Etter, C.L., Garrelts, R.L., Linn, N.C. (1985) Acoustic noise associated with the MOD-1 wind turbine: its source, impact, and control, US Department of Energy, Contract No. DE-AC02-83CH-10093.

[9] Gotz, T., Janik V.M. (2011) Repeated elicitation of the acoustic startle reflex leads to sensitisation in subsequent avoidance behaviour and induces fear conditioning. *BMC Neuroscience*, 12, 30.

[10] Lousã, N., Monteiro, E., Alves-Pereira, M., Castelo Branco, N.A.A. (2003) Rat cochlea exposed to low frequency noise. Paper presencted at the ICBEN 2003, Rotterdam, The Netherlands (ISBN: 90-807990-1-7).

[11] Munzel, T., Gori, T., Babisch, W., Basner. M. (2014) Cardiovascular effects of environmental noise exposure. *European Heart Journal*, 35(13), 829–36.

[12] Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A. (2005) Actin and tubulin-based structures under low frequency noise stress. In: Recent Advances in Multidiscipinary Applied Physics, Mendez-Vilas (ed), Elsevier: London (ISBN-10: 0-80-044648-5)

[13] Chapman, S., St. George, A., Waller, K., Cakic, V., (2013) The Pattern of Complaints about Australian Wind Farms Does Not Match the Establishment and Distribution of Turbines: Support for the Psychogenic, 'Communicated Disease' Hypothesis. *PLoS ONE* 8(10): e76584. http://dx.doi.org/10.1371/journal.pone.0076584

[14] Rapley, B., Alves-Pereira, M., Bakker H (2017) The inadequacy of the A-frequency weighting for the assessment of adverse effects on human populations. Paper presented at ICBEN 2017, Zurich, Switzerland (Paper No. 3873).

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[15] Reis Ferreira, J., Albuquerque e Sousa, J., Foreid, P., Antunes, M., Cardoso, S., Alves-Pereira, M., Castelo Branco, N.A.A. (2006) Abnormal respiratory drive in vibroacoustic disease. Revista Portuguesa Pneumologia, XII (4), 369-374.

http://www.elsevier.pt/en/revistas/revista-portuguesa-pneumologia-320/pdf/S2173511506704080/S300/

[16] Castelo Branco, N.A.A., Monteiro, E., Costa e Silva, A., Reis Ferreira, J. Alves-Pereira, M. (2003) Respiratory epithelia in wistar rats born in low frequency noise plus varying amounts of additional exposure. *Revista Portuguesa Pneumologia*, IX (6), 481-92.

http://www.elsevier.pt/en/revistas/revista-portuguesa-pneumologia-320/pdf/S0873215915307029/S350/

[17] Silva, M.J., Carothers, A., Castelo Branco, N.A.A., Dias, A., Boavida, M.G. (1999). Sister chromatid exchanges workers exposed to noise and vibration. *Aviation, Space & Environmental Medicine*, 70 (March Suppl), A40-5.

[18] Silva, M.J., Carothers, A., Castelo Branco, N.A.A., Dias, A., Boavida, M.G. (2002). Increased levels of sister chromatid exchanges in military aircraft pilots. *Mutation Research,* 44(1), 129-34.

[19] M. J. Silva, A. Dias, A. Barreta, P. J. Nogueira, N. A. A. Castelo Branco, M G. Boavida (2002). Low frequency noise and whole-body vibration cause increased levels of sister chromatid exchange in splenocytes of exposed mice. *Teratogenesis, Carcinogenesis & Mutagenesis*, 22(3), 195-203.

[20] Castelo Branco, N.A.A. (2005). Mutagenesis and malignancy in vibroacoustic disease. Paper presented at the 12th International Conference on Sound and Vibration, Lisbon, Portugal (Paper No. 566).

[21] Torres, R., Tirado, G., Roman, A., Ramirez, R., Colon, H., Araujo, A., Pais, F., Lopo Tuna, J.M.C., Castelo Branco, M.S., Alves-Pereira, M., Castelo Branco, N.A.A. (2001). Vibroacoustic disease induced by long-term exposure to sonic booms. Paper presented at Internoise2001, The Hague, The Netherlands. (ISBN 9080655422)

[22] Araujo, A., Alves-Pereira, M., Joanaz de Melo, J., Castelo Branco, N.A.A. (2004) Vibroacoustic disease in a ten-year-old male. Paper presented at Internoise2004, Prague, Czech Republic. (ISBN: 80-01-03055-5)

[23] Alves-Pereira, M., Castelo Branco, N.A.A. (2007) In-home wind turbine noise is conducive to vibroacoustic disease. Paper presented at the Second International Meeting on Wind Turbine Noise, Lyon, France. (Paper No. 3, 11 pages).

[24] Castelo Branco, N.A.A., Alves-Pereira, M., Martinho Pimenta, A., Reis Ferreir,a J. (2015) Low frequency noise-induced pathology: contributions provided by the Portuguese wind turbine case. Paper presented at Euronoise 2015, Maastricht, The Netherlands.

[25] Anamnesis, or case histories, are *sine qua non* components of clinical medicine. These are not to be confused with anecdotal stories that are recounted amongst non-clinically-trained professionals. For more on this topic, please see reference [14].

[26] Castro, A.P., Águas, A.P., Grande, N.R., Monteiro, E., Castelo Branco, N.A.A. (1999) Increase in CD8+ and CD4+ T lymphocytes in patients with VAD. *Aviation, Space & Environmental Medicine*, 70 (March Suppl), A141-4.

[27] Águas, A.P., Esaguy N., Grande, N.R., Castro, A.P., Castelo Branco, N.A.A. (1999) Acceleration of lupus erythematosus-like processes by LFN in hybrid NZB-W mouse model. *Aviation, Space & Environmental Medicine*, 70 (March Suppl), A132-6.

[28] Oliveira, M.J., Sousa Pereira, A., Águas, A.P., Grande, N.R., Monteiro, E., Serrano, I., Castelo Branco, N.A.A. (1999) Effects of LFN upon the reaction of pleural milky spots to mycobacterial infection. *Aviation, Space & Environmental Medicine*, 70 (March Suppl), A137-40.

[29] Foreid P, Martinho Pimenta AJF, Alves-Pereira M, Castelo Branco NAA. Neuropsychological issues in vibroacoustic disease - Current findings. Proceedings 12th International Congress on Sound & Vibration, Lisbon, Portugal, July 11-14, 2005: No. 573 (10 pages).

[30] Martinho Pimenta, A.J.F., Castelo Branco, M.S.N., Castelo Branco, N.A.A. (1999). Balance disturbances in individuals with VAD. *Aviation, Space & Environmental Medicine*, 70 (March Suppl), A96-9.

[31] Cooper, S. (2014) The Results of an Acoustic Testing Program Cape Bridgewater Wind Farm. Prepared for Energy Pacific (Vic) Pty Ltd, Melbourne, Australia.

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Letter to the Editor re: 'How the factoid of wind turbines causing 'vibroacoustic disease' came to be 'irrefutably demonstrated''

Mariana Alves-Pereira,[1]
Nuno A. A. Castelo Branco[2]

1. School of Economic Sciences and Organizations, Universidade Lusófona, Portugal
2. Principal Investigator, VAD Project

As lead researchers in vibroacoustic disease (VAD), we have been made aware of the article by Chapman et al.[1] in which our work was greatly misrepresented and misunderstood. Correction and clarification are, therefore, required.

Chapman et al. reference a 2007 paper discussing *Public health and the importance of low frequency noise* (LFN), in which the LFN content of a 'Grain Terminal home' and a 'Wind Turbine home' are discussed.[2] The grain terminal (GT) case, originally presented in 2004, was first-authored by our cardiologist,[3] and the Wind Turbine (WT) case was first presented in 2007,[4] with a follow-up in 2010.[5] We stand behind the statements published in these papers.

Both families solicited our help. We did not select or procure either case, and we provided our services *pro bono*. Until then, our experience with LFN-induced pathology had been mostly within occupational exposures, not environmental or residential exposures.

It is not our intention herein to replicate the results of the above-mentioned studies, but some data is required to clarify to the issues at hand.

Some acoustical considerations

The owner of the WT-home paid an independent accredited firm for the acoustical measurements, and provided our team with the numerical information for further analyses. No commercial, financial or professional agreements (contractual or otherwise) existed between this firm and the VAD research team. Figure 1 shows acoustical data compiled by VAD researchers.

As seen in Figure 1, levels of LFN are clearly increased in the bedroom when WT are in operation.

Chapman et al. claimed our work was "of abject methodological quality" (p.247) because "The noise measuring equipment used to measure infrasound in the two houses was different" (p.246). However, in that paper, Alves-Pereira et al.,[2] being fully aware of that technical limitation, wrote: "In a perfect world, designed for the most efficient and accurate scientific studies, all noise assessments ought to be conducted with the same equipment and with the same procedures. This is not feasible. So, despite on-site and factory calibrations, a legitimate question will always remain: can the differences between the ILFN levels in the [GT and WT] homes (…) be due to differences in the noise measuring equipment and procedures alone? Despite this legitimate question, these data are sufficient to warrant precautionary measures".[2]

Some clinical considerations

Another criticism emerged because we "took no account of inattention and lack of energy in school children being common" (p.247).

Upon being contacted by these two families, VAD Team researchers provided non-invasive VAD screening tests. The rationale for these specific tests would require significant self-citation.

The 10-year-old residing since gestation in the GT-home disclosed "the most severe cardiovascular condition", scoring the highest values in mitral valve leaflet thickening and pericardial thickening.[3] These echo-imaging findings had only previously been seen in older LFN-exposed workers,[6] and were entirely unexpected in a 10-year-old child.

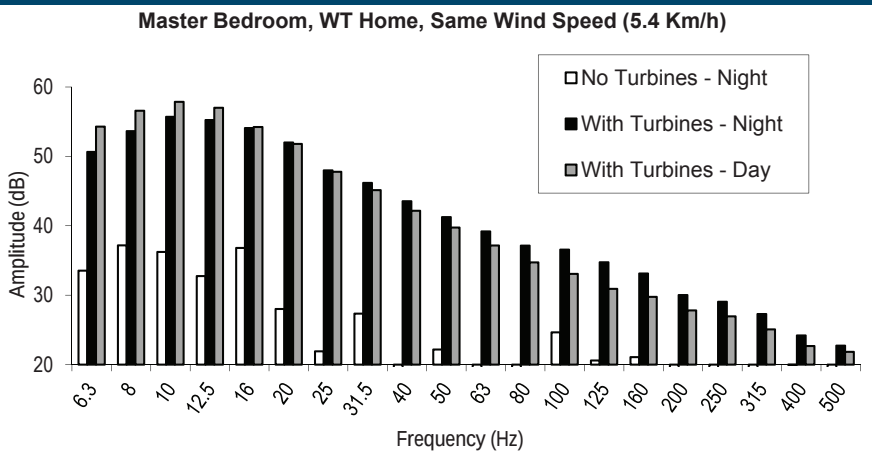
Furthermore, this child had "suffered from asthma until the age of 1 year. At 5–8 months of age, he was medicated for reflux, and then again until he was 1 year old. At 8 months he suffered pneumonia. After the age of 1, he began to develop repeated ear infections that were not responsive to antibiotics. At age 3 he underwent ear surgery. At the age of 5, at school, he suddenly lost his vision, and was taken to the hospital where the EEG revealed a late onset epileptic seizure. Nose bleeds without an apparent cause used to be frequent, but have subsided with age. There is no history of rheumatic fever, radiation or asbestos exposure." (In: Alves-Pereira et al.,[2] citing Araújo et al.[2])

In the WT-case, Evoked Potentials provided to the 12-year-old boy, disclosed "asymmetries in the right and left nerve conduction times, and the right I-V interlatency value was at the threshold of normal values (4.44ms). The endogenous evoked potential P300 recording occurred at 352ms (normal: 300ms)".[4] Taken alone, these values are not relevant, but after a two-month vacation away from the WT home, the child disclosed significant improvement: 322ms.

Neurophysiological tests were provided to this child because four months after WT began operation, the parents received a letter from the child's teacher voicing "concern for the growing difficulties of an otherwise outstanding student, (…) it seems that [the child] has lost interest, makes a lesser effort, as if he were permanently tired. In Physical Education, an abnormal amount of tiredness is also observed. Is [the child] leading a healthy life? Does he sleep sufficient hours during the night?"[2]

These objective medical tests and clinical histories, disclosing morphological and brain potential changes, go well beyond the scope of "common" lack of energy in school children, as Chapman et al. suggest.

Figure 1: Data collected by an independent accredited firm, inside the Master Bedroom of the WT-home. Comparison of values at the same wind speed.[4]



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Some biological considerations

The fundamental histological feature found both in LFN-exposed patients and LFN-exposed laboratory animals was also observed in the horses raised on the WT-home property: thickening of vascular walls due to proliferation of extra-cellular matrices in the absence of an inflammatory process.[5] To fully comprehend the rationale and the highly significant implications of these results, a large amount of self-citation would now be required.

Understanding the pathophysiology of LFN-induced diseases requires knowledge on cellular tensegrity architecture[7] that goes beyond the classical models of general physiopathology.[8,9] Mechanotransduction cellular signaling,[9] a relatively new concept, becomes of paramount importance because it is a major target of the LFN agent.[10] In the absence of this new cellular model, the nature of the biological response prompted by LFN exposure cannot be fully understood.

Final remarks

Given the complexity of this subject, difficulty in recognising the significance of our scientific findings reported over the past three decades is entirely understandable.

Unfortunately, the vast majority of studies concerning health impacts of WT on neighbouring residents do not yet provide an adequate quantification of the physical agent of disease, and are based on highly subjective questionnaires lacking clinical corroboration on relevant endpoints. This is flagrantly perpetuated by the Wind Turbine Health Impact Study, as prepared for the State of Massachusetts.[11] In our ongoing investigations, we have repeatedly pointed out the inadequacy of questionnaires as a valid measure of health effect,[12] a position shared by the Strategic Health Impact Assessment on Wind Energy Development, as prepared for the State of Oregon.[13]

We reiterate that LFN-contaminated homes are a significant public health concern, and substantial health deterioration can be observed in humans and animals dwelling without respite in LFN-rich environments. (Disclaimer: this statement cannot and should not be construed as an argument against the implementation of wind turbines.)

On 30 May 2013, the Supreme Court of Justice in Portugal decided upon the removal of the four WT, initially erected in 2006.[14]

References

1. Chapman S, St George A. How the factoid of wind turbines causing "vibroacoustic disease" came to be "irrefutably demonstrated". *Aust NZ J Public Health* 2013; 37:244-9.
2. Alves-Pereira M, Castelo Branco NAA. Public health and noise exposure: the importance of low frequency noise. *Proceedings of the Inter-Noise 2007 Conference* ; 2007 Aug 28-31; Istanbul, Turkey.
3. Araujo A, Alves-Pereira M, Joanaz de Melo J, Castelo Branco NAA. Vibroacoustic disease in a ten-year-old male. *Proceedings of the Internoise 2004 Conference*; 2004 Aug 22-25; Prague, Czech Republic
4. Alves-Pereira M, Castelo Branco NAA. In-home wind turbine noise is conducive to vibroacoustic disease. *Proceedings of the 2nd International Meet Wind Turbine Noise*; 2007 Sep 20-21; Lyon, France.
5. Castelo Branco NAA, Costa e Curto T, Mendes Jorge L, Cavaco Faísca J, Amaral Dias L, Oliveira P, et al. Family with wind turbines in close proximity to home: follow-up of the case presented in 2007. *Proceedings of the 14th International Meet Low Frequency Noise Vibration Control*; 2010 Jun 9-11; Aalborg, Denmark.
6. Marciniak W, Rodriguez E, Olsowska K, Botvin I, Araujo A, Pais F, et al. Echocardiography in 485 aeronautical workers exposed to different noise environments. *Aviat Space Environ Med*. 1999;70 Suppl 3:A46-53.
7. Ingberg DE. Cellular tensegrity: defining new rules of biological design that governs the cytoskeleton. *J Cell Sci*. 1993;104:613-27.
8. Stamenovic D, Fredberg JJ, Wang N, Butler JP, Ingber DE. A microstructural approach to cytoskeletal mechanics based on tensegrity. *J Theoretical Biol* 1996; 181: 125-136.
9. Wang N, Butler JP, Ingber DE. Mechanotransduction across the cell surface and through the cytoskeleton. *Science* 1993; 260: 1124-27.
10. Alves-Pereira M, Castelo Branco NAA. Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling. *Prog Biophy Molec Biol* 2007; 93: 256-79.
11. Executive Office of Energy and Environmental Affairs. *Wind Turbine Health Impact Study: Report of Independent Expert Panel* [Internet]. Boston (MA): Commonwealth of Massachusetts EEA; 2012 [cited 2014 Jan 15]. Available from: http://www.mass.gov/eea/docs/dep/energy/wind/turbine-impact-study.pdf
12. Alves-Pereira M. *Review of Wind Turbine Health Impact Study: Report of Independent Expert Panel* [Internet]. Boston (MA): Massachusetts Department of Environmental Protection, Massachusetts Department of Public Health; 2012 [cited 2014 Jan 15]. Available from: http://docs.wind-watch.org/MassDEP-wind-health-2-Alves_Pereira.pdf
13. Oregon Health Authority. *Strategic Health Impact Assessment on Wind Energy Development in Oregon* [Internet]. Portland (OR): Government of Oregon Public Health Division; 2013 [cited 2014 Jan 15]. Available from: http://public.health.oregon.gov/HealthyEnvironments/TrackingAssessment/HealthImpactAssessment/Documents/Wnd%20Energy%20HIA/Wind%20HIA_Final.pdf
14. Supreme Court of Justice (Portugal). *Decision No. 2209/08.0TBTVD.L1.S1. Relevant Case Law in the Field of Environment in 2009 - Supreme Administrative Court, 22nd September, 2009, Judgement 161/05.2TBVLG.S1, Sector: Noise* [Internet]. [cited 2014 Jan 15]. Available from: http://www.dgsi.pt/jstj.nsf/954f0ce6ad9dd8b980256b5f003fa814/4559d6d733d1589780257b7b004d464b?OpenDocument

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BEFORE THE PUBLIC UTILITIES COMMISSION
OF THE STATE OF SOUTH DAKOTA

IN THE MATTER OF THE APPLICATION BY PREVAILING WIND PARK, LLC FOR A PERMIT OF A WIND ENERGY FACILITY IN BON HOMME COUNTY, CHARLES MIX COUNTY AND HUTCHINSON COUNTY, SOUTH DAKOTA, FOR THE PREVAILING WIND PARK PROJECT	* * * * * * * * *	**APPLICANT'S RESPONSES TO INTERVENORS' FIRST SET OF DATA REQUESTS EL18-026**

Below, please find the responses of Prevailing Wind Park, LLC ("Applicant" or "Prevailing Wind Park") to Intervenors' First Set of Data Requests to Applicant.

Objections to Definitions

Prevailing Wind Park objects to the definitions of "You" and "Your". For purposes of these responses, "You" and "Your" shall refer to Prevailing Wind Park, LLC, the applicant in this matter and its parent company, sPower Development Company, LLC, and any employees thereof.

1-1) Provide copies of all data requests and responses submitted to or by You.

Lisa Agrimonti: Copies of all data requests and non-confidential responses submitted to or by Prevailing Wind Park are provided at the following link: https://fredriksonandbyron.sharefile.com/d-s62d675400cc4b248. Prevailing Wind Park will provide confidential responses provided in response to Staff Data Requests Set 1 pursuant to a protective agreement.

1-2) Produce all manuals, guides, information sheets, studies, reports, and like documents that refer or relate to the turbine model selected for the Project, namely the GE 3.8-137.

Lisa Agrimonti: Prevailing Wind Park objects to this request to the extent that it seeks information and/or documents not in the possession, custody, or control of Prevailing Wind Park, or that are confidential. Prevailing Wind Park further objects to this request because it is overbroad and vague in its request for "like documents." Subject to and without waiving this objection, Prevailing Wind Park further responds:

Peter Pawlowski: sPower Development Company LLC ("sPower") has a copy of the safety manual for the GE 3.8-137 turbine model that is subject to a confidentiality agreement. sPower is in contact with General Electric to determine under what circumstances the document may be disclosed in this docket. Additional information about the GE turbine can be found on General Electric's publicly available website. See https://www.ge.com/renewableenergy/wind-energy/turbines/3mw-platform.

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1-3) Produce all studies, reports, and like documents that refer or relate to ice-throw from turbines.

Lisa Agrimonti: Prevailing Wind Park objects to this request to the extent that it seeks information and/or documents not in the possession, custody, or control of Prevailing Wind Park, or that is confidential. Prevailing Wind Park further objects to this request because it is overbroad and vague in its request for "like documents." Subject to and without waiving this objection, Prevailing Wind Park further responds:

Peter Pawlowski: See response to 1-2.

1-4) Produce all studies, reports, and like documents analyzing turbines 586 feet or taller.

Lisa Agrimonti: Prevailing Wind Park objects to this request to the extent that it seeks information and/or documents not in the possession, custody, or control of Prevailing Wind Park, or that is confidential. Prevailing Wind Park further objects to this request because it is overbroad and vague in its request for "like documents" and the meaning of "analyzing". Subject to and without waiving this objection, Prevailing Wind Park further responds:

Peter Pawlowski: See the Application for turbine related studies.

1-5) Produce all publications, manuals, guides, information sheets, studies, reports, and like documents that refer or relate to safety distances from turbines and/or danger zones.

Lisa Agrimonti: Prevailing Wind Park objects to this request to the extent that it seeks information and/or documents not in the possession, custody, or control of Prevailing Wind Park, or that is confidential. Prevailing Wind Park further objects to this request because it is overbroad and vague in its request for "like documents." Subject to and without waiving this objection, Prevailing Wind Park further responds:

Scott Creech: See response to Data Request 1-3. Safety manuals for the turbines do not specify safety or danger zones applicable to standard turbine operations. Rather, safety manuals identify specific precautionary measures workers should implement when conducting routine maintenance and in emergency situations.

1-6) Produce all written communications, electronic or otherwise, between You and any representative of Bon Homme, Charles Mix, or Hutchinson counties.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is overbroad regarding the subject matter, time and individuals and not reasonably calculated to lead to the discovery of admissible evidence.

1-7) Produce all written communications, electronic or otherwise, between You and any representative of Planning and Development District III, including but not limited to Brian McGinnis.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is overbroad regarding the subject matter and time and not reasonably calculated to lead to the discovery of admissible evidence.

1-8) Produce all written communications, electronic or otherwise, between You and any representative of the Yankton Sioux Tribe.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is overbroad. Prevailing Wind Park further objects to this request to the extent that it seeks confidential information.

1-9) Explain Erik Johnson's role with the Project.

Peter Pawlowski: Erik Johnson has no role in the Project. Mr. Johnson sits on the board of Prevailing Winds, LLC and otherwise supports and advocates for the Project as a private citizen.

1-10) Explain Ronnie Hornstra's role with the Project.

Peter Pawlowski: Ronnie (Ron) Hornstra has no role in the project. Mr. Hornstra sits on the board of Prevailing Winds, LLC and otherwise supports and advocates for the Project as a private citizen.

1-11) State the number of participating residences located within (a) 1,000 feet of a turbine; (b) 2,000 feet of a turbine; (c) 1/2 mile of a turbine; (d) 1 mile of a turbine; and (e) 2 miles of a turbine.

Bridget Canty: As shown in layout shown in the Application:

(a) There are **0** participating residences within 1,000 feet of a proposed turbine location;

(b) There are **5** participating residences within 2,000 feet of a proposed turbine location;

(c) There are **21** participating residences within ½ mile of a proposed turbine location;

(d) There are **40** participating residences within one mile of a proposed turbine location; and

(e) There are **45** participating residences within two miles of a proposed turbine location.

1-12) State the number of non-participating residences located within (a) 1,000 feet of a turbine; (b) 2,000 feet of a turbine; (c)1/2 mile of a turbine; (d) 1 mile of a turbine; and (e) 2 miles of a turbine.

Bridget Canty: As shown in the layout in the Application:

(a) There are **0** non-participating residences within 1,000 feet of a proposed turbine location;

(b) There is **1** non-participating residence within 2,000 feet of a proposed turbine location;

(c) There are **10** non-participating residences within ½ mile of a proposed turbine location;

(d) There are **46** non-participating residences within one mile of a proposed turbine location; and

(e) There are **82** non-participating residences within two miles of a proposed turbine location.

1-13) How many participating landowners are there for the Project?

Peter Pawlowski: There are 176 land rights agreements related to this Project.

1-14) Assuming all turbines are built, how many participating landowners will have a turbine located on their property?

Peter Pawlowski: Based on the layout in the Application, 35 landowners would have turbines on their properties.

1-15) Identify by county, state, country, or regulatory body all setbacks from non-participating residences greater than 2,000 feet of which you are aware. In doing so, provide the distance of the setback that exists.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is overly broad, unduly burdensome, and not reasonably calculated to lead to the discovery of admissible evidence. Further, since permitting requirements are typically public, this information is as available to the Intervenors as it is to Prevailing Wind Park.

1-16) Admit that the attached Wind Energy Lease and Wind Easement Agreement marked as Exhibit A is an example of an easement agreement provided to landowners for the Project.

Peter Pawlowski: Exhibit A to Intervenors First Set of Data Requests to Applicant is a form of agreement Prevailing Winds, LLC and Prevailing Wind Park, LLC used to acquire easements from landowners. Prevailing Wind Park treats its easement forms as confidential and undertakes efforts to maintain the documents as such, as they do contain

information, the disclosure of which would result in commercial and competitive harm to the company. Prevailing Wind Park maintains that Exhibit A should be treated as a confidential document in this proceeding.

1-17) Identify any Bon Homme, Charles Mix, or Hutchinson county commissioner or planning or zoning board member that has executed any agreement with You. Explain any such agreement.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is not reasonably calculated to lead to the discovery of admissible evidence and to the extent it seeks confidential information. Subject to and without waiving this objection, Prevailing Wind Park further responds:

Peter Pawlowski: Bon Homme County Commissioner Bruce Voigt is a participating landowner in the Prevailing Wind Park Project.

1-18) Identify all other wind projects for which You have been involved with the development thereof in the past 10 years. For each project, identify all applicable setbacks and noise restrictions.

Lisa Agrimonti: Prevailing Wind Park objects to this request as overly broad, unduly burdensome, and not reasonably calculated to lead to the discovery of admissible evidence. The setbacks for a wind farm in a different jurisdiction are not relevant to the South Dakota Public Utilities Commission's decision on the applicable setbacks for the proposed Project. Subject to and without waiving this objection, Prevailing Wind Park further responds:

Peter Pawlowski: sPower has constructed, owns and operates Pioneer Wind Park in Wyoming and Latigo Wind Park in Utah.

1-19) Identify the legal entity from which You are procuring the turbines.

Peter Pawlowski: GE Renewables North America, LLC.

1-20) Please state the difference between Prevailing Winds LLC and Prevailing Winds Park LLC.

Peter Pawlowski: Prevailing Wind Park, LLC is the applicant and holder of the Project assets. Prevailing Winds, LLC sold Prevailing Wind Park, LLC to sPower Development Company, LLC.

1-21) Explain why NEC1, LLC was formed and its role in the Project?

Peter Pawlowski: The entity referenced above is not related to this Project.

1-22) Identify any Bon Homme County, Charles Mix County, or Hutchinson County, South Dakota County Commissioner or Planning or Zoning Board Member that is or was an investor in the Project.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is overly broad and not reasonably calculated to lead to the discovery of admissible evidence.

Peter Pawlowski: Bon Homme County Commissioner Bruce Voight is a participant in the Project.

1-23) Identify the number of incidents that have occurred at Your projects where a turbine has fallen over, started on fire, or had a blade or piece of blade fall off the turbine and briefly describe each incident. Produce any documents, reports, communications, studies, complaints, or the like related to any such incident.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is overly broad and vague.

Peter Pawlowski: No operating wind project owned by sPower has had incidents as listed above.

1-24) Identify the number of complaints You have received regarding ice being thrown or falling from a turbine. Produce any documents, reports, communications, studies, complaints, or the like related to any such complaint.

Peter Pawlowski: None.

1-25) Identify the number of injuries that have occurred at Your projects resulting from a turbine malfunction or ice being thrown or falling from a turbine.

Peter Pawlowski: None.

1-26) State the furthest distance for which you are aware that ice has been thrown from a turbine.

Scott Creech: I do not have an exact measurement, but the farthest distance I am aware of ice being thrown from a turbine is approximately 250 feet.

1-27) State the furthest distance for which you are aware that a blade or piece of blade has been thrown from a turbine.

Scott Creech: During my operations career, I have only observed one blade being dropped from a turbine a distance of approximately 54 meters, about half the blade diameter which was 108 meters.

Dated this 19th day of September, 2018.

By */s/Lisa M. Agrimonti*

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BEFORE THE PUBLIC UTILITIES COMMISSION
OF THE STATE OF SOUTH DAKOTA

IN THE MATTER OF THE APPLICATION BY PREVAILING WIND PARK, LLC FOR A PERMIT OF A WIND ENERGY FACILITY IN BON HOMME COUNTY, CHARLES MIX COUNTY AND HUTCHINSON COUNTY, SOUTH DAKOTA, FOR THE PREVAILING WIND PARK PROJECT	* * * * * * * * * *	**APPLICANT'S RESPONSES TO INTERVENORS' SECOND SET OF DATA REQUESTS EL 18-026**

Below, please find Applicant's responses to Intervenors' Second Set of Data Requests to Applicant.

Objections to Definitions

Prevailing Wind Park objects to the definitions of "You" and "Your". For purposes of these responses, "You" and "Your" shall refer to Prevailing Wind Park, LLC, the applicant in this matter and its parent company, sPower Development Company, LLC, and any employees thereof.

2-1) Provide the application for a Large Wind Energy System Permit You submitted to Bon Homme County.

Peter Pawlowski: The application is available at: https://fredriksonandbyron.sharefile.com/d-sf499da35c754466a

2-2) Provide any application You have submitted to Bon Homme County, Charles Mix County, or Hutchinson County.

Peter Pawlowski: Responsive documents are available at https://fredriksonandbyron.sharefile.com/d-sf499da35c754466a

2-3) What is the modeled noise level and shadow flicker at the Presbyterian-Bohemian Cemetery located at the intersection of 401st Avenue and 295th Street near turbines 48 and 57?

Aaron Anderson: Assuming the figure below shows the Presbyterian-Bohemian Cemetery, the Project will result in approximately 10 hours per year of shadow flicker at the Presbyterian-Bohemian Cemetery using the GE 3.8-137 model.

EXHIBIT
Int. I-27



Chris Howell: The noise modeling for the GE 3.8-137 turbine predicts a sound level from turbines of 33.8 dBA at this location.

2-4) Provide a map that shows the proposed turbines within 2 miles from the residence of Ms. Kelly Pazour (29668 402nd Avenue, Wagner, South Dakota 57380) and the applicable setbacks for those turbines, similar to the map on Page 88 of 156 of Staff Exhibit_JT-1 in Docket EL18-003 for Ms. Teresa Kaaz (http://puc.sd.gov/commission/dockets/electric/2018/EL18-003/exhibits/staff/sl.pdf).

Bridget Canty: See Attachment 2-4 for turbine locations. For setbacks, see Figure 5 in the Application.

2-5) Provide a map that shows the proposed turbines within 2 miles from the residence of Mr. Jerome Powers (40427 294th Street, Wagner, South Dakota 57380) and the applicable setbacks for those turbines, similar to the map on Page 88 of 156 of Staff Exhibit_JT-1 in Docket EL18-003 for Ms. Teresa Kaaz (http://puc.sd.gov/commission/dockets/electric/2018/EL18-003/exhibits/staff/sl.pdf).

Bridget Canty: See Attachment 2-5 for turbine locations. For setbacks, see Figure 5 in the Application.

2-6) Provide a map that shows the proposed turbines within 2 miles from the residence of Mr. Kevin Andersh and the applicable setbacks for those turbines, similar to the map on Page 88 of 156 of Staff Exhibit_JT-1 in Docket EL18-003 for Ms. Teresa Kaaz (http://puc.sd.gov/commission/dockets/electric/2018/EL18-003/exhibits/staff/sl.pdf).

Bridget Canty: See attachment 2-6 for turbine locations. For setbacks, see Figure 5 in the Application.

2-7) Provide a map that shows the proposed turbines within 2 miles from the residence of Mr. Gregg Hubner (29976 406th Avenue, Avon, South Dakota 57315) and the applicable setbacks for those turbines, similar to the map on Page 88 of 156 of Staff Exhibit_JT-1 in Docket EL18-003 for Ms. Teresa Kaaz (http://puc.sd.gov/commission/dockets/electric/2018/EL18-003/exhibits/staff/sl.pdf).

Bridget Canty: See response to Staff Request DR 2-23.

2-8) Provide a map that shows the proposed turbines within 2 miles from the residence of Mr. Paul Schoenfelder (40228 296th Street, Wagner, South Dakota 57380) and the applicable setbacks for those turbines, similar to the map on Page 88 of 156 of Staff Exhibit JT-1 in Docket EL18-003 for Ms. Teresa Kaaz (http://puc.sd.gov/commission/dockets/electric/2018/EL18-003/exhibits/staff/sl.pdf).

Bridget Canty: See response to Staff Request DR 2-24.

2-9) Appendix T, page 84 email from Jennifer Bell to Bridget Canty on the subject of Prevailing Winds Tribal Meeting dated Monday, March 26, 2018 10:02:20 AM. Please provide any additional correspondence between Kip Spotted Eagle and/or the leadership of the Yankton Sioux Tribe, including any agreements made in regards to cultural discoveries during the construction of the Prevailing Winds Park project.

Lisa Agrimonti: Prevailing Wind Park objects to this request because it is overbroad and ambiguous regarding the parties to the requested communications. Prevailing Wind Park further objects to this request to the extent that it seeks confidential information.

Dated this 24th day of September, 2018.

By: */s/ Lisa M. Agrimonti*

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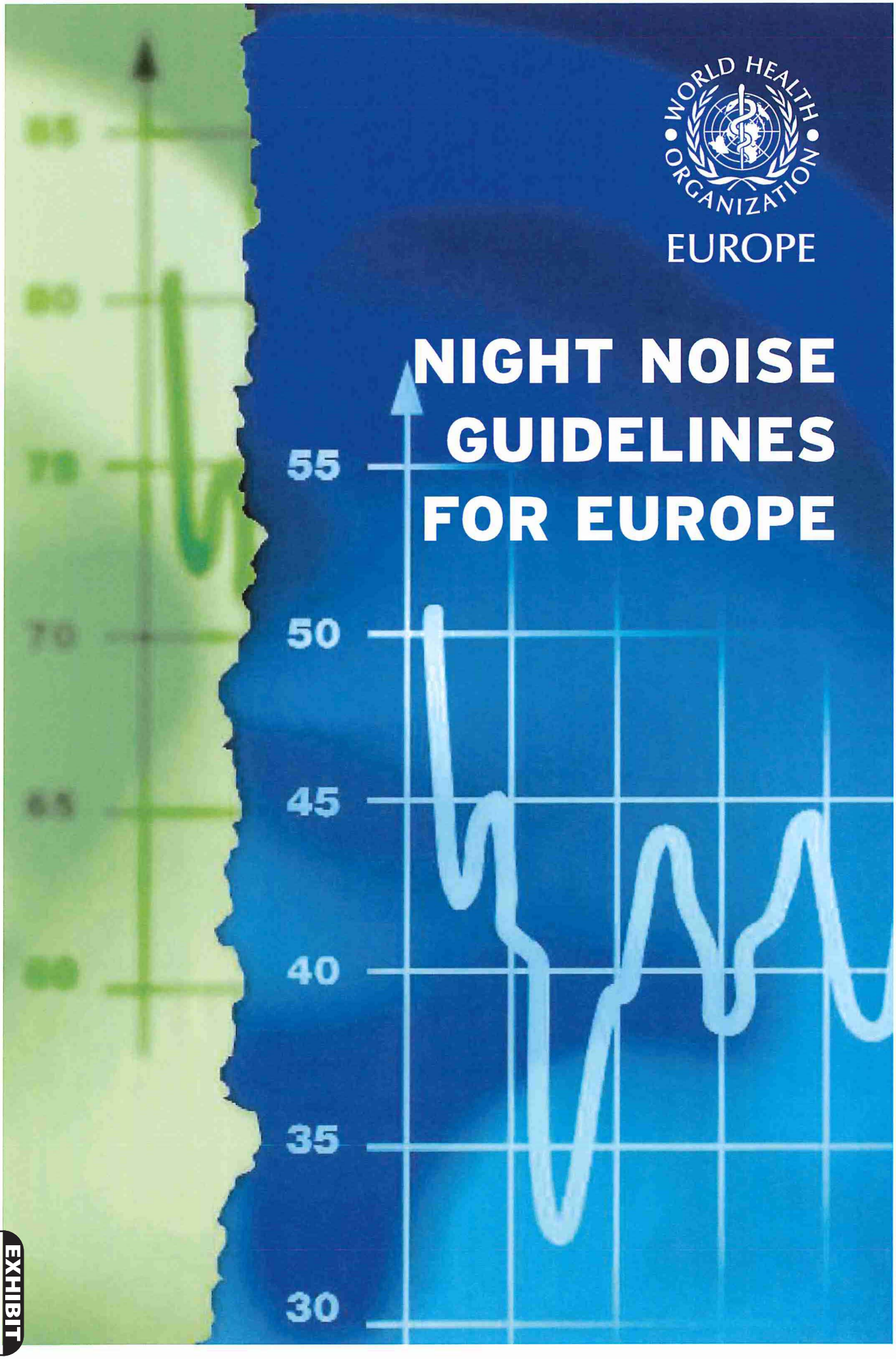


EXHIBIT
Int. I-31

010097



EUROPE

NIGHT NOISE GUIDELINES FOR EUROPE

010099

Keywords

NOISE - ADVERSE EFFECTS - PREVENTION AND CONTROL

SLEEP DEPRIVATION - ETIOLOGY

SLEEP DISORDERS - PREVENTION AND CONTROL

ENVIRONMENTAL HEALTH

HEALTH POLICY - LEGISLATION

GUIDELINES

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ABSTRACT

The WHO Regional Office for Europe set up a working group of experts to provide scientific advice to the Member States for the development of future legislation and policy action in the area of assessment and control of night noise exposure. The working group reviewed available scientific evidence on the health effects of night noise, and derived health-based guideline values. In December 2006, the working group and stakeholders from industry, government and nongovernmental organizations reviewed and reached general agreement on the guideline values and key texts for the final document of the *Night noise guidelines for Europe*.

Considering the scientific evidence on the thresholds of night noise exposure indicated by $L_{night,outside}$ as defined in the Environmental Noise Directive (2002/49/EC), an $L_{night,outside}$ of 40 dB should be the target of the night noise guideline (NNG) to protect the public, including the most vulnerable groups such as children, the chronically ill and the elderly. $L_{night,outside}$ value of 55 dB is recommended as an interim target for the countries where the NNG cannot be achieved in the short term for various reasons, and where policy-makers choose to adopt a stepwise approach. These guidelines are applicable to the Member States of the European Region, and may be considered as an extension to, as well as an update of, the previous WHO *Guidelines for community noise* (1999).

010104

FOREWORD

WHO defines health as a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity, and recognizes the enjoyment of the highest attainable standard of health as one of the fundamental rights of every human being. Environmental noise is a threat to public health, having negative impacts on human health and well-being. In order to support the efforts of the Member States in protecting the population´s health from the harmful levels of noise, WHO issued *Guidelines for community noise* in 1999, which includes guideline values for community noise in various settings based on the scientific evidence available. The evidence on health impacts of night noise has been accumulated since then.

In the WHO European Region, environmental noise emerged as the leading environmental nuisance triggering one of the most common public complaints in many Member States. The European Union tackled the problem of environmental noise with an international law on the assessment and management of environmental noise. The WHO Regional Office for Europe developed the *Night noise guidelines for Europe* to provide expertise and scientific advice to the Member States in developing future legislations in the area of night noise exposure control and surveillance, with the support of the European Commission. This guidelines document reviews the health effects of night time noise exposure, examines exposure-effects relations, and presents guideline values of night noise exposure to prevent harmful effects of night noise in Europe. Although these guidelines are neither standards nor legally binding criteria, they are designed to offer guidance in reducing the health impacts of night noise based on expert evaluation of scientific evidence in Europe.

The review of scientific evidence and the derivation of guideline values were conducted by outstanding scientists. The contents of the document were peer reviewed and discussed for a consensus among the experts and the stakeholders from industry, government and nongovernmental organizations. We at WHO are thankful for those who contributed to the development and presentation of this guidelines and believe that this work will contribute to improving the health of the people in the Region.

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010105

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010106

EXECUTIVE SUMMARY

INTRODUCTION

The aim of this document is to present the conclusions of the WHO working group responsible for preparing guidelines for exposure to noise during sleep. This document can be seen as an extension of the WHO *Guidelines for community noise* (1999). The need for "health-based" guidelines originated in part from the European Union Directive 2002/49/EC relating to the assessment and management of environmental noise (commonly known as the Environmental Noise Directive and abbreviated as END) which compels European Union Member States to produce noise maps and data about night exposure from mid-2007. The work was made possible by a grant from the European Commission and contributions from the Swiss and German governments.

Although a number of countries do have legislation directed at controlling night noise exposure, there is little information on actual exposure and its subsequent effects on the population. Estimates made in some countries of the number of people highly disturbed by noise during sleep (see Fig. 1 for the Netherlands as an example) indicate that a substantial part of the population could be exposed to levels that might risk their health and well-being.

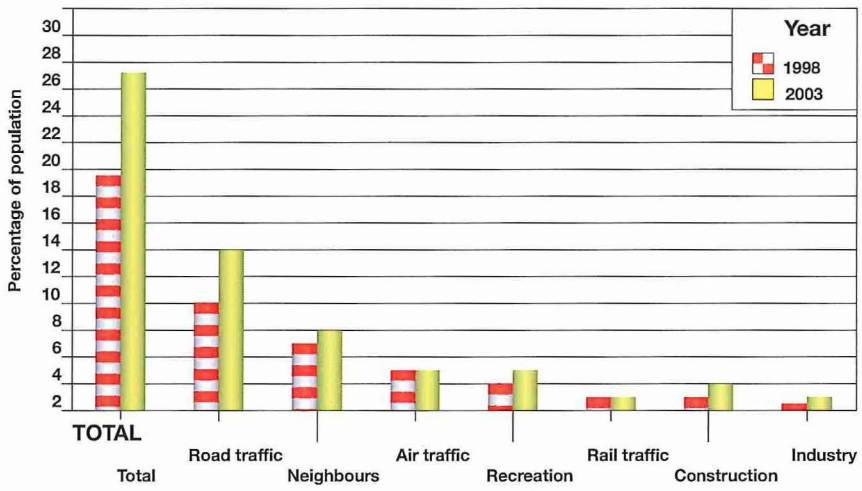


Fig. 1. Percentage of population highly disturbed by noise during sleep in the Netherlands: survey results for 1998 and 2003

As direct evidence concerning the effects of night noise on health is rarely available, these guidelines also use indirect evidence: the effects of noise on sleep and the relations between sleep and health. The advantage of this approach is that a lot of medical evidence is available on the relation between sleep and health, and detailed information also exists on sleep disturbance by noise.

010107

PROCESS OF DEVELOPING GUIDELINES

In 2003, the WHO Regional Office for Europe set up a working group of experts to provide scientific advice to the European Commission and to its Member States for the development of future legislation and policy action in the area of control and surveillance of night noise exposure. The review of available scientific evidence on the health effects of night noise was carried out by an interdisciplinary team who set out to derive health-based guideline values. The contributions from the experts were reviewed by the team and integrated into draft reports following discussion at four technical meetings of the working group. In 2006, all the draft reports were compiled into a draft document on guidelines for exposure to noise at night, which was reviewed and commented on by a number of stakeholders and experts.

At the final conference in Bonn, Germany, on 14 December 2006, representatives from the working group and stakeholders from industry, government and non-governmental organizations reviewed the contents of the draft document chapter by chapter, discussed several fundamental issues and reached general agreement on the guideline values and related texts to be presented as conclusions of the final WHO *Night noise guidelines for Europe*.

NOISE INDICATORS

From the scientific point of view the best criterion for choosing a noise indicator is its ability to predict an effect. Therefore, for different health end points, different indicators could be chosen. Long-term effects such as cardiovascular disorders are more correlated with indicators summarizing the acoustic situation over a long time period, such as yearly average of night noise level outside at the facade ($L_{night,outside}$)[1], while instantaneous effects such as sleep disturbance are better with the maximum level per event (L_{Amax}), such as passage of a lorry, aeroplane or train.

From a practical point of view, indicators should be easy to explain to the public so that they can be understood intuitively. Indicators should be consistent with existing practices in the legislation to enable quick and easy application and enforcement. $L_{night,outside}$, adopted by the END, is an indicator of choice for both scientific and practical use. Among currently used indicators for regulatory purposes, L_{Aeq} (A-weighted equivalent sound pressure level) and L_{Amax} are useful to predict short-term or instantaneous health effects.

SLEEP TIME

Time use studies, such as that undertaken by the Centre for Time Use Research, 2006 (www.timeuse.org/access/), show that the average time adult people are in bed is around 7.5 hours, so the real average sleeping time is somewhat shorter. Due to personal factors like age and genetic make-up there is considerable variation in sleeping time and in beginning and end times. For these reasons, a fixed interval of 8 hours is a minimal choice for night protection.

Though results vary from one country to another, data show (see Fig. 2 as an example) that an 8-hour interval protects around 50% of the population and that it would take a period of 10 hours to protect 80%. On Sundays, sleeping time is consistently 1 hour longer, probably due to people recovering from sleep debt incurred during the week. It should also be borne in mind that (young) children have longer sleeping times.

[1] L_{night} is defined in the END as the outside level. In order to avoid any doubt, the suffix "outside" is added in this document.

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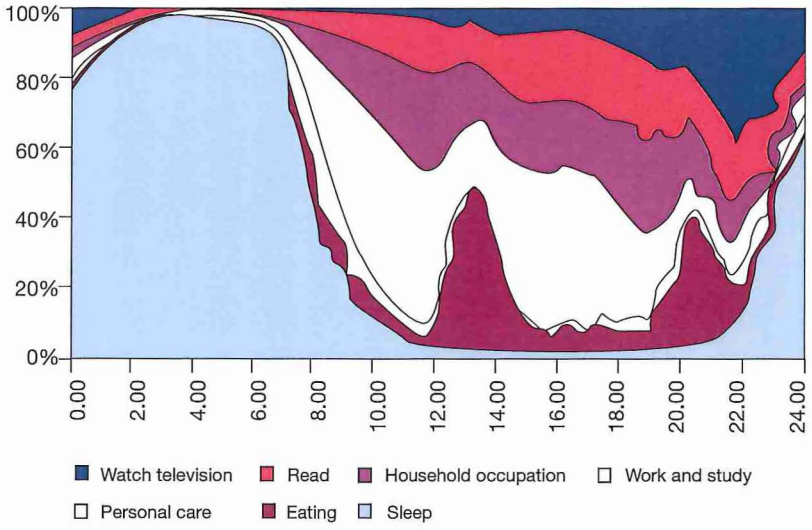


Fig. 2
Percentage of time that the Portuguese population spend asleep or in different activities

Source: http://www.ine.pt/prodserv/destaque/arquivo.asp, based on a study by the Instituto Nacional de Estatistica Portugal, 1999.

NOISE, SLEEP AND HEALTH

There is plenty of evidence that sleep is a biological necessity, and disturbed sleep is associated with a number of health problems. Studies of sleep disturbance in children and in shift workers clearly show the adverse effects.

Noise disturbs sleep by a number of direct and indirect pathways. Even at very low levels physiological reactions (increase in heart rate, body movements and arousals) can be reliably measured. Also, it was shown that awakening reactions are relatively rare, occurring at a much higher level than the physiological reactions.

DEFINITION OF "SUFFICIENT" AND "LIMITED" EVIDENCE

Sufficient evidence: A causal relation has been established between exposure to night noise and a health effect. In studies where coincidence, bias and distortion could reasonably be excluded, the relation could be observed. The biological plausibility of the noise leading to the health effect is also well established.

Limited evidence: A relation between the noise and the health effect has not been observed directly, but there is available evidence of good quality supporting the causal association. Indirect evidence is often abundant, linking noise exposure to an intermediate effect of physiological changes which lead to the adverse health effects.

The working group agreed that there is sufficient evidence that night noise is related to self-reported sleep disturbance, use of pharmaceuticals, self-reported health problems and insomnia-like symptoms. These effects can lead to a considerable burden of disease in the population. For other effects (hypertension, myocardial infarctions, depression and others), limited evidence was found: although the studies were few or not conclusive, a biologically plausible pathway could be constructed from the evidence.

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An example of a health effect with limited evidence is myocardial infarction. Although evidence for increased risk of myocardial infarction related to L_{day} is sufficient according to an updated meta-analysis, the evidence in relation to $L_{night, outside}$ was considered limited. This is because $L_{night, outside}$ is a relatively new exposure indicator, and few field studies have focused on night noise when considering cardiovascular outcomes. Nevertheless, there is evidence from animal and human studies supporting a hypothesis that night noise exposure might be more strongly associated with cardiovascular effects than daytime exposure, highlighting the need for future epidemiological studies on this topic.

The review of available evidence leads to the following conclusions.

- Sleep is a biological necessity and disturbed sleep is associated with a number of adverse impacts on health.
- There is sufficient evidence for biological effects of noise during sleep: increase in heart rate, arousals, sleep stage changes and awakening.
- There is sufficient evidence that night noise exposure causes self-reported sleep disturbance, increase in medicine use, increase in body movements and (environmental) insomnia.
- While noise-induced sleep disturbance is viewed as a health problem in itself (environmental insomnia), it also leads to further consequences for health and well-being.
- There is limited evidence that disturbed sleep causes fatigue, accidents and reduced performance.
- There is limited evidence that noise at night causes hormone level changes and clinical conditions such as cardiovascular illness, depression and other mental illness. It should be stressed that a plausible biological model is available with sufficient evidence for the elements of the causal chain.

VULNERABLE GROUPS

Children have a higher awakening threshold than adults and therefore are often seen to be less sensitive to night noise. For other effects, however, children seem to be equally or more reactive than adults. As children also spend more time in bed they are exposed more to night noise levels. For these reasons children are considered a risk group.

Since with age the sleep structure becomes more fragmented, elderly people are more vulnerable to disturbance. This also happens in pregnant women and people with ill health, so they too are a group at risk.

Finally, shift workers are at risk because their sleep structure is under stress due to the adaptations of their circadian rhythm.

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THRESHOLDS FOR OBSERVED EFFECTS

The no observed adverse effect level (NOAEL) is a concept from toxicology, and is defined as the greatest concentration which causes no detectable adverse alteration of morphology, functional capacity, growth, development or lifespan of the target organism. For the topic of night noise (where the adversity of effects is not always clear) this concept is less useful. Instead, the observed effect thresholds are provided: the level above which an effect starts to occur or shows itself to be dependent on the exposure level. It can also be a serious pathological effect, such as myocardial infarctions, or a changed physiological effect, such as increased body movement.

Threshold levels of noise exposure are important milestones in the process of evaluating the health consequences of environmental exposure. The threshold levels also delimit the study area, which may lead to a better insight into overall consequences. In Tables 1 and 2, all effects are summarized for which *sufficient and limited evidence* exists. For these effects, the threshold levels are usually well known, and for some the dose-effect relations over a range of exposures could also be established.

Effect		Indicator	Threshold, dB
Biological effects	Change in cardiovascular activity	*	*
	EEG awakening	$L_{Amax,inside}$	35
	Motility, onset of motility	$L_{Amax,inside}$	32
	Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep	$L_{Amax,inside}$	35
	Waking up in the night and/or too early in the morning	$L_{Amax,inside}$	42
Sleep quality	Prolongation of the sleep inception period, difficulty getting to sleep	*	*
	Sleep fragmentation, reduced sleeping time	*	*
	Increased average motility when sleeping	$L_{night,outside}$	42
Well-being	Self-reported sleep disturbance	$L_{night,outside}$	42
	Use of somnifacient drugs and sedatives	$L_{night,outside}$	40
Medical conditions	Environmental insomnia**	$L_{night,outside}$	42

Table 1 Summary of effects and threshold levels for effects where *sufficient* evidence is available

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

** *Note that "environmental insomnia" is the result of diagnosis by a medical professional whilst "self-reported sleep disturbance" is essentially the same, but reported in the context of a social survey. Number of questions and exact wording may differ.*

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Table 2
Summary of effects and threshold levels for effects where *limited* evidence is available**

Effect		Indicator	Estimated threshold, dB
Biological effects	Changes in (stress) hormone levels	*	*
Well-being	Drowsiness/tiredness during the day and evening	*	*
	Increased daytime irritability	*	*
	Impaired social contacts	*	*
	Complaints	$L_{night,outside}$	35
	Impaired cognitive performance	*	*
Medical conditions	Insomnia	*	*
	Hypertension	$L_{night,outside}$	50
	Obesity	*	*
	Depression (in women)	*	*
	Myocardial infarction	$L_{night,outside}$	50
	Reduction in life expectancy (premature mortality)	*	*
	Psychic disorders	$L_{night,outside}$	60
	(Occupational) accidents	*	*

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

** *Note that as the evidence for the effects in this table is limited, the threshold levels also have a limited weight. In general they are based on expert judgement of the evidence.*

RELATIONS WITH $L_{NIGHT, OUTSIDE}$

Over the next few years, the END will require that night 'noise' exposures are reported in $L_{night,outside}$. It is, therefore, interesting to look into the relation between $L_{night,outside}$ and adverse health effects. The relation between the effects and $L_{night,outside}$ is, however, not straightforward. Short-term effects are mainly related to maximum levels per event inside the bedroom: $L_{Amax,inside}$. In order to express the (expected) effects in relation to the single European Union indicator, some calculation needs to be done. The calculation for the total number of effects from reaction data on events (arousals, body movements and awakenings) needs a number of assumptions. The first that needs to be made is independence: although there is evidence that the order of events of different loudness strongly influences the reactions, the calculation is nearly impossible to carry out if this is taken into consideration. Secondly, the reactions per event are known in relation to levels at the ear of the sleeper, so an assumption for an average insulation value must be made. In the report a value of 21 dB has been selected. This value is, however, subject to national and cultural differences. One thing that stands out is the desire of a large part of the population to sleep with windows (slightly) open. The relatively low value of 21 dB takes this into account already. If noise levels increase, people do indeed close their windows, but obviously reluctantly, as complaints about bad air then increase and sleep disturbance remains high. This was already pointed out in the WHO *Guidelines for community noise* (1999).

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From source to source the number of separate events varies considerably. Road traffic noise is characterized by relatively low levels per event and high numbers, while air and rail traffic are characterized by high levels per event and low numbers. For two typical situations estimates have been made and presented in graphical form. The first is an average urban road (600 motor vehicles per night, which corresponds roughly to a 24-hour use of 8000 motor vehicles, or 3 million per year, the lower boundary the END sets) and the second case is for an average situation of air traffic exposure (8 flights per night, nearly 3000 per year).

Fig. 3 shows how effects increase with an increase of $L_{night,outside}$ values for the typical road traffic situation (urban road). A large number of events lead to high levels of awakening once the threshold of $L_{Amax,inside}$ is exceeded. To illustrate this in practical terms: values over 60 dB $L_{night,outside}$ occur at less then 5 metres from the centre of the road.

In Fig. 4 the same graph is presented for the typical airport situation. Due to a lower number of events there are fewer awakenings than in the road traffic case (Fig. 3), but the same or more health effects. In these examples the worst case figures can be factors higher: the maximum number of awakenings for an $L_{night,outside}$ of 60–65 dB is around 300 per year.

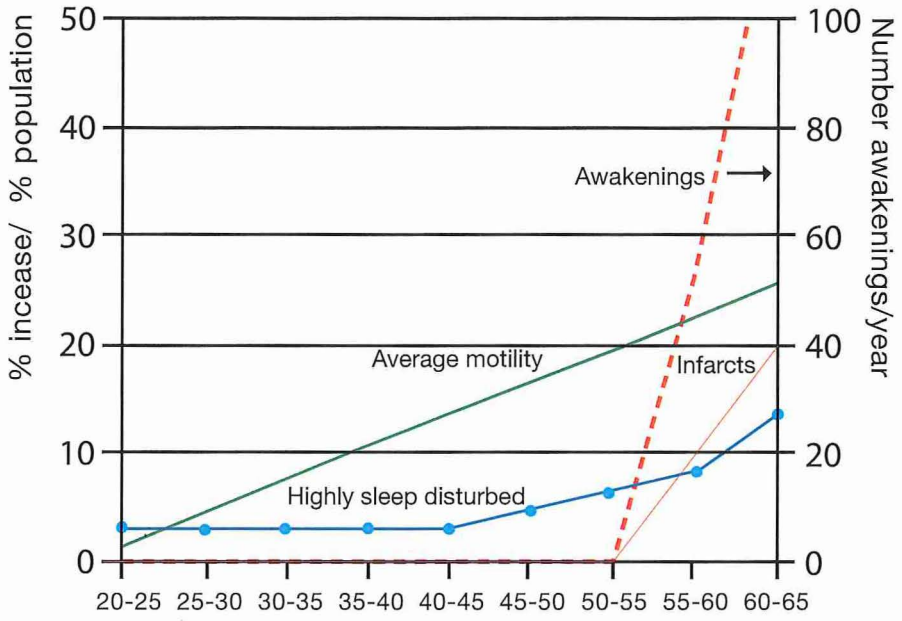


Fig. 3 Effects of road traffic noise at night*

**Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as a percent of the population; awakenings are expressed in number of additional awakenings per year.*

A recent study suggests that high background levels of noise (from motorways) with a low number of separate events can cause high levels of average motility.

Therefore, by using the $L_{night,outside}$ as a single indicator, a relation between effects and indicator can be established. For some effects, however, the relation can be

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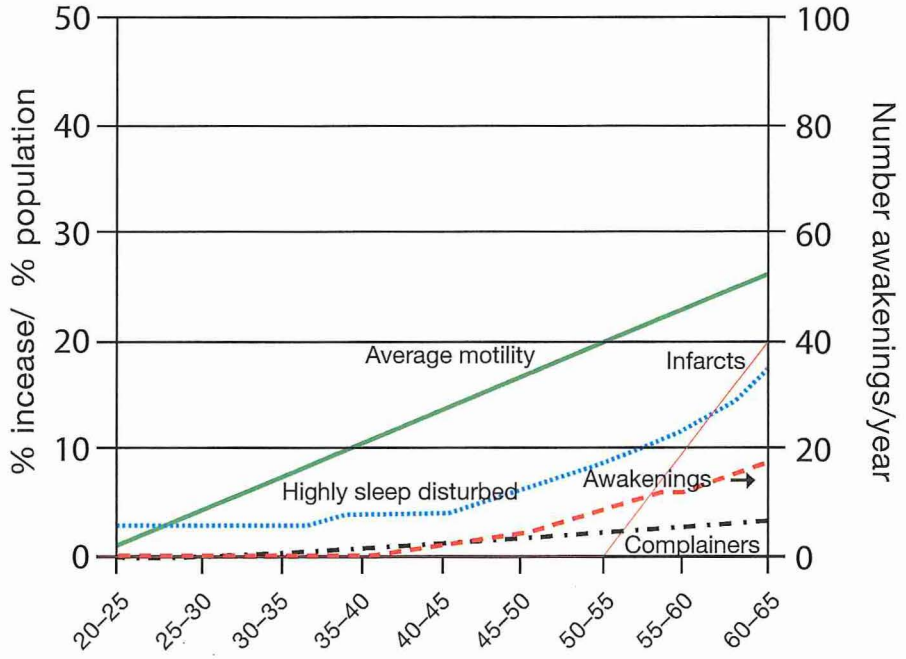


Fig. 4 Effects of aircraft noise at night*

**Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as a percent of the population; complainers are expressed as a % of the neighbourhood population; awakenings are expressed in number of additional awakenings per year.*

source dependent. Although L_{night} gives a good relation for most effects, there is a difference between sources for some. Train noise gives fewer awakenings, for instance. Once source is accounted for, the relations are reasonably accurate.

RECOMMENDATIONS FOR HEALTH PROTECTION

Based on the systematic review of evidence produced by epidemiological and experimental studies, the relationship between night noise exposure and health effects can be summarized as below. (Table 3)

Below the level of 30 dB $L_{night,outside}$, no effects on sleep are observed except for a slight increase in the frequency of body movements during sleep due to night noise. There is no sufficient evidence that the biological effects observed at the level below 40 dB $L_{night,outside}$ are harmful to health. However, adverse health effects are observed at the level above 40 dB $L_{night,outside}$, such as self-reported sleep disturbance, environmental insomnia, and increased use of somnifacient drugs and sedatives.

Therefore, 40 dB $L_{night,outside}$ is equivalent to the lowest observed adverse effect level (LOAEL) for night noise. Above 55 dB the cardiovascular effects become the major public health concern, which are likely to be less dependent on the nature of the noise. Closer examination of the precise impact will be necessary in the range between 30 dB and 55 dB as much will depend on the detailed circumstances of each case.

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Average night noise level over a year $L_{night,outside}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{night,outside}$ of 30 dB is equivalent to the no observed effect level (NOEL) for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. $L_{night,outside}$ of 40 dB is equivalent to the lowest observed adverse effect level (LOAEL) for night noise.
40 to 55 dB	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

Table 3
Effects of different levels of night noise on the population's health

A number of instantaneous effects are connected to threshold levels expressed in L_{Amax}. The health relevance of these effects cannot be easily established. It can be safely assumed, however, that an increase in the number of such events over the baseline may constitute a subclinical adverse health effect by itself leading to significant clinical health outcomes.

Based on the exposure-effects relationship summarized in Table 3, the night noise guideline values are recommended for the protection of public health from night noise as below.

Night noise guideline (NNG)	$L_{night,outside}$ = 40 dB
Interim target (IT)	$L_{night,outside}$ = 55 dB

Table 4
Recommended night noise guidelines for Europe

[1] $L_{night,outside}$ is the night-time noise indicator (L_{night}) of Directive 2002/49/EC of 25 June 2002: the A-weighted long-term average sound level as defined in ISO 1996-2: 1987, determined over all the night periods of a year; in which: the night is eight hours (usually 23.00 – 07.00 local time), a year is a relevant year as regards the emission of sound and an average year as regards the meteorological circumstances, the incident sound is considered, the assessment point is the same as for L_{den}. See *Official Journal of the European Communities*, 18.7.2002, for more details.

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For the primary prevention of subclinical adverse health effects related to night noise in the population, it is recommended that the population should not be exposed to night noise levels greater than 40 dB of $L_{night,outside}$ during the part of the night when most people are in bed. The LOAEL of night noise, 40 dB $L_{night,outside}$, can be considered a health-based limit value of the night noise guidelines (NNG) necessary to protect the public, including most of the vulnerable groups such as children, the chronically ill and the elderly, from the adverse health effects of night noise.

An interim target (IT) of 55 dB $L_{night,outside}$ is recommended in the situations where the achievement of NNG is not feasible in the short run for various reasons. It should be emphasized that IT is not a health-based limit value by itself. Vulnerable groups cannot be protected at this level. Therefore, IT should be considered only as a feasibility-based intermediate target which can be temporarily considered by policy-makers for exceptional local situations.

RELATION WITH THE GUIDELINES FOR COMMUNITY NOISE (1999)

Impact of night-time exposure to noise and sleep disturbance is indeed covered in the 1999 guidelines, as below (WHO, 1999):

> "If negative effects on sleep are to be avoided the equivalent sound pressure level should not exceed 30 dBA indoors for continuous noise. If the noise is not continuous, sleep disturbance correlates best with L_{Amax} and effects have been observed at 45 dB or less. This is particularly true if the background level is low. Noise events exceeding 45 dBA should therefore be limited if possible. For sensitive people an even lower limit would be preferred. It should be noted that it should be possible to sleep with a bedroom window slightly open (a reduction from outside to inside of 15 dB). To prevent sleep disturbances, one should thus consider the equivalent sound pressure level and the number and level of sound events. Mitigation targeted to the first part of the night is believed to be effective for the ability to fall asleep."

The 1999 guidelines are based on studies carried out up to 1995 (and a few meta-analyses some years later). Important new studies (Passchier-Vermeer et al., 2002; Basner et al., 2004) have become available since then, together with new insights into normal and disturbed sleep. New information has made more precise assessment of exposure-effect relationship. The thresholds are now known to be lower than L_{Amax} of 45 dB for a number of effects. The last three sentences still stand: there are good reasons for people to sleep with their windows open, and to prevent sleep disturbances one should consider the equivalent sound pressure level and the number of sound events. The present guidelines allow responsible authorities and stakeholders to do this. Viewed in this way, the *night noise guidelines for Europe* are complementary to the 1999 guidelines. This means that the recommendations on government policy framework on noise management elaborated in the 1999 guidelines should be considered valid and relevant for the Member States to achieve the guideline values of this document.

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CHAPTER 1

INTRODUCTION: METHODS AND CRITERIA

With regard to sleep and waking, we must consider what they are: whether they are peculiar to soul or to body, or common to both; and if common, to what part of soul or body they appertain: further, from what cause it arises that they are attributes of animals, and whether all animals share in them both, or some partake of the one only, others of the other only, or some partake of neither and some of both.

(Aristotle, On sleep and sleeplessness, 350 BC)

1.1 INTRODUCTION

1.1.1 EXISTING POLICY DOCUMENTS FOR NIGHT-TIME NOISE

The aim of this document is to present guidance for exposure to noise during sleep. What is already available?

There are three related documents at the international level:

- *Guidelines for community noise* (WHO, 1999)
- *Directive 2002/49/EC relating to the assessment and management of environmental noise* (European Commission, 2002b)
- *Position Paper on dose-effect relationships for night-time noise* (European Commission, 2004).

In Chapter 5 the relation with the *Guidelines for community noise* (1999) will be explained.

The European Union (EU) Directive relating to the assessment and management of environmental noise (or, as it is commonly known, the Environmental Noise Directive – END), establishes that Member States should create noise maps (2007) and action plans (2008) for parts of their territory. The noise maps should present noise levels expressed in the harmonized indicators L_{den} and L_{night}. Although in the first round only between 20% and 30% of the population will be covered, it is expected that through the use of harmonized methods and indicators a deeper insight will be gained into the exposure of the population to noise. The END does not, however, set any limit values: on the basis of the subsidiarity principle this is left to the Member States. The Directive does, however, require Member States to report on their limit values and express them in the standard indicators. On the CIRCA web site (Communication and Information Resource Centre Administrator, European Commission, 2006) an overview of the data reported to the Commission can be found. Out of the 25 Member States, 10 reported on the L_{night} limits. In Table 1.1 some of these data are summarized.

Due to differences in legal systems it is hard to predict what the actual effect of a certain limit value will be. It could be a relatively high value but rigidly enforced, or a very low value with no legal binding whatsoever.

The *Position Paper* on dose-effect relationships for night-time noise is foreseen in the END (Annex III) and aims to give the competent authorities a tool to evaluate the

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impact on the population. However, it neither provides limit values nor guidelines. The same information that was used in the *Position Paper* also plays a role in these guidelines.

Table 1.1
Reported L_{night} limit values for road traffic noise in new residential areas

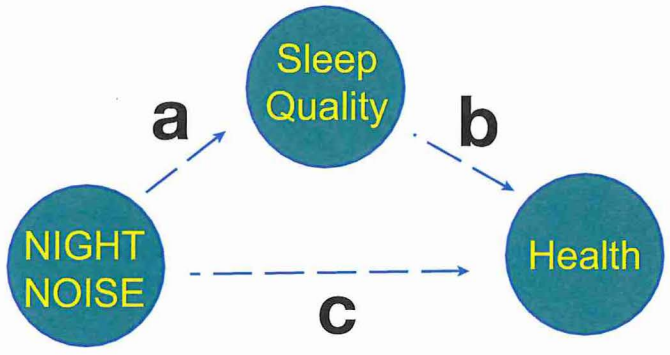
EU Member State	$L_{night,outside}$
France	62
Germany	49
Spain	45
Netherlands	40
Austria	50
Sweden	51 (converted from L_{Aeq} limit 30 dB(A) inside bedroom)
Finland	46
Hungary	55
Latvia	40
Estonia	45
Switzerland	50

Source: European Commission, 2006.

1.1.2 GENERAL MODEL

There is no doubt that a relation exists between sleep and health and well-being, as most of us know from personal experience. That does not mean, however, that this relation is simple. People who do not sleep well may not feel well the day after, but the reverse is also true: unfit people may have a disturbed sleep. Untangling the relations between health and disturbed sleep (night-time noise is only one of many causes) proved difficult, and Fig. 2.1 at the end of Chapter 2 shows why.

Fig. 1.1.
General structure of the report on the effects of night noise



The general structure of the report is given in Fig. 1.1: evidence for the effects of night-time noise on health (c) is supported by evidence on the indirect route via (a) and (b). In Chapter 2 the relations between sleep and health are examined (relation (b) in Fig. 1.1), and this involves clinical evidence from sleep laboratories, but also evidence from animal experiments. In Chapter 3 it is shown how noise disturbs sleep from the basic, autonomous level up to conscious awakenings: relation (a). Chapter 4 presents the evidence between night-time noise and health and well-being: relation (c) in Fig. 1.1. The last chapter, Chapter 5, then provides guidance on reducing health impacts caused by night-time noise exposure.

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1.1.3 PROCESS OF DEVELOPING GUIDELINES

The WHO Regional Office for Europe started the night noise guidelines (NNGL) project with a grant from the European Commission's Directorate-General for Health and Consumer Affairs. In 2003, the WHO Regional Office for Europe set up a working group of experts to provide scientific advice for the development of guidelines for future legislation and policy action in the area of control and surveillance of night noise exposure. The review of available scientific evidence on the health effects of night noise was carried out by the working group to derive health-based guideline values. The contributions from the experts were reviewed by the team and integrated into draft reports following discussion at four technical meetings of the working group.

The first meeting of the working group was held in Bonn, June 2004. It was agreed that the experts would produce background papers on a number of topics identified and assigned at the meeting.

The second meeting in Geneva, December 2004, concentrated on such technical issues as exposure assessment, metrics, health effects and guideline set-up. The topic-specific experts presented the first drafts for the identified topics for detailed discussions at the meeting. The discussions concentrated on central issues such as exposure assessment and guideline derivation.

The third meeting in Lisbon, April 2005, reviewed the revised background papers, and discussed in detail the overall structure of the guidelines document, and the process of consensus building among the working group and stakeholders.

At the workshop of acoustics experts in The Hague, September 2005, a consensus was made on the use of L_{night} as the single indicator for guideline values as it effectively combines the information on the number of events and the maximum sound levels per event over a year.

In 2006, all the draft reports collected at previous meetings were compiled by Mr. Martin van den Berg into a coherent document on guidelines for exposure to noise at night. The latter was revised according to the comments collected though a peer-review by the working group experts.

At the concluding meeting in Bonn, December 2006, the working group and stakeholders from industry, government and nongovernmental organizations reviewed the contents of the draft document chapter by chapter, discussed several fundamental issues and reached general consensus on the guideline values. The final implementation report of NNGL project was submitted to the EU in early 2007.

The following countries and institutes contributed to the development of *Night noise guidelines for Europe* as project partners.

AUSTRIA:	Institute of Hygiene and Social Medicine, University of Innsbruck
CZECH REPUBLIC:	Charles University in Prague
DENMARK:	National Institute of Public Health
FRANCE:	INRETS/LTE–Laboratoire Transports et Environnement CNRS–Centre National de Recherche Scientifique
GERMANY:	Umweltbundesamt–Federal Environmental Agency Landesgesundheitsamt Baden-Württemberg

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ITALY:	ARPAT–Environmental Protection Agency, Tuscany Region University of Rome "La Sapienza"– Center for Pediatric Sleep Disorders
NETHERLANDS:	TNO–Netherlands Organisation for Applied Scientific Research RIVM–National Institute of Public Health and the Environment
POLAND:	University of Warsaw, Clinic of Psychiatry of the Medical Academy
PORTUGAL:	IST–Instituto Superior Técnico
SLOVENIA:	Institute of Public Health of the Republic of Slovenia
SWEDEN:	University of Gävle, Centre for Built Environment
UNITED KINGDOM:	Queen Mary and Westfield College, University of London

In addition, WHO received advice and support from a number of national experts who participated in the working group. The affiliations of these additional expert advisers include:

CANADA:	Health Canada
GERMANY:	Forschungs- und Beratungsbüro Maschke
SWITZERLAND:	Bundesamt für Umwelt, Wald und Landschaft Universität St. Gallen, Institut für Wirtschaft und Ökologie
NETHERLANDS:	Ministry of Housing, Spatial Planning and Environment
UNITED KINGDOM:	Casella Stanger Environmental Consultants

Since the project report was published on the EU web site, various comments were received from experts who have not participated in the working group. The most critical points were regarding the achievability of guideline values in practice. Responding to these feedbacks, the WHO Regional Office for Europe, prepared a revision of guidelines and recommendations, and consulted with international experts and stakeholders including the EU. As of late 2008, it was agreed that the guideline should be based on the lowest observed adverse effects level (LOAEL) rather than the no observed effects level (NOEL). Interim target was also introduced as a feasibility-based level.

1.2 STRENGTH OF EVIDENCE

1.2.1 BASIC CONCEPTS

This document uses well-established practices from other disciplines and policy fields. Of main interest here are evidence-based medicine, the use of epidemiological evidence for environmental risk assessment and experiences with – principally– air quality guidelines.

The concept of "evidence" is further formalized, as variations in wording and scope are currently in use.

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1.2.2 RISK ASSESSMENT AND RISK CONTROL

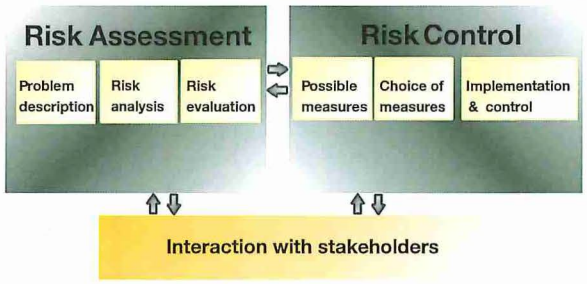


Fig. 1.2
Elements of risk control

Source:
Health Council of the Netherlands, 2004.

Fig. 1.2 outlines a general approach for risk assessment and control. This approach consists of the following steps:

1. problem description: assessing the impact on the population
2. risk analysis: evaluation of impact
3. risk evaluation: assessing impact considered undesirable
4. assessment of options to avoid or reduce impact
5. cost–benefit analysis of the options or of the mix of options
6. assessment of the preferred option
7. implementation and control.

It is important to observe that guideline values can be an input to, as well as an output of this process. At lower levels of decision (a particular infrastructure project, for instance) a preset guideline value reduces – intentionally – the degrees of freedom in the process. At the highest national or international level a guideline value is the outcome. As the scope of this document is to present the health consequences of night-time noise exposure (and not so much the economic outcomes of the choice of a certain value) it concentrates on the first three elements in the risk assessment block.

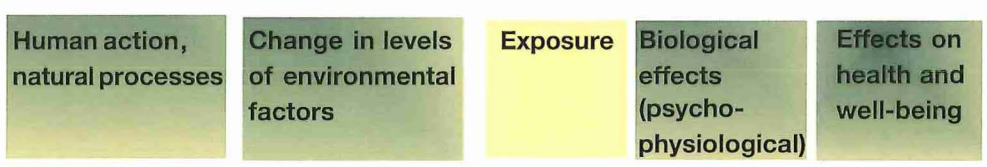
The following questions need to be addressed.

- What is the strength of the available evidence – what are the uncertainties?
- What is the health significance for the effects found?
- How serious is the impact on health?
- Does every instance of exposure lead to an effect and how are they related?
- How can the number of affected people be established?

1.2.3 CAUSE–EFFECT CHAIN

Underlying this approach is the notion of a cause–effect chain between environmental factors and health, symbolically simplified in Fig. 1.3.

Fig. 1.3
Cause-effect chain



Source: Health Council of the Netherlands, 2004.

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There are important questions that need to be asked.

- Is there a causal relation between one link in the chain and the next?
- What are the intervening factors in that relation?
- How strong is the evidence for the relations?

The last question is the hardest to answer, as "strength of evidence" is not easy to express in simple numbers or labels. There are two forms of uncertainty: uncertainty because of variability of outcomes and uncertainty due to a lack of knowledge.

For the purpose of this document the following classification will be used, largely based on the IARC (International Agency for Research on Cancer) criteria accessible at http://monographs.iarc.fr/ENG/Preamble/currentb6evalrationale0706.php (see Table 1.2).

Table 1.2 Classification of evidence

Grade of evidence	Criteria
Sufficient evidence	A causal relation has been established between exposure to night-time noise and an effect. In studies where coincidence, bias and distortion could reasonably be excluded, the relation could be observed and it is plausible that the effect is (also) caused by the exposure.
Limited evidence	A relation was observed between exposure to night-time noise and an effect in studies where coincidence, bias and distortion could not reasonably be excluded. The relation is, however, plausible. A direct relation between cause and effect has not been observed, but there is indirect evidence of good quality and the relation is plausible. Indirect evidence is assumed if exposure leads to an intermediate effect and other studies prove that the intermediate effect leads to the effect.
Insufficient evidence	Available studies are of low quality and lack significance to allow conclusions about causality of the relation between exposure and effect. Plausibility of the relation is limited or absent.

1.2.4 PROCEDURE FOR DERIVING GUIDELINES

The following procedure was followed in order to derive an ordering of guideline values:

1. collection of relevant data
2. evaluation of data in terms of strength of evidence
3. evaluation of data in terms of biological effects, health and well-being
4. ranking of guideline values.

This procedure is essentially the same as in other guideline documents, although steps are more explicitly formalized. A major difference is that sound is a natural environmental quality, which makes defining a no-effect level a futile exercise. Therefore the choice was made for a series of levels with increasing severity of effects.

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1.3 CONSIDERATIONS WITH REGARD TO NIGHT-TIME NOISE INDICATORS

Briefly, the fundamental choices of night-time noise indicators with respect to length of night, use of single event descriptors and long-term average are commented on to assist the reader in understanding the relations presented in later chapters.

1.3.1 LENGTH OF NIGHT

Time use studies (Centre for Time Use Research, 2006) show that the average time adult people are in bed is around 7.5 hours, so the real average sleeping time is somewhat shorter. Due to personal factors such as age and genetic factors there is considerable variation in sleeping time and in beginning and end times. For these reasons, a fixed interval of 8 hours is a minimal choice for night-time protection. From Fig. 1.4 it can be noted that around 50% of the population is protected with an interval of 8 hours and it would take a period of 10 hours to protect 80%. On Sundays, sleeping time is consistently one hour longer, probably due to people recovering from sleep debt incurred during the week. Data for other countries are readily available but this is the only study covering a long period in a consistent way. Fig. 1.5 (from a time use study in Portugal) shows that the stable pattern found in the Netherlands (Fig. 1.4) is not only typical for northern Europe, but also for the southern part. The pattern, however, seems to have shifted slightly. These figures stress that sleep times might be biologically fixed in humans, and culture has almost no influence.

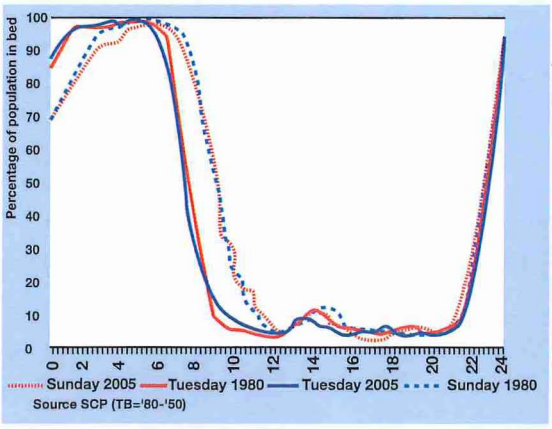


Fig. 1.4
Sleep pattern of Dutch population on weekdays and Sundays, 1980-2005

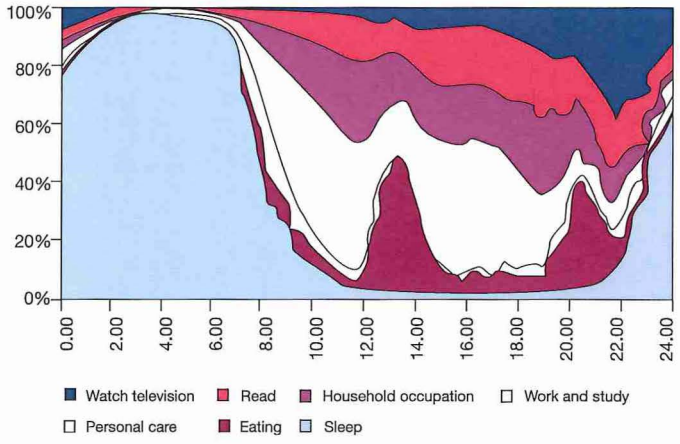


Fig. 1.5
Percentage of time that the Portuguese population spend asleep or in different activities

Source: http://www.ine.pt/prodserv/destaque/arquivo.asp, based on a study by the Instituto Nacional de Estatistica Portugal, 1999

1.3.2 EVENT OR LONG-TERM DESCRIPTOR

Much attention has been paid to the use of single event descriptors such as L_{Amax} (maximum outdoor sound pressure level) and SEL (sound exposure level). As the *Position Paper* on EU noise indicators (European Commission, 2000) points out, this is an important laboratory tool to describe instantaneous reactions to noise. But when it comes to long-term protection, the number of events is equally important. The possibility of predicting after-effects like sleepiness, reaction time, sleeping pill

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use and health complaints, in particular, requires a combination of a number of events and their level instead of just the average L_{Amax} or average SEL. For events with a similar time pattern there is a relatively simple relation between L_{Amax} and SEL, and therefore between L_{Amax} and L_{night} (night-time noise indicator as defined

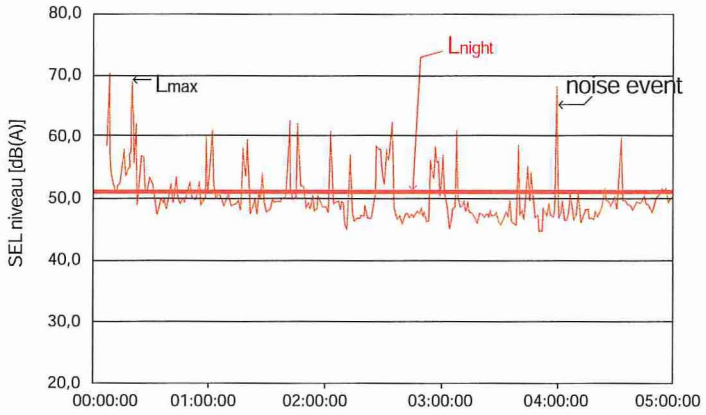


Fig. 1.6
Relation between L_{night}, L_{Amax} and SEL

Source: European Commission, 2000

by the END – see paragraph 1.3.4 below). Appendix 2 describes this in detail. For now let it suffice to say that a choice for an L_{night} level ties the L_{Amax} related effects to a maximum and therefore allows for a protective/conservative approach.

Fig. 1.6 is based on a sound recording in a bedroom for one night. The top of the peaks are the L_{Amax} levels, the total energy is the L_{night} (thick horizontal line). The sound energy in one event is the SEL (not represented). In reality the L_{night} is the average over all nights in one year. This reasoning applies also to the issue of long-term average. A value for an arbitrary single night will, except in extreme cases, bear no relationship to an individual's long-term health status, whereas a sustained sufficiently high level over a long period may.

1.3.3 NUMBER OF EVENTS

There is no generally accepted way to count the number of (relevant) noise events. Proposals range from the number of measured L_{Amax}, the number of units (vehicles, aeroplanes, trains) passing by, to the number exceeding a certain L_{Amax} level (commonly indicated by NAxx; NA70 is the number of events higher than 70 dB).

1.3.4 CONVERSION BETWEEN INDICATORS

1.3.4.1 Introduction

L_{night} is defined as the 1 year L_{Aeq} (exposure to noise) over 8 hours outside at the most exposed facade. For the purpose of strategic noise mapping and reporting the height is fixed at 4 metres. As L_{night} is a relatively new definition and because the studies rarely cover such a long period, the research data are rarely expressed in L_{night}. The most frequently used noise descriptor in sleep research is the L_{Amax} or SEL near the sleeper. This means that a considerable amount of conversion work needs to be done if relations are to be expressed in L_{night}. There are four issues:

- conversion between SEL and L_{Amax}
- conversion from instantaneous to long-term
- conversion from inside to outside
- conversion from (outside) bedroom level to most exposed façade.

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Further background information on these issues is provided in section 1.3.5. This section details the conversions that are actually carried out.

1.3.4.2 SEL to L_{Amax}

SEL is only used for aircraft noise in this report and, according to Ollerhead et al. (1992) from ground-based measurements, the following relation was found:

$$SEL = 23.9+0.81*L_{Amax} \quad [1].$$

A more general approach can be used to estimate SEL for transportation noise.

If the shape of the time pattern of the sound level can be approximated by a block form, then $SEL \approx L_{Amax} + 10lg\ t$, where t (in seconds) is the duration of the noise event. This rule can be used, inter alia, for a long freight train that passes at a short distance. When t is in the range from 3 to 30 seconds, then SEL is 5–15 dB higher than L_{Amax}. For most passages of aircraft, road vehicles or trains, the shape of the time pattern of the sound level can be better approximated with a triangle. If the sound level increases with rate a (in dB per second), and thereafter is at its maximum for a short duration before it decreases with rate -a, then $SEL \approx L_{Amax} - 10lg(a) + 9.4$. Depending on the distance to the source, for most dwellings near transportation sources the rate of increase is in the order of a few dB per second up to 5 dB per second. When (a) is in the range from 9 dB to 1 dB per second, then SEL is 0–9 dB higher than L_{Amax}.

1.3.4.3 Events to long-term

When the SEL values are known (if necessary after converting from L_{Amax}) they can be converted to L_{night}. In general terms, the relation between L_{night} and SEL is:
$L_{night} = 10*lg \sum_i 10^{SEL_i/10} - 10*lg\ (T)$.

If all (N) events have approximately the same SEL level, this may be reduced to:

$$L_{night} = SEL + 10*lg(N) - 70.2 \quad [2],$$

in which:
N = the number of events occurring in period T;
T = time during which the events occur in seconds. For a (night) year 10lg(T) is 70.2.

The notation adheres to the END where the L_{night} is defined as a year average at the most exposed facade. Any reference to an inside level is noted as such, that is, as $L_{night,inside}$. In order to avoid any doubt the notation $L_{night,outside}$ may be used, for instance in tables where both occur.

1.3.4.4 Inside to outside

As the L_{night} is a year value, the insulation value is also to be expressed as such. This means that if the insulation value is 30 dB with windows closed and 15 dB with windows open, the resulting value is 18 dB if the window is open 50% of the time. If these windows are closed only 10% of the time, the result is little more than 15 dB. The issue is complicated by the fact that closing behaviour is, to a certain extent, dependent on noise level. When results about effects are expressed with indoor (that is, inside bedrooms) exposure levels, they need to be converted to L_{night}, in accordance with the END definition. The most important assumption is the correction for inside levels to outside levels. An average level difference of 21 dB has been chosen, as this takes into account that even in well-insulated houses windows may be open a large part of the year. In general:

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$$L_{night} = L_{night,inside} + Y \text{ dB} \quad [3].$$

Y is the year average insulation value of the (bedroom) facade. In this report a default value of 21 dB is used (see also section 1.3.5). It should be stressed that this conversion is thought to be highly dependent on local building habits, climate and window opening behaviour.

1.3.4.5 Most exposed facade

If an inside level is converted to an outside level with [3], it is assumed that this is equivalent to an L_{night} value on the most exposed facade. No information is available on bedroom position and use, so no explicit conversion factor can be given in this report.

This means that the effect estimated on the basis of L_{night} corresponds to an upper limit, because part of the bedrooms will be on a less exposed facade. If an estimate of the exposed population is based on a relation derived with [3], the actual prevalence will be less. From a practical point of view the most exposed facade safeguards protection in cases where there is a possibility that rooms can be swapped.

It should be pointed out that the above does not apply if a relation is based on L_{night} values which are directly measured or computed. These relations will show a large variation because of a misclassification effect, but they give a "correct" estimate of the prevalence of effects in the population. In other words, in some cases a low effect may be attributed to a high L_{night} because the bedroom is on the quiet side.

1.3.5 INSIDE/OUTSIDE DIFFERENCES

Night-time environmental noise affects residents mainly inside their homes. In order to protect residents inside their homes from noise from outside sources, attention should be focused on windows since they are generally the weakest points in the sound propagation path. Roofs must also be considered with regard to aircraft noise.

There are many types of window in the EU, varying from single thin panes within frames without additional insulation, to four-pane windows within insulated frames. The simplest types of facade have a sound reduction (from outside to inside) of usually less than 24 dB, and the most elaborate facades (built to cope with cold climates, for example), have sound reductions of more than 45 dB. In central Europe, most windows are double-glazed, mounted in a rigid and well-insulated frame. Their range of sound reduction is between 30 dB and 35 dB when closed.

When night-time environmental noise reaches high levels, residents tend to close their bedroom windows (cf. Langdon and Buller, 1977; Scharnberg et al., 1982; Schreckenberg et al., 1999; Diaz et al., 2001). The studies by Scharnberg et al. and Schreckenberg et al. found that more than 50% of bedroom windows are closed when outside road traffic noise levels exceed 55 dB (L_{Aeq}). These findings have been replicated in Sweden, according to recent results from the Swedish soundscape research programme on road traffic noise (Fig. 1.7). Nevertheless, while residents with closed windows reported a reduction of sleep disturbances due to noise, they also reported an increase in sleep disturbances due to poor ventilation. Schreckenberg et al. (1999) report a much steeper increase in the incidence of closed windows when road traffic noise reaches high levels than is the case with increased levels of railway noise. Even when night-time noise levels reach 55 B, only 35% of the residents exposed to railway noise reported that they closed their windows at night.

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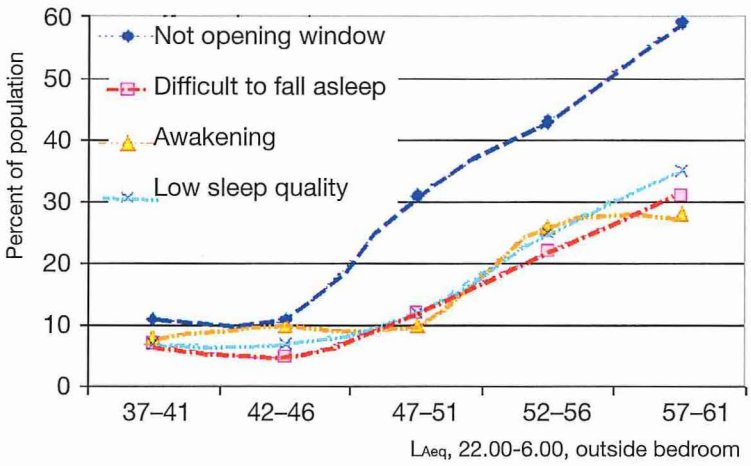


Fig. 1.7
Results from Swedish soundscape research programme on road traffic noise

Source: Öhrström, in European Commission, 2002a.

When windows are slightly open, outside sound levels are usually reduced by 10–15 dB. It should be kept in mind that most European residents want to keep their bedroom windows slightly open at night in order to provide proper ventilation (Scharnberg et al., 1982; Lambert and Plouhinec, 1985; Lambert and Vallet, 1994), and the WHO paper on community noise (WHO, 1999) also recommends that people should be able to sleep with their bedroom windows open.

Passchier-Vermeer et al. (2002) carried out detailed noise measurements inside and outside the bedroom and at the same time measured window position with sensors. The results (Table 1.3) showed that windows are fully closed only in 25% of the nights.

Window position	% nights
Closed	25
Slightly open	43
Hand width	23
Half open	5
Fully open	4

Table 1.3
Window positions during research period (April–November)

This results in average inside/outside differences of around 21 dB, with there being only a slight difference between single- and double-glazed windows (Table 1.4). The survey did not include dwellings which had been specifically insulated against noise. Nevertheless, there was a large variation in insulation values.

	Single-glazed window	Double-glazed window
Average difference at night	21.3	22.2

Table 1.4
Average inside/outside differences in dB

It should be stressed that this figure only applies to facades that have not been fitted with special appliances to reduce noise impact. To give an extreme example of where this general finding does not apply, rooms may be equipped with air conditioning so that windows can stay closed or could even be sealed. Less drastic provisions are sound-attenuated ventilation openings. Little is known, however, about the inhabitants' experiences (long-term use, appreciation) of these and other solutions. For example, sound-attenuated ventilation openings are sometimes blocked in order to cut out draughts.

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1.3.6 BACKGROUND LEVEL

A simple definition of background level or "ambient noise" level is the noise that is not targeted for measurement or calculation. Background noise can interfere with the target noise in a number of ways. It can:

- mask the signal
- interact physically
- interact psychologically.

As this report is often dealing with low-level target noise, masking is an important issue. The other two interactions are more important in the domain of annoyance. Masking, however, is a complex process. The human auditory system is uncannily good at separating signals from "background". Microphones (and the software behind them) have been slow to catch up, as the unsatisfactory results show when it comes to automatically recognizing aircraft in long-term unmanned measuring stations.

The rule of thumb that a noise can be considered masked if the signal is 10 dB below the background is only valid if the noises have the same frequency composition and if they actually occur at the same time. This is particularly important to stress where L_{Aeq} levels are compared: even a relatively continuous motorway of 50 dB cannot mask aircraft noise of 30 dB, because this may be composed of five aircraft arriving at an L_{Amax} of 57 dB. Neither can birdsong, because the frequency domains do not overlap.

Another factor relevant for this report is that background levels are lower at night-time than they are in the daytime. This is true for most man-made noises, but also for the natural background levels as wind speeds at night slow down.

Most levels mentioned in this report do not take background levels into account – explicitly. Where long-term L_{Aeq} levels are related to effects like hypertension and self-reported sleep disturbance, background levels are ignored, but they could obscure the effect at the lower end of the scale. This then influences the lowest level where an effect starts to occur.

In sleep laboratory studies the background level is kept as low as possible, around 30 dB. The background of the instrumentation is 20 dB.

In semi-field experiments it has been found that background noise levels inside bedrooms are very low, partly because people tend to choose their bedrooms on the quiet side of the building. This may have the side-effect of exposing children to higher levels.

1.3.7 CHOICE OF INDICATORS FOR REGULATORY PURPOSES

From the scientific point of view the correct choice for a noise indicator is its performance in predicting the effect. There are, however, a number of additional criteria which may influence the choice. Firstly, for different health end points different indicators could be suitable. Further considerations are of a more political nature, as mentioned in the *Position Paper* on EU noise indicators (European Commission, 2000). Indicators should also be easy to explain to the public – intuitively understandable, avoiding unnecessary breaks with current practice and enforceable. This is probably why in many countries L_{Amax} is a popular indicator: it has undeniable qualities in these areas.

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This is also the case for L_{Aeq} indicators for short periods of, for example, one or a few hours in the middle of the night. Other fashionable indicators are those looking at numbers above a threshold.

For these indicators the relation between health end points and their values is either not well established, or the correlation between them and current indicators is high, or the correlation between the indicator and an effect is low.

1.4 EXPOSURE IN THE POPULATION

1.4.1 NOISE LEVELS

Surprisingly little information is available on the exposure of houses to night-time noise. It is possible that, in a few years time, the END will lead to the creation of a substantial database on these levels, but up till now only two countries have detailed data available (Table 1.5).

	L_{night} in dB				
Country	40–45	46–50	51–55	56–60	61–65+
Switzerland (Müller-Wenk, 2002)	–	24%	14%	7%	2%
Netherlands (Nijland and Jabben, 2004)	25%	31%	19%	6%	1%

Table 1.5 Percentage of dwellings per noise class of L_{night} in dB

Notwithstanding the obvious differences between these two countries, the data show a remarkable similarity.

A first result of the END (see Table 1.6) comes from a study into night regulations for (large) airports (Wubben and Busink, 2004).

Airport	Number of inhabitants	Number of night operations per year	Night operations as percentage of daytime operations
Amsterdam	21 863	23 462	5.8%
Frankfurt	134 651	46 662	10.1%
London	477 289	26 465	5.7%
Paris	180 184	51 683	10.3%

Table 1.6 Number of inhabitants within 45 L_{night} contour

1.4.2 REPORTED NIGHT-TIME NOISE DISTURBANCE

Complaints about night-time exposure to noise are widespread and not exactly new: Roman writers used to complain about the racket in the streets at night (Juvenal, 160). Surprisingly, little detailed information is available today.

Nevertheless, data collected from a few Member States can help to give an impression of the order of magnitude of effects.

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Fig. 1.8 shows the relative contributions to overall sleep disturbance caused by noise from different sources in the Netherlands. These data were derived from surveys in 1998 and 2003 (van Dongen et al., 2004) in which 4000 and 2000 people, randomly selected, were asked: "To what extent is your sleep disturbed by noise from [source mentioned]..." on a scale from 1 to 10. People recording the three highest points in the scale were considered "highly disturbed", according to an international convention. The totals are calculated from the number of people reporting serious sleep disturbance from one or more sources.

Unfortunately, comparable research data from other countries or regions is not available, and there is reason to believe that there may be considerable differences in the figures. Since this study is based on a survey conducted in the Netherlands, it is not representative for other Member States in the EU. General (not specific for night-time) annoyance data from Germany and the United Kingdom give an indication that similar numbers of people are affected.

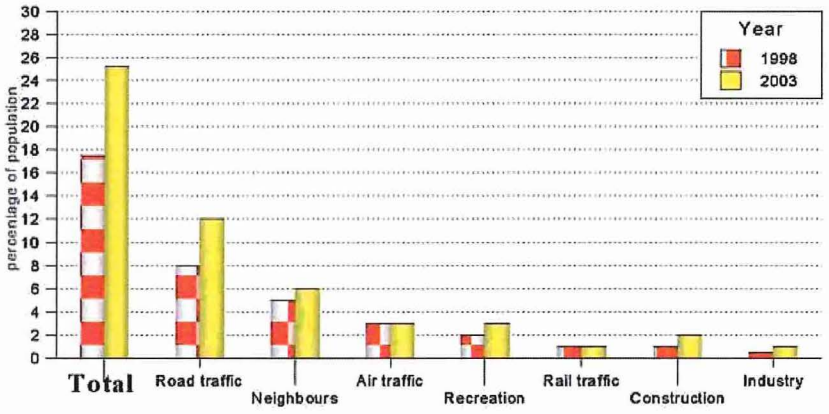


Fig. 1.8 Percentage of population highly disturbed by noise during sleep in the Netherlands

However, the fact that other noise nuisances may contribute significantly to overall sleep disturbance should not be overlooked. Further research on this topic is needed in order to gain an insight into the contribution of various noise sources to sleep disturbance.

1.5 CONCLUSIONS

The methods and criteria for deriving guidelines rest on well-established procedures from epidemiology. To relate the effects to the dose, standard metrics will be used wherever available. If possible, the values found in literature will be converted to avoid confusion. Most of the conversions are relatively straightforward and depend on physical laws; others, in particular the conversion between outside and inside levels, depend on local factors and should be used only if no other information is available.

Information about night-time noise exposure is relatively scarce, despite 10 EU Member States having limit values for night-time noise. The END could substantially increase this information (large-scale noise mapping is foreseen in 2007), increasing the demand for guidance.

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CHAPTER 2

THE RELATION BETWEEN SLEEP AND HEALTH

A night of quiet and repose in the profound silence of Dingley Dell, and an hour's breathing of its fresh and fragrant air on the ensuing morning, completely recovered Mr Pickwick from the effects of his late fatigue of body and anxiety of mind.

(Charles Dickens, The Pickwick Papers, 1836)

2.1 SLEEP, NORMAL SLEEP, DEFINITIONS OF SLEEP DISTURBANCE, CHARACTERISTICS MECHANISMS, THE INSOMNIA MODEL

2.1.1 NORMAL SLEEP (OBJECTIVE MEASUREMENTS)

Sleep is part of living and, along with being awake, forms an inherent biological rhythm (Cooper, 1994). Normal sleep can be defined in an objective or subjective manner. The objective criteria are defined using a polysomnographic recording (PSG) of sleep, the method that measures different physiological functions during sleep. Minimal polygraphic requirements to measure sleep adequately include two channels of electroencephalography (EEG), one channel for the electrooculogram (EOG), and one channel for the submental electromyography (EMG). In routine PSG, additional channels are used to assess respiration, leg movements, oxygenation and cardiac rhythm (Ebersole and Pedley, 2003).

Scoring of sleep stages is usually done on an epoch-by-epoch basis, with a 30-second length used as a standard. Epochs are scored according to the guidelines of Rechtschaffen and Kales (1968). Each epoch is scored as the stage that occupies more than 50% of that epoch. Sleep can be divided into the following stages.

- **Arousal** is not a uniform concept and has been defined differently by different researchers. Commonly, the occurrence of alpha rhythms is required for EEG arousal. Depending on the additional requirements and on the length of time that the slower cortical rhythms are interrupted, arousals have been called, for instance, micro-arousal, minor arousal, EEG awakening or transient activation phases. The American Sleep Disorders Association (1992, 1997) devised a scoring system, taking sequences of 3–15 seconds into account for transient arousals which are not transferred to macroscopic behavioural awakening. Eleven further criteria must be met (see also Chapter 3, section 3.1.2).

- **Vegetative arousals** are activations of the sympathic nervous system.

- **Stage W** corresponds to the waking stage and is characterized by alpha activity or low-voltage, mixed-frequency EEG activity. Rapid eye movements (REMs), eye blinks, and tonic EMG activity are usually present.

- **Stage 1** is scored when more than 50% of an epoch is low-voltage, 2–7 Hertz (Hz) activity. Vertex waves may occur in late stage 1. Slow rolling eye movements lasting several seconds are routinely seen early in stage 1, but K complexes and sleep spindles are absent by definition. Tonic EMG activity is usually less than that of relaxed wakefulness.

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- **Stage 2** requires the presence of sleep spindles or K complexes, and less than 20% of the epoch contains delta activity. Bursts of sleep spindles must last at least 0.5 seconds before they can be scored. K complexes are defined as biphasic vertex sharp waves with a total duration of greater than 0.5 seconds.

- **Stage 3** is scored when 20–50% of an epoch consists of delta activity that is 2 Hz or slower and is greater than 75 μV in amplitude. Sleep spindles may or may not be present.

- **Stage 4** is scored when more than 50% of an epoch consists of delta activity that is 2 Hz or slower and is more than 75 μV in amplitude. Reliable differentiation of stage 3 and stage 4 sleep is difficult by visual inspection, and most laboratories combine stages 3 and 4 into a single determination of slow-wave sleep (SWS).

- **Stage REM** is characterized by relatively low-voltage, mixed-frequency EEG activity with episodic REMs and absent or markedly reduced axial EMG activity. Phasic EMG activity may occur, but tonic activity must be at a level that is as low as, or lower than, that during any other time in the study. Sleep spindles and K complexes are absent. Series of 2- to 5- Hz vertex-negative "saw-tooth waves" occur, particularly just before phasic REM activity. The requirements to score sleep as REM sleep are: REMs, low or absent axial EMG, and typical mixed-frequency EEG recording that does not preclude the scoring of REM.

Movement time is scored when more than 50% of an epoch is obscured by movement artefact. Movement time must be preceded or followed by sleep and is thus distinguished from movement occurring during wakefulness.

Additional sleep values are determined from each sleep study and contribute to the clinical interpretation of the study. These additional variables include the following.

- **Recording time** is the time elapsed between "lights out" and "lights on" at the end of the study.

- **Total sleep time** (TST) is the total time occupied by stage 1, stage 2, SWS and REM sleep.

- **Sleep efficiency** (SE) is defined as total sleep time divided by recording time and is expressed as a percentage.

- **Sleep latency** (SL) is the time from "lights off" to the first epoch scored as sleep. Some authors prefer to use the first epoch of stage 2 in order to be more confident about identifying the onset of sustained sleep. However, when sleep is very disrupted, there may be an extended interval from recognition of stage 1 until an epoch that can be scored as stage 2.

- **REM latency** is the time from sleep onset (as described earlier) to the first time period scored as REM, minus any intervening epochs as wakefulness.

- **Sleep stage percentages** (% in stage 1, stage 2, SWS and REM sleep) are determined by dividing time recorded in each sleep stage by total sleep time.

- **Wake after sleep onset** (WASO) is time spent awake after sleep onset.

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The objective criteria defining normal sleep are based on: sleep latency, total sleep time, sleep efficiency and the number of awakenings, including cortical arousals. However, all these parameters are age-related, sometimes also gender-related, and may vary from one individual to another.

Normal sleep has a clearly defined architecture that is relatively stable. Predictable changes in sleep architecture occur with age. Beginning in middle age, SWS becomes less prominent, the number of awakenings increase, and sleep efficiency decreases. Published information on normal sleep can serve as an outline for normal values in PSG (Williams, Karacan and Hursch, 1974; see also Table 2.1), but each laboratory must study control subjects to identify any significant effects on sleep that result from differences in technique or environment (Ebersole and Pedley, 2003).

Table 2.1
Average normal values for adults of different ages

Sleep parameter (normal values)	20–29 years	40–49 years	60–69 years
TST (min)	419	389	407
Sleep efficiency (TST/TIB[a])	95%	91%	90%
WASO	1%	6%	8%
Stage 1 (% of TST)	4%	8%	10%
Stage 2 (% of TST)	46%	55%	57%
SWS (% of TST)	21%	8%	2%
REM (% of TST)	28%	23%	23%
Sleep latency (min)	15	10	8

[a] *Time in bed*
Source: Williams, Karacan and Hursch, 1974.

Passchier-Vermeer (2003a) reports that subjects not exposed to loud night noise typically report waking up one and a half to two times during an average sleep period, while the number of EEG awakenings including cortical arousals averages 10–12 per night (Table 2.2).

Table 2.2
Parameters of normal sleep

Subjects not exposed to loud night noise	Subjective report of number of awakenings	Number of EEG awakenings
Normal adult subjects	1.5–2	10–12

Source: Passchier-Vermeer, 2003a.

Night arousals result in fragmented sleep, which in turn leads to excessive daytime sleepiness (EDS). The gold standard for the assessment of EDS is the multiple sleep latency test (MSLT) (see Table 2.3), which provides an objective quantification of "sleepiness". The preceding night's sleep requires the PSG to ensure adequate sleep and to exclude sleep disruption. During the day, four or five nap times are scheduled every two hours. For each scheduled nap time the patient lies down and assumes a comfortable sleep position with the technician's instructions to "close your eyes and attempt to sleep". Each nap is terminated 20 minutes after the nap time started if no sleep occurred; or after 15 minutes of continuous sleep as long as sleep onset (SO) criteria are met before the end of 20 minutes; or after 20 minutes if the patient awakens, even if the patient has been asleep less than 15 minutes. The patient is instructed to stay awake between the nap periods.

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Table 2.3 Mean sleep latency

Group	MSLT (min)	No REM SO (% of group)	1 REM SO (% of group)	2 or more REM SO (% of group)
Narcoleptics	2.9 ± 2.7	2	2	96
EDS (non-narcoleptic, non-sleep apnoeic)	8.7 ± 4.9	92	8	0
Controls	13.4 ± 4.3	100	0	0

Source: Ebersole and Pedley, 2003.

2.1.2 DEFINITIONS OF DISTURBED SLEEP

Sleep disorders are described and classified in the International Classification of Sleep Disorders (ICSD) (American Academy of Sleep Medicine, 2005).

When sleep is permanently disturbed and becomes a sleep disorder, it is classified in the ICSD 2005 as "environmental sleep disorder". Environmental sleep disorder (of which noise-induced sleep disturbance is an example) is a sleep disturbance due to a disturbing environmental factor that causes a complaint of either insomnia or daytime fatigue and somnolence. Secondary deficits may result, including deficits in concentration, attention and cognitive performance, reduced vigilance, daytime fatigue, malaise, depressed mood and irritability. The exact prevalence is not known. Fewer than 5% of patients seen at sleep disorder centres receive this diagnosis. The sex ratio is not known. The disorder may occur at any age, although the elderly are at more risk for developing this condition (American Academy of Sleep Medicine, 2005).

2.1.2.1 Insomnia

In the ICSD 2005 the section on insomnia includes a group of sleep disorders all of which have in common the complaint of insomnia (adjustment insomnia, psychophysiological insomnia, paradoxical insomnia, idiopathic insomnia, etc.), defined as repeated difficulty with sleep initiation, duration, consolidation or quality that occurs despite adequate time and opportunity for sleep and results in some form of daytime impairment. Insomnia is a symptom that often arises from primary medical illness, mental disorders and other sleep disorders, but may also arise from abuse or exposure. However, the general criteria for insomnia are the same for all subgroups of insomnias.

2.1.2.2 General criteria for insomnia

A. A complaint is made concerning difficulty initiating sleep, difficulty maintaining sleep, waking up too early or sleep that is chronically non-restorative or poor in quality. In children, the sleep difficulty is often reported by the carer and may consist of observed bedtime resistance or inability to sleep independently.

B. The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep.

C. At least one of the following forms of daytime impairment related to the nighttime sleep difficulty is reported by the patient:

- fatigue or malaise
- attention, concentration, or memory impairment
- social or vocational dysfunction or poor school performance
- mood disturbance or irritability

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- daytime sleepiness
- motivation, energy, or initiative reduction
- proneness to errors or accidents at work or while driving
- tension, headaches, or gastrointestinal symptoms in response to sleep loss
- concerns or worries about sleep.

Defining the cause of a sleep/wake disturbance in an insomnia patient is a complex task since it is often multifactorial. In fact, a confluence of factors that support multiple insomnia diagnoses may be judged important in many patients with insomnia. Although selection of a single diagnosis is preferable and this selection may be appropriate, such a selection should not necessarily imply the absence of a subset of factors relevant to an alternate diagnosis. When criteria for multiple insomnia diagnosis are met, all relevant diagnosis should be assigned.

2.1.2.3 Environmental sleep disorder

In the ICSD 2005, environmental sleep disorder is listed in the category of "other sleep disorders". Noise-induced sleep disturbance is one of the disturbing environmental factors that cause a complaint of either insomnia or daytime fatigue and somnolence.

The diagnostic criteria for environmental sleep disorder are the following.

A. The patient complains of insomnia, daytime fatigue or a parasomnia. In cases where daytime fatigue is present, the daytime fatigue may occur as a result of the accompanying insomnia or as a result of poor quality of nocturnal sleep.

B. The complaint is temporally associated with the introduction of a physically measurable stimulus or environmental circumstance that disturbs sleep.

C. It is the physical properties, rather than the psychological meaning of the environmental factor, that accounts for the complaint.

D. The sleep disturbance is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, medication use or substance use disorder.

The prevalence of environmental sleep disorder is not known. Fewer than 5% of patients seen at sleep disorder centres receive this diagnosis.

International standardization and quantification for measurement of the depth of sleep is based on Rechtschaffen and Kales criteria from 1968. Sleep is divided into 30-second epochs, and a phase is only assessed if the specific features are evident for more than 50% of the epoch length. For example, wakefulness is scored when at least 15 seconds of continuous awakening is present. Arousal reactions not leading to macroscopic awakening were not included in the definition by Rechtschaffen and Kales. With the arousals as described by the American Sleep Disorders Association (1992) it is possible to display subvigilant sleep fragmentation, caused by intrinsic sensory and autonomic alarm reactions. An arousal index providing the arousal density (events per hour of sleep) was taken as a measure of the degree of severity. In one hour, 10–20 arousals are considered as normal in healthy adults. However, the use of EEG arousals with the American Sleep Disorders Association definition provides no sufficient explanation of daytime sleepiness (Ali, Pitson and Stradling 1996; Ayas et al., 2001) unless they are accompanied by vegetative arousals.

Regarding noise, different vigilance level assessments in various functional systems are important. Dumont, Montplaisir and Infante-Rivard (1988) proposed investigations of

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vegetative, motor and sensory functions independently of each other. One of the possible factors indicating disturbed sleep is a vegetative arousal index. A vegetative arousal index of more than 30 per hour is certainly considered as serious, more than 20 per hour as intermediate and more than 10 as a light form of sleep disorder.

With respect to insomnia (section 2.1.2), there is the possibility of misclassification if the general practitioner (GP) overlooks excessive noise as the possible cause of the complaint. There is also the possibility that the insomnia is aggravated by noise.

2.1.3 CONCLUSIONS

Published information on normal sleep can serve as an outline for normal values in PSG. However, these values are only informative, because each sleep laboratory must study control subjects to identify any significant effects on sleep that result from differences in technique or environment. Excessive daytime sleepiness is a consequence of disturbed night sleep and can be objectively assessed by MSLT, which provides an objective quantification of "sleepiness".

2.2 LONG-TERM HEALTH RISK MEDIATED BY SLEEP DISTURBANCES

2.2.1 STRESSORS, NEUROBEHAVIOURAL DATA AND FUNCTIONAL NEUROIMAGING

It is generally accepted that insufficient sleep and particularly sleep loss has a great influence on metabolic and endocrine functions (Spiegel, Leproult and van Cauter, 1999), as well as on inflammatory markers, and contributes to cardiovascular risk. C-reactive protein (CRP) as a major marker of the acute phase response to inflammatory reaction promotes secretion of inflammatory mediators by vascular endothelium and may be therefore directly involved in the development of atherosclerotic lesions. CRP as a risk predictor of strokes and heart attacks linearly increases with total and/or partial sleep loss (Meier-Ewert et al., 2004).

An additional factor, closely linked to cardiovascular health, glucose regulation and weight control, is leptin. Leptin is one of the major regulators of energy homeostasis and its circadian profile interacts closely with sleep.

Secretion of leptin increases at night and decreases during the day. A decreased leptin level, that is connected with sleep loss, increases appetite and predisposes to weight gain, impaired glucose tolerance and impaired host response.

Other studies have focused on how sleep loss affects neurobehavioural functions, especially neurocognitive performance. Functional brain imaging and EEG brain mapping studies show that the patterns of functional connectivity between brain regions, evident in the performance of specific cognitive tasks, are altered by sleep loss (NCSDR, 2003). According to this finding, the maintenance of sustained performance during sleep loss may depend upon regional functional plasticity.

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Cumulative waking, neurocognitive deficits and instability of state that develop from chronic sleep loss have a basis in a neurobiological process that can integrate homoeostatic pressure for sleep across days. Increased efforts have helped to determine the roles of REM and non-REM sleep in memory.
Functional brain imaging techniques, such as positron emission tomography (PET), functional magnetic resonance imaging (fMRI), magnetic resonance spectroscopy (MRS), single photon emission computed tomography (SPECT) and magneto-electroencephalography (MEG), have recently been analysed in a study of sleep and waking (NCSDR, 2003). These techniques allow the measurement of metabolic and neurochemical activity throughout the brain, and can reveal dynamic patterns of regional cerebral activity during various brain states, including stages of sleep and levels of alertness during wakefulness or during functional challenge. These techniques can also help identify both normal and abnormal sleep/wake processes.

In the last five years, functional neuroimaging techniques (particularly PET) have revealed that non-REM sleep is associated with the deactivation of central encephalic regions (brainstem, thalamus, basal ganglia) and multimodal association cortices (for instance, prefrontal and superior temporal/inferior parietal regions). REM sleep is characterized by reactivation of all central encephalic regions deactivated during non-REM sleep except the multimodal association areas. PET studies during sleep-deprived wakefulness have revealed regional cerebral deactivations that are especially prominent in prefrontal and inferior parietal/superior temporal cortices, and in the thalamus. This pattern is consistent and helpful in explaining the nature of cognitive performance deficits that occur during sleep loss. As revealed by means of fMRI techniques during cognitive task performance, the maintenance of performance following sleep loss may be a function of the extent to which other cortical brain regions can be recruited for task performance in the sleep-deprived state.

PET, SPECT and fMRI studies have revealed, in depressed patients, initially elevated activation in anterior cingulate and medial orbital cortices (NCSDR, 2003). In these patients, sleep deprivation reduces this regional hyperactivation, and improvements in mood are a function of the extent to which this activity is reduced. These studies point to possible mechanisms by which antidepressant drugs may exert their effects. Further research should be oriented towards neuroimaging and measurements of changes in the brain's metabolic activity at the neurotransmitter level.

2.2.2 SIGNALS MEDIATED BY A SUBCORTICAL AREA (THE AMYGDALA), THE ROLE OF STRESS HORMONES IN SLEEP DISTURBANCE AND THEIR HEALTH CONSEQUENCES

Experimental as well as clinical studies (Waye et al., 2003; Ising and Kruppa, 2004) showed that the first and fastest signal of stressors introduced by noise is detected and mediated by a subcortical area represented by the amygdala while the stress response to noise is mediated primarily by the hypothalamus-pituitary-adrenal (HPA) axis. A major intrinsic marker of the circadian rhythm is in the level of circulating corticosteroids derived from activity within the HPA axis. A protracted stress response with activation of the HPA axis is a major physiological response to environmental stressors. The cortisol response to awakening is an index of adrenocortical activity, and long-term nocturnal noise exposures may lead, in persons liable to be stressed by noise, to permanently increased cortisol concentration above the nor-

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mal range. The hypothesis that an increased risk of cardiovascular diseases is connected with stress concepts is generally accepted (Ekstedt, Åkerstedt and Soderstrom, 2004; Ising and Kruppa, 2004). Stress reactions may lead to derangement of normal neurovegetative and hormonal processes and influence vital body functions. Cardiovascular parameters such as BP, cardiac function, serum cholesterol, triglycerides, free fatty acids and haemostatic factors (fibrinogen) impede the blood flow through increased viscosity and presumably blood sugar concentration as well. Insulin resistance and diabetes mellitus, stress ulcers and immune system deficiency are also frequent consequences of stress reaction. Disturbed sleep may lead to immunosuppression and diminished protein synthesis (Horne, 1988).

As well as nonspecific effects of the stress response on the functioning of the immune system, there is considerable evidence for a relation between sleep, especially SWS, and the immune system (Brown, 1992). This evidence includes surges of certain immune parameters and growth hormones at onset of SWS, correlation of non-REM sleep, total sleep time and sleep efficiency with natural killer cell activity, and correlation of SWS with recovery from infections. These data, taken together with information on the effect of intermittent transportation noise on SWS during the first sleep cycles and overnight, suggest that the immune response could also be impacted directly by environmental noise during sleep (Carter, 1996).

2.2.3 SLEEP RESTRICTION, ENVIRONMENTAL STRESSORS (NOISE) AND BEHAVIOURAL, MEDICAL AND SOCIAL HEALTH CONSEQUENCES OF INSUFFICIENT SLEEP: RISK OF MORBIDITY AND MORTALITY

Sleep restriction due to environmental stressors leads to primary sleep disorders, but health is also influenced by the consequence of stress response to noise mediated by the HPA axis and/or by restriction of specific sleep stages (see above).

Sleep restriction leads, in approximately 40% of affected subjects, to daytime sleepiness that interferes with work and social functioning. Excessive daytime sleepiness is thus a major public health problem, as it interferes with daily activities, with consequences including cognitive problems, motor vehicle accidents (especially at night), poor job performance and reduced productivity (Lavie, Pillar and Malhotra, 2002). In the last decade, experimentally based data have been collected on chronic restriction of sleep (by 1–4 hours a night), accumulating daytime sleepiness and cognitive impairment. Most individuals develop cognitive deficits from chronic sleep debt after only a few nights of reduced sleep quality or quantity. New evidence suggests additional important health-related consequences of sleep debt related to common viral illnesses, diabetes, obesity, heart disease, depression and other age-related chronic disorders.

The effects and consequences of sleep deprivation are summarized in Table 2.4 (Lavie, Pillar and Malhotra, 2002).

The relationship between sleep quantity and quality and estimates of morbidity and mortality remains controversial. Epidemiological data (NCSDR, 2003) suggest that habitually short sleep (less than 6 hours sleep per night) is associated with increased mortality. Epidemiological studies in recent years elucidated, however, that too much sleep is a problem as well. Kripke et al. (2002) evaluated a questionnaire study of 1.1 million men and women aged 30–102 years and found the lowest mortality risk between respondents sleeping 7 hours per night.

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Type	Short-term	Long-term
Behavioural	Sleepiness Mood changes Irritability and nervousness	Depression/mania Violence
Cognitive	Impairment of function	Difficulty in learning new skills Short-term memory problems Difficulty with complex tasks Slow reaction time
Neurological	Mild and quickly reversible effects	Cerebellar ataxia, nystagmus, tremor, ptosis, slurred speech, increased reflexes, increased sensitivity to pain
Biochemical	Increased metabolic rate Increased thyroid activity Insulin resistance	Decreased weight despite increased caloric intake (in animals) Diabetes, obesity (in humans)
Others	Hypothermia Immune function impairment	Susceptibility to viral illness

Table 2.4 Consequences of sleep deprivation

Mortality risk significantly increased when sleep duration was less than 6 or higher than 8 hours per night. Other authors have also published similar results (Patel et al., 2004; Tamakoshi and Ohno, 2004). Patel et al. (2004) in a prospective study of sleep duration and mortality risk in 5409 women confirmed previous findings that mortality risk is lowest among those sleeping 6–7 hours per night. The mortality risk for death from other causes significantly increased in women sleeping less than 5 and more than 9 hours per night. It is not clear how the length of sleep can increase this risk, although animal evidence points to a direct link between sleep time and lifespan (see section 2.5 in this chapter). Up to now, no epidemiological prospective study has been published that examines the relationship between sleep and health outcomes (morbidity and mortality) with subjective and objective estimates. Recent studies, however, show that sleep duration of least 8 hours is necessary for optimal performance and for prevention of daytime sleepiness and accumulation of sleep debt.

Environmental stressors, including noise, mostly cause insomnia. Insomnia also involves daytime consequences, such as tiredness, lack of energy, difficulty concentrating and irritability. A reasonable prevalence estimate for chronic insomnia in the general population is about 10%; for insomnia of any duration or severity this rises to between 30% and 50%, and incidence increases with ageing. In the course of perimenopausal time, women are particularly vulnerable to developing this complaint. The major consequences and co-morbidity of chronic insomnia (see Table 2.5) consist of behavioural, psychiatric and medical problems. Several studies also report a higher mortality risk (Zorick and Walsh, 2000).

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Table 2.5 Consequences of chronic insomnia

Type	Consequence
Behavioural	Poor performance at work, fatigue, memory difficulties, concentration problems, motor vehicle accidents
Psychiatric	Depression, anxiety conditions, alcohol and other substance abuse
Medical	Cardiovascular, respiratory, renal, gastrointestinal, musculoskeletal disorders Obesity Impaired immune system function
Mortality	Increased risk is reported

2.2.3.1 Behavioural consequences

Transient (short-term) insomnia is usually accompanied by spells of daytime sleepiness and performance impairment the next day. Persistent (long-term) insomnia tends to be associated with poor performance at work, fatigue, memory difficulties, concentration problems and twice as many fatigue-related motor vehicle accidents as in good sleepers.

2.2.3.2 Psychiatric conditions

Epidemiological research indicates that the prevalence of any psychiatric disorder is two or three times higher in insomniacs. The risk of depression as a co-morbid state appears to be particularly strong, being approximately four times more likely in insomnia patients. Furthermore, insomnia may be an early marker for psychiatric disorders such as depression, anxiety conditions and alcohol abuse. Anxiety has been quite commonly found in insomniacs compared with the general population. About 25–40% of insomnia patients are estimated to have significant anxiety, and the abuse of alcohol and other substances is increased in insomniacs relative to good sleepers (Ford and Kamerow, 1989). Samples of unselected psychiatric patients have about a threefold increase in the frequency of insomnia compared with healthy control subjects, and the severity of the condition correlates with the intensity of the psychiatric symptoms. Among samples of outpatients who consulted their GPs for insomnia, about 50% presented with psychiatric conditions, and about half of these patients were probably depressed (Zorick and Walsh, 2000).

2.2.3.3 Medical consequences

Insomnia has been statistically associated with various medical conditions, including disorders of the cardiovascular, respiratory, gastrointestinal, renal and musculoskeletal systems. A large series of insomniac patients showed that poor sleepers are more than twice as much at risk of ischaemic heart disease (IHD) as good sleepers (Hyyppa and Kronholm, 1989). Insomnia patients were also shown (Irwin, Fortner and Clark, 1995) to have impaired immune system function. Keith et al. (2006) hypothesize a connection between sleep deficit as one of the possible factors to explain the rise in obesity. Hormone changes and animal experiments apparently support this.

2.2.3.4 Mortality risk

Only a few epidemiological studies deal with mortality in insomniacs. According to Kripke et al. (1979), reduced sleep time is a greater mortality risk than smoking, hypertension and cardiac disease. Higher death rates are also reported among short sleepers. In this respect, however, further systematic investigation of the link between insomnia, short sleep and death is desirable.

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2.3 RISK GROUPS

Risk groups are people who may be either sensitive (showing more reaction to a stimulus than the average), are more exposed (also called vulnerable) or both.

2.3.1 HEALTH EFFECTS OF DISTURBED SLEEP IN CHILDREN

Although children appear to tolerate a single night of restricted sleep with no detrimental effect on performance of brief tasks, perhaps more prolonged restriction and prolonged tasks similar to those required in school would show negative effects. In addition, as children seem to require more time to recuperate fully from nocturnal sleep restriction than adults (Carskadon, Harvey and Dement, 1981a), with additional nights of partial sleep deprivation, cumulative sleepiness might become a significant problem.

Empirical data that directly address the effects of repeated sleep loss on children's mood or cognitive function are sparse. A range of clinical and observational data support a general picture that inadequate sleep results in tiredness, difficulties in focusing attention, low thresholds for negative reactions (irritability and easy frustration), as well as difficulty in controlling impulses and emotions. In some cases, these symptoms resemble attention-deficit hyperactivity disorder (ADHD).

Environmental noise experienced at home during night-time is a sometimes unpredictable and most often discontinuous event (for example traffic noise, aircraft or train noise, a noisy environment for other reasons, for instance proximity with a discotheque, etc.), that might lead to sleep disruption without leading to behavioural awakenings through the alteration of sleep microstructure, in a similar manner as other sleep disturbing events such as respiratory disturbances.

Therefore, in respect of clinical settings, we can assume that, in children, an experimental model for the consequences of noise can be represented by respiratory disturbances during sleep, such as snoring, upper airway resistance syndrome (UARS) or obstructive sleep apnoea syndrome (OSAS), either for the noise produced by snoring or for the effects on the arousal system and sleep microstructure.

For this reason, this section describes the well-studied effects of sleep breathing disorders on children's health and then evaluates the indicators of sleep disruption from the point of view of sleep microstructure.

In the literature few data on the medium- and long-term effects of disturbed sleep in children are available from the longitudinal point of view. Most reports focused on respiratory disturbances during sleep as a theoretical model to evaluate the long-term effects of disturbed sleep in children. This review reports on the medium- and long-term negative consequences of disturbed sleep on cognitive functioning, behaviour, mental health, growth and the cardiovascular system.

2.3.1.1 Sleep deprivation in children

The effects of sleep deprivation were evaluated in children. The findings only indirectly pertain to this general report, although repeated noise-induced sleep disruption favours sleep deprivation.

In another study, 15 healthy infants aged 78+/-7 days were studied during two nights: one night was preceded by sleep deprivation (kept awake for as long as pos-

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sible beyond their habitual bedtime: median onset 150 min; range 0–210 min) (Thomas et al., 1996). Of the 15 children, 13 slept supine, 12 were breastfed and 4 were from smoking parents. Following sleep deprivation, infants maintained a greater proportion of quiet sleep (44% vs. 39%; p=.002). There was no measurable change in arousal propensity by either graded photic (stroboscope) or auditory stimuli (1 kHz pure tone, delivered in the midline of the cot, from 73 dB and increased in 3 dB steps to 100 dB) during quiet sleep.

Forty-nine Finnish children (26 boys/23 girls) aged 7–12 years were interviewed together with their parents and school teachers, and recorded for 72 hours with a belt-worn activity monitor during weekdays.

The objectively measured true sleep time was associated with psychiatric symptoms reported by a teacher. The decreased amount of sleep was associated more with externalizing than internalizing types of symptoms (aggressive and delinquent behaviour, attention, social, and somatic problems) (Aronen et al., 2000).

In a survey, it was also shown that out of 100 Belgian school children, aged between 9 and12 years, those with poor sleep (insomnia) were also showing more frequent poor school performance (failure to comply with expected grades) than good sleepers. The relation between poor sleep and a noisy environment was, however, not evaluated (Kahn et al., 1989).

2.3.1.2 Neurocognitive manifestations

Several studies in adults have shown that sleep fragmentation and hypoxaemia can result in daytime tiredness and loss of concentration, retrograde amnesia, disorientation, morning confusion, aggression, irritability, anxiety attacks and depression. One could hypothesize that sleep fragmentation and hypoxaemia would affect the neuropsychological and cognitive performance also in children, where the impact of abnormal sleep may be even greater than in adults. In fact, neurocognitive and behavioural deficits and school problems have been reported recently in children with sleep-related obstructive breathing disorders (SROBD).

2.3.1.3 Attention capacity

This represents the ability to remain focused on a task and appropriately attend to stimuli in the environment. Taken together the studies to date indicate that children with SROBD are less reflective, more impulsive, and show poorer sustained and selective attention. Blunden et al. (2000) reported that, compared to 16 controls, 16 children with mild SROBD showed reduced selective and sustained attention. Owens-Stively et al. (1997) suggested a dose–response in attention–impulsivity with moderate to severe obstructive sleep apnoea syndrome (OSAS) children showing greater deficits than mild OSAS children. Importantly, early treatment showed that attention deficits in children with OSAS are reversible (Guilleminault et al., 1982b). In another study, 12 children with moderate to severe OSAS showed a significant reduction in inattention and an improvement in aggressive and hyperactive behaviours and vigilance after surgical treatment (Ali, Pitson and Stradling, 1996).

2.3.1.4 Memory

Rhodes et al. (1995) found inverse correlations between memory and learning performance and the apnoea hypopnea index in 14 morbidly obese children. Smaller deficits were observed by Blunden et al. (2000), who found in their sample of children with mild SROBD that mean global memory performance was in the lower end of the normal range compared to controls.

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A recent study using actigraphy in normal school-age children showed that lower sleep efficiency and longer sleep latency were associated with a higher percentage of incorrect responses in working memory tasks; shorter sleep duration was associated with performing tasks at the highest load level only. Also, controlling for age, gender, and socioeconomic status, sleep efficiency and latency were significantly associated with the mean incorrect response rate in auditory working memory tasks. This study showed that sleep quality (evaluated as sleep efficiency = 100* [sleep + light sleep]/duration) is more strongly associated with performance in working memory tasks than sleep duration, suggesting that in assessing sleep, attention should be directed not only at the amount of sleep but also at sleep quality.

2.3.1.5 Intelligence

Inspection of the mean IQ scores reported in the study by Rhodes et al. (1995) suggested that their sample of five obese children with moderate to severe OSAS performed in the borderline range whereas controls performed in the normal range. Blunden et al. (2000) showed smaller deficits in children with mild SROBD whose mean verbal and global IQ were in the lower end of the normal range.

It remains unclear as to whether the putative negative effects of SROBD on intelligence are global in nature or confined to specific areas such as verbal rather than performance or visuospatial intelligence and whether these impairments can be reversed.

2.3.1.6 Learning and school performance

It has been widely reported (Stradling et al., 1990; Guilleminault et al., 1996; Richards and Ferdman, 2000) that children with SROBD show reduced academic performance and learning.

Weissbluth et al. (1983) found that poor academic achievers had a higher prevalence of night-time snoring (38% vs. 21%) and breathing difficulties (13% vs. 6%). Out of 297 children with SROBD (22% snorers and 18% sleep-associated gas exchange abnormalities), 40% were in the lowest 10th percentile of academic performance (Gozal, 1998) and SROBD in early childhood may continue to adversely affect learning in later years (Gozal and Pope, 2001). Gozal (1998) found in his sample of poor academic achievers that school grades improved post-adenotonsillectomy in treated but not untreated children.

As well as those with SROBD, healthy normal children with fragmented sleep (measured by actigraphy) also showed lower performance on neurobehavioural functioning (NBF) measures, particularly those associated with more complex tasks, and also had higher rates of behavioural problems (Sadeh, Gruber and Raviv, 2002). Furthermore, in normal children without sleep disorders, modest sleep restriction can also affect children's NBF. Sadeh, Gruber and Raviv (2003) monitored 77 children for 5 nights with activity monitors. On the third evening, the children were asked to extend or restrict their sleep by an hour on the following three nights. Their NBF was reassessed on the sixth day following the experimental sleep manipulation and showed that sleep restriction led to improved sleep quality and to reduced reported alertness.

These studies suggest that fragmented sleep or insufficient sleep is highly relevant during childhood and that children are sensitive to modest alterations in their natural sleep duration.

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Early reports documented that untreated OSAS can have long-term negative effects, such as failure to thrive, cor pulmonale and mental retardation. These severe consequences are less common now due to early diagnosis and treatment, but recent reports have focused on other long-term effects mainly related to neurocognitive deficits, such as poor learning, behavioural problems and ADHD (Marcus, 2001).

Gozal and Pope (2001) tried to determine the potential long-term impact of early childhood snoring. Analysing questionnaires of 797 children in a low academic performance group (LP) and 791 in a high academic performance (HP) group, they found that frequent and loud snoring during early childhood was reported in 103 LP children (12.9%) compared with 40 HP children (5.1%). Therefore, children with lower academic performance in middle school are more likely to have snored during early childhood and to require surgery for snoring compared with better performing schoolmates. These findings suggest that children who experienced sleep-disordered breathing during a period traditionally associated with major brain growth and substantial acquisition of cognitive and intellectual capabilities may suffer from a partially irreversible compromise of their a priori potential for academic achievement. Three major components that result from the intermittent upper airway obstruction that occurs during sleep in children could theoretically contribute to such neurocognitive deficits, namely episodic hypoxia, repeated arousal leading to sleep fragmentation and sleep deprivation, and periodic or continuous alveolar hypoventilation.

Schooling problems may underlie more extensive behavioural disturbances such as restlessness, aggressive behaviour, EDS and poor neurocognitive test performances. Nearly 20–30% of children affected by OSAS or loud and frequent snoring show important signs of behavioural problems such as inattention and hyperactivity. Problems similar to symptoms of ADHD are linked to the presence of repeated sleep arousals, and intermittent hypoxic events, inducing a lack of behavioural inhibition with negative implications for working memory, motor control and self-regulation of motivation and affect.

In contrast with these data, Engle-Friedman et al. (2003) recently found a significant improvement of functions, at least in mild to moderate OSAS, when measured several months following an adenotonsillectomy, but they confirmed that their results could not rule out the possibility, even after treatment, of partial irreversible damage to academic function that may be detected only later in life. In addition, they stated that adults who also had deficits of neurocognitive executive functions related to the prefrontal area failed to improve significantly after treatment.

The negative long-term effects may be mediated by the irreversible alteration of the prefrontal cortex (PFC) and be related to structural changes of the brain as a consequence of both hypoxaemia and sleep fragmentation induced by OSAS or other pathologies affecting sleep.

In a recent report concerning OSAS adults, Macey et al. (2002) demonstrated grey matter loss in cerebral sites involved in motor regulation of the upper airway as well as in areas contributing to cognitive function (frontal and parietal cortex, temporal lobe, anterior cingulate, hippocampus and cerebellum). It can be argued that, in critical stages of brain development (that is, in childhood), these effects can lead to even more severe consequences, which could explain the negative long-term effects.

It is speculative to think that the remodelling of the brain could also be mediated by sleep and, therefore, sleep fragmentation could affect the process of brain plasticity (that is, the capacity of the brain to modify its structure and function over time). Recent studies show-

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ing experience-dependent gene-expression of gene *zif-268* during paradoxical sleep in rats exposed to a rich sensorimotor environment, and the role of sleep in enhancing the remodelling of ocular dominance in the developing visual cortex are also in line with the hypothesis that sleep affects neuronal plasticity and memory processes (Peigneux et al., 2001).

2.3.1.7 Neurobehavioural manifestations

Behavioural disturbances are common in children with SRODB, with higher prevalence rates of both internalized (for instance being withdrawn, shy, anxious and psychosomatic) and externalized (for instance impulsivity, hyperactivity, aggression and delinquency) problematic behaviours (Blunden, Lushington and Kennedy, 2001). The most frequently documented problematic behaviour in children with SROBD is attention deficit hyperactivity with a prevalence rate of 20–40% (Weissbluth et al., 1983; Ali, Pitson and Stradling, 1993). Conversely, children with ADHD showed a high prevalence rate of snoring (Chervin et al., 1997) and a co-diagnosis of ADHD has been reported in 8–12% of children with OSAS (O'Brien and Gozal, 2002).

A few studies have documented that children with sleep disorders tend to have behavioural problems similar to those observed in children with ADHD. A survey of 782 children documented daytime sleepiness, hyperactivity, and aggressive behaviour in children who snored, with 27% and 38% of children at high risk for a sleep or breathing disorder displaying clinically significant levels of inattention and hyperactive behaviour, respectively (Ali, Pitson and Stradling, 1994).

At 3 years of age children with persistent sleep problems (n = 308) were more likely to have behaviour problems, especially tantrums and behaviour management problems (Zuckerman, Stevenson and Bailey, 1987).

In a study of 16 children with a mean age of 12+/-4 years suffering from chronic pain due to juvenile rheumatoid arthritis and secondary poor sleep, polysomnographic recordings showed poorer night-time sleep, longer afternoon naptime and more daytime sleepiness than normal values from the literature (Zamir et al., 1998). In a school survey of children aged 9–12 years (n = 1000), those with poor sleep (insomnia for more than 6 months) had poorer school performance, defined as failure to comply with expected grades, than good sleepers. Their learning problems were tentatively attributed to the long-term effect of poor sleep (Kahn et al., 1989).

A questionnaire administered to children aged 4–12 years (n = 472) showed a relation between sleep problems and tiredness during the day (Stein et al., 2001).

In children aged 9–12 years (n = 77), shortening sleep by one hour was associated with reduced alertness and significant lowering of neurobehavioural functioning (Sadeh, Gruber and Raviv, 2003). In school-age children (n = 140) recorded at home with an actigraph, a significant relation was shown between the presences of fragmented sleep, daytime sleepiness and lower performance in neurobehavioural functioning evaluated by various performance tests (Sadeh, Gruber and Raviv, 2000). These children also had higher rates of behavioural problems, as reported by their parents (Sadeh, Gruber and Raviv, 2002).

In Finland, children aged 7–12 years (n = 49) were interviewed together with their parents and schoolteachers and recorded for 72 hours with a belt-worn activity monitor during weekdays. The decreased amount of sleep was associated with symptoms such as aggressive and delinquent behaviour, attention, social and somatic problems. The findings of this research were better associated with the teachers' than the par-

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ents' reports, suggesting that parents may be unaware of their child's sleep deficiencies as the behavioural problems may be more evident at school than at home (Aronen et al., 2000).

A prospective long-term study conducted in Sweden on 2518 children revealed that within a subgroup of 27 children with severe and chronic sleep problems, 7 children developed symptoms that met the criteria for ADHD by the age of 5.5 years (Thunström, 2002). Compared to the other children with sleep problems, these subjects had more frequent psychosocial problems in the family, bedtime struggles and long sleep latency at bedtime.

A population-based, cross-sectional questionnaire survey was conducted in Massachusetts on 30 195 children aged 5 years (Gottlieb et al., 2003). Children described by their parents as having sleep-disordered breathing (snoring, noisy breathing, apnoea) were significantly more likely to have daytime sleepiness and problem behaviours, including hyperactivity, inattention and aggressiveness (all with an odds ratio >2.0). These problem behaviours were suggestive of ADHD.

Similar findings were found in a group of children aged 5–7 years with periodic limb movement disorder who were studied polygraphically and their recording compared with those of age-matched children with ADHD. Their repeated sleep fragmentation resulting from the periodic limb movement disorder favoured the development of symptoms similar to those seen in ADHD (Crabtree et al., 2003).

The parents of a group of children with an average age of 8.6 years (range 2–17 years) reported that their children had difficult behaviours on the day that followed a 4-hour night-time sleep restriction (Wassmer et al., 1999). In one study, a 2-hour sleep reduction induced by delayed bedtime has been shown to increase daytime sleepiness, mainly during morning hours (Ishihara and Miyke, 1998; Ishihara, 1999).

Following one night of 4 hours of sleep deprivation imposed on children (aged 11–13 years), a decrease in performance tests has been observed (Carskadon, Harvey and Dement, 1981a).

Following one night's sleep loss, adolescents showed increased sleepiness, fatigue and reaction time. They selected less difficult academic tasks during a set of tests, but the percentages of correct responses were comparable to those seen following a normal night's sleep (Engle-Friedman et al., 2003).

Another study has been conducted on 82 children, aged 8–15 years. They were assigned an optimized, 10-hour night of sleep, or a restricted 4-hour night of sleep. Sleep restriction was associated with shorter daytime sleep latency, increased subjective sleepiness, and increased sleepy and inattentive behaviours, but was not associated with increased hyperactive-impulsive behaviour or impaired performance in tests of response inhibition and sustained attention (Fallone et al., 2001).

2.3.1.8 Mental health

A recent longitudinal study on the outcomes of early life sleep problems and their relation to behaviour problems in early childhood stressed the importance of studying the natural history of sleep problems and their consequences in order to identify whether persistent or recurrent sleep problems at age 3–4 years are associated with co-morbidities such as child behaviour problems, maternal depression and poor family functioning (Peiyoong, Hiscock and Wake, 2003).

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The authors found that night waking at 3–4 years of age continued to be common. Seventy eight percent of mothers reported that their child awoke during the night at least once during the week, and of these waking children, 43% were reported to have awakenings 4 or more nights per week. Children with early sleep problems had significantly higher mean scores on internalizing and externalizing behaviour and the aggressive behaviour and somatic problems subscales of the Child Behavior Checklist (CBCL).

It has been noted that within groups of children and adolescents with psychiatric, behavioural or emotional problems, rates of sleep disorders are elevated (Sadeh et al., 1995). On the other hand, children and adolescents with disturbed sleep report more depression, anxiety, irritability, fearfulness, anger, tenseness, emotional instability, inattention and conduct problems, drug use and alcohol use.

Only a few longitudinal studies in adolescents have evaluated the impact of insomnia on future functioning. In a large sample of 11–17-year-old adolescents, followed for one year, using symptoms of DSM-IV criteria for insomnia, Roberts, Roberts and Chen (2002) found that nearly 18% of the youths 11–17 years of age reported non-restorative sleep almost every day in the past month, over 6% reported difficulty in initiating sleep, over 5% waking up frequently during the night, another 3% had early-morning awakening almost every day, over 7% reported daytime fatigue and 5% daytime sleepiness. Combining "often" and "almost every day" response categories dramatically increases prevalence, ranging from 60% for non-restorative sleep to 23% for daytime fatigue and 12% for waking up at night with difficulty going back to sleep. The re-evaluation of the sample at follow-up showed that insomnia predicted two indicators of psychological functioning: self-esteem and symptoms of depression (Roberts, Roberts and Chen, 2002).

2.3.1.9 Growth impairment

Failure to thrive is a well-known complication of disturbed sleep and childhood OSAS. The cause of poor growth is not known, although many different reasons have been implicated: (a) poor caloric intake associated with adenotonsillar hypertrophy; (b) excessive caloric expenditure secondary to increased work of breathing; (c) abnormal growth hormone (GH) release secondary to loss of deep non-REM sleep. The relative roles of these factors are unclear (Marcus et al., 1994; ATS, 1999). Circulating concentrations of insulin-like growth factor-I (IGF-I) and IGF-binding protein 3 (IGFBP-3) reflect mean daily GH levels, and seem to correlate well with physiological changes in GH secretion. In the operated children with initial OSAS a highly significant reduction in the apnoea-hypopnea index (AHI) was found and both the IGF-I and the IGFBP-3 concentrations increased significantly. GH is released in a pulsatile fashion; the initial secretion is synchronized with the onset of SWS and strongly correlated with slow-wave activity, within 90 to 120 minutes from the onset of sleep (Nieminen et al., 2002). In OSAS children, the sleep architecture is relatively well-preserved, but the microstructural alteration of SWS due to micro-arousals induced by respiratory disturbance could play a role in the abnormal profile of GH secretion.

2.3.1.10 Cardiovascular complications

Children with OSAS had a significantly higher diastolic blood pressure (BP) than those with primary snoring. Multiple linear regression showed that BP could be predicted by apnoea index, body mass index and age. The aetiology of OSAS-related hypertension is thought to be due to a number of factors, particularly sympathetic nervous system activation secondary to arousal and, to a lesser degree, hypoxaemia.

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Although cortical arousals at the termination of obstructive apnoeas are less common in children than in adults, children may manifest signs of subcortical arousal, including autonomic changes such as tachycardia. It is therefore possible that these subcortical arousals are associated with elevations of BP. A correlation between the frequency of obstructive apnoea and BP, but no correlation between SaO2 (arterial oxygen saturation) and BP was found, suggesting that respiratory-related subcortical arousals rather than hypoxaemia may be a major determinant of BP elevation in children (Marcus, Greene and Carroll, 1998). Similarly to BP variations induced by OSAS, other studies suggest that chronic exposure to environmental noise during sleep could contribute to a permanent increases in BP in otherwise healthy individuals and that no habituation to noise was apparent over three consecutive sleep sessions (Carter et al., 2002). This is further elaborated in Chapter 4, section 4.5.

2.3.1.11 Risk of accidents

Only one study was found that evaluated the association between sleep and duration of wakefulness and childhood unintentional injury (Valent, Brusaferro and Barbone, 2001).

Two hundred and ninety-two injured children who attended the Children's Emergency Centre in Udine, Italy, or their parents were interviewed following a structured questionnaire. The sleeping time and wakefulness of the child was assessed retrospectively for each of the 48 hours before injury. For each child, the authors compared the 24 hours immediately before the injury (hours 1–24; case period) with hours 25–48 (control period).

Overall, more children had longer hours of sleep during the control period than during the case period. A direct association between injury risk and sleeping less than 10 hours was found among boys (RR: 2.33; 95% CI: 1.07–5.09) but not among girls (RR: 1.00; 95% CI: .29–3.45). The study also found a direct association between injury occurring between 16.00 and midnight, and being awake for at least 8 hours before injury occurred (both sexes, RR: 4.00; 95% CI: 1.13–14.17). Sleeping less than 10 hours a day was associated with an 86% increase in injury risk. A significantly increased risk did not emerge in all subgroups of patients but it was evident among children aged 3–5 years, boys in particular. A fourfold increase in injury risk was also associated with being awake for at least 8 hours among males only. These findings demonstrated that inadequate sleep duration and lack of daytime naps are transient exposures that may increase the risk of injury among children. Results of a study on sleep disturbance and injury risk in young children show inadequate sleep duration and lack of daytime naps. A lack of daytime naps means transient exposures that may increase the risk of injury among children. Among children (boys in particular) aged 3–5 years, sleeping less than 10 hours a day was associated with an 86% increase in injury risk. A fourfold increase in injury risk was also associated with being awake for at least 8 hours.

Daytime sleepiness in children is often manifested by externalizing behaviours noted by parents or teachers, such as increased activity levels, aggression, impulsivity, as well as by poor concentration, instantiation irritability and moodiness (Fallone, Owens and Deane, 2002).

Analysing attendance at school, data show that accidents took place at school (25.6%) and at home (22.0%), and statistics show that there is a highly significant greater total accident rate among boys than among girls. The most frequent injuries happening at school are fractures and dislocation of joints, head injuries being more common among school injuries compared with spare-time injuries. Most injuries

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occurred when children were in sports areas and it is noteworthy that 25% of all injuries were caused through intentional violence by other pupils.

2.3.1.12 Use of sleeping pills

Several studies demonstrated that the use of sleeping pills is common among children and that paediatricians are prone to prescribe these medications. Twenty-five percent of firstborn infants had been given "sedatives" by 18 months (Ounsted and Hendrick, 1977). A research study into parental reports of 11 000 preschool children showed that 12% took psychoactive drugs, most commonly for sleep: 39% daily and 60% intermittently for 1–2 years (Kopferschmitt et al., 1992). Another study (Trott et al., 1995) revealed that 35% of prescriptions for children less than a year old were for sleep disturbances and that sleep disturbances were also the most common reason for prescribing medications to preschool children (23%). Two French surveys on adolescents showed that 10–12% of the respondents reported use of prescription or over-the-counter drugs for sleep disturbances (Patois, Valatz and Alperovitch, 1993; Ledoux, Choquet and Manfredi, 1994). Recently it has been reported that of 671 community-based United States paediatricians, 75% had recommended over-the-counter and 50% prescription medicines for insomnia during the past 6 months (Owens, Rosen and Mindell, 2003). In addition, an Italian survey showed that pharmacological treatment for sleep problems was prescribed during the past 6 months by 58.54% of paediatricians and by 61.21% of child neuropsychiatrists (Bruni et al., 2004).

2.3.2 BASIC INDIVIDUAL FACTORS: GENDER AND AGE

Gender shows itself to be an important predictor of disturbed sleep in virtually all epidemiological studies (Karacan et al., 1976; Bixler, Kales and Soldatos, 1979; Ancoli-Israel and Roth, 1999; Leger et al., 2000; Sateia et al., 2000). On the other hand, there does not seem to be much of a difference in polysomnographical parameters between males and females, except for the former losing SWS with increasing age and having slightly reduced sleep efficiency also with increasing age (Williams, Karacan and Hursch, 1974; Hume, Van and Watson, 1998). Ehlers and Kupfer (1997) timed the start of differences between genders to between 20 and 40 years. Spectral analysis also indicates slightly larger amounts of low frequency activity in females (Dijk, Beersma and Bloem, 1989; Dijk, Beersma and Van den Hofdakker, 1989). In addition, men seem to run a higher risk of morbidity and mortality related to sleep problems than women (Nilsson et al. 2001). The inconsistency between polysomnography and subjective measures has not been resolved but it may be important that most polysomnographical studies have controlled for anxiety and depression. Thus, it is conceivable that the higher level of subjective complaints in women reflects a higher prevalence of anxiety. The latter is a speculation, however. A confounding factor in gender comparisons is that phases in female biological cycles are also usually controlled for in polysomnographical studies, meaning that potential effects of, for example, menstruation, may not receive their proper weight. A recent review has gone through the literature in this area (Moline et al., 2003). It found that the luteal phase of the menstrual cycle is associated with subjective sleep problems, but polysomnographical studies have not supported this. Pregnancy affects sleep negatively as early as in the first trimester and the effects mainly involve awakenings and difficulties getting back to sleep. Napping is a frequent coping method. The post-partum period is often associated with severe sleep disruption, mainly due to feeding and comforting the infant. There seems to be some relation between sleep disruption and post-partum mood, but nothing is known about the

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causal relations. Menopause seems to involve disrupted sleep in relation to hot flushes, depression/anxiety and sleep-disordered breathing. Oestrogen is associated with improved sleep quality but it is not clear whether the effects are due to a reduction of hot flushes. Oestrogen also improves sleep-disordered breathing.

With respect to background factors, age is an established predictor of disturbed sleep (Karacan et al., 1976; Bixler, Kales and Soldatos, 1979; Ancoli-Israel and Roth, 1999; Ribet and Derriennic, 1999; Leger et al., 2000; Sateia et al., 2000). Interestingly, however, older age may be related to a lower risk of impaired awakening (Åkerstedt et al., 2002c), that is, in this study it was easier to wake up and one felt better rested with increasing age, while at the same time sleep quality was lower. The increased risk of disturbed sleep is consistent with the increasingly strong interference of the circadian morning upswing of the metabolism with increasing age (Dijk and Duffy, 1999). Thus sleep maintenance is impaired and when sleep is interrupted "spontaneously", the awakening is, by definition, easily accomplished and will be lacking in inertia. This ease of awakening may be interpreted as "being well-rested", and obviously the need for sleep is not great enough to prevent an effortless transition into wakefulness.

In addition, sleep homeostasis seems to be weakened with age in the sense that sleep becomes more fragmented and SWS or power density in the delta bands decrease (Williams, Karacan and Hursch, 1974; Bliwise, 1993; Dijk et al., 1999). As mentioned above, the effects are more pronounced in males, a fact that may be linked to reduced levels of growth hormone and testosterone.

2.3.3 PERSONS EXPOSED TO STRESSORS AS A RISK GROUP

A number of epidemiological studies point to a strong link between stress and sleep (Åkerstedt, 1987; Urponen et al., 1988; Ancoli-Israel and Roth, 1999). In fact, stress is considered the primary cause of persistent psychophysiological insomnia (Morin, Rodrigue and Ivers, 2003). That stress can affect proper sleep seems obvious, but Vgontzas et al. (2001) at Pennsylvania State University College of Medicine have found another reason why middle-aged men may be losing sleep. It is not just because of what they worry about; rather, it is due to "increased vulnerability of sleep to stress hormones".

As men age, it appears they become more sensitive to the stimulating effects of corticotropin-releasing hormones (CRH). When both young and middle-aged men were administered CRH, the older men remained awake longer and slept less deeply. (People who don't get enough of this "slow-wave" sleep may be more prone to depression.)

The increased prevalence of insomnia in middle age may, in fact, be the result of deteriorating sleep mechanisms associated with increased sensitivity to arousal-producing stress hormones, such as CRH and cortisol. In another study, the researchers compared patients with insomnia to those without sleep disturbances. They found that "insomniacs with the highest degree of sleep disturbance secreted the highest amount of cortisol, particularly in the evening and night-time hours", suggesting that chronic insomnia is a disorder of sustained hyperarousal of the body's stress response system. Also, recent epidemiological studies have shown a connection between disturbed sleep and later occurrence of stress-related disorders such as cardiovascular diseases (Parish and Shepard, 1990; Nilsson et al., 2001;

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Leineweber et al., 2003) and diabetes type II (Nilsson et al., 2002). The mechanism has not been identified but both lipid and glucose metabolisms are impaired in relation to experimentally reduced sleep (Åkerstedt and Nilsson, 2003). Burnout is another result of long-term stress and a growing health problem in many industrialized countries (Weber and Jaekel-Reinhard, 2000). In Sweden, burnout is thought to account for most of the doubling of long-term sickness absence since the mid-1990s (RFV, 2003). The characteristic clinical symptoms of the condition are excessive and persistent fatigue, emotional distress and cognitive dysfunction (Kushnir and Melamed, 1992; Melamed, Kushnir and Sharom, 1992). Self-reports of disturbed sleep are pronounced in subjects scoring high on burnout (Melamed et al., 1999; Grossi et al., 2003). Since shortened and fragmented sleep is related to daytime sleepiness and impaired cognitive performance (Bonnet, 1985, 1986a, 1986b; Dinges et al., 1997; Gillberg and Åkerstedt, 1998; Åkerstedt, 1990), disturbed sleep might provide an important link between the state of chronic stress and the complaints of fatigue and cognitive dysfunction seen in burnout.

Partinen, Eskelinen and Tuomi (1984) investigated several occupational groups and found disturbed sleep to be most common among manual workers and much less so among physicians or managing directors. Geroldi et al. (1996) found in a retrospective study of older individuals (above the age of 75) that former white-collar workers reported better sleep than blue-collar workers. Kupperman et al. (1995) reported fewer sleep problems in subjects satisfied with work.

In what seems to be the most detailed study so far, Ribet and Derriennic (1999) studied more than 21 000 subjects in France, using a sleep disturbance index and logistic regression analysis. They found that shift work, a long working week, exposure to vibrations, and "having to hurry" appeared to be the main risk factors, controlling for age and gender. Disturbed sleep was more frequent in women (Karacan et al., 1976; Bixler, Kales and Soldatos, 1979; Ancoli-Israel and Roth, 1999) and in higher age groups.

The particular stressor linked to disturbed sleep may be linked to pressure of work (Urponen et al., 1988; Ancoli-Israel and Roth, 1999; Ribet and Derriennic, 1999; Åkerstedt et al., 2002b). The demands of work are a classical work stress factor and, when combined with low decision latitude, a relation has been shown to cardiovascular diseases (Theorell et al., 1998) and absenteeism (North et al., 1996). Interestingly, when "persistent thoughts about work" was added to the regression in the study by Åkerstedt et al. (2002b) this variable took over part of the role of work demands as a predictor. This suggests that it may not be work demands per se that are important, but rather their effect on unwinding after work. In two studies it has been demonstrated that even moderate worries about being woken during the night or having a negative feeling about the next day will affect sleep negatively, mainly reducing SWS (Torsvall and Åkerstedt, 1988; Kecklund and Åkerstedt, 1997). On the other hand, there is very little data to connect real life stress with polysomnographical indicators of disturbed sleep. Most studies have used rather innocuous and artificial stressors in a laboratory environment. Field studies of stress are virtually lacking, with some exceptions (Hall et al., 2000).

A lack of social support at work is a risk indicator for disturbed sleep (Åkerstedt et al., 2002b). Few previous data of this type have been found, but poor (general) social support has been associated with sleep complaints in Vietnam veterans (Fabsitz, Sholinsky and Goldberg, 1997). On the other hand, there are several studies indicating a close connection with poor social support for, for example,

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cardiovascular diseases (Arnetz et al., 1986) or muscle pain (Ahlberg-Hultén, Theorell and Sigala, 1995).

Interestingly, the metabolic changes seen after sleep curtailment in normal sleepers or in insomniacs and sleep apnoeics are similar to those seen in connection with stress. That is, lipid and glucose metabolisms are increased, as are cortisol levels (Spiegel, Leproult and van Cauter, 1999; Vgontzas et al., 2000, 2001). Together with the prospective links to stress-related diseases such as diabetes type II, to cardiovascular diseases as discussed above and with mortality (Kripke et al., 1979, 2002; Åkerstedt et al., 2002a; Dew et al., 2003), the findings could suggest that disturbed sleep may be an important mediator in the development of stress-related diseases.

2.3.4 SHIFT WORK AS A RISK FACTOR FOR SLEEP DISTURBANCE AND HEALTH EFFECTS

The dominating health problem reported by shift workers is disturbed sleep and wakefulness. At least three quarters of the shift working population is affected (Åkerstedt, 1988). When comparing individuals with a very negative attitude to shift work with those with a very positive one, the strongest discriminator seems to be the ability to obtain sufficient quality of sleep during the daytime (Axelsson et al., 2004). EEG studies of rotating shift workers and similar groups have shown that day sleep is 1–4 hours shorter than night sleep (Foret and Lantin, 1972; Foret and Benoit, 1974; Matsumoto, 1978; Tilley, Wilkinson and Drud, 1981; Torsvall et al., 1989; Mitler et al., 1997). The shorter time is due to the fact that sleep is terminated after only 4–6 hours without the individual being able to return to sleep. The sleep loss is primarily taken out of stage 2 sleep and stage REM sleep (dream sleep). Stages 3 and 4 ("deep" sleep) do not seem to be affected. Furthermore, the time taken to fall asleep (sleep latency) is usually shorter. Night sleep before a morning shift is also reduced but the termination is through artificial means and the awakening usually difficult and unpleasant (Dahlgren, 1981a; Tilley et al., 1982; Åkerstedt, Kecklund and Knutsson, 1991; Kecklund, 1996).

Interestingly, day sleep does not seem to improve much across a series of night shifts (Foret and Benoit, 1978; Dahlgren, 1981b). It appears, however, that night workers sleep slightly better (longer) than rotating workers on the night shift (Kripke, Cook and Lewis, 1971; Bryden and Holdstock, 1973; Tepas et al., 1981). The long-term effects of shift work on sleep are rather poorly understood. However, Dumont, Montplaisir and Infante-Rivard (1988) found that the amount of sleep/wake and related disturbances in present day workers were positively related to their previous experience of night work. Guilleminault et al. (1982a) found an over-representation of former shift workers with different clinical sleep/wake disturbances appearing at a sleep clinic. Recently, we have shown that in pairs of twins with different night work exposure, the exposed twin reports somewhat deteriorated sleep quality and health after retirement (Ingre and Åkerstedt, 2004).

The main reason for short daytime sleep is the influence exerted by the circadian rhythm. The more sleep is postponed from the evening towards noon next day, the more truncated it becomes and when noon is reached the trend reverts (Foret and Lantin, 1972; Åkerstedt and Gillberg, 1981). Thus, sleep during the morning hours is strongly interfered with, despite the sizeable sleep loss that, logically, should enhance the ability to maintain sleep (Czeisler et al., 1980). Also, homeostatic influences control sleep. For example, the expected 4–5 hours of daytime sleep, after a

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night spent awake, will be reduced to 2 hours if a normal night's sleep precedes it and to 3.5 hours if a 2-hour nap is allowed (Åkerstedt and Gillberg, 1986). Thus, the time of sleep termination depends on the balance between the circadian and homeostatic influences. The circadian homeostatic regulation of sleep has also been demonstrated in great detail in studies of forced or spontaneous desynchronization under conditions of temporal isolation and ad lib sleeping hours (Czeisler et al., 1980; Dijk and Czeisler, 1995).

2.3.4.1 Alertness, performance and safety

Night-oriented shift workers complain as much of fatigue and sleepiness as they do about disturbed sleep (Åkerstedt, 1988). The sleepiness is particularly severe on the night shift, hardly appears at all on the afternoon shift and is intermediate on the morning shift. The maximum is reached towards the early morning (05.00–07.00). Frequently, incidents of falling asleep occur during the night shift (Prokop and Prokop, 1955; Kogi and Ohta, 1975; Coleman and Dement, 1986). At least two thirds of the respondents report that they have experienced involuntary sleep during night work.

Ambulatory EEG recordings verify that incidents of actual sleep occur during night work in, for example, process operators (Torsvall et al., 1989). Other groups, such as train drivers or truck drivers show clear signs of incidents of falling asleep while driving at night (Caille and Bassano, 1977; Torsvall and Åkerstedt, 1987; Kecklund and Åkerstedt, 1993). This occurs towards the second half of the night and appears as repeated bursts of alpha and theta EEG activity, together with closed eyes and slow undulating eye movements. As a rule the bursts are short (1–15 seconds) but frequent, and seem to reflect lapses in the effort to fend off sleep. Approximately a quarter of the subjects recorded show the EEG/EOG patterns of fighting with sleep. This is clearly a larger proportion than what is found in the subjective reports of episodes of falling asleep.

As may be expected, sleepiness on the night shift is reflected in performance. One of the classics in this area is the study by Bjerner, Holm and Swensson (1955) who showed that errors in meter readings over a period of 20 years in a gas works had a pronounced peak on the night shift. There was also a secondary peak during the afternoons. Similarly, Brown (1949) demonstrated that telephone operators connected calls considerably slower at night. Hildebrandt, Rohmert and Rutenfranz (1974) found that train drivers failed to operate their alerting safety device more often at night than during the day. Most other studies of performance have used laboratory type tests and demonstrated, for example, reduced reaction time or poorer mental arithmetic on the night shift (Tepas et al., 1981; Tilley et al., 1982). Flight simulation studies have furthermore shown that the ability to "fly" a simulator (Klein, Bruner and Holtman, 1970), or to carry out a performance test (Dawson and Reid, 1997) at night may decrease to a level corresponding to that after moderate alcohol consumption (>0.05% blood alcohol) Interestingly, Wilkinson et al. (1989) demonstrated that reaction time performance on the night shift (nurses) was better in permanent than rotating shift workers.

If sleepiness is severe enough, interaction with the environment will cease and if this coincides with a critical need for action an accident may ensue. Such potential performance lapses due to night work sleepiness were seen in several of the train drivers discussed earlier (Torsvall and Åkerstedt, 1987). The transport area is where most of the available accident data on night shift sleepiness has been obtained (Lauber and Kayten, 1988). Thus, Harris (1977) and Hamelin (1987) demonstrated that single vehicle accidents have by far the greatest probability of occurring at night.

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So do fatigue-related accidents (Reyner and Horne, 1995) but also most other types of accidents, for example head-on collisions and rear-end collisions (Åkerstedt, Kecklund and Horte, 2001). The National Transportation Safety Board ranks fatigue as one of the major causes of heavy vehicle accidents (NTSB, 1995).

For conventional industrial operations very little relevant data is available but fatal work accidents show a higher risk in shift workers (Åkerstedt et al., 2002a) and accidents in the automotive industry may exhibit night shift effects (Smith, Folkard and Poole, 1994). An interesting analysis has been put forward by the Association of Professional Sleep Societies' Committee on Catastrophes, Sleep and Public Policy (Mitler et al., 1988). Their consensus report notes that the nuclear plant meltdown at Chernobyl occurred at 01.35 and was due to human error (apparently related to work scheduling). Similarly, the Three Mile Island reactor accident occurred between 04.00 and 06.00 and was due not only to the stuck valve that caused a loss of coolant water but, more importantly, to the failure to recognize this event, leading to the near meltdown of the reactor. Similar incidents, although with the ultimate stage being prevented, occurred in 1985 at the Davis Besse reactor in Ohio and at the Rancho Seco reactor in California. Finally, the committee also states that the NASA Challenger space shuttle disaster stemmed from errors in judgement made in the early morning hours by people who had had insufficient sleep (through partial night work) for days prior to the launch. Still, there is very limited support for the notion that shift work outside the transport area actually carries a higher overall accident risk.

As with sleep, the two main factors behind sleepiness and performance impairment are circadian and homeostatic factors. Their effects may be difficult to separate in field studies but are clearly discernible in laboratory sleep deprivation studies (Fröberg et al., 1975) as well as in studies of forced desynchronization (Dijk, Duffy and Czeisler, 1992). Alertness falls rapidly after awakening but gradually levels out as wakefulness is extended. The circadian influence appears as a sine-shaped superimposition upon this exponential fall in alertness. Space does not permit a discussion of the derivation of these functions, but the reader is referred to Folkard and Åkerstedt (1991) in which the "three-process model of alertness regulation" is described. This model has been turned into computer software for predicting alertness and performance and to some extent accident risk.

2.3.4.2 Health effects

Gastrointestinal complaints are more common among night shift workers than among day workers. A review of a number of reports covering 34 047 persons with day or shift work found that ulcers occurred in 0.3–0.7% of day workers, in 5% of people with morning and afternoon shifts, in 2.515% of persons with rotating shift systems with night shifts, and in 10–30% of ex-shift workers (Angersbach et al., 1980). Several other studies have come to similar conclusions (Thiis-Evensen, 1958; Segawa et al., 1987; Harrington, 1994). Other gastrointestinal disorders, including gastritis, duodenitis and dysfunction of the digestive system are more common in shift workers than in day workers (Koller, 1983).

The pathophysiologic mechanism underlying gastrointestinal disease in shift workers is unclear, but one possible explanation is that intestinal enzymes and intestinal mobility are not synchronized with the sleep/wake pattern. Intestinal enzymes are secreted according to the circadian rhythm, and shift workers' intake of food is irregular compared with intestinal function (Suda and Saito, 1979; Smith, Colligan and Tasto, 1982). A high nightly intake of food may be related to increased lipid levels (Lennernäs, Åkerstedt and Hambraeus, 1994) and eating at the circadian low point

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may be associated with altered metabolic responses (Hampton et al., 1996). In addition, reduced sleep affects lipid and glucose metabolism (Spiegel, Leproult and van Cauter, 1999).

A number of studies have reported a higher incidence of cardiovascular disease, especially coronary heart disease, in male shift workers than in men who work days (for review see Kristensen, 1989; Boggild and Knutsson, 1999). A study of 504 paper mill workers followed for 15 years found a dose–response relationship between years of shift work and incidence of coronary heart disease in the exposure interval 1–20 years of shift work (Knutsson et al., 1986). A study of 79 000 female nurses in the United States gave similar results (Kawachi et al., 1995) as did a study with more than 1 million Danish men (Tüchsen, 1993) and a cohort of Finnish workers (Tenkanen et al.,1997). As with gastrointestinal disease, a high prevalence of smoking among shift workers might contribute to the increased risk of coronary heart disease, but smoking alone cannot explain the observed excess risk (Knutsson, 1989b). Another possibility is disturbances of metabolic parameters such as lipids and glucose for which there is some support as discussed above.

Only a few studies have addressed the issue of pregnancy outcome in shift workers. In one study of laboratory employees, shift work during pregnancy was related to a significantly increased risk of miscarriage (RR: 3.2) (Axelsson, Lutz and Rylander, 1984). Another study of hospital employees also demonstrated an increased risk of miscarriage (RR: 1.44, 95% CI: 0.83–2.51) (Axelsson and Rylander, 1989). Lower birth weight in infants of mothers who worked irregular hours has been reported (Axelsson and Rylander, 1989; Nurminen, 1989). No teratogenic risk associated with shift work was reported (Nurminen, 1989).

The mortality of shift and day workers was researched by Taylor and Pocock (1972), who studied 8603 male manual workers in England and Wales between 1956 and 1968. Day, shift, and ex-shift workers were compared with national figures. The Standardized Mortality Ratio (SMR) can be calculated from observed and expected deaths reported in the paper. SMRs for deaths from all causes were 97, 101 and 119 for day, shift, and ex-shift workers respectively. Although the figures might indicate an increasing trend, the differences were not statistically significant. However, the reported SMR close to 100 is remarkable because the reference population was the general male population. Most mortality studies concerned with occupational cohorts reveal SMRs lower than 100, implying a healthy workers' effect (Harrington, 1978). The same study showed a significantly increased incidence of neoplastic disease in shift workers (SMR 116). A Danish study of 6000 shift workers failed to demonstrate any excess mortality in shift workers (Boggild et al., 1999). Not much evidence exists on the connection between shift work and cancer. The mortality study by Taylor and Pocock (1972) reported an increased incidence of neoplasms in shift workers compared with the general population. A recent Danish case–control study reported an increased risk of breast cancer among 30–45-year-old women who worked mainly nights (Hansen, 2001). Among 75 000 nurses those with more than 15 years of night work showed an increased risk of colorectal cancer (Schernhammer et al., 2003). If the results are confirmed, a possible mechanism may be the low levels of the hormone melatonin, due to light exposure during the night with a subsequent suppression of melatonin.

Very few studies are available but Koller, Kundi and Cervinka (1978) found a prevalence of endocrine and metabolic disease of 3.5% in shift workers and 1.5% in day workers. Kawachi et al. (1995) found in a prospective study of shift work-

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ers that the age-standardized prevalence was 5.6% at 15 years of shift work experience compared with 3.5% for no exposure. Nagaya et al. (2002) found that markers of insulin resistance were more frequent in shift workers above the age of 50 than in day workers. Other indicators, such as body mass index, glucose levels and so forth, give a rather inconclusive impression as indicated in a review by Boggild and Knutsson (1999).

Another contributing factor to gastrointestinal diseases might be the association between shift work and smoking. A number of studies have reported that smoking is more common among shift workers (Angersbach et al., 1980; Knutsson, Åkerstedt and Jonsson, 1988). Studies concerned with alcohol consumption comparing day workers and shift workers have produced conflicting results (Smith, Colligan and Tasto, 1982; Knutsson, 1989a; Romon, Nuttens and Fievet, 1992), probably due to local cultural habits. One study, which used g-glutamyltransferase as a marker of alcohol intake, did not indicate that the shift workers had a higher intake of alcohol than the day workers (Knutsson, 1989a).

Sickness absence is often used as a measure of occupational health risks. However, sickness leave is influenced by many irrelevant factors and cannot be considered as a reliable measure of true morbidity. Studies on sickness absence in day and shift workers have revealed conflicting results and there is no evidence that shift workers have more sickness absence than day workers (for review, see Harrington, 1978).

2.3.4.3 Conclusion

Shift work or similar arrangements of work hours clearly affects sleep and alertness and there is a moderate risk of cardiovascular and gastrointestinal disease. Other diseases such as cancer or diabetes may be related to shift work but the evidence is as yet rather weak.

The present review suggests that the risk of disturbed sleep increases with age but there also seems to be a recent stress-related increase in sleep disturbance in young adults. The long-term health consequences are not yet understood.

The relation between gender and disturbed sleep is confusing. Females, as a rule, complain more of sleep problems, but do not exhibit any objective indications of more disturbed sleep, at least not among otherwise healthy women. With increasing age the sleep of males deteriorates whereas that of women is relatively well upheld. Pregnancy, however, is a period of increased risk of disturbed sleep, whereas the menstrual cycle and menopause show less evidence of sleep disturbance. Clearly there is a great need for longitudinal research on gender and sleep and, in particular, on the possible health consequences connected with pregnancy.

Stress due to work or family seems to be one of the major causes of disturbed sleep. The link to the risk of insomnia is well-established, but reduced sleep in itself seems to yield the same physiological changes as stress. This suggests that several of the major civilization diseases in Europe and the United States (diabetes, cardiovascular diseases and burnout) could be mediated via disturbed sleep. This link clearly warrants longitudinal studies with interventions.

Shift workers constitute a group that suffers from disturbed sleep for most of their occupational life. The reason is the interference of work hours with the normal timing of sleep. This leads to an increased risk of accidents, directly due to excessive sleepiness, but also to cardiovascular and gastrointestinal diseases, although it is

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not clear whether the latter effects are sleep related or due to circadian factors – or to a combination. Recent studies also suggest that breast cancer may result from shift work due to the effects of light on melatonin secretion. This still needs verification, however. Future research needs to identify countermeasures, the reasons for large individual differences in tolerance and the possible carcinogenic and other effects.

The conclusions above should be seen against the profound effects of reduced or fragmented sleep on the neuroendocrine (including glucose and lipid regulation) and immune systems as well as the effects on mortality, diabetes and cardiovascular disease.

2.3.5 CONCLUSION

Children, the elderly, pregnant women, people under stress and shift workers are vulnerable to (noise) disturbance of their sleep.

2.4 ACCIDENTS RELATED TO SLEEP QUALITY

As already stated in the earlier section on cardiovascular complications, children with disturbed sleep present cognitive dysfunction and behavioural disturbances, abnormal growth hormone release, increase of diastolic BP and an increased risk of accidents and use of sleeping pills.

Regarding sleep disturbance and accidents in adults, data show that 15–45% of all patients suffering from sleep apnoea, 12–30% of all patients suffering from narcolepsy and 2–8% of all patients suffering from insomnia have at least one accident (in a lifetime) related to sleepiness (statistics from the Stanford Sleep Disorders Clinic).

As already discussed in section 2.3.4, the biggest industrial catastrophes, such as the Three Mile Island, Bhopal, Chernobyl and Exxon Valdez disasters, have occurred during the night shift. The shift schedules, fatigue and sleepiness were cited as major contributing factors to each incident.

The LARES study (Large Analysis and Review of European housing and health Status) is one of the few studies analysing this issue directly. The results show that the likelihood of home accidents is significantly greater when the individual is tired all the time or most the time and there is an association between sleep disturbance and accidents, with 22% of those reporting an accident also reporting having their sleep disturbed during the previous four weeks.

The data available to document the impact of environmental noise on sleep deprivation and accidents are largely inadequate. There is no estimation of relative risk. Further research is needed in order to identify the accident-related burden of diseases attributable to noise during the night-time.

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2.5 ANIMAL STUDIES

As pet owners know, cats sleep (most of the time it seems) and so do dogs. But do fish sleep? And flies? Yes, most animals sleep, and they even show the same phenomena as in humans; from deep sleep, dream sleep to sleep disorders. There are also many differences and weird behaviour, such as sleeping with only one half of the brain at a time (dolphins and ducks).

As Ising points out (Appendix 3), in animal experiments it is possible to assess the complete causal chain from noise exposure via physiological reactions and biological risk factors to morbidity or even mortality. However, a quantitative application of the results to humans is not possible. Instead, the method is useful in studying the pathomechanisms qualitatively. Rechtschaffen and Bergmann (1965) studied sleep deprivation in rats, showing that total sleep deprivation leads to mortality in 16 to 20 days. As the animals in the last stage died from microbial infection, Everson and Toth (2000) proceeded to show early infection of the lymph nodes and other tissues and hypothesized that daily sleep of some amount is necessary to maintain an intact immune system that will prevent bacterial invasion, a view that has been challenged.

Surprisingly, sleep in the common fruit fly – *Drosophila melanogaster* – has many similarities with mammalian sleep, including sleep deprivation leading to impaired performance. Genetic studies in fruit flies (Cirelli et al., 2005) led to mutant flies that can get by on 30% less sleep than their normal counterparts, thanks to a single mutation in one gene. While they sleep 30% less they show no immediate ill effects. The lifespan of the flies is, however, reduced by 30%.

These animal models certainly lead one to believe that sleep is a biological necessity, and tampering with it is dangerous for survival.

As Ising shows (Appendix 3) noise may play a role in this. Under stressful circumstances the death rate of rats is increased when noise levels are increased from "ambient" to L_{eq}=69 dB(A). Are noise and sleep deprivation stressors that both lead to early death? Is the noise effect due to sleep deprivation? A carefully planned study may sort this out. The question still remains, however, as to how far this is relevant to humans.

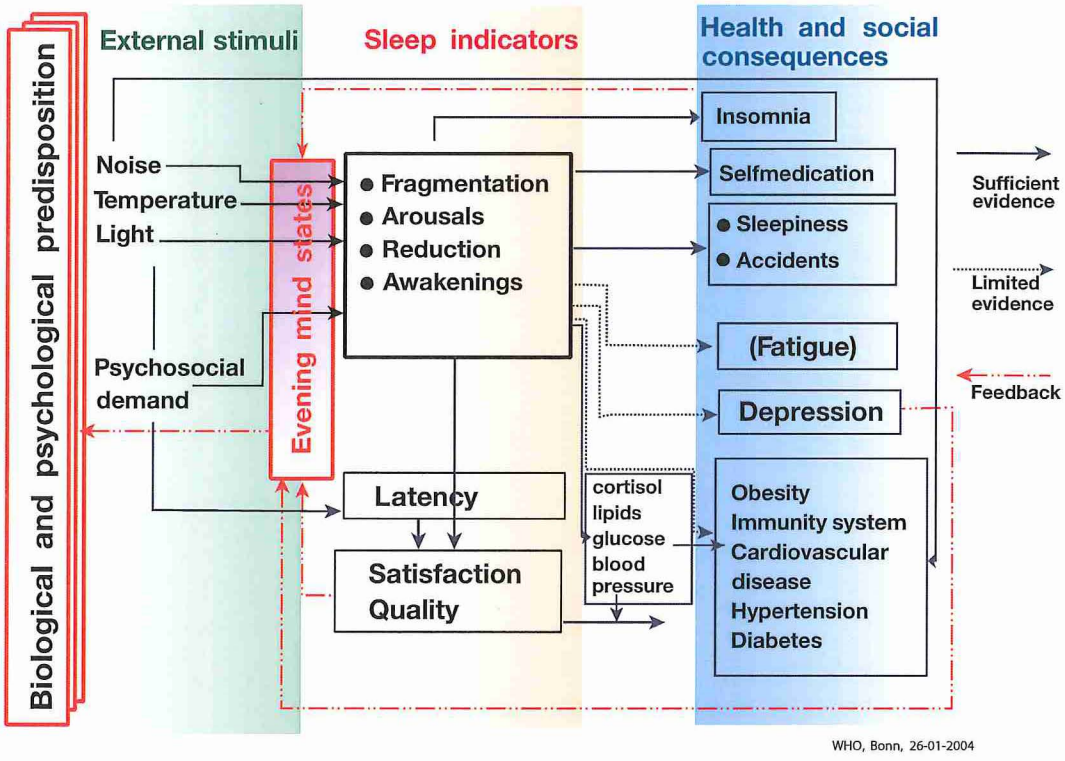
2.6 CONCLUSIONS

From the evidence presented so far it can be deduced that sleep is important for human functioning. Why exactly is less evident, but it is clear that disturbed sleep (either from internal factors or from external factors) leads to or is at least associated with fatigue, lower cognitive performance, depression, viral illness, accidents, diabetes, obesity and cardiovascular diseases. Animal experiments show that sleep deprivation shortens lifespan. The fact that – in comparison– relatively mild effects turn up in human sleep deprivation experiments could be due to the short period (about 10 days in controlled experiments) and the limitation to young and healthy adults. The central position of sleep in human functioning is summarized in Fig. 2.1. In this figure relations with sufficient evidence are indicated with solid lines, while relations for which limited evidence exists are indicated with interrupted lines. Feedback connections are in red and double-dotted.

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The presence of feedback loops in the system is an indication that it may be difficult to prove direct cause–effect relations. One example is the relation between sleep quality and depression. They are strongly associated, but it is uncertain if depression causes bad sleep, or bad sleep causes depression (see also Chapter 4, section 4.8.11). This may also depend on one of the many other factors, so it could be different for different personality types.

Fig.2.1
Expert view on the relations between sleep and health



Impaired sleep is widely considered as a health problem per se, and this chapter has shown that there are many internal and external causes. In the next chapter the relation between noise and sleep quality is further unravelled.

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CHAPTER 3

EFFECTS OF NIGHT-TIME NOISE ON SLEEP

Best travel tip: Never ever forget to pack ear plugs.
(Virginia Jealous, Lonely Planet author)

3.1 SHORT-TERM EFFECTS OF TRANSPORTATION NOISE ON SLEEP WITH SPECIFIC ATTENTION TO MECHANISMS AND POSSIBLE HEALTH IMPACT

3.1.1 INTRODUCTION

In this section reactions to single events are presented. In Chapter 2 normal sleep and sleep disorders are described in medical terms, but here the focus is on the mechanisms underlying the relation between noise and sleep quality. How does a sound penetrate the brain and cause a disruption of sleep?

3.1.2 HOW NOISE INFLUENCES NORMAL SLEEP

Noise can induce changes in the EEG or in autonomic variables that are called arousals or phasic activations. Similar brief episodes of activity also occur without noise in normal sleep, and, more frequently, in sleep that is otherwise disturbed, for example by apnoea. Arousal during sleep is not a uniform concept and has been defined differently by different researchers. Commonly, the occurrence of alpha rhythms is required for EEG arousal. Depending on the additional requirements and on the length of time that the slower cortical rhythms are interrupted, arousals have been called, for instance, micro-arousal, minor arousal, EEG awakening or transient activation phases. EEG awakening requires an interruption of the sleep patterns of at least 15 seconds (half the period) when sleep staging is scored by periods of 30 seconds, but need not be experienced consciously. Because normal REM sleep is a state characterized by cerebral arousal with frequently occurring alpha rhythms, additional criteria are needed to define arousal from REM. The criteria used are increased heart rate, EMG, or irregular respiration. However, since the mechanisms of such autonomic responses appear to be at least partly different from the causal mechanisms of EEG arousal, such definitions seem to make arousal from sleep a heterogeneous concept that may not have simple relationships with noise exposure.

EEG arousals lasting at least 30 seconds have been found to occur as often as 4 times (95% CI: 1–15) per hour during sleep on average, while micro-arousals occurred 21 times (95% CI: 7–56) per hour (Mathur and Douglas, 1995). Since these figures are from a laboratory study, they almost certainly are higher than the figures that hold for the natural situation at home. Sleep pressure decreases the density of micro-arousals (Sforza, Jouny and Ibanez, 2000). While the number of EEG arousals (dur-

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ing sleep stages 1 and 2) increases with age (Mathur and Douglas, 1995; Boselli et al., 1998) possibly only for men (Hume, Van and Watson, 2003), their average length is stable and circa 15 seconds (Boselli et al., 1998). Also the threshold for auditory arousal decreases with age (Zepelin, McDonald and Zammit, 1984; Busby and Pivik, 1985; Busby, Mercier and Pivik, 1994) and towards the end of the night (Basner et al., 2004). Recovery after EEG awakenings takes longer for noise-induced awakenings than for spontaneous awakenings (Basner et al., 2004). The time required for falling asleep again depends on the sound level and especially for loud events this latency is considerably longer than after spontaneous awakenings. Thus, in general, noise-induced EEG awakenings are more disruptive than spontaneous awakenings and therefore will more often be experienced consciously and remembered afterwards. In common situations with aircraft overflight noise at home, (minor) arousals were found in 10.3% of the 64-second intervals without aircraft noise and this percentage was found to be increased by circa 4% up to 14.3% in intervals with an aircraft noise event (Hume, Van and Watson, 2003). Thus, in that particular (exposure) situation, about 1 in 24 aircraft overflights caused a (minor) arousal.

3.1.3 MECHANISMS

Activity in the auditory system up to the brainstem nucleus inferior colliculus occurs within 10 milliseconds after the onset of a sound. This early activity appears to be obligatory and is hardly affected by the state (sleeping or awake). Being asleep or awake does influence later activity. The auditory pathways proceed from the inferior colliculus to the thalamus and from there to the auditory cortex. The state (asleep or awake) affects the activity in the thalamocortical circuits, which occurs after 10–80 milliseconds. In particular, during SWS the transmission of auditory information through the thalamus is suppressed. This is not the case during REM sleep or when awake.

Thus, while in all (sleep) stages, sound activates the auditory system up to the inferior colliculus, the sound-induced activation of higher areas is suppressed in SWS. Therefore, further activation depending on those higher areas (for example, extracting meaning) is not likely to occur as a primary reaction to sound during SWS. For understanding arousal during SWS, it is important that the inferior colliculus and the earlier auditory nucleus of the lateral lemniscus, and also the (dorsal and ventral) cochlear nuclei project to reticular arousal system. Presumably, sound is always capable of arousing the sleeping subject through these connections. The ascending arousal system is heterogeneous and encompasses mono-aminergic, glutamatergic, and cholinergic nuclei that can directly or indirectly activate the thalamus and cortex. An important indirect route is the activation of the basal forebrain, which can activate the cerebral cortex through widespread, mainly cholinergic projections. The activation of the thalamus and cortex is indicated by an increase in EEG rhythm frequency and a reduction of the inhibition in the thalamic sensory relay nuclei. As a consequence of the latter effect, subsequent sound-induced activation may pass the thalamus and may be subject to more elaborate processing than initial sound. It can be speculated that sound in this way also reduces the threshold for somatosensory information that initiates body movements so that more body movements are observed when exposed to sound. The occurrence of habituation of cortical responses suggests an active role played by a part of the brain that blocks or at least limits the impact of the activated ascending pathways.

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The parasympathetic autonomic nervous system seems to be responsible for the bradycardia observed in non-REM sleep and mainly in tonic REM sleep through the increase in vagal activity (Guazzi et al., 1968). The variability of heart rate in REM sleep could be placed under the same control, as vagotomy strongly reduced the heart rate instability (Baust and Bohnert, 1969). During falling asleep, respiration is unstable and alternates between hypo- and hyperventilation episodes. This respiration, called "periodic respiration" (Mosso, 1886), disappears when stable sleep occurs (stage 2). The main hypotheses concerning this periodic ventilation refer to metabolic control and chemoreceptor responses to levels of $PaCO_2$ and PaO_2 (Chapman et al., 1988). In stable non-REM sleep, respiration is regular in amplitude and frequency, although ventilation per minute is lower than during awakening. In REM sleep, respiration appears irregular with sudden variations in amplitude and frequency. This irregularity appears to be not modifiable by metabolic factors and, therefore, it is possibly linked to mechanisms leading to REM expression. The non-habituation of the cardiovascular responses would be explained by the absence of an inhibitory influence on the part of the arousal system that affects the centres regulating the autonomous response.

3.1.4 EEG RESPONSE

The sleep polygraph continuously records EEG activity, eye movement (EOG) and muscle tone (EMG). These data are used to classify sleep into various stages, and to assess times of falling asleep and waking up. Also, sleep variables such as total sleep time and total time spent in SWS (consisting of sleep stages 3 and 4, the stages of deep sleep) and in the REM stage (also called dream or paradoxical sleep) can be assessed on the basis of sleep polygraph recordings. Polygraphic indicators of responses to individual noise events are changes from a deeper to a less deep sleep and EEG awakening. Several field studies (Pearsons, Bennett and Fidell, 1973; Vernet, 1979; Vallet et al., 1983; Hume, Van and Watson, 2003; Basner et al., 2004) have been conducted regarding noise-induced changes in sleep stage and awakening using EEG recordings. Transition from a deep stage of sleep to a shallower sleep stage can be the direct consequence of a nocturnal noise event. Although not perceived by the sleeper, these transitions modify the sleep architecture and may reduce the amount of SWS (Carter, 1996; Basner et al., 2004) and the amount and rhythmicity of REM sleep may be markedly affected (Naitoh, Muzet and Lienhard, 1975; Thiessen, 1988). In addition to their results from a laboratory study, Basner et al. (2004) present results from a field study with valid data for 63 subjects (aged 18–65 years) with 15 556 aircraft noise events included in the final analyses. They established a curve that gives the probability of awakening as a function of L_{Amax} with a model that assumed a background noise level just prior to the aircraft noise event of 27 dB(A). The L_{Amax} threshold for noise-induced awakenings was found to be about 35 dB(A). Above this threshold the probability of noise-induced awakenings increases monotonically up to circa 10% when L_{Amax} = 73 dB(A). This is the extra probability of awakening associated with the aircraft noise event, on top of the probability of awakening spontaneously in a 90 second interval.

Some arousals provoked by noise events are so intense that they induce awakening. Frequent awakening leads to sleep fragmentation and overall sleep disturbance. The noise threshold for awakening is particularly high in deep SWS (stages 3 and 4) while it is much lower in shallower sleep stages (stages 1 and 2). In REM sleep the awakening threshold is variable and depends on the significance of the stimulus. Total

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sleep time can be reduced by both a longer time to fall asleep and premature final awakening. It has been reported that intermittent noises with maximum noise levels of 45 dB(A) and above can increase the time taken to fall asleep by a few to 20 minutes (Öhrström, 1993). In the morning hours, the sleeper can be more easily awakened by ambient noise and has more difficulty going back to sleep because sleep pressure is progressively reduced with time (Rechtschaffen, Hauri and Zeitlin, 1966; Keefe, Johnson and Hunter, 1971).

Terzano et al. (1990, 1993) showed that with increasing intensity of sound pressure level (white noise at 45, 55, 65 and 75 dB(A), white noise induced a remarkable enhancement of cyclic alternating patterns (CAP)/non-REM, characterized by a linear trend from the lowest to the highest intensities, revealed by a significant increase in the CAP rate already at 45 dB(A). Noise decreased mainly SWS, REM and total sleep time, and increased waking after sleep onset, stage 1 non-REM and CAP rate (Terzano et al., 1993). For CAP/non-REM values between 45% and 60%, subjects generally recalled a moderate nocturnal discomfort and values of CAP/non-REM over 60% corresponded to a severe complaint.

This result corroborates previous findings described by Lukas (1972a) who reported that reactions less intense than a sleep stage change correlate better to the noise intensity than awakening reactions.

3.1.5 CARDIOVASCULAR RESPONSE

For sleeping persons, mean heart rate, mean systolic and diastolic BP and variability in heart rate are usually assessed. Indicators of responses to individual noise events are instantaneous changes in (variability of) heart rate and changes in systolic BP. Several field studies (Carter et al., 1994) have been conducted regarding momentary change in heart rate. Intermittent noise during sleep has been found to induce a biphasic cardiac response and a transient constriction of peripheral vessels together with a short phasic activation in the EEG, while no other behavioural effect can be seen (Muzet and Ehrhart, 1978). This biphasic cardiac response starts with an increase in heart rate, probably due to a phasic inhibition of the parasympathetic cardio-inhibitory centre, followed by a compensatory decrease due to a phasic decrease in orthosympathetic activity (Keefe, Johnson and Hunter, 1971; Muzet and Ehrhart, 1980). The vasoconstrictive response was reported to be due to the sympathetic peripheral stimulation provoked by the auditory reflex (Kryter and Poza, 1980). More recently, Carter et al. (2002) have shown that beat-by-beat BP changes can be induced by suddenly occurring noises. Although habituation in some effect parameters can occur in a few days or weeks, this habituation is not complete and the measured modifications of the cardiovascular functions remain unchanged over long periods of exposure time (Muzet and Ehrhart, 1980; Vallet et al., 1983). Most striking is that none of the cardiovascular responses show habituation to noise after a prolonged exposure, while subjective habituation occurs within a few days. In people that are used to sleep in a noisy surrounding, noise-induced changes in heart rate are dependent on the maximum sound level of a noise event, but not on the EEG sleep stage).

3.1.6 BODY MOVEMENT

Motility is the term used for accelerations of the body or body parts during movement. It is measured with actimeters, usually worn on the wrist in field research and

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the laboratory. Brink, Müller and Schierz (2006) describe a more sophisticated method which is based on the bed being placed on accelerometers. This allows the tracking of whole body movements.

Motility is related to many variables of sleep and health (Reyner, 1995; Reyner et al., 1995; Passchier-Vermeer et al., 2002). Clinical research shows that the sleep/wake cycle (assessed by polysomnography, EEG, EOG, EMG) passes through the 24-hour period synchronously with the rest/activity cycle (assessed by actimetry) (Borbely et al., 1981). A number of investigations have compared the results of polysomnographic recordings (number of EEG-awakenings during sleep period, duration of sleep period, sleep onset time, wake-up time) with results of actimetry. The correlation between actimetrically assessed duration of sleep period, sleep onset time, wake-up time and similar variables assessed with polysomnography was found to be very high (correlation coefficients between individual test results in the order of 0.8–0.9). Measures of instantaneous motility are the probability of motility and the probability of onset of motility in a fixed time interval, for example a 15-, 30- or 60-second interval. Increased instantaneous motility during sleep is considered to be a sensitive behavioural marker of arousal, but the relation with arousal is not simple. Also other factors, such as the need to relieve the pressure on body parts for better blood circulation, cause motility, and spontaneously occurring arousals are part of the normal sleep process. The noise-induced probability of (onset of) motility is the difference between the probability of (onset of) motility during noise events minus the probability in the absence of noise.

Onset of motility and minor arousal found on the basis of EEG recordings are highly correlated. In the United Kingdom sleep disturbance study, Ollerhead et al. (1992) found for their study population that during sleep there is on average an EEG (minor) arousal in 40% of the 30-second intervals with onset of motility. Unfortunately, it is unknown whether this 40% is also valid for noise-induced awakenings. In 12% of the 30-second intervals with an EEG (minor) arousal, motility does not occur. Several field studies (Horne et al., 1994; Fidell et al., 1998, 2000; Flindell, Bullmore and Robertson, 2000; Griefahn et al., 2000; Passchier-Vermeer et al., 2002, 2004) have been conducted regarding noise-induced instantaneous motility. For this effect, relationships have been established with SEL or L_{Amax}, for aircraft noise only. In Passchier-Vermeer et al. (2002) relationships between noise-induced increase in motility or noise-induced increase in onset of motility in the 15-second interval with the maximum noise level of an overflight, and L_{Amax} or SEL have been approximated by quadratic functions (see, for instance, Fig. 3.2). It may be noted that the threshold of motility (L_{Amax} = 32 dB(A)) is in the same range as the threshold found by Basner et al. (2004) for EEG awakenings, with a definition that also encompassed transitions to steep stage 1 (L_{Amax} = 35 dB(A)). The probability of motility at 70 dB(A) of about 0.07 is lower than the probability of noise-induced EEG awakening at L_{Amax} = 73 dB(A) of about 0.10. There is no a priori reason to expect the above threshold probabilities to be the same for these two measures of sleep disturbance, but, taking into account that motility is assessed for shorter intervals (15 seconds vs. 90 seconds), the differences in probabilities above threshold appear to be limited.

One of the variables influencing the relationships between noise-induced instantaneous motility and L_{Amax} or SEL, is long-term aircraft noise exposure during sleep. The probability of instantaneous aircraft noise-induced motility is lower when the long-term exposure is higher. This may be partly due to the higher base rate motility in quiet intervals in higher long-term exposure, which is used as a reference for

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the instantaneous noise-induced motility. Other factors influencing the relationships between instantaneous motility and L_{Amax} or SEL are the point of time in the night, and time since sleep onset. For example, after 7 hours of sleep, noise-induced motility is about 1.3 larger than in the first hour of sleep. Age has only a slight effect on noise-induced motility, with younger and older people showing a lower motility response than persons in the age range of 40–50 years.

3.1.7 BEHAVIOURAL AWAKENING IN ADULTS

Passchier-Vermeer (2003a) published a review of nine studies on awakening by noise. It was found that these studies had different definitions of what constituted an "awakening". In this review, however, all awakening data were collected on "behavioural awakening": these are awakenings that were followed by an action (like pressing a button) from the sleeper. The number of awakenings defined in this manner is much smaller than the number of sleep stage changes which lead to EEG patterns similar to wakefulness.

Data were available for rail traffic noise, ambient (probably road) noise, civil aviation noise and military aviation noise.

The rail traffic noise study is very small (only 20 subject nights), but showed no awakenings. The study states that "there is some evidence, be it very limited, that railway noise events, in the range of SEL_{indoor} considered (up to 80 dB(A)), do not increase [the] probability of awakening".

Ambient noise also showed no effect on the probability of awakening, but as it is uncertain exactly what noise is meant, no firm conclusions could be drawn.

Military aircraft noise showed a very strong effect, but this study is of limited applicability since the few subjects (military) lived near the end of the runway.

For civil aviation noise there were sufficient data to derive a dose-effect relation:

$$\text{Percentage of noise-induced awakenings} = -0.564 + 1.909*10^{-4}*(SEL_{inside})^2 \quad [4],$$

where SELinside is the sound exposure level of an aircraft noise event in the bedroom.

This relation is confined to commercial aircraft noise over the intervals $54<SEL<90$ ($37<L_{Amax}<82$) and the number of events per night $1< N< 10$.

With this relation, it is possible to calculate for an individual L_{night} the expected number of noise-induced behavioural awakenings. This requires all single contributions over the year to this L_{night} to be known. Alternatively (if, for instance, a future situation has to be estimated for which no exact data are available) a worst case scenario can be calculated. Fig. 3.1 represents the results of this worst case approach (converted to L_{night}, see Chapter 1, section 1.3.4), and so gives the maximum number of awakenings n_{max} that may be expected.

$$n_{max}= 0.3504*10^{(L_{night}-35.2)/10} \quad [5].$$

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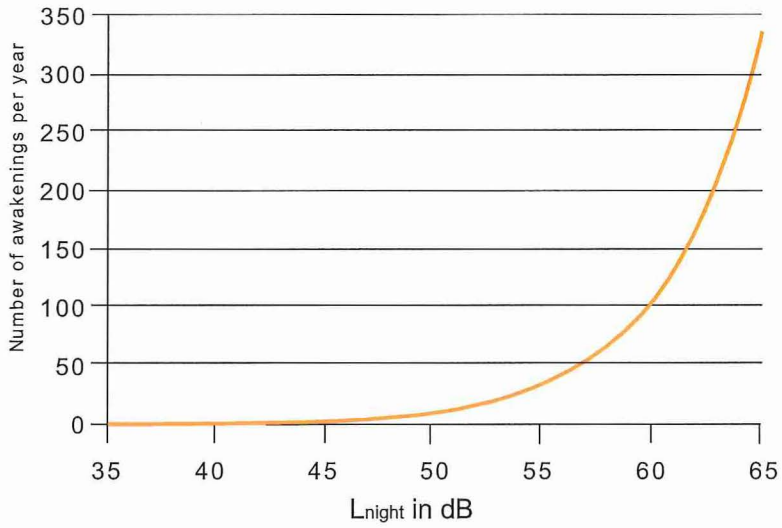


Fig. 3.1
Worst case prediction of noise-induced behavioural awakenings.
L_{night}, converted from inside relation with [3] on page 10.

Source: Miedema, Passchier-Vermeer and Vos, 2003

It can be demonstrated that the number of awakenings reaches a maximum when the SEL_{inside} value is 58.8 dB(A).

It should be noted that, on average, 600 spontaneous awakenings per person are reported per year. This also explains why so many more awakenings are reported than can be attributed directly to aircraft noise. At 55 L_{night}, nearly 100 overflights per night with SEL_{inside} = 58.8, or 1 every 5 minutes are possible. It is, therefore, very likely that an overflight coincides with a spontaneous awakening.

3.1.8 DOSE–EFFECT RELATIONS FOR BODY MOVEMENTS DURING SLEEP

In Passchier-Vermeer et al. (2002) motility is registered in 15-second intervals. A distinction is made between two variables:

- the *presence of motility* in the interval (indicated by m) and
- the onset *of motility*, meaning the presence of motility when there was no motility in the preceding interval (indicated by k).

Relations between a noise-induced increase in motility (m) or a noise-induced increase in the onset of motility (k) in the 15-second interval with the maximum sound level of an overflight, and $L_{Amax,inside}$ or SEL_{inside} have been approximated by quadratic functions with the following format:

$$m = b^*(L_{Amax,inside} - a) + c^*(L_{Amax,inside} - a)^2 \quad [6].$$

The coefficients a, b and c are given in Table 3.1. The value of a is the value below which m or k is zero. Fig. 3.2 shows the relationship between m and $L_{Amax,inside}$ together with the 95% confidence interval. Relations apply to $L_{Amax,inside}$ and SEL_{inside} values of at most 70 and 80 dB(A), respectively.

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Table 3.1.
Coefficients of the quadratic equation (formula [6]) of m and k as a function of $L_{Amax,inside}$ or SEL_{inside} for the 15-second interval in which an indoor maximum sound level of an aircraft noise event occurs. The equations are applicable in the $L_{Amax,inside}$ range from a up to 70 dB(A), or SEL_{inside} range from a up to 80 dB(A). Below a, m and k are zero.

	(Aircraft) noise-induced increase of probability of motility (m)	(Aircraft) noise-induced increase of probability of onset of motility (k)
range	$32 < L_{Amax,inside} < 70$ dB(A) (see Fig. 3.2)	$32 < L_{Amax,inside} < 70$ dB(A)
a	32	32
b	0.000633	0.000415
c	3.14×10^{-5}	8.84×10^{-6}
range	$38 < SEL_{inside} < 80$ dB(A)	$40 < SEL_{inside} < 80$ dB(A)
a	38	40
b	0,000532	0.000273
c	2.68×10^{-5}	3.57×10^{-6}

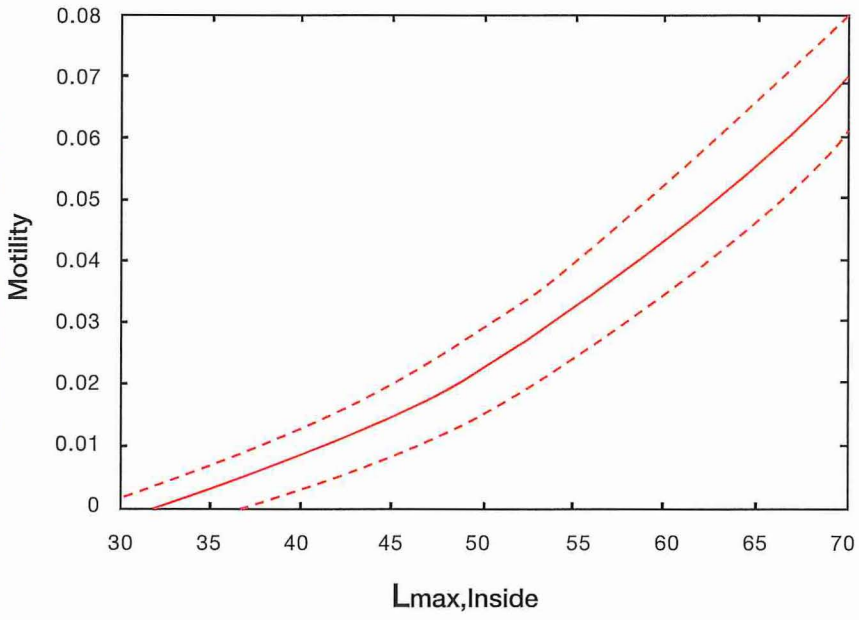


Fig. 3.2
Probability of (aircraft) noise-induced motility (m) at the 15-second interval in which the indoor maximum sound level occurs (solid line) and the 95% confidence interval, as a function of $L_{Amax,inside}$ bedroom

Source: Miedema, Passchier-Vermeer and Vos, 2003.

The study report also gives the upper boundaries for motility, based on the relationship between L_{Amax}, SEL and L_{night} (Fig. 3.3). This figure is mathematically derived from relation [6] as described in Appendix 2.

This area of study is still under development. Miedema, Passchier-Vermeer and Vos (2003) give a detailed account in their study report of the relation between the study used for the data presented here (Passchier-Vermeer et al., 2002), earlier studies like the much quoted Civil Aviation Authority study (Ollerhead et al., 1992; Ollerhead, 1994) and earlier work done in the United States.

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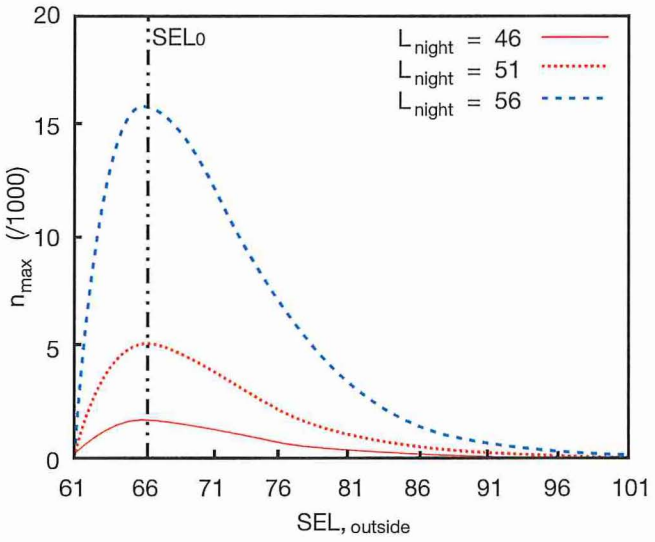


Fig. 3.3
Maximum number of noise-induced motility for three values of L_{night}. Converted from inside relation with [3] on page 10

Source: Miedema, Passchier-Vermeer and Vos, 2003.

3.1.9 INDIVIDUAL SENSITIVITY

Sensitivity to noise may vary greatly from one individual to another. Primary self-evaluation of sensitivity to noise has been used as a factor to evaluate highly sensitive and non-sensitive groups and to compare their reactions to noise exposure during daytime and night-time (Di Nisi et al., 1990). In this study, self-declared highly sensitive individuals had a higher cardiovascular response rate to noise than non-sensitive people during their waking exposure, while there was no difference in sensitivity to noise between these two groups during their night-time exposure while they were asleep.

The physiological sensitivity to noise depends also on the age of the sleeper. Thus, while EEG modifications and awakening thresholds are, on average, 10 dB(A) higher in children than in adults, their cardiovascular sensitivity to noise is similar, if not higher, than the older group (Muzet and Ehrhart, 1980; see also Appendix 4). Elderly people complain much more than younger adults about environmental noise. However, their spontaneous awakenings occurring during night sleep are also much more numerous. Therefore, it is difficult to conclude if elderly people are more sensitive to noise or if they hear noise because they are often awake during the night. This natural fragmentation of their night sleep tends also to lengthen their return to the sleeping state and this accounts for a significant part of their subjective complaints.

Differences in sensitivity to noise have been found between the sexes. Thus, young men seem to complain more about noise-disturbed sleep than young females (Muzet et al., 1973). However, this difference seems to reverse for populations over 30 years of age and then females (often mothers) appear to be more sensitive to noise than males (Lukas, 1972b).

3.1.10 USE OF INSTANTANEOUS EFFECTS IN PREDICTIONS OVER A LONGER TIME PERIOD

It is tempting to use the relations between single exposures and measured effects in long-term predictions. Although this is perhaps possible, a word of caution is appropriate.

In general, the reactions are calculated by looking at a certain time frame around an exposure, usually in the order of a few minutes. The second limitation is that order and follow-up effects are neglected. Time and order effects of identical events on

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motility have been described by Brink, Wirth and Schierz (2006). Only if the situation that is modelled resembles the one that was used in the single exposure analysis, are no major deviations to be expected. Reactions to noise events are generally not independent from each other. Each event may alter a subject's tendency to awake at the next event, even if no awakening reaction is detected for that particular event. If, for example, each event would additionally increase the probability of awakening at the next event, the total probability of awakening per night would be greater than predicted by mere summation of the single event probabilities. Most likely, this underestimation of probability will occur when events in the real situation follow in close succession, whereas events in the single exposure analysis did not. Such limitations can to some degree be overcome through applying advanced statistical methods such as those put forward by Basner (2006). A third limitation is that an overall increase in the base line could go undetected.

If the situation that is calculated resembles the one that was used in the single exposure analysis, probably no major deviations are to be expected. Care should be taken to extrapolate outside the boundaries given in the number of events or L_{Amax}. Calculations for Amsterdam Schiphol Airport show a good agreement between the number of calculated awakenings per year (based on the actual SEL data) and the self-reported number of awakenings. This number is a factor 2 lower than the worst case scenario presented in section 3.1.7 above.

3.2 CHRONIC EFFECTS: CHRONIC INCREASE OF MOTILITY

Mean motility – all body movements counted together – during sleep is strongly related to age and is also a function of noise exposure during the sleep period. The relationships between mean motility and $L_{night,inside}$ are shown in Fig. 3.4. Mean motility during sleep is lowest at the age of 45 years, and greater at higher and lower ages. The relation between mean motility, $L_{night,inside}$ and age is:

$$\text{Mean motility} = 0.0587 + 0.000192*L_{night,inside} - 0.00133*\text{age} + 0.0000148*\text{age}^2 \quad [7].$$

The relation between the increase in mean noise-induced motility, m_{night}, and $L_{night,inside}$ is:

$$m_{night} = 0.000192*L_{night,inside} \quad [8],$$

assuming, as described in Chapter 1, section 1.3.4, that $L_{night,inside} = L_{night}-21$:

$$m_{night} = 0.000192*L_{night}-0.004032 \quad [8a].$$

Source: Miedema, Passchier-Vermeer and Vos, 2003.

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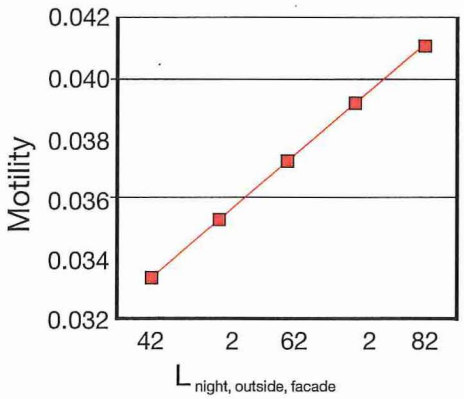


Fig. 3.4
Increase in mean motility (body movements during sleep). Converted from inside relation with [3]

The increase in m_{night} is 22% over the baseline motility (0.03 on average) if indoor $L_{night,inside}$ increases from 0 (absence of aircraft noise) to 35 dB(A) (living close to a runway). This increase is independent of age, although the absolute level varies.

Other chronic effects like the use of sleeping pills, changes in BP and changes in levels of stress hormones are discussed in the next chapter.

3.3 CONCLUSIONS

During sleep the auditory system remains fully functional. Incoming sounds are processed and evaluated and although physiological changes continue to take place, sleep itself is protected because awakening is a relatively rare occurrence. Adaptation to a new noise or to a new sleeping environment (for instance in a sleep laboratory) is rapid, demonstrating this active protection. The physiological reactions do not adapt, as is shown by the heart rate reaction and the increase of average motility with sound level. The autonomous physiological reactions are a normal reaction to these stimuli, but the question is whether prolonged "abuse" of this system leads to adverse consequences for the organism. The next chapter tries to answer that.

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CHAPTER 4

EFFECTS OF NIGHT-TIME NOISE ON HEALTH AND WELL-BEING

The sick die here because they can't sleep,

For when does sleep come in rented rooms?
It costs a lot merely to sleep in this city!
That's why everyone's sick: carts clattering
Through the winding streets, curses hurled
At some herd standing still in the middle of the road,
Could rob Claudius or a seal of their sleep!

(Juvenal, 1st century AD)

4.1 INTRODUCTION

In Chapters 2 and 3, sufficient evidence was presented to support the hypothesis for the simplified model presented in Chapter 1: sleep disturbance is connected to health impairment, and noise is an important factor that causes sleep disturbance. The full model (Fig. 2.1, Chapter 2) showed why it is difficult to find evidence for a direct relation between noise exposure at night and health outcomes. Noise is but one of the internal and external factors that cause sleep disturbance and feedback loops obscure the view of the cause and effect chain. In this chapter the evidence for the direct relation is presented.

4.2 SELF-REPORTED (CHRONIC) SLEEP DISTURBANCES

Self-reported sleep disturbance is investigated by means of a questionnaire containing questions regarding sleep disturbance. Often, sleep disturbance is not the main focus of the questionnaires used in studies of self-reported noise effects. This means that considerable effort is needed to harmonize the different response categories. The relationships for self-reported sleep disturbance are based on analyses of the 15 data sets with more than 12 000 individual observations of exposure–response combinations, from 12 field studies (Miedema, 2003; Miedema, Passchier-Vermeer and Vos, 2003).

The curves are based on data in the L_{night} (outside, most exposed facade) range 45–65 dB(A). The polynomial functions are close approximations of the curves in this range and their extrapolations to lower exposure (40–45 dB(A)) and higher exposure (65–70 dB(A)). The formulae of these polynomial approximations are as follows (SD = sleep disturbance; H = high; L = low):

for road traffic:

$$\%HSD = 20.8 - 1.05*L_{night} + 0.01486*(L_{night})^2 \quad [9]$$

$$\%SD = 13.8 - 0.85*L_{night} + 0.01670*(L_{night})^2 \quad [10]$$

$$\%LSD = -8.4 + 0.16*L_{night} + 0.01081*(L_{night})^2 \quad [11]$$

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for aircraft:

$$\%HSD = 18.147 - 0.956*L_{night} + 0.01482*(L_{night})^2 \quad [12]$$

$$\%SD = 13.714 - 0.807*L_{night} + 0.01555*(L_{night})^2 \quad [13]$$

$$\%LSD = 4.465 - 0.411*L_{night} + 0.01395*(L_{night})^2 \quad [14]$$

and for railways:

$$\%HSD = 11.3 - 0.55*L_{night} + 0.00759*(L_{night})^2 \quad [15]$$

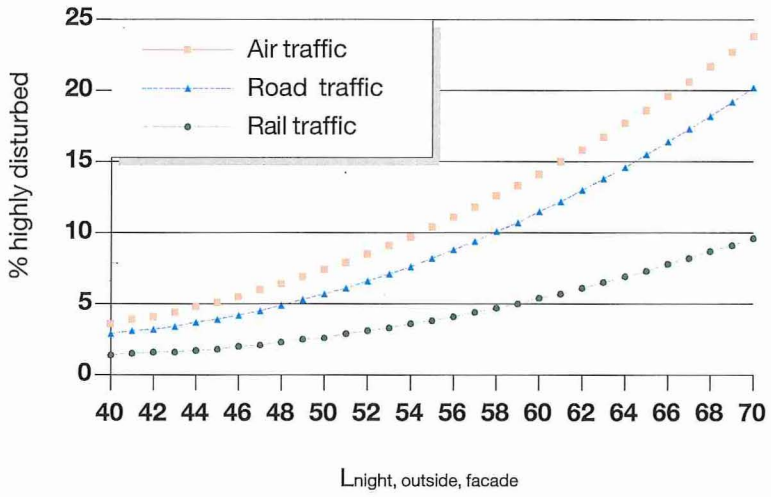
$$\%SD = 12.5 - 0.66*L_{night} + 0.01121*(L_{night})^2 \quad [16]$$

$$\%LSD = 4.7 - 0.31*L_{night} + 0.01125*(L_{night})^2 \quad [17].$$

The above relations represent the current best estimates of the influences of L_{night} on self-reported sleep disturbance for road traffic noise and for railway noise, when no other factors are taken into account. Fig. 4.1 illustrates the relations [9] [12] and [15] for persons highly disturbed by road, aircraft and rail noise.

Fig. 4.1
High sleep disturbance by noise at night

Source:
European Commission, 2004.



With regard to the relations for aircraft noise it should be noted that the variance in the responses is large compared to the variance found for rail and road traffic. This means that the uncertainty regarding the responses for night-time aircraft noise is large, and such responses can be considered as indicative only. Miedema (2003) suggests the following causes.

- The time pattern of noise exposures around different airports varies considerably due to specific night-time regulations.
- The sleep disturbance questions for aircraft noise show a large variation.
- The most recent studies show the highest self-reported sleep disturbance at the same L_{night} level. This suggests a time trend.

For industrial noise there is an almost complete lack of information, although there are some indications (Vos, 2003) that impulse noise may cause considerable disturbance at night.

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4.3 COMPLAINTS

According to the Health Council of the Netherlands (2004), the submission of a complaint about noise is symptomatic of reduced well-being.

Complaints about noise are widespread, and night noise seems to cause more complaints than daytime noise at the same level. Hume, Morley and Thomas (2003) found that around Manchester Airport complaints per 1000 aircraft traffic movements rose from an average of 10 in daytime hours to up to 80 in the night. When linking part of the complaints to measured noise levels, they found an increase from an average of 1 complaint at 70 PNLdB (circa 58 L_{Amax}) to 2 at 114 PNLdB (circa 102 L_{Amax}).

Due to differences in complaint cultures and registration practices, it is difficult to make comparisons between complaint registrations. Around Amsterdam Schiphol Airport a relation between complaints and L_{Aeq} was found (Ministerie Verkeer en Waterstaat, 2005). The threshold for complaints is around 45 L_{den}, and increases to 7% of the population at 72 L_{den}. Night-time complaints follow the same pattern, and the threshold for night complaints is 35 L_{night}. In Fig. 4.2 the mean percentage shows a definite relationship with L_{night}. The 95 percentile indicates that the threshold is 35 L_{night}.

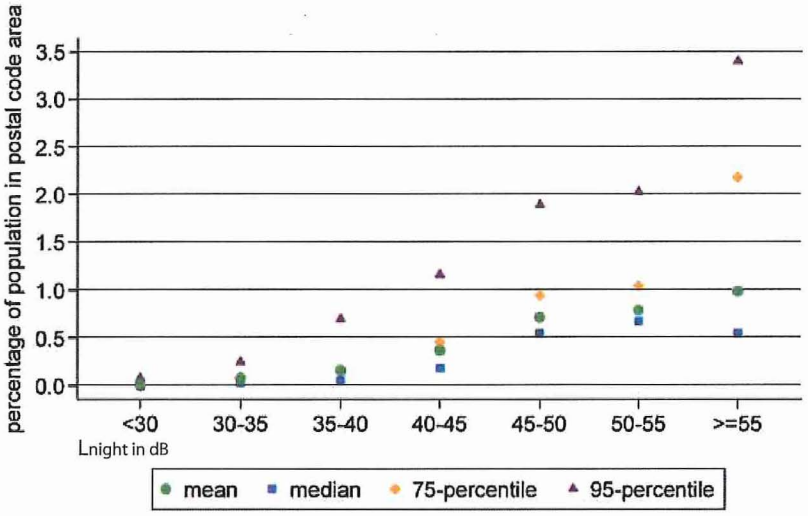


Fig. 4.2 Dose-effect relation for persons having complained at least once during a year between 1994 and 2004

Source: Ministerie Verkeer en Waterstaat (2005).

4.4 NEIGHBOURHOOD NOISE AND NOISE FROM NEIGHBOURS

Inventory studies in the Netherlands indicate that sleep disturbance attributable to the most annoying forms of neighbourhood noise and noise from neighbours (contact noise and human noises in the environment) is on a similar scale to disturbance attributable to the most annoying sources of road traffic noise (mopeds and passenger cars). It is reasonable to assume that chronic sleep disturbance is, in the long term, liable to have consequences for health and well-being. The sound pressure level and other noise characteristics are liable to determine the nature of the influence to some extent, but certain other factors play a more prominent role than is the case with traffic noise. These factors include appreciation of the noise and of the party responsible for the noise, as well as the hearer's personal circumstances. However, scientific understanding of the relative importance of and interaction between

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acoustic and non-acoustic factors is not sufficient for the committee to draw any definitive conclusions regarding the relationship between, on the one hand, exposure to night-time neighbourhood noise and noise from neighbours and, on the other, health and well-being.

Leidelmeijer and Marsman (1997) carried out an interview-based study of 1242 households in the Netherlands, in which subjects were asked about daytime and night-time noise from neighbours and any associated annoyance. A distinction was made on the basis of the part of the house in which the noises were audible and any associated annoyance was experienced. Subjects proved least tolerant of noise from their neighbours that was audible in the master bedroom. The researchers distinguished five types of noise, which are listed below (Table 4.1), along with the percentage of subjects who indicated hearing the relevant type of noise from a neighbouring dwelling at night in the master bedroom.

Table 4.1 Daytime and night-time noise from neighbours

Source: Leidelmeijer and Marsman, 1997.

Type of noise	% subjects hearing noises at night in the bedroom
Contact noise	22%
Noise from sanitary fittings, central heating, etc.	19%
Noise from radio, TV and hi-fi	12%
Do-it-yourself noises	8%
Pets	6%

Where each of the five investigated types of noise was concerned, roughly 10–15% of subjects indicated that they felt it was unacceptable for the noise to be audible during the day. Overall, nearly 30% of subjects said that sanitary fittings should not be audible at night, while approximately 50% felt each of the other four types of noise was unacceptable at night.

In 1993, Kranendonk, Gerretsen and van Luxemburg produced a synthesis of the research conducted up to that point in time into the annoyance associated with noise from neighbours. Subsequently, in 1998, van Dongen et al. published a report on the relationship between noise from neighbouring dwellings and the airborne and contact noise attenuating indices I_{lu}, $I_{lu;k}$, and I_{co}, drawing on data from a questionnaire-based survey of the residents of 600 dwellings, whose acoustic quality was determined in 202 cases. The results of the two studies are reasonably consistent. Both found that the chief causes of annoyance were loud radios, hi-fis and TVs, audible and sometimes intelligible voices, the slamming of doors and footsteps on floors and staircases. In both cases, it proved that, when Ilu had a value of 0 (the minimum requirement for new homes), 10% of subjects reported high annoyance and 15% reported annoyance caused by noise from neighbouring dwellings. These figures are not specific to night-time noise, but apply to annoyance over a 24-hour period.

On the basis of the findings outlined above, the committee concludes that the standard of inter-dwelling sound attenuation presently required does not provide sufficient protection to prevent annoyance caused by noise from neighbours. Since people are less tolerant of the noise their neighbours make at night-time than of their neighbours' evening or daytime noise, it may be assumed that much of the annoyance associated with noise from neighbours relates to the influence of such noise on sleep.

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4.5 CARDIOVASCULAR EFFECTS OF NOISE – FINDINGS FROM EPIDEMIOLOGICAL STUDIES

4.5.1 INTRODUCTION

It is a common experience that noise is unpleasant and affects the quality of life. It disturbs and interferes with activities of the individual including concentration, communication, relaxation and sleep (WHO Regional Office for Europe, 2000; Schwela, 2000). Besides the psychosocial effects of community noise, there is concern about the impact of noise on public health, particularly regarding cardiovascular outcomes (Suter, 1992; Passchier-Vermeer and Passchier, 2000; Stansfeld, Haines and Brown, 2000). Non-auditory health effects of noise have been studied in humans for a couple of decades using laboratory and empirical methods. Biological reaction models have been derived, which are based on the general stress concept (Selye, 1956; Henry and Stephens, 1977; Ising et al., 1980; Lercher, 1996). Amongst other non-auditory health end points, short-term changes in circulation including BP, heart rate, cardiac output and vasoconstriction, as well as stress hormones (epinephrine, norepinephrine and corticosteroids) have been studied in experimental settings for many years (Berglund and Lindvall, 1995; Babisch, 2003). Various studies have shown that classical biological risk factors are higher in subjects who were exposed to high levels of traffic noise (Arguelles et al., 1970; Eiff et al., 1974; Verdun di Cantogno et al., 1976; Algers, Ekesbo and Strömberg, 1978; Knipschild and Sallé, 1979; Manninen and Aro, 1979; Eiff et al., 1981a; Rai et al., 1981; Marth et al., 1988; Babisch and Gallacher, 1990; Babisch et al., 1990; Lercher and Kofler, 1993; Schulte and Otten, 1993; Dugué Leppänen and Gräsbeck, 1994; Yoshida et al., 1997; Goto and Kaneko, 2002). Although controls for other risk factors were not consistent in all these studies, the hypothesis emerged that persistent noise stress increases the risk of cardiovascular disorders including high BP (hypertension) and IHD.

- Sound/noise is a psychosocial stressor that activates the sympathetic and endocrine system.
- Acute noise effects do not only occur at high sound levels in occupational settings, but also at relatively low environmental sound levels when, more importantly, intended activities such as concentration, relaxation or sleep are disturbed.

The following questions need to be answered.

- Do these changes observed in the laboratory habituate or persist under chronic noise exposure?
- If they habituate, what are the physiological costs? If they persist, what are the long-term health effects?

The answers to these questions come from epidemiological noise research. Large-scale epidemiological studies have been carried out for a long time (Babisch, 2000). The studies suggest that transportation noise is associated with adverse cardiovascular effects, in particular IHD. The epidemiological evidence is constantly increasing (Babisch, 2002, 2004a). The biological plausibility of the association derives from the numerous noise experiments that have been carried out in the laboratory. There is no longer any need to prove the noise hypothesis as such. Decision-making and risk management, however, rely on a quantitative risk assessment which requires an established dose–response relationship. Since many of the stress indicators and risk factors that have been investigated in relation to

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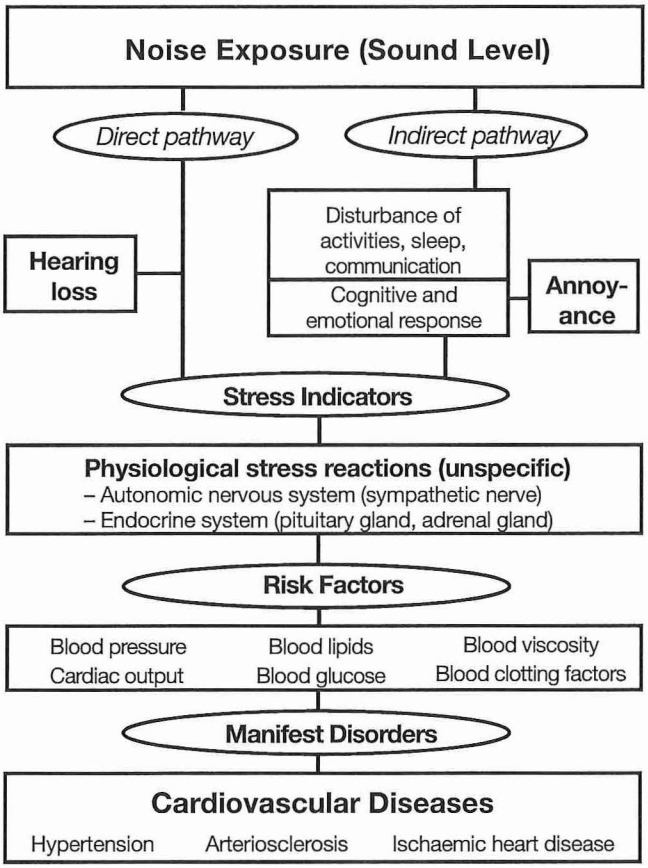


Fig. 4.3
Noise effects reaction scheme

Source:
Babisch, 2002.

noise are known to be classical cardiovascular risk factors, the hypothesis has emerged that chronic noise exposure increases the risk of hypertension, arteriosclerosis and IHD. Its relevance for public health comes from the high prevalence of cardiovascular diseases in developed and industrialized countries. It is unclear as to what extent chronically repeated noise-induced sleep disturbance contributes to the development of somatic health disorders. Only a few epidemiological studies address this particular issue. Epidemiological noise research has seldom distinguished between day and night exposures, or between the exposure of the living room and the bedroom. However, some deduction can be made from daytime to night-time exposure.

4.5.2 NOISE AND STRESS-REACTION MODEL

The auditory system is continuously analysing acoustic information, which is filtered and interpreted by different cortical and subcortical brain structures. The limbic system, including the hippocampus and the amygdala, plays an important role in the emotional processing pathways (Spreng, 2000). It has a close connection to the hypothalamus that controls the autonomic nervous system and the hormonal balance of the body. Laboratory studies found changes in blood flow, BP and heart rate in reaction to noise stimuli as well as increases in the release of stress hormones including the catecholamines adrenaline and noradrenaline, and the corticosteroid

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cortisol (Berglund and Lindvall, 1995; Maschke, Rupp and Hecht, 2000; Babisch, 2003). Such changes also occur during sleep without the involvement of cortical structures. The amygdala has the capacity to learn due to its plasticity, particularly with respect to the meaning of sound stimuli (for example, danger of an approaching lorry) (Spreng, 2000, 2004). Acoustic stimulation may act as an unspecific stressor that arouses the autonomous nervous system and the endocrine system. The generalized psychophysiological concept given by Henry and Stephens can be applied directly to noise-induced stress reaction (Henry, 1992). The stress mechanism as such is genetically determined but it may be modified by experience and environmental factors. Its biological function is to prepare the organism to cope with a demanding stressor. The arousal of the sympathetic and endocrine system is associated with changes in physiological functions and the metabolism of the organism, including BP, cardiac output, blood lipids (cholesterol, triglycerides, free fatty acids, phosphatides), carbohydrates (glucose), electrolytes (magnesium, calcium), blood clotting factors (thrombocyte aggregation, blood viscosity, leukocyte count) and others (Friedman and Rosenman, 1975; Cohen, Kessler and Underwood Gordon, 1995; Lundberg, 1999). In the long term, functional changes and dysregulation may occur, thus increasing the risk of manifest diseases.

Fig. 4.3 shows the principal reaction schema used in epidemiological noise research for hypothesis testing (Babisch, 2002). It simplifies the cause–effect chain, that is: sound – annoyance (noise) – physiological arousal (stress indicators) – (biological) risk factors – disease – and mortality (the latter is not explicitly considered in the graph). The mechanism works "directly" through synaptic nervous interactions and "indirectly" through the emotional and the cognitive perception of the sound. It should be noted that the "direct" pathway is relevant even at low sound levels particularly during sleep, when the organism is at its nadir of arousal. The objective noise exposure (sound level) and the subjective noise "exposure" (annoyance) may serve independently as exposure variables in the statistical analyses of the relationship between noise and health end points.

Principally, the effects of environmental noise cannot directly be extrapolated from results of occupational noise studies. The two noise environments cannot simply be merged into one sound energy-related dose–response model (for example, a simple 24-hour average noise level measured with a dose-meter). Noise effects are not only dependent on the sound intensity but also on the frequency spectrum, the time pattern of the sound and the individuals' activities which are disturbed. Therefore, epidemiological studies carried out under real-life conditions can provide the basis for a quantitative risk assessment provided that there is adequate control over confounding and exposure variables. Other noise sources might act as confounders and/or effect modifiers on the association of interest. The effects of road traffic noise (at home) were shown to be stronger in subjects who were also exposed to high noise levels at work (Babisch et al., 1990).

4.5.3 PREVIOUS REVIEWS ON ENVIRONMENTAL NOISE AND CARDIOVASCULAR RISK

Causality in epidemiology can never be completely proven (Schlesselman, 1987; Christoffel and Teret, 1991; Weed, 2000). It is a gradual term for which evidence is increasing with the increasing number of facts. However, the magnitude of effect, the presence of a dose–response relationship and consistency with other studies in different populations and with different methodology and biological plausibility are

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commonly accepted arguments for a causal relationship (Bradford Hill, 1965; Evans, 1976; Morabia, 1991; Weed and Hursting, 1998). Classical, systematic and quantitative reviews have been published in the past, summarizing the results of studies that have been carried out up to the end of the last century, and assessing the evidence of the relationship between community noise and cardiovascular disease outcomes (Health Council of the Netherlands, 1994, 1999, 2004; Berglund and Lindvall, 1995; IEH, 1997; Morrell, Taylor and Lyle, 1997; Porter, Flindell and Berry, 1998; Babisch, 2000; Passchier-Vermeer and Passchier, 2000), including a classical review and synthesis report by Babisch (2000) and a systematic review (meta-analysis) by van Kempen et al. (2002).

In a meta-analysis it was concluded that the risk of hypertension due to aircraft noise was 1.26 per increase of 5 dB(A) (95% CI: 1.14–1.39, L_{day} = 55–72 dB(A)) (van Kempen et al., 2002). But, only one study (Knipschild, 1977a) was considered in the meta-analysis. With respect to road traffic noise and hypertension a pooled estimate of 0.95 per 5 dB(A) (95% CI: 0.84–1.08, L_{day} = <55–80 dB(A)) was calculated (van Kempen et al., 2002). Two cross-sectional studies (Knipschild and Sallé, 1979; Knipschild, Meijer and Sallé, 1984) were considered in this calculation. The highest degree of evidence was for the association between community noise and IHD. Across the studies there was not much indication of an increased risk for subjects who lived in areas with a daytime average sound pressure level of less than 60 dB(A). For higher noise categories, however, higher risks were relatively consistently found amongst the studies (Babisch, 2004a). Statistical significance was rarely achieved.

Some studies permit reflections on dose–response relationships. These mostly prospective studies suggest an increase in risk for outdoor noise levels above 65–70 dB(A) during the daytime, the relative risks ranging from 1.1 to 1.5. Noise effects were larger when mediating factors like years in residence, room orientation and window-opening habits were considered in the analyses. In a meta-analysis it was concluded that the risk of IHD increased by 1.09 per 5 dB(A) of the road traffic noise level (95% CI: 1.05–1.13, L_{day} = 51–70 dB(A)) (van Kempen et al., 2002), when two cross-sectional studies (Babisch et al., 1993a) were considered. However, the pooled estimate of two prospective studies (Babisch et al., 1999) was calculated to be 0.97 per 5 dB(A) (95% CI: 0.90–1.04, L_{day} = 51–70 dB(A)) (van Kempen et al., 2002). When the diagnosis of IHD was limited to myocardial infarction, three studies (Babisch et al., 1999, 1994) were considered in this meta-analysis. Then the linear effect estimate was 1.03 per 5 dB(A) increase in road traffic noise level (95% CI: 0.99–1.09, L_{day} = 51–80 dB(A)). New studies have appeared in the meantime which are included in the present updated review (Matsui et al., 2001; Bluhm, Nordling and Berglind, 2001; Evans et al., 2001; Rosenlund et al., 2001; Belojevic and Saric-Tanaskovic, 2002; Goto and Kaneko, 2002; Lercher et al., 2002; Maschke, 2003; Franssen et al., 2004; Matsui et al., 2004; Niemann and Maschke, 2004; Babisch et al., 2005). Others are on their way or have not yet been finalized and published, for instance the pan-European HYENA project (Jarup et al., 2003).

4.5.4 UPDATED REVIEW OF EPIDEMIOLOGICAL STUDIES

Sixty epidemiological studies were recognized as having either objectively or subjectively assessed the relationship between transportation noise and cardiovascular end points. The identification of studies was based on the author's expert

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knowledge of the topic and respective literature. Details are given in the major report (Babisch, 2006). Information particularly on night-time exposure (L_{night}: 22.00–06.00 or 23.00–07.00) was seldom available. Newer studies used non-weighted or weighted averages of the 24-hour exposure (L_{eq}, L_{dn}, L_{den}). Some aircraft noise studies used national calculation methods (for example, Dutch Kosten Units). For comparisons of study results and the pooling of data (meta-analysis), sound levels were converted on the basis of best guess approximations to L_{day} (Matschat and Müller, 1984; Passchier-Vermeer, 1993; Bite and Bite, 2004; Franssen et al., 2004). It should be noted in this context that doubling/halving of road traffic volume results in a 3 dB(A) higher/lower average sound pressure level. Not all studies allowed dose–response reflections because some of them considered very broad exposure categories. Besides objective noise measurements, subjective measurements of exposure have been used in some epidemiological noise studies, which is in accordance with the noise–stress model. Type of road (for example, busy street, side street, etc.), disturbances and annoyance were rated by the study subjects from given scales.

4.5.5 MEAN BP

Table A2 of the major report (Babisch, 2006) lists the major findings of epidemiological traffic noise studies in which mean BP was considered as the outcome. It indicates mean systolic and diastolic BP differences as obtained from extreme group comparisons of noise exposure. The effects in children and in adults are discussed separately. The findings in children are difficult to interpret with regard to possible health risks in their later life. The effect may be of a temporary nature and may not be relevant to permanent health damage. There is evidence during childhood (Gillman et al., 1992), adolescence (Yong et al., 1993) and adulthood (Tate et al., 1995) that the BP level at an early age is an important predictor of the BP level at a later age. Studies over the full age range are missing (tracking). Growth and body weight are important factors in BP development. The impact of body size was not adequately considered in some of the studies. A crude hint regarding reversible effects on BP came from one study (Morrell et al., 2000). Results of the Munich intervention study on the effects of a reduction of aircraft noise have only been reported regarding cognitive performance but not with respect to change of BP (Hygge, Evans and Bullinger, 2002). It was concluded from the available data on the length of exposure that children do not seem to adapt to high levels of road traffic noise but to some extent to aircraft noise (Passchier-Vermeer, 2000; Bistrup et al., 2001). However, the database appears to be too poor to draw final conclusions. Aircraft noise studies focused on exposure at school, while road traffic noise studies mostly considered noise exposure at home. The conclusions given by Evans and Lepore (1993) seem still to hold true:

> "We know essentially nothing about the long-term consequences of early noise exposure on developing cardiovascular systems. The degree of blood pressure elevations is small. The clinical significance of such changes in childhood blood pressure is difficult to determine. The ranges of blood pressure among noise-exposed children are within the normal levels and do not suggest hypertension. The extent of BP elevations found from chronic exposure are probably not significant for children during their youth, but could portend elevations later in life that might be health damaging."

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Regarding mean BP, no consistent findings in the relationship between traffic noise level and mean systolic or diastolic BP can be seen in adults across the studies. In longitudinal studies, problems arose from migration of subjects, which had a considerable impact on sample size. The latter problem also applies to cross-sectional studies, in general. Sensitive subjects may tend to move out of the polluted areas, which dilutes the effect of interest. Medication due to high BP may affect the BP readings. However, the exclusion of subjects with hypertension or hypertension treatment dilutes the true effect on BP differences, if the hypothesis (noise causes high BP) is true. In principle, hypotension – a fall in BP – can also be a stress reaction. All this makes it more reasonable to look at manifest hypertension (defined by a cut-off criterion) as a clinical outcome rather than at mean BP readings (Ising, 1983; Winkleby, Ragland and Syme, 1988). To date, there is no evidence from epidemiological data that community noise increases mean BP readings in the adult population. However, this does not discard the noise hypothesis as such. Studies suffered from insufficient power, narrow exposure range or other difficulties in the study design.

4.5.6 HYPERTENSION

Table A3 of the major report (Babisch, 2006) gives the results of epidemiological traffic noise studies on the relationship between community noise level and the prevalence or incidence of hypertension. Hypertension in these studies was either defined by WHO criteria (WHO-ISH Guidelines Subcommittee, 1999), similar criteria based on measurements of systolic and diastolic BP, from information which was obtained from a clinical interview, or a social survey questionnaire about hypertension diagnosed by a doctor. Most studies refer to road traffic noise. However, in recent years some new aircraft noise studies entered the database. The subjects studied were the adult male and female population, sometimes restricted to certain age ranges. With regard to the association between community noise and hypertension, the picture is heterogeneous. With respect to aircraft noise and hypertension, studies consistently show higher risks in higher exposed areas. The evidence has improved since a previous review (Babisch, 2000). The relative risks found in four significantly positive studies range between 1.4 and 2.1 for subjects who live in high exposed areas, with approximate daytime average sound pressure level in the range of 60–70 dB(A) or more. Swedish studies found a relative risk of 1.6 at even lower levels >55 dB(A). With respect to road traffic noise, the picture remains unclear. New studies, more than older studies, tend to suggest a higher risk of hypertension in subjects exposed to high levels of road traffic noise, showing relative risks between 1.5 and 3.0. However, the earlier studies cannot be neglected in the overall judgement process. Across all studies no consistent pattern of the relationship between community noise and prevalence of hypertension can be seen. Dose–response relationships were considered in new studies. Subjective ratings of noise or disturbances due to traffic noise seem to consistently show a positive association with prevalence of hypertension. The relative risks found here range from 0.8 to 2.3. These studies, however, are of lower validity due to principal methodological issues regarding over-reporting (Babisch et al., 2003).

4.5.7 IHD

Table A5 of the major report (Babisch, 2006) gives the results of cross-sectional epidemiological traffic noise studies on the relationship between noise level and prevalence of IHD. Table A6 of the major report gives the results of case-control

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and cohort studies on the association between noise level and incidence of IHD. In cross-sectional studies, IHD prevalence was assessed by clinical symptoms of angina pectoris, myocardial infarction, ECG abnormalities as defined by WHO criteria (Rose and Blackburn, 1968), or from self-reported questionnaires regarding doctor-diagnosed heart attack. In longitudinal studies, IHD incidence was assessed by clinical myocardial infarction as obtained from hospital records, ECG measurements or clinical interviews. The majority of studies refer to road traffic noise. With regard to IHD, the evidence of an association between community noise and IHD risk has increased since a previous review (Babisch, 2000). There is not much indication of a higher IHD risk for subjects who live in areas with a daytime average sound pressure level of less than 60 dB(A) across the studies. For higher noise categories, a higher IHD risk was relatively consistently found amongst the studies. Statistical significance was rarely achieved. Some studies permit reflections on dose–response relationships. These mostly prospective studies suggest an increase in IHD risk at noise levels above 65–70 dB(A), the relative risks ranging from 1.1 to 1.5 when the higher exposure categories were grouped together. Noise effects were larger when mediating factors like residence time, room orientation and window-opening habits were considered in the analyses. This accounts for an induction period (Rose, 2005) and improves exposure assessment. The results appear as consistent when subjective responses of disturbance and annoyance are considered, showing relative risks ranging from 0.8 to 2.7 in highly annoyed/disturbed/affected subjects. However, these findings may be of lower validity due to methodological issues.

4.5.8 MEDICATION AND DRUG CONSUMPTION

Table A8 of the major report (Babisch, 2006) gives the results of studies on the relationship between drug consumption and community noise. Medication was primarily investigated with respect to aircraft noise. A significant prevalence ratio for medication with cardiovascular drugs of 1.4 was found in the sample of Amsterdam Schiphol Airport (Knipschild, 1977a). The results of the "drug survey", where the annual data of the pharmacies regarding the purchase of cardiovascular drugs were analysed (repeated cross-sectional survey), supported this finding. An increase in drug purchase over time in the exposed areas and not in the less exposed was found. This refers to the purchase of cardiovascular and antihypertensive drugs, as well as the purchase of hypnotics, sedatives and antacids (Knipschild and Oudshoorn, 1977). Furthermore a dependency with changes in night flight regulations was found (decrease after reduction of night flights). A large recent study around Amsterdam Schiphol Airport found only a slightly higher risk of self-reported medication with cardiovascular drugs, including antihypertensive drugs (relative risk 1.2), in subjects exposed to aircraft noise where the noise level L_{den} exceeded 50 dB(A) (Franssen et al., 2004). Dose–response relationships across noise levels (L_{den} = <50–65 dB(A)) with respect to prescribed and non-prescribed sedatives/sleeping pills were found (relative risk 1.5 and 2.0, respectively) in the highest noise category of L_{den} = 61–65 dB(A). The preliminary results of an ongoing aircraft noise study from Sweden carried out around Stockholm's airport are in line with the Dutch studies (Bluhm et al., 2004). A significant relative risk of 1.6 for the use of antihypertensive drugs was found in male subjects, where the noise level according to the Swedish calculation standard exceeded FBM = 55 dB(A). The road traffic noise studies, where medication/purchase of drugs was investigated also tend to show a higher use in higher exposed subjects (Eiff and Neus, 1980; Schulze et al., 1983; Lercher,

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1992). The relative risk for cardiovascular drugs was 1.3 in the Bonn study and 5.0 in the Erfurt study. The results for other drugs including sleeping pills, sedatives, tranquillizers and hypnotics ranged between 1.2 and 3.8 in these studies. All in all, the studies on the relationship between the use of medication or purchase of drugs and community noise support the general hypothesis of an increase in sleep disturbance and cardiovascular risk in noise-exposed subjects.

4.5.9 EVALUATION OF STUDIES

This section refers only to studies where the prevalence or the incidence of manifest cardiovascular diseases was considered as a potential health outcome of chronic exposure to environmental noise. The focus here is on a quantitative risk assessment with respect to manifest diseases. Furthermore, studies on the effects of low-altitude jet-fighter noise are also excluded, because this type of noise includes other dimensions of stress (for instance, fear). Thirty-seven studies have assessed the prevalence or incidence of manifest diseases, including hypertension and IHD (angina pectoris, myocardial infarction, ECG abnormalities).

4.5.9.1 Criteria

Epidemiological reasoning is largely based on the magnitude of effect estimates, dose–response relationships, consistency of findings, biological plausibility of the effects and exclusion of possible bias. Internal (the role of chance) and external validity (absence of bias and confounding) are important issues in the evaluation of studies (Bradford Hill, 1965). Analytic studies (for example, cohort or case-control studies) are usually considered as having a higher validity and credibility than descriptive studies (for example, cross-sectional or ecological studies) (Hennekens and Buring, 1987), although many of the reservations against cross-sectional studies seem to be of minor importance when considering noise. For example, it does not appear to be very likely that diseased subjects tend to move differentially more often into exposed areas. Rather the opposite may be true, if noise stress is recognized as a potential cause of the individual's health problem. Thus, a cross-sectional study design may act conservatively on the results. The presence of a dose–response relationship is not a necessary criterion of causality. Non-linear relationships, including "u-" or "j-" shaped, saturation and threshold effects may reflect true associations (Calabrese and Baldwin, 2003; Rockhill, 2005). With respect to the derivation of guideline values in public health policy, the assessment of a dose–response relationship enables a quantitative risk assessment on the basis of continuous or semi-continuous (for instance 5 dB(A) categories) exposure data. Dichotomous exposure data, on the other hand, that refer to a cut-off criterion which splits the entire exposure range into two halves, can be used to evaluate the hypothesis of an association (qualitative interpretation), but not a quantitative assessment. The objective or subjective assessment of exposure and/or health outcomes is an important issue when judging the validity of a study (Malmström, Sundquist and Johansson, 1999; Cartwright and Flindell, 2000; Hatfield et al., 2001). The objective prevalence of hypertension was found to be higher in a population sample than the subjective prevalence of hypertension (Schulte and Otten, 1993). In a telephone survey more than half of the hypertensives classified themselves as normotensive (sensitivity 40% for men and 46% for women) (Bowlin et al., 1993). In a representative health survey, the validity of the self-reported assessment of morbidity (subjective morbidity) was found to be "low" with respect to hypercholesterolaemia, "intermediate" with respect to angina pectoris, hypertension and stroke and "high" with respect to

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myocardial infarction (Bormann et al., 1990). Myocardial infarction is a very definite and severe health outcome which subjects would clearly know about if they had experienced it. Its assessment by questionnaire tends to be more credible than that regarding hypertension. Test–retest reliability was found to be good with respect to "harder" outcomes, including high BP and heart attack (Lundberg and Manderbacka, 1996; Lipworth et al., 2001). Over-reporting, on the other hand, may be a source of potential bias, particularly when both exposure and outcome are assessed on a subjective basis (Winkleby, Ragland and Syme, 1988; Babisch et al., 2003). The subjects may be more prone to blame their environment for their health problems, or may even tend to exaggerate adverse effects or exposure in order to influence noise policy. Therefore, a higher credibility and ranking was given to studies where exposure and outcome were assessed objectively (for example sound level versus subjective ratings, and measurement of BP or a clinical interview versus self-reported hypertension in a self-administered questionnaire). This means that the sound level must have been measured or calculated on the basis of the traffic counts, and clinical interviews or measurements must have been carried out by medically trained personnel (no self-administered questionnaire data) to give a study a high ranking. Studies which have been adequately controlled (for instance stratification, model adjustment (regression), matching) for a reasonable set of confounding variables in the statistical analyses, besides age and sex, were given a high ranking.

4.5.9.2 Assessment

The evaluation concerning the epidemiological studies was made with respect to the identification of good quality studies that can be feasibly considered for the derivation of guideline values. These studies can either be used for a statistical meta-analysis, for a combined interpretation (synthesis) or for singular interpretations. All the studies were evaluated with respect to the following criteria for inclusion or exclusion in the synthesis process. Necessary criteria were: (1) peer-reviewed in the international literature; (2) reasonable control of possible confounding; (3) objective assessment of exposure; (4) objective assessment of outcome; (5) type of study; and (6) dose–response assessment. All six criteria were fulfilled by the two prospective cohort studies carried out in Caerphilly and Speedwell (Babisch et al., 1999; Babisch, Ising and Gallacher, 2003), the two prospective case-control studies carried out in the western part of Berlin ("Berlin I" and "Berlin II") (Babisch et al., 1992, 1994), and the new prospective case-control study carried out in the whole of Berlin ("NaRoMI" = "Berlin III") (Babisch, 2004b; Babisch et al., 2005). The studies refer to road traffic noise and the incidence of myocardial infarction. They were also the only ones considered in an earlier meta-analysis on this issue (van Kempen et al., 2002), with the exception of the "NaRoMI" study, which was not available at that time. All these studies are observational analytic studies (Hennekens and Buring, 1987). If descriptive studies on individuals – namely cross-sectional studies – are allowed, another two studies from Caerphilly and Speedwell on the association between road traffic noise and the prevalence of IHD, myocardial infarction and angina pectoris can be taken into account (Babisch et al., 1988, 1993a, 1993b). These studies were also considered in the meta-analysis by van Kempen et al. (2002). However, the results of the Berlin study on the prevalence of myocardial infarction (Babisch et al., 1994) – which was also considered in that meta-analysis – are not considered here, because the outcome was assessed subjectively with a self-administered questionnaire (an exclusion criterion). All the studies suggest an increase in IHD, in particular myocardial infarction. These studies are used for a new meta-analysis (section 4.5.10).

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Regarding aircraft noise, the cross-sectional Okinawa study (Matsui et al., 2001; Matsui et al., 2004) on the association between aircraft noise and hypertension fulfils the inclusion criteria. When studies are included that did not assess dose–response relationships but only compared dichotomous categories of exposure in the analyses, two more studies appear on the list. The studies were carried out in the vicinity of Amsterdam Schiphol Airport. They suggest a higher risk of cardiovascular diseases in general (Knipschild, 1977b), and – specifically – for hypertension and IHD (angina pectoris, ECG abnormalities, heart trouble) (Knipschild, 1977a) in subjects from areas exposed to high aircraft noise. These studies were considered in the meta-analysis by van Kempen et al. (2002). However, they do not fulfil the strict criteria set here. Finally, if the inclusion criteria are widened to include peer-reviewed studies that assessed dose–response relationships between objective indicators of exposure and the subjective (self-reported) prevalence of diseases, a further two studies can be considered. These are the cross-sectional study carried out in Stockholm regarding the association between aircraft noise and hypertension (Rosenlund et al., 2001), and the cross-sectional part of the study in Berlin regarding the association between road traffic noise and myocardial infarction (Babisch et al., 1994). Fig. 4.4 shows the results of the three aircraft noise studies carried out in Amsterdam, Okinawa and Stockholm (Knipschild, 1977a; Rosenlund et al., 2001; Matsui et al., 2004). The graph clearly indicates that the results are too heterogeneous to derive a pooled dose–response curve. However, all three studies show an increase in risk with increasing noise level.

Studies that are not given a high ranking according to the above mentioned criteria, however, may serve as additional sources of information to support the evidence of the conclusions being made on the basis of this review. This is illustrated in Fig. 4.5. The entries are relative risks (centre of the bars) with 95% confidence intervals (the bars) for dichotomous comparisons of noise exposure (extreme groups or high vs. low). A relative consistent shift of the bars to relative risks greater than 1 can be seen. The dark-shaded bars in the diagram refer to studies where the noise exposure was determined objectively (noise levels), the

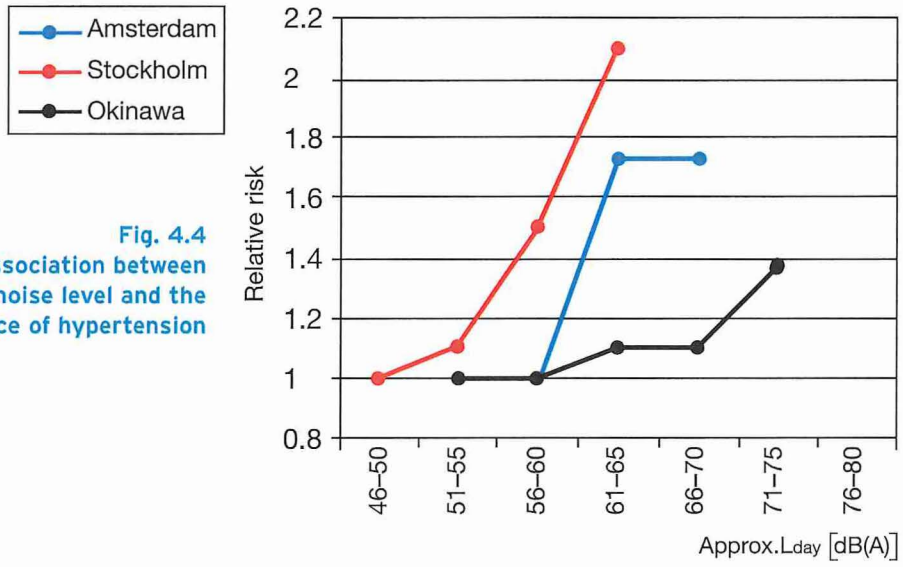


Fig. 4.4
Association between aircraft noise level and the prevalence of hypertension

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light-shaded bars where it was determined subjectively (annoyance). Road traffic and aircraft noise studies are here viewed together. No corresponding results are available for rail traffic studies. If different subgroups of the population (males/females) or different health end points were taken into account, specific studies appear more than once in the illustration.

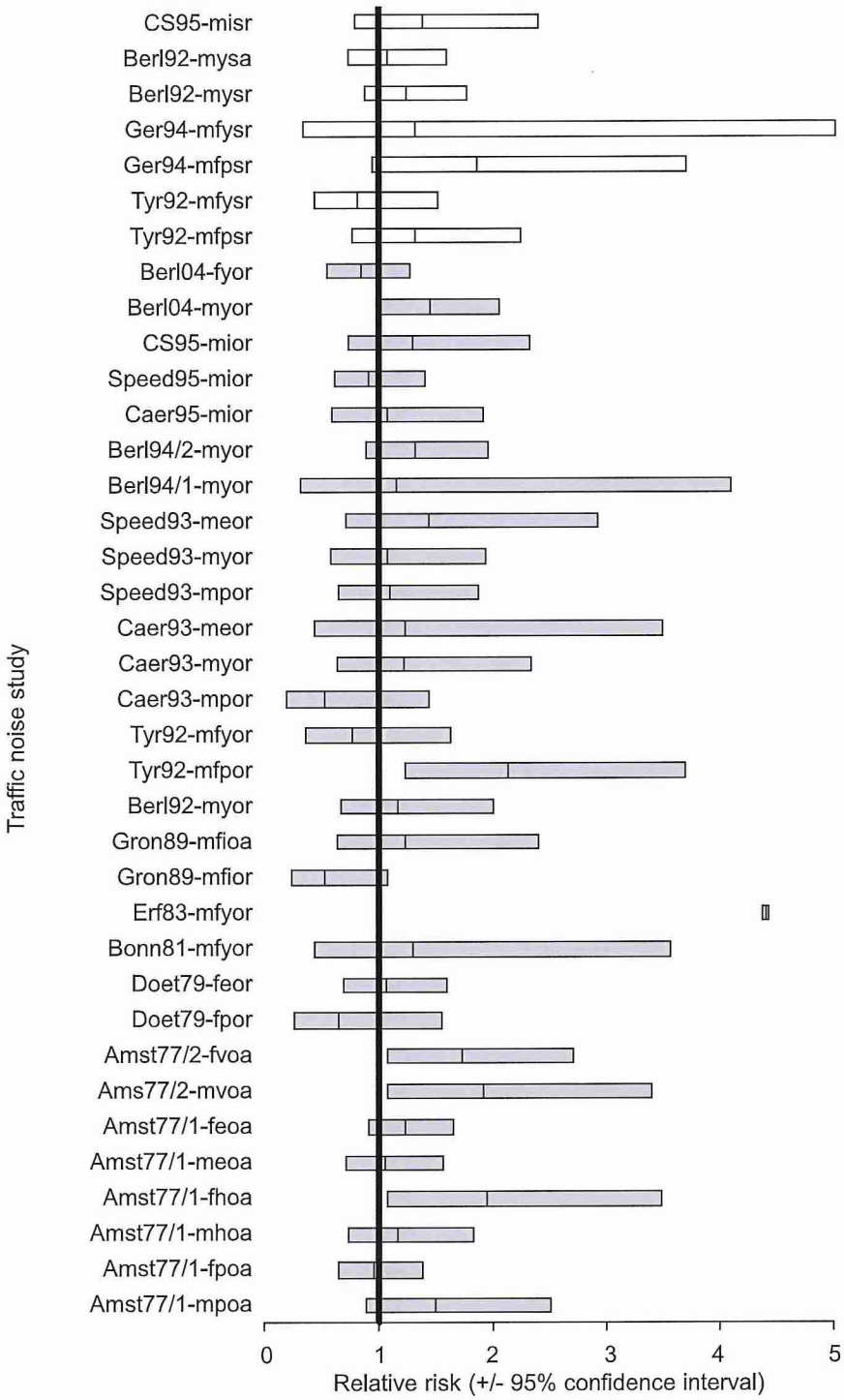


Fig. 4.5
Results of epidemiological studies on the association between traffic noise and ischaemic heart disease

Captions: sex:
f = female,
m = male;
noise measurement:
o = objective (sound level), dark-shaded beam;
s = subjective (annoyance), light-shaded beam;
type of noise:
a = aircraft noise;
r = road traffic noise;
ischaemic heart disease:
e = ECG-ischaemic signs,
h = heart complaints,
i = ischaemic heart disease,
p = angina pectoris,
v = cardiovascular complaints in general, y = heart attack.

Source: Babisch, 2002, modified according to the results of Babisch, 2004b.

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4.5.10 DOSE-RESPONSE CURVE: META-ANALYSIS

For a quantitative risk assessment and the derivation of guidelines for public health noise policy a common dose–response curve is required. The risk estimates obtained from different noise studies can be summarized using the statistical approach of a meta-analysis. Based on the judgement criteria discussed in section 4.5.9.2, five analytic and two descriptive studies emerged that can be used to derive a common dose–response curve for the association between road traffic noise and the risk of myocardial infarction. Two separate meta-analyses were made by considering the analytic studies that were carried out in Caerphilly and Speedwell (cohort studies) and Berlin (case-control studies) on the one hand, and the descriptive studies that were carried out in Caerphilly and Speedwell (cross-sectional studies) on the other hand. It turned out, as a result of the evaluation, that all these studies referred to road traffic noise during the day (L_{day}: 06.00–22.00) and the incidence or prevalence of myocardial infarction as the outcome. Study subjects were men. In all analytic studies the orientation of rooms was considered for the exposure assessment (facing the street or not). With respect to the Caerphilly and Speedwell studies, the six years of pooled follow-up data provided the respective information. In all descriptive studies the traffic noise level referred to the facades that were facing the street and did not consider the orientation of rooms/windows. All individual effect estimates were adjusted for the covariates considered in each of the studies. Different sets of covariates were considered in each study. However, this pragmatic approach accounts best for possible confounding in each study and provides the most reliable effect estimates derived from each study. The concept of meta-analysis was used to aggregate and summarize the findings of the different studies (Olkin, 1995; Blettner et al., 1999). The program "meta" was downloaded from the "STATA" web site for use in the statistical package STATA (version 8.0), and for calculating the pooled random effect estimates.

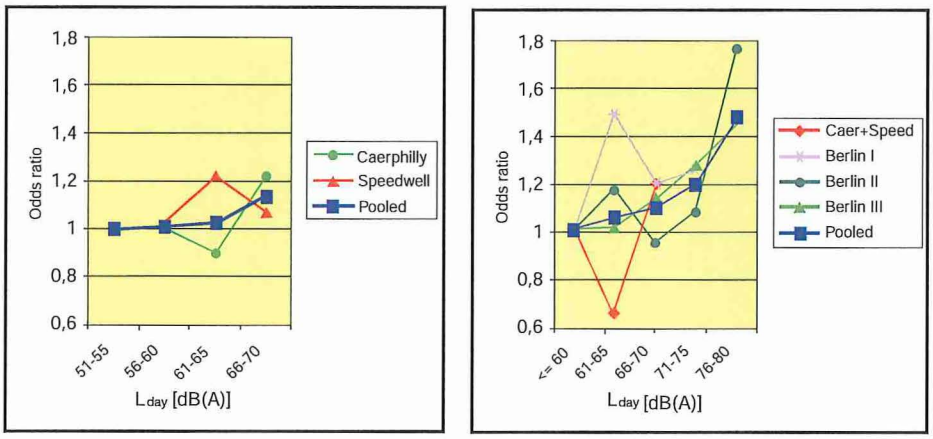
Table 4.2
Single and pooled (meta-analysis) effect estimates (odds ratios and 95% confidence intervals) of descriptive and analytic studies on the relationship between road traffic noise level (L_{day}) and the incidence/prevalence of myocardial infarction

	Road traffic noise level - L_{day} [dB(A)]					
Descriptive studies	**51-55**	**56-60**	**61-65**	**66-70**		**N**
Caerphilly	1.00	1.00 (0.58-1.71),[13.29]	0.90 (0.56-1.44),[17.23]	1.22 (0.63-2.35),[8.98]		2512
Speedwell	1.00	1.02 (0.57-1.83),[11.19]	1.22 (0.70-2.21),[12.62]	1.07 (0.59-1.94),[10.94]		2348
Pooled	1.00	1.01 (0.68-1.50)	1.02 (072-1.47)	1.14 (0.73-1.76)		
Q-Test		p=0.96	p=0.41	p=0.77		
Analytic studies	**60**	**61-65**	**66-70**	**71-75**	**76-80**	**N**
Caerphilly + Speedwell	1.00	0.65 (0.27-1.57),[4.95]	1.18 (0.74-1.89),[17.48]	---	---	3950
Berlin I	1.00	1.48 (0.57-3.85),[4.21]	1.19 (0.49-2.87),[4.94]	1.25 (0.41-3.81),[3.09]	1.76 (0.11-28.5),[0.50]	243
Berlin II	1.00	1.16 (0.82-1.65),[31.43]	0.94 (0.62-1.42),[22.76]	1.07 (0.68-1.68),[18.92]	1.46 (0.77-2.78),[9.27]	4035
Berlin III	1.00	1.01 (0.77-1.32),[54.42]	1.13 (0.86-1.49),[50.87]	1.27 (0.88-1.84),[28.24]	---	4115
Pooled	1.00	1.05 (0.86-1.29)	1.09 (0.90-1.34)	1.19 (0.90-1.57)	1.47 (0.79-2.76)	
Q-Test		p=0.57	p=0.87	p=0.84	p=0.90	

Table 4.2 shows individual and pooled effect estimates with confidence intervals (rounded brackets), statistical weights (square brackets) for the individual studies, and the Q-test of heterogeneity between studies. According to the Q-test, the nil hypothesis of non-heterogeneity was never discarded. Figs 4.6 and 4.7 show odds ratios of individual studies and the pooled estimates for the descriptive and analytic studies.

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Fig. 4.6 and Fig. 4.7
Single and pooled effect estimates (odds ratios) for the descriptive and analytic studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction



4.5.11 EFFECT MODIFICATION

Support for any noise–effect relationship may come from subgroup analyses that are in line with the noise hypothesis. This refers to effect modification with respect to residence time, window-opening behaviour and other determinants that affect the noise exposure and cumulative noise dose. In the Amsterdam aircraft noise studies, a steady increase in the purchase of cardiovascular and antihypertensive drugs at local pharmacies was found over the period of eight years in a community newly exposed to aircraft noise. No such increase was found in a control community that was not exposed to aircraft noise (Knipschild and Oudshoorn, 1977). Positive associations between the prevalence of cardiovascular diseases and residence time in exposed areas (but not in unexposed) were also found in the road traffic noise studies carried out in Bonn with respect to hypertension (Eiff and Neus, 1980; Neus et al., 1983) and in Caerphilly and Speedwell with respect to IHD (Babisch et al., 1999; Babisch, Ising and Gallacher, 2003). When the analyses of the road traffic noise studies carried out in Berlin, Caerphilly and Speedwell were restricted to subjects who had not moved within a retrospective period of 10–15 years, the effect estimates turned out to be larger than for the total samples of each study (Babisch et al., 1994, 1999, 2005). Similarly, a larger effect was found in the study in Sollentuna with respect to hypertension (Bluhm, Nordling and Berglind, 2001). No such effect was found in the Lübeck study (Hense, Herbold and Honig, 1989; Herbold, Hense and Keil, 1989). The cross-sectional data of the study carried out in Los Angeles on children regarding mean BP indicated some habituation to aircraft noise (Cohen et al., 1980). The longer the children were enrolled in the school, the smaller was the difference in BP between exposed and non-exposed children.

However, the follow-up study suggested that this may also be an effect of attrition (Cohen et al., 1981). The longer the families experienced the noise, the more likely that they moved away from the exposed areas (selection bias). In contradiction to this, BP differences between children exposed and not exposed to road traffic noise increased with school grade (Karsdorf and Klappach, 1968). Intervention studies were conducted with respect to changes in BP and changes in air traffic operation (for example the opening/closing of airports or runways). In the Munich study, a

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larger increase in BP was found in children from a noisy area (Evans, Bullinger and Hygge, 1998). Other studies suggested reversible effects on BP when the exposure was lowered (Wölke et al., 1990; Morrell et al., 1998, 2000). In the Tyrol study, significantly lower BP readings were found in subjects who kept the windows closed throughout the night (Lercher and Kofler, 1993, 1996). When the subjects lived close to the highway (within a distance of approximately 500 metres), the prevalence of hypertension was higher in subjects whose bedroom was facing the main road than in those whose bedroom was not facing the main road. The orientation of rooms and window opening was also found to be an effect modifier of the association between road traffic noise and IHD in the Caerphilly and Speedwell studies (Babisch et al., 1999). The relative risk with respect to the noise level was slightly higher in subjects with rooms facing the street and subjects keeping the windows usually open when spending time in the room. A much greater relative risk of hypertension was found in subjects who slept with open bedroom windows in the Spandau Health Survey (Maschke, 2003; Maschke, Wolf and Leitmann, 2003). Hearing impairment was found to be an effect modifier on the association between aircraft noise and hypertension (Rosenlund et al., 2001). Amongst the exposed subjects, a higher risk associated with the noise was only found in subjects without hearing loss.

4.5.12 EXPOSURE DURING THE NIGHT

Unfortunately, epidemiological noise research provides nearly no information regarding the particular impact of noise exposure during the night on cardiovascular health outcomes. The Spandau Health Survey explicitly distinguished between the exposure of the living room (during the day) and the exposure of the bedroom (during the night). There, a slightly higher relative risk of hypertension was found with respect to the traffic noise level during the night (relative risk 1.9 vs. 1.5) compared with the noise level during the day (Maschke, 2003; Maschke, Wolf and Leitmann, 2003). Furthermore, sleeping with open bedroom windows was associated with a large increase in risk. However, due to the small sample size, the confidence intervals were very large. In the drug survey of the Amsterdam aircraft noise studies, a steady increase in purchase of hypnotics (sleeping pills) and sedatives was found (Knipschild and Oudshoorn, 1977). This trend decreased considerably when night flights were largely banned. Such a decrease was not found regarding cardiovascular drugs for which the purchase also increased over time. However, this may partly be due to the fact that atherosclerotic manifestations of high BP were less reversible (in contrast to vasoconstriction, which is more related to acute or semi-acute effects, for instance in children). It was mentioned in the previous section that closing the windows had a protective effect on BP readings in the Tyrol study (Lercher and Kofler, 1993). This was only found regarding closing the windows during the night and not during the day. Furthermore, subjects who had switched the bedroom and the living room because of the noise had a significantly lower BP than those who did not do so. The findings are discussed in a broader context of coping strategies (Lercher, 1996). When subjective responses to community noise were considered, higher relative risks of cardiovascular diseases were found for noise-related disturbances of sleep and relaxation, rather than for other disturbances or subjective descriptors of noise exposure, which did not refer to the night-time. This was found in the Caerphilly and Speedwell studies (Babisch, Ising and Gallacher, 2003), the NaRoMI study (Babisch et al., 2005), the Spandau Health Survey (Maschke, Wolf and Leitmann, 2003) and a general population sample of Germany (Bellach et al., 1995). The LARES study (Niemann and Maschke,

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2004), in which noise-induced sleep disturbance was assessed, did not show a higher relative risk compared with the general annoyance.

4.5.13 RISK GROUPS

Most epidemiological noise studies looked at the cardiovascular effects of community noise in men. This may simply be due to the fact that the prevalence of cardiovascular diseases in middle-aged subjects is higher in men than in women. Statistical power is an important issue for the design of a study. Furthermore, in noise experiments, physiological reactions controlled by the autonomic nervous system were less pronounced in females than in males (Neus et al., 1980; Ising and Braun, 2000). Improper control for possible differential effects of the intake of sex hormones, including contraceptives, which may prevent or promote adverse (noise) stress effects, may act conservatively on the results (Cairns et al., 1985; Eiff, 1993; Farley et al., 1998). The studies carried out in Lübeck (Hense, Herbold and Honig, 1989; Herbold, Hense and Keil, 1989), Pancevo (Belojevic and Saric-Tanaskovic, 2002), Berlin (Babisch et al., 2005), Stockholm (Rosenlund et al., 2001), a German population sample (Bellach et al., 1995), Bonn (residence time) (Eiff and Neus, 1980; Eiff et al., 1981b) and in Amsterdam (angina pectoris) (Knipschild, 1977a) found higher prevalences of hypertension, IHD and the use of cardiovascular drugs in noise-exposed men than in women. The studies carried out in Bonn (sound level) (Eiff and Neus, 1980; Eiff et al., 1981b), Sollentuna and Amsterdam (heart trouble) (Knipschild, 1977a; Bluhm, Nordling and Berglind, 2001) found the opposite. In the studies carried out in the former Soviet Union, it was reported that noise effects on the cardiovascular system were more pronounced in young and middle-aged subjects (Karagodina et al., 1969). Swedish noise studies (Bluhm, Nordling and Berglind, 2001, 2004) and the LARES study (Niemann and Maschke, 2004) found similar results. The opposite (larger effects in elderly subjects) was reported from the Amsterdam study (Knipschild, 1977a) and the Stockholm study (Rosenlund et al., 2001). The available database on cardiovascular effects of noise in children is poor. No data are available that refer, in particular, to noise and sleep. The quantitative impact of transportation noise on the cardiovascular system is still a matter of research. A quantitative health risk assessment for children cannot be made at the moment.

Based on the available information from noise studies, it must be concluded that children do not appear to be a particular risk group with respect to cardiovascular outcomes, especially BP. This does not mean that the literature does not suggest higher BP readings in children. It only means that the effect in children does not appear to be different than that in adults. However, children may be exposed longer to noise throughout their lifetime than the adults that have been studied. No long-term follow-up studies are known that focus on noise exposure. Most studies on children considered noise in schools rather than noise at home, which implies different mechanisms about how noise could contribute to a rise in BP (raised effort in learning/speech perception vs. disturbed relaxation/sleep). The prospective part of the Caerphilly and Speedwell studies gave a small hint that health status could be a modifying factor. In subjects with prevalent chronic diseases, road traffic noise was associated with a slightly larger increase in the incidence (new cases) of IHD than in subjects without prevalent diseases – when the objective noise level was considered (Babisch et al., 2003). Surprisingly, when annoyance and disturbances due to traffic noise were considered for exposure, the opposite was found. Noise effects were only seen in subjects without prevalent diseases. This was discussed with respect to reporting bias.

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4.5.14 RISK EVALUATION

The process of risk assessment (risk evaluation) comprises hazard identification ("Which health outcome is relevant for the exposure?"), exposure assessment ("How many are affected?") and dose–response assessment ("threshold of effect"). This information is summarized in "risk characterization" ("health hazard characterization"). It involves the interpretation of the available evidence from the available data and other scientific disciplines, and is subject to discussion of the uncertainties. These include chance, bias and validity of studies as well as transparency, replicability and comprehensiveness of reviews. As a result of the risk evaluation process, a quantitative estimate about the likelihood that the hazard will affect exposed people will be derived. Usually, attributable risk percentages are calculated (Walter, 1998). This will serve as key information for any kind of risk management including regulatory options (Jasanoff, 1993). The term "adverse" is essential in this context of environmental standard setting. Risk management should ensure that "adverse" health effects do not occur. The fact that an organism responds to noise does not have to be per se "adverse". The severity of a health outcome is an important determinant of the adversity of an effect and implies variable action levels for public health policy (Babisch, 2002, 2004a; Griefahn et al., 2002; Health Council of the Netherlands, 2003). Since considerable parts of the population are exposed to high noise levels (EEA, 2004), noise policy can have a significant impact on public health (Neus and Boikat, 2000). Due to the increasing number of people affected with the decreasing severity of the effect, even small individual risks and less severe health outcomes can be relevant for public health and decision-making. It has been shown that moderate noise exposures implying a small individual risk may cause more noise-induced cases of health-impaired subjects than higher noise exposures. Franssen et al. (2004) pointed out that the number of people suffering from poor health due to aircraft noise is dominated by the larger number of people that is exposed to relatively moderate-to-low noise levels and not by those exposed to high noise levels. This means that more emphasis should be put on the reduction of noise in moderately exposed areas. However, public health policy cannot only consider population attributable risks (risk percentages), but must also consider individual risks (lifetime risk).

In practice, it seems to be reasonable that noise policy should reduce noise, beginning with the highest exposures and ending with the lowest ones. Decision-making will have to find common standards of acceptable risks, which may vary according to the cost–benefit considerations within and between communities and countries. Such practical standards may, however, vary due to economic development and abilities, cost–benefit considerations and priority settings of a community or country. Health quality targets derived from scientific research are usually intended to minimize risks; decision-making in the political process is only partly scientifically based due to economic limitations and concurring interests (Nijland et al., 2003). Different health outcomes or indicators of well-being and quality of life imply different action levels. Environment and health policy must determine acceptable noise standards that consider the whole spectrum from subjective well-being to somatic health (for example annoyance, physiological arousal, health risk). The evidence for a causal relationship between community or transportation noise and cardiovascular risk appears to have increased over recent years due to new studies that accomplish the database.

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4.5.15 CONCLUSIONS

The evaluation process used in this paper considered the "necessary" criteria: peer-reviewed publication in an international journal, reasonable quantitative control of possible confounding, objective assessment of exposure and outcome, type of study (analytic vs. descriptive), and dose–response assessment (not only dichotomous "high" vs. "low"). The approach differs from that of an earlier meta-analysis (van Kempen et al., 2002) in that there regression coefficients were calculated for the entire dose–response curve within a single study (for instance the increase in risk per 5 dB(A)), which were then pooled between studies. Since higher exposure categories usually consist of smaller numbers of subjects than the lower categories, regression coefficients across noise levels tend to be influenced by the lower categories. This may lead to an underestimation of the risk in higher noise categories. The approach presented here pooled the effect estimates of single studies within each noise category, thus giving more weight to the higher noise categories and accounting for possible non-linear associations.

Fig. 4.8 and Fig. 4.9
Pooled effect estimates (meta analysis) of descriptive and analytic noise studies of the association between road traffic noise level and the prevalence (left graph) and incidence (right graph), respectively, of myocardial infarction (odds ratio ± 95% confidence interval).

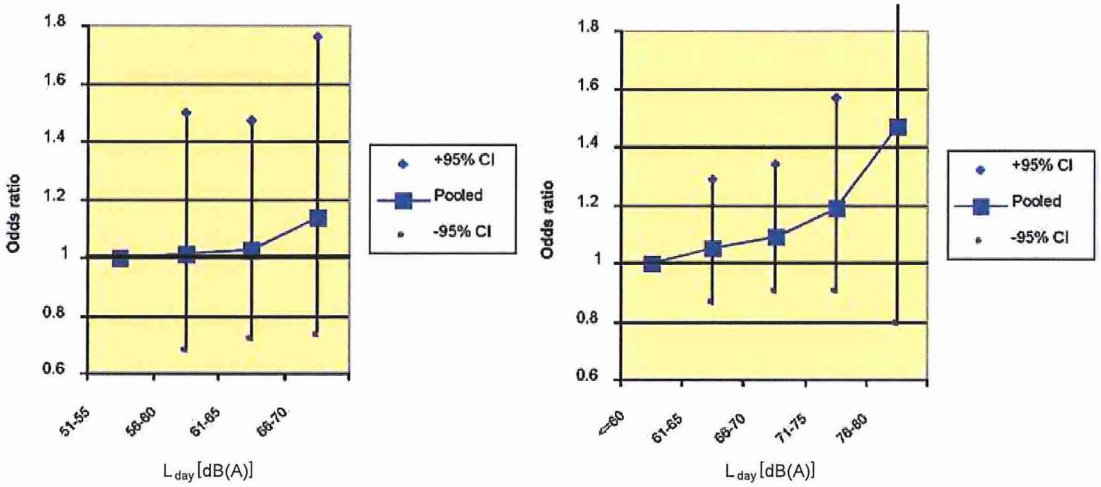


Fig. 4.8 and Fig. 4.9 show the two risk curves for descriptive and analytic studies (Hennekens and Buring, 1987). The graphs show the pooled effect estimates (odds ratios) and the 95% confidence intervals for each noise category. Whereas the cross-sectional studies (Fig. 4.9) cover the sound level range of L_{day} from >50 to 70 dB(A), the cohort and case-control studies (Fig. 4.8) cover the range from ≤60 to 80 dB(A). Both curves together can serve as a basis for a quantitative risk assessment. From Fig. 4.8 it can be seen that below 60 dB(A) for L_{day} no noticeable increase in risk of myocardial infarction is to be detected. Therefore for the time being, L_{day} = 60 dB(A) can be seen as NOAEL (no observed adverse effect level) for the relationship between road traffic noise and myocardial infarction (Babisch, 2002). For noise levels greater than 60 dB(A), the risk of myocardial infarction increases continuously, and is greater than 1.2 for noise levels of 70 dB(A). This can be seen in Fig. 4.9. It should be mentioned that the risk estimates, in general, were found to be higher in subjects that had lived in the exposed areas for a longer time (Babisch et al., 1994, 1999, 2005). This is in accordance with the noise hypothesis and the effects of chronic noise stress (Lercher and Kofler, 1996; Thompson, 1997). However, for the calculation of population attributable risks the figures for the whole population are relevant due to unknown information about residence time.

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No particular risk groups could be identified on the basis of epidemiological research on cardiovascular effects of community noise. The assessment of dose-effect relationships sometimes suggested a cut-off level, above which the risk tends to increase. From a biological point of view, one would expect a continuous increase in risk with increasing noise level. However, adaptation, habituation and coping may be reasons for an empirical threshold of effect. Decisions with respect to guideline values usually refer to a quantitative risk assessment of populations (for example population attributable risk percentage). However, prevention strategies – for ethical reasons – should not ignore the individual risks of highly exposed subjects, even if their number may be small.

With respect to night noise exposure, nearly no information is available from epidemiological studies on the cardiovascular effects of long-term noise exposure of the bedroom during the night. Only one study distinguished between the exposures of the bedroom and the living room in the statistical analyses (Maschke, Wolf and Leitmann, 2003). The results suggested slightly higher effect estimates for the prevalence of hypertension with respect to the noise exposure of the bedroom (during the night) compared with the exposure of the living room (during the day). However, the difference was small (odds ration 1.9 vs. 1.5), which means that it still remains an open question whether the night exposure or the overall exposure throughout the whole day is the driving force. The study has some methodological limitations that were addressed in the summary of the major technical report and in a recent advisory report of the Health Council of the Netherlands (2004). They are mainly concerned with the fact that the study population consisted of a selected, predominantly older and health conscious group of persons that might have already suffered from regular health problems (risk group). A few studies that looked at the association between subjective responses to community noise and cardiovascular outcomes suggest a closer relationship with sleep-related annoyance/disturbance reaction rather than with non-sleep-related annoyance/disturbance (Bellach et al., 1995; Babisch et al., 1999, 2005; Maschke, Wolf and Leitmann, 2003; Niemann and Maschke, 2004). Closing the bedroom window or, vice versa, sleeping with the bedroom window open, was associated with a lower or higher risk, respectively (Lercher, 1996). The same was found with respect to changing the bedroom to the living room because of noise. These findings may indicate that night-time noise may be more a determinant of noise-induced cardiovascular effects than daytime exposure. However, daytime activity patterns and expectations of the individuals are much more inhomogeneous than during the night, which tends to dilute the statistical association of true effects with the day noise exposure.

Given the situation that only a few data are available from epidemiological studies with respect to effects on sleep (exposure of the bedroom during the night), there does not seem to be any other way of reasoning than inferring night noise recommendations or guidelines from the results of studies that refer to noise exposure during the daytime period (L_{day}) or the whole day (L_{dn}, L_{24h}). L_{den}, in this context, appears to be a useful noise indicator for decision-making and regulatory purposes. Penalties of 5 dB(A) and 10 dB(A) are usually given to the evening period and the night period, respectively. It can be used for noise mapping and refers normally to the most exposed facade, which incorporates a certain degree of exposure misclassification regarding cause–effect relationships. This weighted indicator was introduced to assess the relationship between sound level and noise annoyance (European Commission, 2002a). However, it may not be adequate for research into (somatic) health-related noise effects. Non-weighted separate exposure indicators, such as L_{day}, $L_{evening}$ or L_{night}, may be more appropriate when assessing physiological responses to the noise. In urban settings, night-time average noise levels (22.00–06.00) for road traffic tend to be approximately 7–10 dB(A) lower than daytime average noise levels – relatively independent (no freeways) of the traffic volume of the street (Utley, 1985; Ullrich, 1998; Evans et al., 2001). In such cases, L_{den} is approximate-

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ly 2–3 dB(A) higher than L_{day} (Bite and Bite, 2004). Therefore, in epidemiological studies in which the relative effects of road traffic noise are studied, the sound emission during the daytime can as well be viewed as an approximate relative measure of the overall sound emission including the night. This seems to be further justified because existing noise regulations usually consider a 10 dB(A) difference between the day and the night. The NOAEL of 60 dB(A) for L_{day} corresponds, in this respect, with 50 dB(A) for L_{night}. This approximation can only be made with respect to road traffic noise.

Aircraft noise has been less intensively studied in noise epidemiology. The studies focused on high BP. Dose–response curves were hardly considered. A large European study on the association between aircraft noise and road traffic noise on BP is currently being conducted (Jarup et al., 2003). Regarding aircraft noise – and particularly the ongoing debate on night flight restrictions in the vicinity of busy airports – no other alternative exists at present than to take the myocardial infarction risk curves derived from road traffic noise studies as an approximate for aircraft noise. Since aircraft noise acts on all sides of a building, that is, different to road traffic noise, the suspicion exists that the effects induced by aircraft noise could be greater than those induced by road traffic (Ortscheid and Wende, 2000; Babisch, 2004a). This may be due to the lack of evasive possibilities within the home and the greater annoyance reactions to aircraft noise, which are usually expressed in social surveys (Miedema and Vos, 1998). More research is needed regarding the association between aircraft noise and cardiovascular end points.

This section is clearly focused on ill health as an outcome of the adverse effect of noise. A common dose-effect curve for the relationship between road traffic noise (outdoors) and the risk of myocardial infarction was developed. This curve can be used for a quantitative risk assessment and the calculation of attributable cases in a community. However, decisions regarding limit values have to be made within the spectrum between discomfort (annoyance) and ill health (disease) (Lindström, 1992; Babisch, 2002). Whereas quality targets at the lower end of the effects scale may be more flexible, quality targets at the upper end may be more obligatory. For example, for ethical reasons (equality principle) it does not seem to be justified if (ill) health-based limit values are varied according to the type of living area as expressed in land development plans (for example residential, mixed or commercial).

4.6 INSOMNIA

A group of Japanese researchers carried out a questionnaire-based survey of 3600 adult Japanese women (aged between 20 and 80) to gather information about the factors that contribute to insomnia (Kageyama et al., 1997). Some 11% of subjects were found to be affected by insomnia (as defined on the basis of WHO's International Statistical Classification and Related Health Problems, 10th revision – ICD10). Analysis of the survey data took account of various distorting variables, such as age, number of (small) children in the family, social status, receipt of medical treatment, regularity of bedtimes, apnoea-like problems and serious unpleasant experiences in the six months prior to completing the questionnaire. When the percentage of insomniacs in each of the three areas with the highest exposures was compared with the percentage in the low-exposure areas, the ratios worked out at, respectively, 1.4 (2100 vehicles per hour, L_{night} of around 65 dB(A)), 2.1 (2400 vehicles per hour, L_{night} of around 67 dB(A)) and 2.8 (6000 vehicles per hour, L_{night} of around 70 dB(A)). The most frequently reported problem was difficulty getting to sleep.

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Research into the effects of exposure to air and road traffic noise has shown that increases in night-time noise exposure or in noise exposure during the sleep latency period have a statistically significant adverse impact on subjects' ability to get off to sleep and on sleep inception periods.

4.7 EFFECTS ON PERFORMANCE

4.7.1 COGNITION AND SWS

Jan Born and co-workers at the University of Lübeck (Wagner, Gais and Born, 2001; Benedict et al., 2004; Born and Wagner, 2004; Gais and Born, 2004; Drosopoulos, Wagner and Born, 2005) have reported interesting research and put forward intriguing hypotheses on the relation between noise exposure, sleep loss and subsequent cognitive performance. They conclude that declarative memory benefits mainly from sleep periods dominated by SWS, while there is no consistent benefit for this memory from periods rich in REM sleep. This points to the importance of SWS for declarative memory.

Since sleep in the early night is dominated by SWS, in contrast to late night when REM sleep dominates, this would imply that noise in the early night, for example aircraft noise before midnight, would be particularly damaging to memory and related cognitive functions. However, this implication has not yet been explicitly tested. That is, there seems to be a certain risk for impoverished memory due to noise in the early night, but there is as yet no graded quantification about whether ordinary pre-midnight noise levels around large airports are sufficient to make a difference to SWS. We also lack graded quantification about the relationship between impoverished SWS and the resulting effect on different aspects of declarative memory.

Thus, in terms of Fig. 1.1 we have evidence for the arrow marked (b), but we do not have enough information to say whether the strength of arrow (a) is sufficient to cause reduced SWS in field settings.

Furthermore, since children's memory systems pass through developmental changes and are not structured in the same way as for adults, it would be interesting to know to what extent the Born group results are also valid for children, and whether the depth of children's sleep counteracts or enhances SWS dominance in the early night.

4.7.2 COMPARING DAYTIME AND NIGHT-TIME NOISE EXPOSURE

As implied by Fig. 1.1, the relation between noise exposure and resulting effects on cognition should be analysed somewhat differently depending on whether the noise exposure takes place during the day or night. Analysing the cognitive effects of daytime noise exposure is fairly straightforward. For night-time noise exposure, however, any effects on cognition can either be a more or less direct effect of the noise exposure, or an indirect effect mediated by reduced sleep or sleep quality.

Also, comparing, for example, memory and learning functions when exposed to

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night-time noise, in contrast to daytime noise, shifts the focus of analysis away from encoding (in memory) or acquisition (in learning) while experiencing noise, to a focus on storing the material to be remembered or learnt while asleep (compare to daytime noise effects on cognition as reported by Hygge, Evans and Bullinger, 2002; Stansfeld et al., 2005). Thus, assuming that people are mainly asleep at night, all cognitive work that relies on the intake of information, listening or reading is not relevant. In all, this suggests that studies of daytime noise levels cannot be used much to give rough estimates of the effects of night-time exposure.

4.7.3 COMPARING CHILDREN AND ADULTS

How far can effects of daytime noise levels on children be generalized to give a rough estimate of the effects on adults? Are children more sensitive? Judging from earlier daytime studies of children and adults doing the same cognitive tasks while exposed to noise, children are not more sensitive than adults to noise (Boman, Enmarker and Hygge, 2004), but they perform at a lower level than the adults both in noisy and in quiet environments. Thus, it could be said that children are not more vulnerable to (daytime) noise in relation to cognitive performance, but since so much more cognitive work is expected from children while in school, their learning environment and their cognitive tasks can be said to be more noise vulnerable than corresponding environments for adults.

4.7.4 NOISE AND AFTER-EFFECTS

An argument can be made for noise as a stressor leading to reduced motivation (Glass and Singer, 1972), which in turn may act as a mediator of impaired cognitive performance. Along this line of reasoning, night-time noise may be more potent in inducing reduced motivation than daytime noise, but for the time being this is only a conjecture and has not been tested.

4.7.4.1 The role of restoration

Noise can be viewed both as a source of stressful demands and as a constraint on restoration. Noise levels and noise sources that are not by themselves particularly demanding during the waking hours of the day, may nevertheless be quite effective in blocking and constraining when they appear in periods meant to be restorative, such as sleep (Hartig, 2004). To what extent this idea is applicable to night-time noise exposure has not yet been explored.

4.7.4.2 Noise and communication

Some of the difficulties with children's responses to noise are related to problems in speech perception. A metric that weights night-time exposure more heavily is, in fact, less useful since children's auditory processing with parents and teachers is obviously more critical during waking hours.

4.8 EFFECTS ON PSYCHIC DISORDERS

Noise exposure at night may be more disturbing than daytime noise because it interferes with rest and sleep at a time when people want to relax. It seems plausible that night-time noise might have a particular effect on mental health. However, there is lit-

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tle direct research into night-time noise and mental health and it is first necessary to consider the evidence for environmental noise and mental health in general. The association between noise and mental health has been examined using a variety of outcomes including (at the simplest level) individual symptoms, as well as psychiatric hospital admission rates, use of health services and psychotropic medication, and community surveys.

4.8.1 TRANSPORTATION NOISE AND MENTAL HEALTH

Sources of transportation noise that have been studied in relation to mental health include road traffic noise and aircraft noise. Studies relating to each type of noise will be considered in turn.

4.8.1.1 Road traffic noise

The association between road traffic noise exposure and psychological distress has been studied in the small town of Caerphilly, South Wales. In the cross-sectional results, no association was found between the initial level of road traffic noise based on traffic noise maps, in terms of L_{eq} referring to the period 06.00–22.00, and minor psychological distress, measured by the General Health Questionnaire (GHQ), a screening questionnaire for depression and anxiety, even after adjustment for sociodemographic factors (Stansfeld et al, 1993). In longitudinal analyses in the Caerphilly Study, no association was found between road traffic noise and psychological distress, even after adjustment for sociodemographic factors and baseline psychological distress, although there was a small non-linear association of noise with increased anxiety scores (Stansfeld et al, 1996).

The disadvantage of the Caerphilly study is that it relied on one location with not very high levels of traffic noise. In a secondary analysis of a large British road traffic noise study, which took into account multiple noise exposure sites, the noise level in dB(A) exceeded for 10% of the time was weakly associated with a mental health symptoms scale of five items adjusting for age, sex, income and length of residence (Halpern, 1995). Weaker associations between traffic density and the mental health symptoms scale may relate to the skewed distribution of this traffic density variable. It seemed that traffic noise was more important than traffic flow. The scale used included some clear mental health items but also some that were less obviously related to mental health. It may be questioned whether the reported association between noise level and mental health symptoms was actually due to noise exposure; adjustment for the amount of "noise heard" reduced the association very little, suggesting no causal association with noise, but it is likely that there was a good deal of error in the measurement of this variable, reducing its validity.

It may be that the peak noise level is a better indicator of environmental noise heard indoors than noise measures averaged over time and that peak levels are a crucial indicator for mental health. Furthermore, in a road traffic noise study in Belgrade, 253 residents exposed to road traffic noise levels of >65dB(A), with high levels both day and night (L_{eq} 76.5 in the day, 69.5 at night in the noise-exposed area), experienced significantly more fatigue, depression, nervousness and headaches, compared to residents exposed to <55dB(A) (Belojevic and Jakovljevic, 1997). Sleep quality was also found to be worse among the inhabitants of noisy streets, compared to inhabitants of quiet streets, and those living in noisy streets had more difficulties falling asleep, more night awakenings and more pronounced tiredness after sleep. However, there were no differences in time taken to fall asleep or to go back to sleep,

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duration of sleep or consumption of sleeping pills between noise-exposed and non-exposed residents. A great methodological advantage of this study was that the high and low noise exposure areas were homogenous for age, sex, employment and subjective noise sensitivity. A community study in 366 Japanese women suggests that road traffic noise only has effects on depression, fatigue and irritability above a threshold of 70 dB(A) (Yoshida et al., 1997). However, it is difficult to be confident of the results of these analyses as they were unadjusted for age or social deprivation. Milder psychological states such as health functioning and well-being have also been examined in the first stage of an intervention study on the effect of introducing a bypass to relieve traffic congestion in a small town in North Wales (Stansfeld, Haines and Brown, 2000). Health functioning was measured by the SF-36 General Health Survey (Ware and Sherbourne, 1992), including dimensions of general health status, physical functioning, general mental health and social functioning. Ninety-eight respondents were studied who lived on a busy high street with traffic noise levels varying between 72 and 75 dBA outdoor L_{eq}. These respondents were compared with 239 control subjects living in adjacent quieter streets (noise level 55–63 dB(A) outdoor L_{eq}). Although subjects were well-matched on age, sex, housing insulation, car ownership and employment status, they were not so well-matched on proportion of manual workers, household crowding, deprivation and home ownership. There was no evidence that respondents exposed to higher levels of road traffic noise had worse health functioning than those exposed to lower levels of the noise, adjusting for levels of deprivation.

Another method of assessing mental health effects related to noise exposure is to use an indirect indicator such as medication use. In five rural Austrian communities exposed to road traffic noise, noise levels above 55 dB(A), including increasing night-time exposure to noise from trucks, were associated with increased risk of taking sleeping tablets (OR = 2.22 [CI: 1.13–4.38]) and overall prescriptions (OR = 3.65 [CI: 2.13–6.26]) relative to road traffic noise exposure less than 55 dB(A) (Lercher, 1996). This suggested effects at fairly low noise levels. In this case mental ill health may be secondary to sleep disturbance, which is likely to occur at lower nocturnal noise levels than mental health symptoms resulting from daytime noise exposure. As this occurred in a rural setting where road traffic was the predominant source of noise it would be interesting to replicate these findings in other settings.

4.8.1.2 Road traffic noise and mental health in children

Noise exposure and mental health has also been studied in children where child self-reported mental health on a standard scale and teacher ratings of classroom adjustment in response to motorway, road and rail noise were measured in a large sample of 8–11-year-old Austrian primary school children and in a second stage sample of extreme noise-exposed groups. Noise exposure was significantly associated with classroom adjustment scores but, intriguingly, child self-reported mental ill health was only impaired in noisy settings for children of low birth weight and preterm birth (Lercher et al., 2002).

4.8.1.3 Aircraft noise

Community surveys have found that high percentages of people reported "headaches", "restless nights", and "being tense and edgy" in high aircraft noise areas (Kokokusha, 1973; Finke et al., 1974; Öhrström, 1989). An explicit link between aircraft noise and symptoms emerging in such studies raises the possibility of a bias towards over-reporting of symptoms (Barker and Tarnopolsky, 1978). Notably, a study around three Swiss airports (Grandjean et al., 1973), did not mention that it was related to aircraft noise and did not find any association between the

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level of exposure to aircraft noise and symptoms. In the West London Survey, "tinnitus", "burns, cuts and minor accidents", "ear problems" and "skin troubles" were all more common in areas of high noise exposure (Tarnopolsky, Watkins and Hand, 1980). Acute symptoms such as "depression", "irritability", "difficulty getting off to sleep", "night waking", "skin troubles", "swollen ankles" and "burns, cuts and minor accidents" were particularly common in high noise areas. However, apart from "ear problems" and "tinnitus", 20 out of 23 chronic symptoms were more common in low noise environments. Symptoms did not increase with increasing levels of noise. This is possibly related both to more social disadvantage and associated ill health among residents in low aircraft noise exposure areas and the possible unwillingness of chronically unhealthy individuals to move into potentially stressful high noise exposure areas. Nevertheless, it would not exclude an effect of noise in causing some acute psychological symptoms. As the majority of aircraft noise exposure is during the day, daytime exposure is likely to have greater effects than nighttime exposure. Many of the effects of noise in industrial and teaching settings may be related primarily to disturbances in communication.

4.8.2 NOISE EXPOSURE AND MENTAL HOSPITAL ADMISSION RATES

Much of the concern with the possible effects of noise on mental health began with the study of admissions to psychiatric hospitals from noisy areas. Early studies found associations between the level of aircraft noise and psychiatric hospital admissions, both in London (Abey Wickrama et al., 1969) and Los Angeles (Meecham and Smith, 1977). These results have been criticized on methodological grounds (Chowns, 1970; Frerichs, Beeman and Coulson, 1980) and a replication study by Gattoni and Tarnopolsky (1973) failed to confirm these findings. Jenkins et al., (1979) found that age-standardized admission rates to a London psychiatric hospital over four years were higher as the level of noise of an area decreased, but lower noise areas were also central urban districts, where high admission rates would be expected. In a further extensive study of three hospitals (Jenkins, Tarnopolsky and Hand, 1981), high aircraft noise was associated with higher admission rates in two hospitals, but in all three of them, admission rates seemed to follow non-noise factors more closely; the effect of noise, if any, could only be moderating that of other causal variables but not overriding them. Kryter (1990), in a re-analysis of the data, found "a more consistently positive relation between level of exposure to aircraft noise and admissions rates". Undoubtedly, the route to hospital admission is influenced by many psychosocial variables that are more potent than exposure to noise. Therefore, whether or not noise causes psychiatric disorder is more suitably answered by studying a community sample.

4.8.3 NOISE EXPOSURE AND PSYCHIATRIC MORBIDITY IN THE COMMUNITY

In a community pilot study carried out in West London, Tarnopolsky et al. (1978) found no association between aircraft noise exposure and either GHQ scores (Goldberg, 1972) (dichotomized 4/5, low scorers/high scorers) or estimated psychiatric cases (Goldberg et al., 1970). This was the case even when exposure to road traffic noise was controlled, except in three subgroups: persons "aged 15–44 of high education" (41%, 14% $p<0.05$), "women aged 15–44" (30%, 13% n.s.), and those in "professional or managerial occupations". The authors expressed the guarded opinion that

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noise might have an effect in causing morbidity within certain vulnerable subgroups. In the subsequent West London Survey of Psychiatric Morbidity (Tarnopolsky, Morton-Williams and Barker, 1980), 5885 adults were randomly selected from within four aircraft noise zones, according to the Noise and Number Index. No overall relationship was found between aircraft noise and the prevalence of psychiatric morbidity either for GHQ scores or for estimated numbers of psychiatric cases, using various indices of noise exposure. However, there was an association between noise and psychiatric morbidity in two subgroups: "finished full-time education at age 19 years +", and "professionals". These two categories, which had a strong association with each other, were combined and then showed a significant association between noise and psychiatric morbidity ($X2 = 8.18$, df 3 $p<0.05$), but only for the proportion of high GHQ scorers. Tarnopolsky, Morton-Williams and Barker (1980) concluded that their results "show so far that noise per se in the community at large, does not seem to be a frequent, severe, pathogenic factor in causing mental illness but that it is associated with symptomatic response in selected subgroups of the population".

More recent studies have examined the effects of higher levels of military aircraft noise. Exposure to higher levels of military aircraft noise around the busy Kadena military airport in Japan was related in an exposure–effect association to depressiveness and nervousness measured by questionnaire using the Todai Health Index, based on the Cornell Medical Index (Ito et al., 1994; Hiramatsu et al., 1997). Mental health subscales included in this study measured depressiveness, nervousness, neurosis, and mental instability. Noise level was expressed as WECPNL (the power average of the maximum perceived noise exposure level in dB(A)) from 75–79, 80–84, 85–89, 90–94 and over 95). In unadjusted analyses, statistically significant differences were found in scores of depressiveness, nervousness and neurosis between the non-noise exposed control group and the pooled group exposed to 75–95 WECPNL. In multivariate analysis adjusting for age, sex, marital status, type of house and length of residence, noise exposure greater than 95 WECPNL was associated with higher scores on depressiveness and neurosis (Hiramatsu et al., 1997). Clear exposure–effect relationships were not found between scale scores and noise exposure, as expressed in five unit steps. However, using more broadly defined groups, an exposure–effect association was evident. This highlighted differences between the highest noise exposure group and lower exposure groups and indicated a threshold effect rather than a linear relationship – that mental health effects are more likely to be found at higher noise levels. In general, psychological rather than somatic symptoms were more related to noise in this study. Further analyses of the Japanese studies suggest that high levels of military aircraft noise may have effects on mental health. In a cross-sectional study of 5963 inhabitants around two air bases in Okinawa, those exposed to noise levels of L_{dn} 70 or above had higher rates of "mental instability" and depressiveness (Hiramatsu et al., 2000). Those who were more annoyed showed a higher risk of mental or somatic symptoms. A further survey using similar methodology on 6486 respondents found exposure–effect associations between aircraft noise exposure, nervousness and mental health (Miyakita et al., 1998). These are important studies because of the opportunity to examine the effect of high noise-exposure levels and the probability that vulnerable people migrating out of noisy areas and thus biasing the sample was small.

The use of health services has also been taken as a measure of the relationship between noise and psychiatric disorder. Grandjean et al. (1973) reported that the proportion of the Swiss population taking drugs was higher in areas with high levels of aircraft noise and Knipschild and Oudshoorn (1977) found that the purchase of sleeping pills, antacids, sedatives and antihypertensive drugs all increased in a vil-

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lage newly exposed to aircraft noise, but not in a "control" village where the noise level remained unchanged. In both studies, there was also an association between the rate of contact with general practitioners and level of noise exposure. In the Heathrow study (Watkins, Tarnopolsky and Jenkins, 1981), various health care indicators were used – use of drugs, particularly psychiatric or self-prescribed, visits to the GP, attendance at hospital, and contact with various community services – but none of these showed any clear trend in relation to levels of noise. A recent study found that the use of sleeping tablets and sedatives was elevated with increasing night-time noise exposure, especially in the elderly (Passchier-Vermeer et al., 2002). This has been judged to be "sufficient" evidence of a noise effect (Health Council of the Netherlands, 2004).

4.8.4 AIRCRAFT NOISE EXPOSURE AND MENTAL HEALTH IN CHILDREN

Poustka, Eckermann and Schmeck (1992) studied the psychiatric and psychosomatic health of 1636 children aged 4–16 in two geographical regions that differed according to the noise made by jet fighters frequently exercising at low altitude. Psychological and neurological outcomes were not related to noise exposure. They found that associations between noise exposure and depression and anxiety could be demonstrated, but only beneath the threshold of clinical significance. These results are less convincing because the areas differed socioeconomically and the results were not adjusted for these factors and also because of lack of precision of the measures of noise exposure. However, in Munich, children living in areas exposed to high aircraft noise had lower levels of psychological well-being than children living in quieter environments (Evans, Hygge and Bullinger, 1995). The longitudinal data from around Munich showed that after the inauguration of the new airport, the newly noise-exposed communities demonstrated a significant decline in self-reported quality of life measured on the Kindl scale, after being exposed to the increased aircraft noise for 18 months (third wave of testing), compared with a control sample (Evans, Bullinger and Hygge, 1998). Impairment of "quality of life" is a less severe disturbance than impairment of mental health. Further studies have examined the effects of noise on child psychiatric disorders.

Chronic aircraft noise exposure was not associated with anxiety and depression (measured with psychometrically valid scales), after adjustment for socioeconomic factors, in the Schools Health and Environment Study around Heathrow Airport (Haines et al., 2001a). In a further larger study of children's health around Heathrow Airport – the West London Schools Study (Haines et al., 2001b) – an association was found between aircraft noise exposure level and increased hyperactivity scores on the hyperactivity subscale of the Strength and Difficulties Questionnaire (Goodman, 1997). These studies suggest that noise influences child mental health in terms of hyperactivity and that it may affect child stress responses and sense of well-being.

4.8.5 NEIGHBOURHOOD NOISE AND MENTAL HEALTH

Noise from neighbours is the commonest source of noise complaints to local authorities in the United Kingdom (Chartered Institute of Environmental Health, 1999). Noise which is continuous, apparently indefinite, of uncertain cause or source, emotive or frightening or apparently due to thoughtlessness or lack of consideration is most likely to elicit an adverse reaction (Grimwood, 1993). In the 1991 BRE survey,

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people most objected to barking dogs, banging doors, noise from radio, television, or hi-fi and human voices (Grimwood, 1993). In this survey, two types of emotional response to noise were observed: outwardly directed aggression, characterized by feelings of annoyance, aggravation, bitterness and anger towards the source of the noise, and a more emotional response of tension, anxiety and feelings of pressure. These responses are reminiscent of the distinction between internalizing and externalizing disorders. Whether noise from neighbours can induce psychiatric disorder has been little studied in community research, but this is an area that deserves further study (Stansfeld, Haines and Brown, 2000).

Undoubtedly, prolonged exposure to noise can be very upsetting, intrusive and interfering for sleep and everyday activities. In poorly built dwellings, especially apartments, even low intensity noises may be clearly audible through walls, floors, or ceilings (Raw and Oseland, 1991). In this situation, noise is destructive of privacy, especially for those living alone, and may be associated with perceptions of threat or increase a sense of isolation. This may be especially the case among people who are chronically anxious and likely to complain of sensitivity to noise; prolonged noise exposure may make them more anxious and unhappy. Often, this leads to arguments with neighbours, leading to a breakdown of neighbourly relationships and further isolation which may well in itself have a bad effect on mental health. Occasionally, this may be a sign of feelings of persecution associated with psychotic illness in which noise exposure is just an external trigger of an internally generated condition.

4.8.6 MECHANISMS FOR CAUSAL LINKS BETWEEN NOISE AND MENTAL HEALTH

What might the mechanism be for the effects of noise on mental health? One way to approach this is through the effects of noise on cognitive performance where the laboratory evidence of effects is fairly robust (Smith and Broadbent, 1992). Effects of noise on mental health might be expected because there is evidence that noise impairs other aspects of human functioning, such as performance (Loeb, 1986) and sleep, that are important in maintaining normal functioning, and that noise causes adverse emotional reactions such as annoyance. In general, it seems that noise exposure increases arousal, and decreases attention through distraction (Broadbent, 1953), increases the need for focusing attention to cut out irrelevant stimuli (Cohen and Spacapan, 1978), as well as altering choice of task strategy (Smith and Broadbent, 1981). Even relatively low levels of noise may have subtle ill effects, and in this respect, the state of the person at the time of performance may be as important as the noise itself (Broadbent, 1983). Individuals' perception of their degree of control over noise may also influence whether it impairs memory (Willner and Neiva, 1986) while perception of lack of control over environmental conditions may be an important mediator of health effects.

Additionally, noise may also affect social performance as: (1) a stressor causing unwanted aversive changes in affective state; (2) by masking speech and impairing communication; and (3) by distracting attention from relevant cues in the immediate social environment (Jones, Chapman and Auburn, 1981). It may be that people whose performance strategies are already limited for other reasons (for instance through high anxiety) and who are faced with multiple tasks may be more vulnerable to the masking and distracting effects of noise.

The mechanism for the effects of noise on health is generally conceptualized as fit-

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ting the stress–diathesis model, in which noise exposure increases arousal, and chronic exposure leads to chronic physiological change and subsequent health effects. It is not clear, however, whether this model is appropriate for mental health effects. A more sophisticated model (Biesiot, Pulles and Stewart, 1989; Passchier-Vermeer, 1993) incorporates the interaction between the person and their environment. In this model, the person readjusts their behaviour in noisy conditions to reduce exposure. An important addition is the inclusion of the appraisal of noise (in terms of danger, loss of quality, meaning of the noise, challenges for environmental control, etc.) and coping (the ability to alter behaviour to deal with the stressor). This model emphasizes that dealing with noise is an active not a passive process.

4.8.7 HABITUATION TO NOISE AND MENTAL HEALTH

It is likely that mental health effects arise from persistent exposure to noise over a long period of time. But do people habituate or adapt to noise over time? In some studies people do seem to adapt to noise and no longer notice noise that they are frequently exposed to. On the other hand, in some studies of annoyance there seems to be little evidence of adaptation (Cohen and Weinstein, 1981). It may be that, as in physiological studies, a failure of adaptation occurs if the stimulus is novel, salient or implies threat. The development of mental health symptoms implies a failure to habituate to noise, or at least to adapt to noise. In some studies control over noise or active coping with noise rather than passive emotion-focused coping is related to lower levels of symptom (van Kamp, 1990). Habituation has not been formally studied in relation to noise and mental health.

4.8.8 RISK GROUPS FOR MENTAL HEALTH EFFECTS FROM NOISE

One way to look at susceptibility to noise is to think about groups in the population who may be more susceptible to noise, for instance people with existing physical or mental illness tend to be more highly annoyed by noise and potentially could be vulnerable to mental health effects. Similarly, people with hearing impairment may be vulnerable to communication difficulties in noisy environments that could increase the risk of mental health symptoms. People who report that they are sensitive to noise tend to be more prone to noise annoyance and may be more at risk for common mental disorders (Stansfeld et al., 2002).

4.8.9 POPULATION GROUPS AT RISK FOR MENTAL HEALTH EFFECTS FROM NOISE

There is some evidence that children are more vulnerable to the mental health effects of noise than adults in terms of prematurity, low birth weight and through scoring higher on hyperactivity. There is no consistent evidence of age, social class, ethnic or gender differences in susceptibility to mental health effects from environmental noise.

4.8.10 NOISE SENSITIVITY

Noise sensitivity, based on attitudes to noise in general (Anderson, 1971; Stansfeld, 1992), is an intervening variable which explains much of the variance between exposure and individual annoyance responses (Weinstein, 1978; Langdon, Buller and

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Scholes, 1981; Fields, 1993). Individuals who are noise-sensitive are also likely to be sensitive to other aspects of the environment (Broadbent, 1972; Weinstein, 1978; Thomas and Jones, 1982; Stansfeld et al., 1985a). This raises the question as to whether noise-sensitive individuals are simply those who complain more about their environment. Certainly, there is an association between noise sensitivity and neuroticism (Thomas and Jones, 1982; Öhrström, Bjorkman and Rylander, 1988; Jelinkova, 1988; Belojevic and Jakovljevic, 1997; Smith, 2003), although it has not been found in all studies (Broadbent, 1972). On the other hand, Weinstein (1980) hypothesized that noise sensitivity is part of a critical/uncritical dimension, showing the same association as noise sensitivity to measures of noise, privacy, air pollution and neighbourhood reactions. He suggested that the most critical subjects, including noise-sensitive people are not uniformly negative about their environment, but more discriminating than the uncritical group, who comment uniformly on their environment.

Noise sensitivity has also been related to current psychiatric disorder (Bennett, 1945; Tarnopolsky, Morton-Williams and Barker, 1980; Iwata, 1984). Stansfeld et al. (1985) found that high noise sensitivity was particularly associated with phobic disorders and neurotic depression, measured by the Present State Examination (Wing, Cooper and Sartorius, 1974). Similar to this association with phobic symptoms, noise sensitivity has also been linked to a coping style based on avoidance, which may have adverse health consequences (Pulles, Biesiot and Stewart, 1988) and a tendency to report health complaints rather than take a more active coping approach to noise (Lercher and Kofler, 1996). Noise sensitivity may be partly secondary to psychiatric disorder: depressed patients followed over four months became less noise-sensitive as they recovered (Stansfeld, 1992). These "subjective" psychological measurements were complemented by an "objective" psychophysiological laboratory investigation of reactions to noise in a subsample of depressed patients. Noise-sensitive people tended to have higher levels of tonic physiological arousal, more phobic and defence/startle responses and slower habituation to noise (Stansfeld, 1992). Thus, noise-sensitive people attend more to noises, discriminate more between noises, find noises more threatening and out of their control, and adapt to noises more slowly than people who are less sensitive. Through its association with greater perception of environmental threat and its links with negative affectivity and physiological arousal, noise sensitivity may be an indicator of vulnerability to minor psychiatric disorder, although not necessarily psychiatric disorder caused by noise (Stansfeld, 1992).

In analysis of a subset of noise-sensitive women, compared to less sensitive women in the West London survey, there was no evidence that aircraft noise exposure predicted psychiatric disorder in the sensitive women (Stansfeld et al., 1985). In the Caerphilly study, noise sensitivity predicted psychological distress at follow-up after adjusting for baseline psychological distress, but did not interact with the noise level, suggesting that noise sensitivity does not specifically moderate the effect of noise on psychological distress (Stansfeld et al., 1993). However, in further analyses, a statistically significant association between road traffic noise exposure and psychological distress, measured by the General Health Questionnaire (GHQ), was found in noise-sensitive men, that was not found in men of low noise sensitivity (Stansfeld et al., 2002). In the original analyses, after adjusting for trait anxiety at baseline, the effect of noise sensitivity was no longer statistically significant. This suggests that much of the association between noise sensitivity and psychological distress may be accounted for by the confounding association with trait anxiety. Constitutionally anxious people may be both more aware of threatening aspects of their environment and more prone to future psychiatric disorder. It seems possible that these traits might be linked.

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In a United Kingdom community study, associations were examined between noise exposure, noise sensitivity, subjective symptoms and sleep disturbance in a random sample of 543 adults (Smith, 2003). Perceived noise exposure was related to subjective health, but this association became non-significant after adjustment for negative affectivity. In a similar way, adjustment for negative affectivity eliminated the association between noise sensitivity and subjective health. Thus, it was suggested that noise sensitivity was merely a proxy measure of negative affectivity or neuroticism. However, although this means that noise sensitivity is not specific to noise, the more recent analyses suggest that high levels of trait anxiety or neuroticism may be an indicator of vulnerability to noise effects and could put people at risk of adverse psychological effects from noise, even if they do not increase the risk of physical ill health.

4.8.11 MENTAL HEALTH CONSEQUENCES OF INSOMNIA

Transient insomnia is usually accompanied by reports of daytime sleepiness and performance impairment the next day. Chronic insomnia is generally associated with poorer emotional and physical health. Several large-scale epidemiological studies of the general adult population have shown that between one third and one half of people who complain of chronic insomnia are also diagnosable with primary psychiatric disorders, mostly anxiety and mood disorders. Mellinger, Balter and Uhlenhuth (1985) found that 17% of adults reported "a lot" of trouble falling asleep or staying asleep over the past year; 47% of them had high levels of psychological distress, with symptom complexes suggestive of depression and anxiety disorders. In contrast, only 11% of individuals with no history of insomnia showed elevated levels of psychiatric symptoms. In a survey of almost 8000 individuals, Ford and Kamerow (1989) reported that 10% had suffered from significant insomnia for at least a two-week period during the previous six months; 40% of the insomniacs met criteria for psychiatric disorders, with the majority being anxiety disorders and depression; only 16% of those with no sleep complaints had psychiatric illness.

Breslau et al. (1996) found a strong correlation between lifetime prevalence of sleep problems and psychiatric disorders, with anxiety, depression, and substance abuse disorders being the most common. Similar results have been found by Vollrath, Wicki and Angst (1989), Chang et al. (1997) and Dryman and Eaton (1991). In a large-scale European population-based study (Ohayon and Roth, 2003), it was found that insomnia more often precedes rather than follows incident cases of mood disorders.

Insomniacs not only have higher rates of psychiatric disorders, but they also have increased rates of various kinds of psychological symptoms: patients with insomnia reported increased psychological stress and/or decreased ability to cope with stress according to surveys of the American (Roth and Ancoli-Israel, 1999) and Japanese (Kim et al., 2000) population. Almost 80% of insomniacs had a significant increase on one or more clinical scales on the Minnesota Multiphasic Personality Inventory (MMPI) (Kalogjera-Sackellares and Cartwright, 1997). Even people whose insomnia was due to identified medical factors showed elevation on the MMPI, suggesting a possible causal relationship or specific association between insomnia and psychiatric symptomatology. Compared to good sleepers, severe insomniacs reported more medical problems, had more GP office visits, were hospitalized twice as often and used more medication. Severe insomniacs had a higher rate of absenteeism, missing work twice as often as did good sleepers. They also had more problems at work (including decreased concentration, difficulty performing duties and more work-related accidents) (Leger et al., 2002).

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4.8.12 INSOMNIA AS A MENTAL HEALTH SYMPTOM

Insomnia is a symptom of many psychiatric disorders, especially depression and anxiety. In studies of depressed patients compared to control subjects, there was prolonged latency to sleep, increased wakefulness during sleep, early morning wakening, decreased sleep efficiency and reduced total sleep time. There is also evidence that insomnia may be a risk factor for developing depression (Riemann, Berger and Voderholzer, 2001; Roberts, Roberts and Chen, 2002). This raises the question as to whether prolonged noise exposure leading to insomnia provokes the onset of depression in susceptible people? This seems theoretically possible, but there is little evidence to support it. In a longitudinal study of adolescents, it was the other way round – that depressive symptoms preceded the onset of insomnia (Patton et al, 2000). Delayed sleep latency in children has been linked to increased externalizing symptoms including aggressive behaviour, and impaired attention and social problems (Aronen et al., 2000). In this cross-sectional study, the direction of association was uncertain, but it seems most plausible that the sleep disturbance is a feature of the behavioural disturbance rather than a cause of it. Three criteria have been suggested for sleep disturbance to be environmentally determined: (1) the sleep problem is temporally associated with the introduction of a physically measurable stimulus or definable set of environmental circumstances; (2) the physical rather than the psychological properties of the environmental factors are the critical causative elements; and (3) removal of the responsible factors results in an immediate or gradual return to normal sleep and wakefulness (Kraenz et al., 2004). Most studies do not fulfil these criteria. In a German school-based study of 5–6-year-old children, sleep disturbance by noise, largely from road traffic, was reported "sometimes" in 10% by parents of children and 2% "often". Children's reports were slightly higher: "sometimes" in 12% and 3% "often" (Kraenz et al., 2004). Further longitudinal research is needed to ascertain whether noise-induced insomnia leads on to overt psychiatric disorder.

In summary, population as well as clinic-based studies have demonstrated a high rate of psychiatric morbidities in patients with chronic insomnia. It has traditionally been assumed that insomnia is secondary to the psychiatric disorders; however, it is possible that in some cases the insomnia preceded the psychiatric disorder.

4.8.13 DEPRESSIVE EPISODE AND ANXIETY DISORDERS

A mild depressive episode is diagnosed by clinical interview. The criteria for a mild depressive episode include two or more symptoms of depressed mood, loss of interest or fatigue lasting at least two weeks, with two or three symptoms such as reduced concentration, reduced self-esteem, ideas of guilt, pessimism about the future, suicidal ideas or acts, disturbed sleep, diminished appetite and social impairment, and fewer than four symptoms including lack of normal pleasure/interest, loss of normal emotional reactivity, waking =>2 hours early, loss of libido, diurnal variation in mood, diminished appetite, loss of =>5% body weight, psychomotor agitation or psychomotor retardation.

Anxiety disorders are similarly diagnosed by clinical interview. The criteria for "generalized anxiety disorders" include duration of at least six months of free-floating anxiety and autonomic overactivity.

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4.8.14 ASSOCIATIONS BETWEEN INSOMNIA AND PSYCHIATRIC DISORDERS

At the present time, exposure–effect associations have not been established between parameters of sleep disturbance (number of behavioural awakenings, body movements or EEG awakenings) and the onset of depressive and anxiety disorders, although there is some evidence that insomnia is a risk factor for developing depression (Riemann, Berger and Voderholzer, 2001; Roberts, Roberts and Chen, 2002). A number of longitudinal prospective studies in different age groups have found associations between self-reports of insomnia and the subsequent onset of psychiatric disorder, in particular major depression. A selection of the most important studies and their findings are outlined in Table 4.3 below.

Table 4.3 Insomnia as a predictor of psychiatric disorder

Study	Sample size	Sample	Follow-up interval	Depression measure	Results
Ford and Kamerow, 1989	7954	Community sample	1 year	Diagnostic interview schedule	Risk of developing new depression for insomnia on two occasions: [OR=39.8, 95% CI 19.8-80.0]
Breslau et al., 1996	1200	21–30 years members of health maintenance organization	3 years	Diagnostic interview schedule	RR for new onset major depression associated with baseline insomnia [RR=4.0, 95% CI 1.5-5.6]
Chang et al., 1997	1053	Male medical students	34 years (median)	Clinical depression	RR for clinical depression for those who reported insomnia at medical school [RR= 2.0, 95% CI 1.2-3.3]
Roberts, Roberts and Chen, 2002	3136	11–17 years from managed care rosters	1 year	Diagnostic interview schedule for children major depression module	Fully adjusted OR for insomnia in waves 1 and 2 for depression at follow-up [OR=1.92, 95% CI 1.30-2.82]

4.8.15 CONCLUSIONS: ASSOCIATIONS BETWEEN NOISE AND PSYCHIATRIC DISORDERS

The effects of noise are strongest for those outcomes that, like annoyance, can be classified under "quality of life" rather than illness. What they lack in severity is made up for in numbers of people affected, as these responses are very widespread.

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Current evidence does seem to suggest that environmental noise exposure, especially at higher levels, is related to mental health symptoms and possibly raised anxiety and consumption of sedative medication, but there is little evidence that it has more serious effects. Further research is needed on mental health effects at very high noise levels. Existing studies may be confounded either by prior selection of subjects out of (or into) noisy areas as a result of noise exposure, or by confounding between noise exposure, socioeconomic deprivation, and psychiatric disorder. It is also possible that people underestimate or minimize the effects of noise on health through optimism bias (Hatfield and Soames Job, 2001) and that this is particularly protective for mental health.

The evidence is not strong for the association between noise exposure and mental ill health. What evidence there is suggests that noise exposure may be responsible for psychological symptoms above 70 dB(A) L_{eq}. Almost all studies have only examined the effects of daytime noise on mental health, but it is possible that night-time noise, during sleep time, may have effects on mental health at lower levels than daytime noise.

The most powerful evidence of noise on mental health comes from studies of military aircraft noise. There is also some evidence that intense road traffic noise may lead to psychological symptoms. There is no evidence of any effects of railway noise on mental health.

4.9 THE SEVERITY OF SELF-REPORTED SLEEP DISTURBANCE

4.9.1 INTRODUCTION

In section 2.1.2 of Chapter 2 of this report, it is stated that sleep disturbance caused by noise may either be diagnosed (Environmental Sleep Disorder: ICSD 780-52-6) or self-reported. Although self-reported sleep disturbance is subjective by definition, its observed occurrence correlates with noise levels as well as with important diagnostic criteria for ICSD 780-52-6. It appears justified to consider self-reported sleep disturbance as an impairment of health, especially if indicated by representative population samples in social surveys. Furthermore, section 4.1 of Chapter 4 of this report gives a quantitative relationship between noise level L_{night} and the percentage of population that reports a disturbed sleep of high, medium or low disturbance intensity.

But an open question concerns severity: even if night-time noise causes large percentages of the population to declare themselves as highly sleep-disturbed, this could nevertheless represent an almost negligible loss of health, if the mean severity of self-reported sleep disturbance were negligible in comparison with commonly accepted diseases. Attempts have been made to give an answer to this important question, using WHO's concept of disability weights (Murray et al., 1996) as a basis for severity comparisons.

4.9.2 AN ASSESSMENT OF DISABILITY WEIGHTS

A Swiss study (Müller-Wenk, 2002) aimed at determining a disability weight for sleep disturbance due to road traffic noise. For this purpose, a description of road-

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noise-related sleep disturbance was set up: essentially, this state of health was assumed to be present if a person indicated that, due to traffic noise, he or she, almost every night, had problems with falling asleep, with continuing sleep during the night or with early or non-restorative waking in the morning. In addition, a list was established with already available disability weights (Murray et al., 1996; Stouthard et al., 1997) for a selection of 28 diseases of various types, covering a range from very light severity to high severity (Müller-Wenk 2002:65–66). All 64 members of the medical staff of the Swiss Accident Insurance Institute (SUVA) were then asked in a written questionnaire to determine the hitherto unknown disability weight of sleep disturbance by interpolation, that is, by inserting sleep disturbance at the appropriate place between the presented 28 diseases that were sorted according to ascending disability weight. These participants were chosen because the physicians of the SUVA, besides being medical doctors, have a particularly high professional know-how in comparing the severity of different types of disability. Forty-two questionnaires were completed, of which 41 were usable.

From these questionnaires, an arithmetical mean of 0.055 of the disability weight for sleep disturbance could be calculated, with a 95% confidence limit of 0.039 at the low end and 0.071 at the high end. This result can be illustrated by mentioning diseases from the catalogues of Murray et al. (1996) or Stouthard et al. (1997) with the same disability weight: hence the disability weight of the road-noise-related sleep disturbance is roughly the same as the disability weight of "chronic hepatitis B infection without active viral replication", the latter having a mean disability weight of 0.06 and a 95% confidence interval from 0.034 to 0.087. The low-end estimate of 0.039 for sleep disturbance severity would correspond to the mean disability weight of "benign prostatic hypertrophy (symptomatic cases)", whilst the high estimate of 0.071 would correspond to the mean disability weight of "uncomplicated diabetes mellitus". The conclusion is that the mean disability weight of road-noise-related sleep disturbance is not smaller (= less severe) than the disability weight of health impairments commonly recognized as diseases, and there is a strong overlap amongst the probability distributions of these disability weights. On the basis of the chosen disability weight 0.055 for self-reported sleep disturbance, and taking into account the current traffic noise levels during the night in many European states, it is justified to consider noise-related sleep disturbance as a substantial loss of public health.

4.9.3 COMPARISON BETWEEN INSOMNIA AND SELF-REPORTED SLEEP DISTURBANCE

The original list of disability weights (Murray et al., 1996) did not contain any kind of non-normal sleep. In the meantime, WHO has published an extended list (Mathers et al., 2003, Annex Table 5a) containing a disability weight of 0.100 for insomnia (diagnostic code 307.42). This has opened a way to recheck the disability weight of 0.055 (Müller-Wenk, 2002), by asking a panel of medical professionals to compare, on the basis of disability weights, the mean severity of self-declared sleep disturbance due to road noise at night with the mean severity of insomnia. It may be debated whether it is more straightforward to compare two types of sleep anomalies with similar symptoms, or to compare self-declared sleep disturbance with various types of completely different diseases. But it makes sense anyway to use the comparison with insomnia as a second approach for determining the disability weight of self-reported sleep disturbance.

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This severity comparison between different sleep anomalies was made in 2005 by structured oral interviews, executed by a medical staff member of the sleep clinic of Kantonsspital St. Gallen (Switzerland), with 14 GPs selected at random from all GPs who had admitted patients to the sleep clinic during the nine preceding months. These patients were mainly suffering from OSAS. The question was as follows:

> "Could you please give us your opinion on the relative severity of three different cases of insomnia:
>
> 1. (primary) insomnia, in our region usually called psychophysiological insomnia
> 2. Obstructive Sleep Apnoea Syndrome (OSAS)
> 3. traffic-noise-related sleep disturbance, that may occur with persons who are forced to sleep along through roads with nocturnal motor traffic.
>
> Your opinion should be based on the patients you have seen in your office lately, or on other persons of your social environment. When comparing the severity of the health impairment, the focus should be above all on the person's condition during the day after the sleep-disturbed night. The absolute value of the severity is less important for the current study than the relative severity amongst the three cases of insomnia. The opinion of the severity may be expressed on a linear scale from 0 (no impairment at all) to 10 (impairment almost unsupportable). On the scale from 0 to 10, you may give us your mean value of the severity, or you may give us a span from a low to a high for the severity."

All of the interviewed GPs gave their opinions, and the result is presented in Table 4.4.

Table 4.4
Severity ratings (10 = almost insupportably disturbing, 0 = not in the least disturbing) by 14 GPs selected at random

	Primary insomnia				OSAS (sleep apnoea)				Sleep disturb.(noise)				Ratio noise/	Noise/
No	Max	Min	Mean	Rank	Max	Min	Mean	Rank	Max	Min	Mean	Rank	priminsomnia	OSAS
10	6	4	5	3	8	6	7	1	8	6	7	1	1.40	1.00
11	5	3	4	3	9	7	8	1	8	4	6	2	1.50	0.75
12			5	3			10	1	7	8	7.5	2	1.50	0.75
13	2	3	2.5	2	4	5	4.5	1	1	2	1.5	3	0.60	0.33
14			3	2			6	1	1	2	1.5	3	0.50	0.25
15			8	2			9	1			6	3	0.75	0.67
16			8	1			7	2			4	3	0.50	0.57
17			5	1			5	1			3	3	0.60	0.60
18	2	3	2.5	2			6	1	1	2	1.5	3	0.60	0.25
19			8	1			3	2			2	3	0.25	0.67
20			6	2			7	1			4	3	0.67	0.57
21			7	2			8	1			0	3	0.00	0.00
22			4	3			5	2			6	1	1.50	1.20
23			4	3	6	7	6.5	2	8	9	8.5	1	2.13	1.31
Mean			5.143	2.143			6.57	1.286			4.18	2.429	0.89	0.64
Sigma													0.60	
Median													0.63	
Upper value 95% C.I. for mean													1.20	
Lower value 95% C.I. for mean													0.58	

Clearly, the severity judgements vary widely between the participating GPs. Apart from the differences in personal judgement, this variation is certainly influenced by the mix of patients visiting a particular GP. For instance, GP number 15 could have encountered one or two very serious cases of OSAS, whilst his/her experience with noise-related sleep disturbance might refer to persons that were only moderately disturbed by night-time noise in their bedroom. On the other hand, number 22 could

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have had experience with persons suffering very much from sleep disturbance due to high traffic noise exposure, whilst his/her OSAS or primary insomnia patients happened to be light cases. One must accept that even GPs have a limited experience with the whole range of cases of each of the three types of insomnia, so that their opinion on the mean severity of noise-related sleep disturbance, compared to the mean severity of OSAS or insomnia, is influenced by the randomness of their patient mix.

Nevertheless, the table supports the following statements.

- With respect to severity, the majority of GPs rank noise-related sleep disturbance lower than insomnia and OSAS, while three of them put noise-related sleep disturbance in the first rank. Only one of the participants (number 21) considers noise-related sleep disturbance as a fully negligible disturbance.
- The severity ratio between noise-related sleep disturbance and insomnia varies between 0 and 2.1. Seven of the fourteen GPs indicate a severity ratio between 0.5 and 0.75, that is to say that half the participants are of the opinion that the severity of noise-related sleep disturbance amounts to 50–75% of the severity of insomnia.
- The mean of this severity ratio is 0.89, with a standard deviation (sigma) of 0.60. The confidence interval (CI) for the mean goes from 0.58 to 1.20. The median of the severity ratio is 0.63. The distribution is skewed to the right.

The severity ratio developed above can be used as a proportionality factor between the known disability weight for insomnia and the required disability weight for self-reported sleep disturbance. Bearing in mind that the already existing WHO disability weight for insomnia is 0.10, a best guess for the mean disability weight for self-reported sleep disturbance due to road traffic noise at night is therefore 0.089, with a CI from 0.058 to 0.12.

4.9.4 CONCLUSIONS

According to the two groups of interviewed medical professionals, persons that declare themselves to be chronically deprived of normal sleep by road traffic noise have a health state whose mean disability weight is comparable to "chronic hepatitis B infection without active viral replication" or higher. Irrespective of the question whether self-reported sleep disturbance is formally recognized as a disease or not, its severity is comparable to commonly accepted diseases.

The best estimate for a mean disability weight for self-reported sleep disturbance due to road traffic noise was 0.055 (CI: 0.039; 0.071) according to Müller-Wenk (2002), whilst our recheck based on a comparison with insomnia resulted in a disability weight of 0.09 (CI: 0.06; 0.12). The higher disability weight according to the second approach might be caused by the fact that in this second approach, there was a stronger focus on "the person's condition during the day after the sleep-disturbed night".

The above figures compare reasonably with a study published by van Kempen (1998), cited in Knol and Staatsen (2005:46), where a severity weight of 0.10 for severe sleep disturbance was found, based on the judgement of 13 medical experts according to the protocol of Stouthard et al. (1997).

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In conclusion, a mean disability weight of 0.07 is proposed for self-reported sleep disturbance due to road noise or similar ambient noise. This disability weight can be used in connection with the equations of section 4.1 of this chapter for highly sleep-disturbed persons.

4.10 DISCUSSION: CAN CHRONIC SHORT-TERM EFFECTS CAUSE LONG-TERM EFFECTS ON HEALTH?

EEG modifications, cardiovascular responses, body movements and awakenings due to noise occur within a few seconds after the stimulus. In addition to the instantaneous effects related to single events, large field studies on aircraft (Passchier-Vermeer et al., 2002) and road traffic noise exposure during night-time (Griefahn et al., 2000; Passchier-Vermeer et al., 2004) show that also sleep latency and average motility during the sleep period increased monotonously as a function of the noise exposure level. The increase in average motility was substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002) suggesting persistent arousal during the sleep related to aircraft noise. Furthermore, an international field study (Jurriëns et al., 1983) found slightly reduced REM sleep, increased time being awake according to the EEG, increased average heart rate, and reduced performance on a reaction time test in people when exposed during the night to higher road traffic noise levels.

The relationship between instantaneous effects and more global modifications of one night sleep, as well as chronic changes, is not simple, as illustrated by the findings concerning motility. An increase in average motility that is substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002) suggests a persistent arousal during sleep related in a dose-dependent way to the aircraft noise.

Since EEG arousal and instantaneous motility are correlated, this finding suggests that also the number of (micro-)arousals may increase during noise exposure more than by the sum of the instantaneous (micro-)arousals that occur contingent upon a noise event.

For overall motility during sleep, clear indications have been found of associations with further effects, although the causal direction is not in all cases clear. Mean (onset of) motility during sleep is associated with the following variables based on questionnaires and diaries (Passchier-Vermeer et al., 2002):

- frequency of conscious awakening during the sleep period: the increase is 0.8 conscious;
- awakenings per night, if motility increases from low to high;
- frequency of awakening remembered next morning: the increase is 0.5 remembered;
- awakenings per night, if motility increases from low to high;
- long-term frequency of awakening attributed to specific noise sources assessed with a questionnaire;
- sleep quality reported in a morning diary;
- long-term sleep quality assessed with a questionnaire;

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- number of sleep complaints assessed with a questionnaire;
- number of general health complaints assessed with a questionnaire.

The associations of mean motility with these variables are stronger than the corresponding associations of mean onset of motility.

For evaluating the adverseness of the instantaneous effects, it is important to consider whether they bring the body into a more persistent state of higher arousal or not, although this is not the only criterion. Those effects which are progressively disappearing with the repetition of the stimulus may be less harmful than those which do persist over long exposure time, provided that the suppression of the effects do not require costs in another form. For example, short-term cardiovascular effects that appear not to habituate could lead to permanent cardiovascular system impairment (Carter, 1996, 1998).

The relations presented for motility and conscious awakening imply that motility is sensitive to noise and has a relatively low threshold, while conscious awakening, the strongest instantaneous interference of noise with sleep, has the highest threshold of the instantaneous effects considered.

In one of the most sophisticated field studies (Passchier-Vermeer et al., 2002), increased probability of instantaneous motility was found for events with a maximum sound level L_{Amax} > 32 dB(A), while in a meta-analysis conscious awakening was found for events with L_{Amax} > 42 dB(A) (Passchier-Vermeer, 2003a). Above their threshold, these effects were found to increase monotonously as a function of the maximum sound level during a noise event (aircraft noise). It is important to note that in another recent sophisticated field study (Basner et al., 2004), the threshold found for EEG awakening was L_{Amax} = 35 dB(A), that is, only a little higher than the 32 dB(A) found for noise-induced awakenings. This strengthens the evidence that noise starts to induce arousals at L_{Amax} values in the range 30–35 dB(A). Given the night-time noise levels to which people are exposed, these results imply that instantaneous effects are common. Although most studies concerned aircraft noise, the instantaneous effects can be assumed to occur at similar levels for different types of transportation.

The above observations can be used as a basis for setting limits with respect to night-time transportation noise. For transparency, it is useful to distinguish two steps in choosing actual limits: the first step is the derivation of a health-based limit; the second step is the derivation of an actual limit that takes into account the health-based limit as well as feasibility arguments. Here the concern is with the first step.

When deriving a health-based limit, two points need to be considered: the dose-dependent effects of a single noise event, and the number of events. With respect to the dose-dependent effects of a single event, adverse effects can be distinguished from effects that by themselves need not be adverse but can contribute to an adverse state. It is proposed to classify conscious awakenings as an adverse effect. Conscious awakenings have been estimated to occur at a baseline rate of 1.8 awakening per night. A substantial increment of conscious awakenings over this baseline is thought to be adverse. Since, in general, falling asleep after conscious awakening takes some time, and this latency is longer after noise-induced conscious awakening that will often also induce an emotional reaction (anger, fear), it will also reduce the time asleep and may affect mood and functioning next day. Although additional, more sophisticated analyses could be performed to refine this estimate, we propose L_{Amax}

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= 42 dB(A) is proposed as the currently best estimate of the threshold for conscious awakening by transportation noise. This would mean that the no observed effect level ($NOEL_{Amax}$) for transportation noise events is at most 42 dB(A).The most sensitive instantaneous effect that has been studied extensively in field studies is motility. A single interval with (onset of) noise-induced motility by itself cannot be considered to be adverse.

However, noise-induced motility is a sign of arousal, and frequent (micro-)arousal and accompanying sleep fragmentation can affect mood and functioning next day and lead to a lower rating of the sleep quality. Therefore, motility is relevant for adverse health effects, but more than a few intervals with noise-induced motility are needed for inducing such effects. Although additional, more sophisticated analyses could be performed to refine this estimate, we propose L_{Amax} = 32 dB(A) as the currently best estimate of the threshold for motility induced by transportation noise. The threshold found for EEG awakening was L_{Amax} = 35 dB(A), that is, only a little higher than the 32 dB(A) found for noise-induced awakenings. This would mean that the $NOEL_{Amax}$ for transportation noise events is most likely at most 32 dB(A), and definitely not higher than 35 dB(A). It is important to note that the above given $NOEL_{Amax}$ ~ 32 dB(A) and $NOEL_{Amax}$ ~ 42 dB(A) are indoor levels, in the sleeping room. Although events below 32 dB(A) are audible, and, hence, further research may show more sensitive effects than motility, on the basis of the present available evidence we propose to assume that $NOEL_{Amax}$ = 32 dB(A) and set a health-based night-time noise limit that is tolerant for transportation noise events with L_{Amax} ~ 32 dB(A). On the other hand, since adverse health effects need to be prevented by health-based limits and even though vulnerable groups may require lower limits, on the basis of the present available evidence we propose to assume that $NOAEL_{Amax}$ = 42 dB(A) and set a health-based night-time noise limit that does not tolerate transportation noise events with L_{Amax} > 42 dB(A).

On the basis of the above proposal, it would be possible to derive a night-time noise guideline value in terms of L_{night}. Such a guideline value would indicate the level below which no short-term effects are to be expected that would lead to temporary reduced health or chronic disease. Such a guideline value needs to be compared with guideline values derived directly with a view to preventing temporary reduced health and chronic diseases. In particular, for self-reported sleep disturbance, which is an expression of reduced well-being and may be an indication of effects that could contribute to cardiovascular disease, exposure–effect relationships have been derived on the basis of an extensive set of original data from studies from various countries (Miedema, Passchier-Vermeer and Vos, 2003; Miedema, 2004). The percentage of people reporting high noise-induced sleep disturbance (%HS) levels off at 45 dB(A) but at a non-zero effect level. The remaining effect may be caused by events not incorporated in the exposure assessment and it appears that if all noise contributions would be incorporated in the exposure metric, high noise-induced sleep disturbance would vanish between 40 dB(A) and 45 dB(A), say at 42 dB(A). Since values found for other temporary reduced health effects or chronic diseases, in particular cardiovascular diseases, will be higher, and considering self-reported sleep disturbance as an adverse effect, this would suggest L_{night} = 42 dB(A) as the NOAEL to be compared with the value derived from the short-term effects. Note that this is an outdoor level, which would, assuming partly opened windows and an actual insulation of 15 dB(A), correspond to an indoor equivalent night-time sound level of 27 dB(A). The above discussion is based on motility, EEG awakenings, and conscious awakening. In addition, EEG micro-/minor arousals, and autonomic reactions have been discussed above.

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Furthermore, there are potential instantaneous effects, such as effects on memory consolidation or restoration of the immune system, for which the information on a possible relation with noise exposure is so limited that they were not considered here. In order to acquire more insight into these effects, more field research is needed. Field research is needed because earlier studies have shown that estimates of effects on the basis of laboratory studies are much higher than estimates from field studies. Methodological differences between the different approaches certainly cannot be the only possible explanation. Research allowing the introduction of some specific but light laboratory technique into the sleeper's own bedroom, should be encouraged, as, for example, used in the Swiss Noise Study 2000 (Brink, Müller, and Schierz, 2006). The key to better insight into effects of night-time noise, leading to mechanistic models describing the relationships between noise exposure, instantaneous effects, effects at the level of a 24-hour period and chronic effects, appears to be epidemiological studies at home with well-designed instrumentation.

The relationships between noise exposure, instantaneous effects, effects at the level of a single 24-hour period and chronic effects is complex because the effects at a smaller time scale do not simply add up to effects at a larger time scale. For example, the noise-related increase in night-time average motility was substantially higher than would be expected on the basis of the instantaneous extra motility at the times of the noise events (Passchier-Vermeer et al., 2002), suggesting persistent arousal during sleep related to aircraft noise. It is likely that such shifts in the basic state are more important for the development of chronic effects than the instantaneous effects per se. A further complication is that some effects habituate. Habituation in some effect parameters can occur in a few days or weeks, but the habituation is not always complete. The measured modifications of the cardiovascular functions remain unchanged over long periods of exposure time (Muzet and Ehrhart, 1980; Vallet et al., 1983). Most striking is that none of the cardiovascular responses show habituation to noise after a prolonged exposure, while subjective habituation occurs within a few days. It appears plausible that, in particular non-habituating effects lead to the development of chronic effects, but also the disappearance of effects with continuing exposure may come at a cost associated with suppressing the effects. A third complication is that daytime noise exposure may contribute to the effects found in relation to night-time noise. Large epidemiological studies are needed that compare populations exposed to similar daytime noise and differ in their night-time noise exposure only. A specific challenge for mechanistic models on the effects of noise on sleep is the identification of factors that make subjects vulnerable to night-time noise. The following groups may be hypothesized to be more vulnerable to noise during sleep: old people, ill people, people with chronic insomnia, shift workers and people resting during daytime, people with a tendency to depression, light sleepers, pregnant women, people with high anxiety and high stress levels. Furthermore, children need attention because of their relatively high exposure during sleep, and because they are in a phase of neurocognitive development for which undisturbed sleep may be particularly important.

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CHAPTER 5

GUIDELINES AND RECOMMENDATIONS

5.1 ASSESSMENT

In Chapter 1 the need for a guideline document for night-time exposure to noise was defended on the basis of the lack of existing guidance, the signs that a substantial part of the population could be exposed to levels of noise that might risk their health and well-being and the EU activities that compel the public and authorities to take notice when noise maps showing L_{night} levels are made public.

Where sufficient direct evidence concerning the effects of night-time noise on health could not be collected, indirect evidence was looked at: the effects of noise on sleep (quality) and the relations between sleep and health.

In Chapter 2 the evidence was presented that sleep is a biological necessity and disturbed sleep is associated with a number of health outcomes. Studies of sleep disturbance in children and in shift workers clearly show the adverse effects. Unravelling the relations between sleep and health (Fig. 2.1) shows that sleep is an essential feature of the organism, so that simple direct relations can hardly be expected.

In Chapter 3 it was shown beyond doubt that noise disturbs sleep through a number of direct and indirect pathways. Even at very low levels physiological reactions (heart rate, body movement and arousals) can be reliably measured. It was also shown that awakening reactions are relatively rare, occurring at a much higher level.

Chapter 4 summarized the known evidence for the direct effects of night-time noise on health. The working group agreed that there is sufficient evidence that night noise is related to self-reported sleep disturbance, use of pharmaceuticals, self-reported health problems and insomnia-like symptoms. These effects can lead to a considerable burden of disease in the population. For other effects (hypertension, myocardial infarctions, depression and others), limited evidence was found: although the studies were few or not conclusive, a biologically plausible pathway could be constructed from the evidence.

An example of a health effect with limited evidence is myocardial infarction. Although evidence for increased risk of myocardial infarction related to L_{day} is sufficient according to an updated meta-analysis, the evidence in relation to $L_{night,outside}$ was considered limited. This is because $L_{night,outside}$ is a relatively new exposure indicator, and few field studies have focused on night noise when considering cardiovascular outcomes. Nevertheless, there is evidence from animal and human studies supporting a hypothesis that night noise exposure might be more strongly associated with cardiovascular effects than daytime exposure, highlighting the need for future epidemiological studies on this topic.

The review of available evidence leads to the following conclusions.

- Sleep is a biological necessity and disturbed sleep is associated with a number of health outcomes.

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- There is sufficient evidence for biological effects of noise during sleep: increase in heart rate, arousals, sleep stage changes and awakening.
- There is sufficient evidence that night noise exposure causes self-reported sleep disturbance, increase in medicine use, increase in body movements and (environmental) insomnia.
- While noise-induced sleep disturbance is viewed as a health problem in itself (environmental insomnia), it also leads to further consequences for health and well-being.
- There is limited evidence that disturbed sleep causes fatigue, accidents and reduced performance.
- There is limited evidence that noise at night causes hormone level changes and clinical conditions such as cardiovascular illness, depression and other mental illness. It should be stressed that a plausible biological model is available with sufficient evidence for the elements of the causal chain.

In the next section threshold levels are presented for the effects, where these can be derived.

5.2 THRESHOLDS FOR OBSERVED EFFECTS

The NOAEL is a concept from toxicology, and is defined as the greatest concentration which causes no detectable adverse alteration of morphology, functional capacity, growth, development or lifespan of the target organism. For the topic of night-time noise (where the adversity of effects is not always clear) this concept is less useful. Instead, the observed effect thresholds are provided: the level above which an effect starts to occur or shows itself to be dependent on the dose. This can also be an adverse effect (such as myocardial infarcts) or a potentially dangerous increase in a naturally occurring effect such as motility.

Threshold levels are important milestones in the process of evaluating the health consequences of environmental exposure. The threshold levels also delimit the study area, which may lead to a better insight into overall consequences. In Tables 5.1 and 5.2 all effects are summarized for which sufficient or limited evidence exists (see Table 1.2 in Chapter 1 for a definition). For the effects with sufficient evidence the threshold levels are usually well known, and for some the dose-effect relations over a range of exposures could also be established.

5.3 RELATIONS WITH $L_{night,outside}$

Over the next few years, the END will require that night exposures are reported in $L_{night,outside}$. It is therefore interesting to look into the relation between $L_{night,outside}$ and the effects from night-time noise. The relation between the effects listed in Tables 5.1 and 5.2 and $L_{night,outside}$ is, however, not straightforward. Short-term effects are mainly related to maximum levels per event inside the bedroom: $L_{Amax,inside}$. In order to express the (expected) effects in relation to the single EU indicator, some calculation needs to be done.

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Table 5.1
Summary of effects and threshold levels for effects where sufficient evidence is available

Effect		Indicator	Threshold, dB	Reference (chapter, section)
	Change in cardiovascular activity	*	*	3.1.5
	EEG awakening	$L_{Amax, inside}$	35	4.10
Biological effects	Motility, onset of motility	$L_{Amax, inside}$	32	3.1.8, dose–effect relation for aircraft
	Changes in duration of various stages of sleep, in sleep structure and fragmentation of sleep	$L_{Amax, inside}$	35	3.1
	Waking up in the night and/or too early in the morning	$L_{Amax, inside}$	42	3.1.7, dose–effect relation for aircraft
Sleep quality	Prolongation of the sleep inception period, difficulty getting to sleep	*	*	3.1
	Sleep fragmentation, reduced sleeping time	*	*	3.1
	Increased average motility when sleeping	$L_{night, outside}$	42	3.2, dose–effect relation for aircraft
Well-being	Self-reported sleep disturbance	$L_{night, outside}$	42	4.2, dose–effect relation for aircraft/road/rail
	Use of somnifacient drugs and sedatives	$L_{night, outside}$	40	4.5.8
Medical conditions	Environmental insomnia**	$L_{night, outside}$	42	3.1; 4.1; 4.2

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

** *Note that "environmental insomnia" is the result of diagnosis by a medical professional whilst "self-reported sleep disturbance" is essentially the same, but reported in the context of a social survey. Number of questions and exact wording may differ.*

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Table 5.2
Summary of effects and threshold levels for effects where limited evidence is available+

Effect		Indicator	Estimated, threshold dB	Reference (chapter, section)
Biological effects	Changes in (stress) hormone levels	*	*	2.5
	Drowsiness/tiredness during the day and evening	*	*	2.2.3
	Increased daytime irritability	*	*	2.2.3
Well-being	Impaired social contacts	*	*	2.2.3
	Complaints	$L_{night,outside}$	35	4.3
	Impaired cognitive performance	*	*	2.2.3
	Insomnia	*	*	4.6
	Hypertension	$L_{night,outside}$	50	2.2.3; 4.5.6
	Obesity	*	*	2.2.3
	Depression (in women)	*	*	4.8
	Myocardial infarction	$L_{night,outside}$	50	4.5.15
	Reduction in life expectancy (premature mortality)	*	*	2.2.3; 2.5
	Psychiatric disorders	$L_{night,outside}$	60	4.8.15
	(Occupational) accidents	*	*	2.2.3; 2.4

+ *Note that as the evidence for the effects in this table is limited, the threshold levels also have a limited weight. In general they are based on expert judgement of the evidence.*

* *Although the effect has been shown to occur or a plausible biological pathway could be constructed, indicators or threshold levels could not be determined.*

The calculation for the total number of effects from reaction data on events (arousals, body movements and awakenings) needs a number of assumptions. The first that needs to be made is independence: although there is evidence (Brink, Müller and Schierz, 2006) that the order of events of different loudness strongly influences the reactions, the calculation is nearly impossible to carry out if this is taken into consideration.

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Secondly, the reactions per event are known in relation to levels at the ear of the sleeper, so an assumption for an average insulation value must be made. In this report a value of 21 dB (see Chapter 1, sections 1.3.4 and 1.3.5) has been selected. This value is, however, subject to national and cultural differences. One thing that stands out is the desire of a large part of the population to sleep with windows (slightly) open. The relatively low value of 21 dB already takes this into account. If noise levels increase, people do indeed close their windows, but obviously reluctantly, as then complaints about bad air increase and sleep disturbance remains high. This was already pointed out in the WHO guidelines on community noise (WHO, 1999).

From source to source the number of separate events varies considerably. Road traffic noise is characterized by relatively low levels per event and high numbers, while air and rail traffic are characterized by high levels per event and low numbers. For two typical situations estimates are made and presented in graphical form. The first is an average urban road (600 motor vehicles per night, which corresponds roughly to a 24-hour use of 8000 motor vehicles, or 3 million per year, the lower boundary the END sets) and the second case is for an average situation of air traffic exposure (8 flights per night, nearly 3000 per year).

Fig. 5.1 shows how effects increase with an increase of $L_{night,outside}$ values for the typical road traffic situation (urban road). A large number of events lead to high levels of awakening once the threshold of $L_{Amax,inside}$ is exceeded. To illustrate this in practical terms: values over 60 dB $L_{night,outside}$ occur at less then 5 metres from the centre of the road.

In Fig. 5.2 the same graph is presented for the typical airport situation. Due to a lower number of events there are fewer awakenings than in the road traffic case (Fig. 5.1), but the same or more health effects.

In these examples the worst case figures can be factors higher: the maximum number of awakenings for an $L_{night,outside}$ of 60–65 dB is around 300 per year.

A recent study suggests that high background levels (from motorways) with low numbers of separate events can cause high levels of average motility (Passchier-Vermeer, to be published). In Table 5.3 the full details are summarized.

5.4 DEALING WITH SITUATIONS EXCEEDING THE THRESHOLDS

Noise exposure data demonstrate that a large part of the population is over the no-effect levels. It is expected that this will extend into the future for quite some time. This means that circumstances may require that a risk assessment must be made. It is then recommended to apply the method laid out in Chapter 1, using the values given in Tables 5.1 and 5.2 and the dose–effect relations given in Chapter 4.

Typical actions requiring risk assessment are:

- new infrastructure projects (if an environmental impact statement is required)
- improvement programmes

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- policy evaluation
- national or international setting of limit values.

In the EU *Position Paper* (European Commission, 2002a) an overview of national night-time noise regulations can be found.

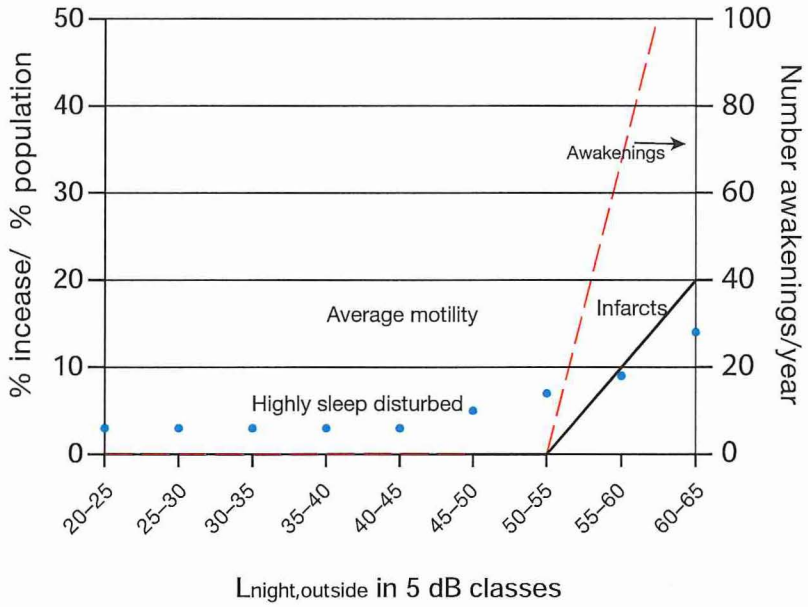


Fig. 5.1
Effects of road traffic noise at night*

Source: European Commission, 2002 a

** Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as a percent of the population; awakenings are expressed in number of additional awakenings per year.*

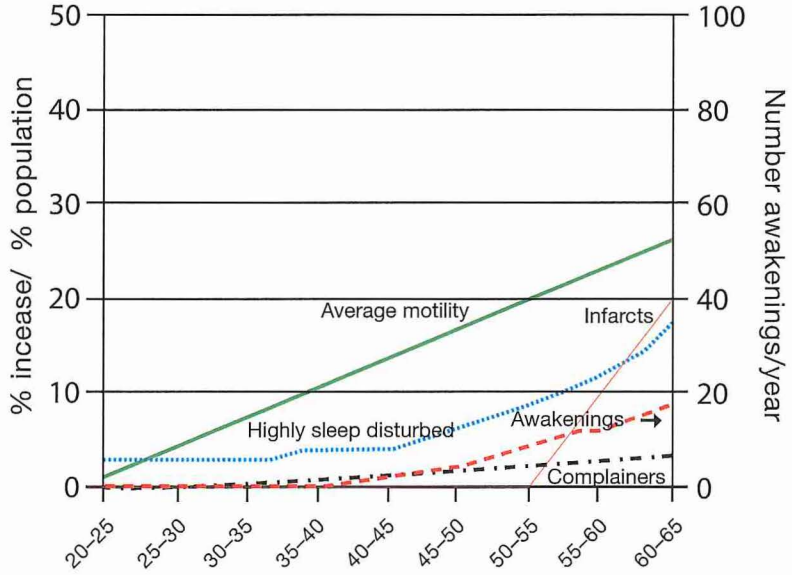


Fig. 5.2
Effects of aircraft noise at night*

Source: European Commission, 2002 a

** Average motility and infarcts are expressed in percent increase (compared to baseline number); the number of highly sleep disturbed people is expressed as percent of the population; complainers are expressed as a percent of the neighbourhood population; awakenings are expressed in number of additional awakenings per year.*

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Table 5.3
Effects (yearly, additional with respect to the normal except odd ratio) 0=below threshold, +=increase)

$L_{night,outside}$	Arousals		Body movements related to single exposures (15 sec intervals)		Average body movements (without single exposures)	Awakenings		% sleep disturbed (% highly sleep disturbed)			Myocardial infarcts
UNIT	number	number air	number average traffic	number average urban road	number	number average air traffic	number average urban road	% of exposed air	road	rail	odd ratio
NORMAL	Children 2 555	Adults 3 650	21 000		21 000	600		0			1
20–25	0	0	0	0	200	0	0	0	0	0	1
25–30	0	0	7	0	875	0	0	<3	<3	<2	1
30–35	+	+	22	0	1 547	0	0	<3	<3	<2	1
35–40	+	+	37	0	2 220	0	0	4	3	2	1
40–45	+	+	58	243	2 900	2	0	4	3	2	1
45–50	+	+	85	635	3 600	5	0	6	5	2	1
50–55	+	+	111	1 145	4 200	9	0	9	7	3	1
55–60	+	+	145	1 770	4 900	12	54	12	9	4	1.1
60–65	+	+	180	2 520	5 500	17	155	17	14	6	1.2

Source: European Commission, 2002 a

5.5 PROTECTION MEASURES AND CONTROL

What is the best strategy to reduce sleep disturbance? The first thought should always be to reduce the impact, either by reducing the number of events or by reducing the sound levels, or both. For some effects reducing the number of events may seem to be more effective (although that depends on the exact composition). Other effects are reduced by lowering overall noise level by either the number of events, the levels per event or by any combination.

In combination with other measures, sound insulation of bedroom windows is an option, but care must be taken to avoid negative impact on inside air quality. Even then, many people may want to sleep with their windows open, thereby making the insulation ineffective. Although good instruction may go some way to helping to overcome this, it is still a matter well worth taking into account. In warmer climates, in particular, insulation is not a serious option for residential purposes and excessive exposure must be avoided either by removing the people exposed or removing the source if source-related measures fail.

Although air conditioning of houses (or just bedrooms) is not commonplace in the EU, there are indications that its use is increasing, especially in the warmer parts of the Region. Although this still leaves the possibility that people may sleep with their windows open outside the summer season, it is something to consider when discussing measures.

Exposed areas could be a good choice for uses such as offices, where there will be no people at night, or where it is a physical impossibility to sleep with the windows open (fully air-conditioned buildings, for example hotels and sometimes hospitals).

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A simple measure is the orientation of noise-sensitive rooms on the quiet side of the dwelling (this applies to road and rail traffic noise).

Zoning is an instrument that may assist planning authorities in keeping noise-sensitive land uses away from noisy areas. In the densely populated areas of the EU this solution must often compete, however, with other planning requirements or a simple lack of suitable space.

5.6 RECOMMENDATIONS FOR HEALTH PROTECTION

Sleep is an essential part of healthy life and is recognized as a fundamental right under the European Convention on Human Rights[1](European Court of Human Rights, 2003). Based on the systematic review of evidence produced by epidemiological and experimental studies, the relationship between night noise exposure and health effects can be summarized as below. (Table 5.4)

Table 5.4
Effects of different levels of night noise on the population's health[2]

Average night noise level over a year $L_{night,outside}$	Health effects observed in the population
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed. $L_{night,outside}$ of 30 dB is equivalent to the NOEL for night noise.
30 to 40 dB	A number of effects on sleep are observed from this range: body movements, awakening, self-reported sleep disturbance, arousals. The intensity of the effect depends on the nature of the source and the number of events. Vulnerable groups (for example children, the chronically ill and the elderly) are more susceptible. However, even in the worst cases the effects seem modest. $L_{night,outside}$ of 40 dB is equivalent to the LOAEL for night noise.
40 to 55 dB	Adverse health effects are observed among the exposed population. Many people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
Above 55 dB	The situation is considered increasingly dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. There is evidence that the risk of cardiovascular disease increases.

[1] "Article 8:1. Everyone has the right to respect for his private and family life, his home and his correspondence." Although in the case against the United Kingdom the Court ruled that the United Kingdom Government was not guilty of the charges, the right to undisturbed sleep was recognized (the Court's consideration 96).

[2] $L_{night,outside}$ in Table 5.4 and 5.5 is the night-time noise indicator (L_{night}) of Directive 2002/49/EC of 25 June 2002: the A-weighted long-term average sound level as defined in ISO 1996-2: 1987, determined over all the night periods of a year; in which: the night is eight hours (usually 23.00 – 07.00 local time), a year is a relevant year as regards the emission of sound and an average year as regards the meteorological circumstances, the incident sound is considered, the assessment point is the same as for L_{den}. See *Official Journal of the European Communities*, 18.7.2002, for more details.

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Below the level of 30 dB $L_{night,outside}$, no effects on sleep are observed except for a slight increase in the frequency of body movements during sleep due to night noise. There is no sufficient evidence that the biological effects observed at the level below 40 dB $L_{night,outside}$ are harmful to health. However, adverse health effects are observed at the level above 40 dB$L_{night,outside}$, such as self-reported sleep disturbance, environmental insomnia, and increased use of somnifacient drugs and sedatives. Therefore, 40 dB $L_{night,outside}$ is equivalent to the LOAEL for night noise. Above 55 dB the cardiovascular effects become the major public health concern, which are likely to be less dependent on the nature of the noise. Closer examination of the precise impact will be necessary in the range between 30 dB and 55 dB as much will depend on the detailed circumstances of each case.

A number of instantaneous effects are connected to threshold levels expressed in L_{Amax} (Table 5.1). The health relevance of these effects cannot be easily established. It can be safely assumed, however, that an increase in the number of such events over the baseline may constitute a subclinical adverse health effect by itself leading to significant clinical health outcomes.

Based on the exposure–effects relationship summarized in Table 5.4, the night noise guideline values are recommended for the protection of public health from night noise as below (Table 5.5).

Table 5.5
Recommended night noise guidelines for Europe

Night noise guideline (NNG)	$L_{night,outside}$ = 40 dB
Interim target (IT)	$L_{night,outside}$ = 55 dB

For the primary prevention of subclinical adverse health effects related to night noise in the population, it is recommended that the population should not be exposed to night noise levels greater than 40 dB of $L_{night,outside}$ during the part of the night when most people are in bed. The LOAEL of night noise, 40 dB $L_{night,outside}$, can be considered a health-based limit value of the night noise guidelines (NNG) necessary to protect the public, including most of the vulnerable groups such as children, the chronically ill and the elderly, from the adverse health effects of night noise.

An interim target (IT) of 55 dB $L_{night,outside}$ is recommended in the situations where the achievement of NNG is not feasible in the short run for various reasons. It should be emphasized that IT is not a health-based limit value by itself. Vulnerable groups cannot be protected at this level. Therefore, IT should be considered only as a feasibility-based intermediate target which can be temporarily considered by policy-makers for exceptional local situations.

All Member States are encouraged to gradually reduce the proportion of the population exposed to levels over the IT within the context of meeting wider sustainable development objectives. It is highly recommended to carry out risk assessment and management activities at local and national levels targeting the exposed population, and aiming at reducing night noise to the level below IT or NNG. IT and NNG can be used for health impact assessment of new projects (for example construction of roads, railways, airports or new residential areas) even before the achievement of IT, as well as for the risk assessment of the whole population. In the long run the NNG would be best achieved by control measures aimed at the sources along with other comprehensive approaches.

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5.7 RELATION WITH THE GUIDELINES FOR COMMUNITY NOISE (1999)

The *Guidelines for community noise* (WHO, 1999) have been quoted a number of times in this paper, so one could rightfully ask what the relation is between the 1999 guidelines and the present NNG.

Impact of night-time exposure to noise and sleep disturbance is indeed covered in the 1999 guidelines, and this is the full statement (WHO, 1999):

> "If negative effects on sleep are to be avoided the equivalent sound pressure level should not exceed 30 dBA indoors for continuous noise. If the noise is not continuous, sleep disturbance correlates best with L_{Amax} and effects have been observed at 45 dB or less. This is particularly true if the background level is low. Noise events exceeding 45 dBA should therefore be limited if possible. For sensitive people an even lower limit would be preferred. It should be noted that it should be possible to sleep with a bedroom window slightly open (a reduction from outside to inside of 15 dB). To prevent sleep disturbances, one should thus consider the equivalent sound pressure level and the number and level of sound events. Mitigation targeted to the first part of the night is believed to be effective for the ability to fall asleep."

It should be noted that the noise indicators of the 1999 guidelines are L_{Aeq} and L_{Amax}, measured inside for continuous and non-continuous noise, respectively. The present night noise guidelines adopt an harmonized noise indicator as defined by Environmental Noise Directive (2002/49/EC): L_{night} measured outside, averaged over a year.

It should also be borne in mind that the 1999 guidelines are based on studies carried out up to 1995 (and a few meta-analyses some years later). Important new studies (Passchier-Vermeer et al., 2002; Basner et al., 2004) have become available since then, together with new insights into normal and disturbed sleep.

Comparing the above statement with the recommendations, it is clear that new information has made more precise statements possible. The thresholds are now known to be lower than L_{Amax} of 45 dB for a number of effects. The last three sentences still stand: there are good reasons for people to sleep with their windows open, and to prevent sleep disturbances one should consider the equivalent sound pressure level and the number of sound events. The present NNG allow responsible authorities and stakeholders to do this. Viewed in this way, the *Night noise guidelines for Europe* complements the 1999 guidelines. This means that the recommendations on government policy framework on noise management elaborated in the 1999 guidelines should be considered valid and relevant for the Member States to achieve the guideline values of this document.

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REFERENCES

Abey Wickrama I et al. (1969). Mental hospital admissions and aircraft noise. *Lancet*, 2 (7633):1275–1277.

Adams S (1991). Prescribing of psychotropic drugs to children and adolescents. *British Medical Journal*, 302:217.

Ahlberg-Hultén GK, Theorell T, Sigala F (1995). Social support, job strain and musculoskeletal pain among female health care personnel, *Scandinavian Journal of Work, Environment and Health*, 21 (6):435–439.

Åkerstedt T (1987). Sleep and stress. In: Peter JH, Podszus T and von Wichert P, eds. *Sleep related disorders and internal diseases*. Springer Verlag, Heidelberg, pp. 183–191.

Åkerstedt T (1988). Sleepiness as a consequence of shift work. *Sleep*, 11:17–34.

Åkerstedt T (1990). Sleep and performance – an introduction. In: Costa G et al., eds. *Shift work: health, sleep and performance*. Peter Lang, Frankfurt am Main, pp. 409–414.

Åkerstedt T, Gillberg M (1981). The circadian variation of experimentally displaced sleep. *Sleep*, 4:159–169.

Åkerstedt T, Gillberg M (1986). A dose-response study of sleep loss and spontaneous sleep termination. *Psychophysiology*, 23:293–297.

Åkerstedt T, Nilsson PM (2003). Sleep as restitution: an introduction. *Journal of Internal Medicine*, 254:6–12.

Åkerstedt T, Kecklund G, Horte L-G (2001). Night driving, season, and the risk of highway accidents. *Sleep*, 24:401–406.

Åkerstedt T, Kecklund G, Knutsson A (1991). Spectral analysis of sleep electroencephalography in rotating three-shift work. *Scandinavian Journal of Work, Environment and Health*, 17:330–336.

Åkerstedt T et al. (2002a). A prospective study of fatal occupational accidents – relationship to sleeping difficulties and occupational factors. *Journal of Sleep Research*, 11:69–71.

Åkerstedt T et al. (2002b). Work load and work hours in relation to disturbed sleep and fatigue in a large representative sample. *Journal of Psychosomatic Research*, 53:585–588.

Åkerstedt T et al. (2002c). Sleep disturbances, work stress and work hours. A cross-sectional study. *Journal of Psychosomatic Research*, 53:741–748.

Algers B, Ekesbo I, Strömberg S (1978). The impact of continuous noise on animal health. *Acta Veterinaria Scandinavica*, 67 (suppl.):1–26.

Ali NJ, Pitson D, Stradling JR (1993). Snoring, sleep disturbance and behaviour in 4–5 year-olds. *Archives of Disease in Childhood*, 68:360–366.

Ali NJ, Pitson D, Stradling JR (1994). Natural history of snoring and related behaviour problems between the ages of 4 and 7 years. *Archives of Disease in Childhood*, 71:74–76.

Ali NJ, Pitson D, Stradling JR (1996). Sleep disordered breathing: effects of adenotonsillectomy on behaviour and psychological functioning. *European Journal of Pediatrics*, 155:56–62.

American Academy of Pediatrics Committee on Environment Health (1997). Noise: a hazard for the fetus and newborn. *Pediatrics*, 100:724–727.

American Academy of Sleep Medicine (2005). *International Classification of Sleep Disorders. Diagnostic and coding manual*. 2nd edition, Westchester, Illinois.

American Sleep Disorders Association (1992). EEG arousals: scoring rules and examples. A preliminary report from the Sleep Disorders Atlas Task Force. *Sleep*, 15 (2):174–184.

American Sleep Disorders Association (1997). *International Classification of Sleep Disorders. Diagnostic and coding manual revised*. Rochester, Minnesota.

010227

Ancoli-Israel S, Roth T (1999). Characteristics of insomnia in the United States: Results of the 1991 National Sleep Foundation survey. *Sleep*, 22 (suppl.2):S347–S353.

Anderson CMB (1971). *The measurement of attitude to noise and noises, National Physical Laboratory Acoustics Report*, AC 52, Teddington, Middlesex.

Angersbach D et al. (1980). A retrospective cohort study comparing complaints and disease in day and shift workers. *International Archives of Occupational and Environmental Health*, 45:i27–i40.

Arguelles A et al. (1970). Endocrine and metabolic effects of noise in normal, hypertensive and psychotic subjects. In: Welch BL and Welch AS, eds. *Physiological effects of noise*. Plenum Press, New York.

Aristotle (350 B.C.E). *On sleep and sleeplessness*, Translated by J. I. Beare, http://classics.mit.edu/Aristotle/sleep.html.

Arnetz B et al. (1986). *Lakares arbetsmiljo: psykosociala, fysikaliska saint fysiologiska data fran en strukturerad djupintervju och biokemisk undersokning av allmanlakare och allmankirurger.* Stressforskningsrapport, IPM, No. 187.

Aronen ET et al. (2000). Sleep and psychiatric symptoms in school-age children. *Journal of the American Academy of Childhood and Adolescent Psychiatry*, 39:502–508.

ATS (American Thoracic Society) (1999) Cardiorespiratory sleep studies in children. Establishment of normative data and polysomnographic predictors of morbidity. *American Journal of Respiratory and Critical Care Medicine*, 160:1381–1387.

Axelsson G, Rylander R (1989). Outcome of pregnancy in relation to irregular and inconvenient work schedules. *British Journal of Industrial Medicine*, 46:306–312.

Axelsson G, Lutz C, Rylander R (1984). Exposure to solvents and outcome of pregnancy in university laboratory employees. *British Journal of Industrial Medicine*, 41:305–312.

Axelsson J et al. (2004). Tolerance to shift work – how does it relate to sleep and wakefulness? *International Archives of Occupational and Environmental Health*, 77(2):121–9.

Ayas NT et al. (2001). Effectiveness of CPAP treatment in daytime function in sleep apnea syndrome. *American Journal of Respiratory and Critical Care Medicine*, 164:608–613.

Babisch W (2000). Traffic noise and cardiovascular disease: epidemiological review and synthesis. *Noise & Health*, 2 (8):9–32.

Babisch W (2002). The noise/stress concept, risk assessment and research needs. *Noise & Health*, 4 (16):1–11.

Babisch W (2003). Stress hormones in the research on cardiovascular effects of noise. *Noise & Health*, 5 (18):1–11.

Babisch W (2004a). Health aspects of extra-aural noise research. *Noise & Health*, 6 (22):69–81.

Babisch W (2004b).The NaRoMI study: Executive summary – traffic noise. In: Umweltbundesamt, ed. *Chronic noise as a risk factor for myocardial infarction, The NaRoMI study (major technical report)*. WaBoLu-Hefte 02/04, Umweltbundesamt, Berlin.

Babisch W (2004c), Die NaRoMI-Studie, Auswertung, Bewertung und vertiefende Analysen zum Verkehrslärm. In: Umweltbundesamt, ed. *Chronischer Lärm als Risikofaktor für den Myokardinfarkt, Ergenbisse der "NaRoMI"-Studie.* WaBoLu-Hefte 03/04, Umweltbundesamt, Berlin.

Babisch W (2006). *Transportation noise and cardiovascular risk. Review and synthesis of epidemiological studies, dose-effect curve and risk estimation* (http://www.umweltdaten.de/publikationen/fpdf-l/2997.pdf, accessed 1 March 2007).

Babisch W, Gallacher JEJ (1990). Traffic noise, blood pressure and other risk factors: the Caerphilly and Speedwell Collaborative Heart Disease Studies. In: Berglund B, Lindvall T, eds. *New advances in noise research, part I. Proceedings of the 5th International Congress on Noise as a Public Health Problem, Stockholm 1988*, Stockholm, Swedish Council for Building Research.

010228

Babisch W, Ising H, Gallacher JEJ (2003). Health status as a potential effect modifier of the relation between noise and incidence of ischaemic heart disease. *Occupational and Environmental Medicine*, 60:739–745.

Babisch W et al. (1988). Traffic noise and cardiovascular risk. The Caerphilly Study, first phase. Outdoor noise levels and risk factors. *Archives of Environmental Health*, 43:407–414.

Babisch W et al. (1990). Traffic noise, work noise and cardiovascular risk factors: the Caerphilly and Speedwell Collaborative Heart Disease Studies. *Environment International*, 16:425–435.

Babisch W et al. (1992). *Verkehrslärm und Herzinfarkt, Ergebnisse zweier Fall-Kontroll-Studien in Berlin*. WaBoLu-Hefte 2/92, Institut für Wasser-, Boden- und Lufthygiene, Umweltbundesamt, Berlin.

Babisch W et al. (1993a). Traffic noise and cardiovascular risk: the Caerphilly and Speedwell studies, second phase. Risk estimation, prevalence, and incidence of ischemic heart disease. *Archives of Environmental Health*, 48:406–413.

Babisch W et al. (1993b). Traffic noise and cardiovascular risk: the Speedwell study, first phase. Outdoor noise level and risk factors. *Archives of Environmental Health*, 48:401–405.

Babisch W et al. (1994). The incidence of myocardial infarction and its relation to road traffic noise – the Berlin case-control studies. *Environment International*, 20:469–474.

Babisch W et al. (1999). Traffic noise and cardiovascular risk: the Caerphilly and Speedwell studies, third phase – 10 years follow-up. *Archives of Environmental Health*, 54:210–216.

Babisch W et al. (2003). Traffic noise and myocardial infarction. results from the NaRoMI study, *ICBEN Proceedings, Schiedam*.

Babisch W et al. (2005). Traffic noise and risk of myocardial infarction. *Epidemiology*, 16:33–40.

Barker SM, Tarnopolsky A (1978). Assessing bias in surveys of symptoms attributed to noise. *Journal of Sound and Vibration*, 59:349–354.

Basner M (2006). *Markov state transition models for the prediction of changes in sleep structure induced by aircraft noise*, Forschungsbericht 2006-07. Deutsches Zentrum für Luft und Raumfahrt (DLR), Köln.

http://www.eid.dlr.de/me/Institut/Abteilungen/Flugphysiologie/Fluglaerm/Lit_Laermw_Reports/FB_2006-07_markov.pdf, accessed 1 March 2007).

Basner M et al. (2004). *Effects of nocturnal aircraft noise*. Deutsches Zentrum für Luft und Raumfahrt (DLR) Bericht 2004-07/E.

Baust W, Bohnert B (1969). The regulation of heart rate during sleep. *Experimental Brain Research*, 7:169–180.

Bellach B et al. (1995). Gesundheitliche Auswirkungen von Lärmbelastung – Methodische Betrachtungen zu den Ergebnissen dreier epidemiologischer Studien. *Bundesgesundheitsblatt*, 38:84–89.

Belojevic G, Jakovljevic B (1997). Subjective reactions to traffic noise with regard to some personality traits. *Environment International*, 23:221–226.

Belojevic G, Saric-Tanaskovic M (2002). Prevalence of arterial hypertension and myocardial infarction in relation to subjective ratings of traffic noise exposure. *Noise & Health*, 4 (16):33–37.

Benedict C et al. (2004). Intranasal insulin improves memory in humans. *Psychoneuroendocrinology*, 29:1326–1334.

Bennett E (1945). Some tests for the discrimination of neurotic from normal subjects, *British Journal of Medical Psychology*, 20:271–277.

Berglund B, Lindvall T (1995). Community noise. Document prepared for the World Health Organization. *Archives of the Center for Sensory Research* 2 (1), Stockholm, Center for Sensory Research.

Biesiot W, Pulles MPJ, Stewart RE (1989). *Environmental noise and health*. VROM, GA-DR-03-03, Leidschendam.

010229

Bistrup ML (2003). Prevention of adverse effects of noise on children. *Noise & Health*, 5:59–64.

Bistrup ML et al. (2001). *Health effects of noise on children – and perception of risk of noise.* National Institute of Public Health, Copenhagen.

Bite M, Bite PZ (2004). Zusammenhang zwischen den Straßenverkehrslärmindizes L_{Aeq}(06-22) und L_{Aeq}(22-06) sowie L_{den}. *Zeitschrift für Lärmbekämpfung*, 51:27–28.

Bixler EO, Kales A, Soldatos CR (1979). Sleep disorders encountered in medical practice: A national survey of physicians. *Behavioral Medicine*, 1–6.

Bjerner B, Holm A, Swensson A (1955). Diurnal variation of mental perfomance. A study of three-shift workers. *British Journal of Industrial Medicine*, 12:103–110.

Blettner M et al. (1999). Traditional reviews, meta-analyses and pooled analyses in epidemiology. *International Journal of Epidemiology*, 28:1–9.

Bliwise Dl (1993). Sleep in normal aging and dementia. *Sleep*, 16:40–81.

Bluhm G, Nordling E, Berglind N (2001). Increased prevalence of hypertension in a population exposed to road traffic noise. In Boone R, ed. *Internoise 2001. Proceedings of the 2001 International Congress and Exhibition on Noise Control Engineering, The Hague, Vol. 3.* Nederlands Akoestisch Genootschap, Maastricht.

Bluhm G, Nordling E, Berglind N (2004). Road traffic noise and annoyance – an increasing environmental health problem. *Noise & Health,* 6 (24):43–49.

Bluhm G et al. (2004). Aircraft noise exposure and cardiovascular risk among men – First results from a study around Stockholm Arlanda airport. In: Czech Acoustoical Society, ed. *Proceedings of the 33rd International Congress and Exhibition on Noise Control Engineering, Prague,* 31.

Blunden S, Lushington K, Kennedy D (2001). Cognitive and behavioural performance in children with sleep-related obstructive breathing disorders. *Sleep Medicine Reviews*, 5:447–461.

Blunden S et al. (2000). Behaviour and neurocognitive function in children aged 5–10 years who snore compared to controls. *Journal of Clinical and Experimental Neuropsychology*, 22:554–568.

Boggild H, Knutsson A (1999). Shift work, risk factors and cardiovascular disease. *Scandinavian Journal of Work, Environment and Health*, 25:85–99.

Boggild H et al. (1999). Shift work, social class, and ischaemic heart disease in middle aged and elderly men: a 22-year follow-up in the Copenhagen Male Study. *Occupational and Environmental Medicine*, 56:640–645.

Boman E, Enmarker I, Hygge S (2004). Strength of noise effects on memory as a function of noise source and age. *Noise & Health*, 7:11.26.

Bonnet MH (1985). Effect of sleep disruption on sleep, performance, and mood. *Sleep*, 8:11–19.

Bonnet MH (1986a). Performance and sleepiness as a function of frequency and placement of sleep disruption. *Psychophysiology*, 23:263–271.

Bonnet MH (1986b). Performance and sleepiness following moderate sleep disruption and slow wave sleep deprivation. *Physiology and Behavior*, 37:915–918.

Borbely AA et al. (1981). Sleep deprivation: effect on sleep stages and EEG power density in man. *Electroencephalography and Clinical Neurophysiology*, 51 (5):483–495.

Bormann C et al. (1990). *Subjektive Morbidität.* BGA Schriften 4/90, MMV Medizin Verlag, Munich.

Born J, Wagner U (2004). Awareness in memory: being explicit about the role of sleep. *Trends in Cognitive Sciences*, 8:242–244.

Boselli M et al. (1998). Effects of age on EEG arousals in normal sleep. *Sleep*, 21 (4):351–357.

Bowlin SJ et al. (1993). Validity of cardiovascular disease risk factors assessed by telephone survey: the behavioral risk survey. *Journal of Clinical Epidemiology*, 46:561–571.

010230

Bradford Hill AS (1965). *The environment and disease: association or causation? Proceedings of the Royal Society of Medicine*, 58:295–300.

Breslau N et al. (1996). Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biological Psychiatry*, 39 (6):411–418.

Brink M, Müller CH, Schierz C (2006). Contact-free measurement of heart rate, respiration rate, and body movements during sleep. *Behavior Research Methods*, 38 (3):511–521.

Brink M, Wirth K, Schierz C (2006). Effects of early morning aircraft overflights on sleep and implications for policy making. *Euronoise 2006*, Tampere.

Broadbent DE (1953). Noise, paced performance and vigilance tasks. *British Journal of Psychology*, 44:295–303.

Broadbent DE (1972). Individual differences in annoyance by noise. *Sound*, 6:56–61.

Broadbent DE (1983). Recent advances in understanding performance in noise. In: Rossi G, ed. *Proceedings of 4th International Congress, Noise as a Public Health Problem*. Centro Ricerche e Studi Amplifon, Milan, pp. 719–738.

Brown RC (1949). The day and night performance of teleprinter switchboard operators. *Journal of Occupational Psychology*, 23:121–126.

Brown R (1992). Sleep deprivation and the immune response to pathogenic and non-pathogenic antigens. In: Husband AJ, ed. *Behaviour and immunity*. CRC Press, Bocaton, Florida.

Bruni O et al. (2004). The sleep knowledge of pediatricians and child neuropsychiatrists. *Sleep and Hypnosis*, 6 (3):130–138.

Bryden G, Holdstock TI (1973). Effects of night duty on sleep patterns of nurses. *Psychophysiology*, 10:36–42.

Busby K, Pivik R (1985). Auditory arousal threshold during sleep in hyperkinetic children. *Sleep*, 8:332–341.

Busby K, Mercier L, Pivik R (1994). Ontogenetic variations in auditory arousal threshold during sleep. *Psychophysiology*, 31:182–188.

Caille EJ, Bassano JL (1977). Validation of a behavior analysis methodology: variation of vigilance in night driving as a function of the rate of carboxyhemoglobin. In: Mackie RR, ed. *Vigilance*. Plenum Press, New York, pp. 59–72.

Cairns V et al. (1985). Oral contraceptive use and blood pressure in a German metropolitan population. *International Journal of Epidemiology*, 14:389–395.

Calabrese EJ, Baldwin LA (2003). Toxicology rethinks its central belief. Hormesis demands a reappraisal of the way risks are assessed. *Nature*, 421:691–692.

CALM (2004). Research for a quieter Europe in 2020 (http://www.calm-network.com/index_stratpap.htm, accessed 11 November 2006)

Carskadon MA, Harvey K, Dement WC (1981a). Acute restriction of nocturnal sleep in children. *Perceptual and Motor Skills*, 53:103–112.

Carskadon MA, Harvey K, Dement WC (1981b). Sleep loss in young adolescents. *Sleep*, 4:299–321.

Carter NL (1996). Transportation noise, sleep, and possible after-effects. *Environment International*, 22:105–116.

Carter NL (1998). Cardiovascular response to environmental noise during sleep. In: *7th International Congress on Noise as a Public Health Problem, Sydney, Australia*, 2:439–444.

Carter NL et al. (1994). Environmental noise and sleep – a study of arousals, cardiac arrhythmia and urinary catecholamines. *Sleep*, 17 (4):298–307.

Carter NL et al. (2002). Cardiovascular and autonomic response to environmental noise during sleep in night shift workers. *Sleep*, 25:444–451.

010231

Cartwright J, Flindell I (2000). Research methodology – noise annoyance and health. In: Cassereau D, ed. *Internoise 2000. Proceedings of the 29th International Congress on Noise Control Engineering, Nice.* Société Française d'Acoustique (SFA), Nice.

Centre for Time Use Research (2006). (http://www.timeuse.org/access/, accessed 11 November 2006).

Chang PP et al. (1997). Insomnia in young men and subsequent depression. The Johns Hopkins Precursors Study, *American Journal of Epidemiology*, 146:105–114.

Chapman KR et al. (1988). Possible mechanisms of periodic breathing during sleep. *Journal of Applied Physiology*, 64:1000–1008.

Chartered Institute of Environmental Health (1999). *Environmental health report 1997/8.*

Chervin RD et al. (1997). Symptoms of sleep disorders, inattention, and hyperactivity in children. *Sleep*, 20:1185–1192.

Chowns RH (1970). Mental hospital admissions and aircraft noise. *Lancet*, 1:467–468.

Christoffel T, Teret SP (1991). Public health and law. Epidemiology and the law: courts and confidence intervals. *American Journal of Public Health*, 81:1661–1666.

Cirelli C et al. (2005). Reduced sleep in *Drosophila shaker* mutants. *Nature*, 434:1087–1092.

Cohen S, Spacapan S (1978). The after-effects of stress: an attentional interpretation, *Environmental Psychology and Non-verbal Behaviour*, 3:43–57.

Cohen S, Weinstein N (1981). Non-auditory effects of noise on behaviour and health. *Journal of Social Issues*, 37:36–70.

Cohen S, Kessler, RC and Underwood Gordon L (1995). Strategies for measuring stress in studies of psychiatric and physical disorders. In: Cohen S, Kessler RC and Underwood Gordon L, eds. *A guide for health and social scientists.* Oxford University Press, New York.

Cohen S et al. (1980). Physiological, motivational, and cognitive effects of aircraft noise on children. *American Psychologist*, 35:231–243.

Cohen S et al. (1981). Aircraft noise and children: longitudinal and cross-sectional evidence on adaptation to noise and the effectiveness of noise abatement. *Journal of Personality and Social Psychology*, 40:331–345.

Coleman RM and Dement WC (1986). Falling asleep at work: a problem for continuous operations. *Sleep Research*, 15:265.

Cooper R, ed. (1994). *Sleep.* Chapman and Hall Medical, London.

Crabtree VM et al. (2003). Periodic limb movement disorder of sleep in children. *Journal of Sleep Research*, 12:73–81.

Czeisler CA et al. (1980). Human sleep: its duration and organization depend on its circadian phase. *Science*, 210:1264–1267.

Dahlgren K (1981a). Adjustment of circadian rhythms and EEG sleep functions to day and night sleep among permanent night workers and rotating shift workers. *Psychophysiology*, 18:381–391.

Dahlgren K (1981b). Long-term adjustment of circadian rhythms to a rotating shiftwork schedule. *Scandinavian Journal of Work, Environment and Health*, 7:141–151.

Dawson D, Reid K (1997). Fatigue, alcohol and perfomance impairment. *Nature*, 388:235.

Dew MA et al. (2003). Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. *Psychosomatic Medicine*, 65:63–73.

Diaz J et al. (2001). *Traffic noise pollution. Similarities and differences between European regions. A state-of-the-art-review.* Technical University, Berlin.

Dickens. *Gutenberg Electronic Library,* http://www.gutenberg.org/files/580/580.txt.

010232

Dijk D-J, Czeisler CA (1995). Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *Journal of Neuroscience*, 15:3526–3538.

Dijk D-J, Duffy JF (1999). Circadian regulation of human sleep and age-related changes in its timing. *Annals of Medicine*, 31:130–140.

Dijk D-J, Beersma DGM and Bloem GM (1989). Sex differences in the sleep EEG of young adults: Visual scoring and spectral analysis. *Sleep*, 12:500–507.

Dijk D-J, Beersma DGM, van den Hofdakker RH (1989). All night spectral analysis of EEG sleep in young adult and middle-aged male subjects. *Neurobiology of Aging*, 10:677–682.

Dijk D-J, Duffy JF, Czeisler CA (1992). Circadian and sleep-wake dependent aspects of subjective alertness and cognitive performance. *Journal of Sleep Research*, 1:112–117.

Dijk D-J et al. (1999). Ageing and the circadian and homeostatic regulation of human sleep during forced desynchrony of rest, melatonin and temperature rhythms. *Journal of Physiology*, 516:611–627.

Dinges DF et al. (1997). Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. *Sleep*, 20:267–277.

Di Nisi J et al. (1990). Comparison of cardiovascular responses to noise during waking and sleeping in humans. *Sleep*, 13:108–120.

Dongen JEF (van) et al. (1998) *Dosis-effect relaties voor geluid van buren*. TNO PG, Leiden, Publication No. 98.002.

Dongen, JEF (van) et al. (2004). *Hinder door milieufactoren en de beoordeling van de leefomgeving in Nederland*. RIVM, Bilthoven, TNO-INRO, Delft.

Drosopoulos S, Wagner U, Born, J (2005). Sleep enhances explicit recollection in recognition memory. *Learning & Memory*, 12:44–51.

Dryman A, Eaton WW (1991). Affective symptoms associated with the onset of major depression in the community: findings from the US National Institute of Mental Health Epidemiologic Catchment Area Program. *Acta Psychiatrica Scandinavica*, 84 (1):1–5.

Dugué B, Leppänen E, Gräsbeck R (1994). Preanalytical factors and standardized specimen collection: the effects of industrial noise. *Stress Medicine*, 10:185–189.

Dumont M, Montplaisir J, Infante-Rivard C (1988). Insomnia symptoms in nurses with former permanent nightwork experience. In: Koella WP et al., eds. *Sleep '86*. Gustav Fischer Verlag, Stuttgart, pp. 405–406.

Ebersole JS, Pedley TA, eds. (2003). *Current practice of clinical electroencephalography*. 3rd edition, Lippincott Williams & Wilkins, Philadelphia.

EEA (2004). *Traffic noise: exposure and annoyance*. European Environmental Agency, Copenhagen.

Ehlers CL, Kupfer DJ (1997). Slow-wave sleep: Do young adult men and women age differently? *Sleep*, 6:211–215.

Eiff AWV (1993). Selected aspects of cardiovascular responses to acute stress. In: Ising H, Kruppa B, eds. *Lärm und Krankheit – Noise and Disease. Proceedings of the International Symposium, Berlin 1991*. Gustav Fischer Verlag, Stuttgart.

Eiff AWV, Neus H (1980). Verkehrslärm und Hypertonie-Risiko. 1. Mitteilung: Verkehrslärm und Hypertonie-Risiko. *Münchener Medizinische Wochenschrift*, 122:893–896.

Eiff AWV et al. (1974). Kapitel 7: Der medizinische Untersuchungsteil. In: Forschungsgemeinschaft D, eds. *Fluglärmwirkungen I, Hauptbericht*. Harald Boldt Verlag KG, 32, Boppard.

Eiff AWV et al. (1981a). Verkehrslärm und Hypertonie-Risiko. 2. Mitteilung: Hypothalamus-Theorie der essentiellen Hypertonie. *Münchener Medizinische Wochenschrift*, 123:420–424.

Eiff AWV et al. (1981b). *Feststellung der erheblichen Belästigung durch Verkehrslärm mit Mitteln*

010233

der Streßforschung (Bonner Verkehrslärmstudie). Umweltforschungsplan des Bundesminister des Innern, Lärmbekämpfung, Forschungsberichts Nr. 81-10501303, Umweltbundesamt, Berlin.

Ekstedt M, Åkerstedt T, Soderstrom M (2004). Arousals during sleep are associated with increased levels of lipids, cortisol and blood pressure. *Psychosomatic Medicine*, 66:925–931.

Engle-Friedman M et al. (2003). The effect of sleep loss on next day effort. *Journal of Sleep Research*, 12:113–124.

European Commission (2000). *Position paper on EU noise indicators*. Office for Official Publications of the European Communities, Luxembourg.

European Commission (2002a). *Position paper on dose–response relationships between transportation noise and annoyance*. Office for Official Publications of the European Communities, Luxembourg.

European Commission (2002b). *Directive 2002/49/EC relating to the assessment and management of environmental noise*. Office for Official Publications of the European Communities, Luxembourg.

European Commission (2004). *Position paper on dose-effect relationships for night time noise* (http://ec.europa.eu/environment/noise/home.htm#1, accessed 11 November 2006).

European Commission (2006). Communication and Information Resource Centre Administrator (CIRCA) (http://forum.europa.eu.int/Public/irc/env/d_2002_49/home, accessed 11 November 2006).

European Court of Human Rights (2003). *Case of Hatton and others vs. the United Kingdom* (Application No. 36022/97).

Evans AS (1976) .Causation and disease: the Henle-Koch postulates revised. *Yale Journal of Biology and Medicine*, 49:175–195.

Evans A, Hygge S, Bullinger M (1995). Chronic noise exposure and physiological stress. *Psychological Science*, 6:333–338.

Evans GW, Bullinger M, Hygge S (1998). Chronic noise exposure and psychological response: A prospective study of children living under environmental stress. *Psychological Science*, 9:75–77.

Evans GW, Johnson D (2000). Stress and open-office noise. *Journal of Applied Psychology*, 85 (5):779–783.

Evans GW, Lepore SJ (1993). Nonauditory effects of noise on children: a critical review. *Children's Environments*, 10 (1):31–51.

Evans GW et al. (2001). Community noise exposure and stress in children. *Journal of the Acoustical Society of America*, 109:1023–1027.

Everson CA, Toth LA (2000). Systemic bacterial invasion induced by sleep deprivation. *American Journal of Physiology, Regulatory, Integrative and Comparative Physiology*, 278:R905–R916.

Fabsitz RR, Sholinsky P, Goldberg J (1997). Correlates of sleep problems among men: the Vietnam era twin registry. *Journal of Sleep Research*, 6:50–60.

Fallone G et al. (2001). Effects of acute sleep restriction on behavior, sustained attention, and response inhibition in children. *Perceptual and Motor Skills*, 93:213–229.

Fallone G, Owens JA, Deane J (2002). Sleepiness in children and adolescents: clinical implications. *Sleep Medicine*, 6 (4):287–386.

Farley TMM et al. (1998). Combined oral contraceptives, smoking, and cardiovascular risk. *Journal of Epidemiology and Community Health*, 52:775–785.

Fidell S et al. (1998). *Field studies of habituation to change in night-time aircraft noise and of sleep motility measurement methods*. BBN Technologies, California, Report No. 8195.

Fidell S et al. (2000). Effects on sleep disturbance of changes in aircraft noise near three airports. Journal of the Acoustical Society of America, 107 (5:1):2535–2547.

010234

Fields JM (1993). Effect of personal and situational variables on noise annoyance in residential areas. *Journal of the Acoustical Society of America*, 93 (5):2753–2763.

Finke HO et al. (1974). *Effects of aircraft noise on man. Proceedings of the Symposium on Noise in Transportation, Section III, paper 1*. Institute of Sound and Vibration Research, Southampton, United Kingdom.

Flindell IH, Bullmore AJ, Robertson KA (2000). *Aircraft noise and sleep*. 1999 UK trial methodology study, Southampton, ISVR, Report 6131 RO1.

Folkard S, Åkerstedt T (1991). A three process model of the regulation of alertness and sleepiness. In: Ogilvie R, Broughton R, eds. *Sleep, arousal and performance: problems and promises*. Birkhauser, Boston, pp. 11–26.

Ford DE, Kamerow DB (1989). Epidemiologic study of sleep disturbances and psychiatric disorders: an opportunity for prevention. *Journal of the American Medical Association*, 262:1479–1484.

Foret J, Benoit O (1974). Structure du sommeil chez des travailleurs a horaires alternants. *Electroencephalography and Clinical Neurophysiology*, 37:337–344.

Foret J, Benoit O (1978). Shiftwork: the level of adjustment to schedule reversal assessed by a sleep study. *Waking and Sleeping*, 2:107–112.

Foret J, Lantin G (1972). The sleep of train drivers: an example of the effects of irregular work schedules on sleep. In: Coiqloun WP, ed. *Aspects of human efficiency. Diurnal rhythm and loss of sleep*. The English Universities Press Ltd, London, pp. 273–281.

Franssen EA, Staatsen BAM, Lebret E (2002). Assessing health consequences in an environmental impact assessment. The case of Amsterdam Airport Schiphol. *Environmental Impact Assessment Review*, 22:633–653.

Franssen EA et al. (2004). Aircraft noise around a large international airport and its impact on general health and medication use. *Journal of Occupational and Environmental Medicine*, 61 (5):405–413.

Frerichs RR, Beeman BL, Coulson AH (1980). Los Angeles Airport noise and mortality – faulty analysis and public policy. *American Journal of Public Health*, 3 (70):357–362.

Friedman M, Rosenman RH (1975). *Der A-Typ und der B-Typ*. Rowohlt Verlag Gmbh, Reinbek bei Hamburg.

Froberg J et al. (1975). Circadian variations of catecholamine excretion, shooting range performance and self-ratings of fatigue during sleep deprivation. *Biological Psychology*, 2:175–188.

Gais S, Born J (2004). Declarative memory consolidation: mechanisms acting during human sleep. *Learning & Memory*, 11:679–685.

Gattoni F, Tarnopolsky A (1973). Aircraft noise and psychiatric morbidity, *Psychological Medicine*, 3 (4):516–520.

Geroldi C et al. (1996). Principal lifetime occupation and sleep quality in the elderly. *Gerontology*, 42:163–169.

Gillberg M, Åkerstedt T (1998). Sleep loss and performance. No "safe" duration of a monotonous task. *Physiology and Behavior*, 64:599–604.

Gillman MW et al. (1992). Assessing the validity of childhood blood pressure screening: unbiased estimates of sensitivity, specificity, and predictive values. *Epidemiology*, 3:40–46.

Glass DC, Singer JE (1972). *Urban stress. Experiments on noise and social stressors*. Academic Press, New York.

Goldberg DP (1972). *The detection of psychiatric illness by questionnaire*. Oxford University Press, London.

Goldberg DP et al. (1970). A standardized psychiatric interview for use in community surveys. *British Journal of Preventive and Social Medicine*, 24 (1):18–23.

Goodman R (1997). The Strengths and Difficulties Questionnaire: a research note, *Journal of Child Psychology and Psychiatry*, 38 (5):581–586.

010235

Goto K, Kaneko T (2002). Distribution of blood pressure data from people living near an airport. *Journal of Sound and Vibration*, 250:145–149.

Gottlieb DJ et al. (2003). Symptoms of sleep-disordered breathing in 5-year-old children are associated with sleepiness and problem behaviors. *Pediatrics*, 112:870–877.

Gozal D (1998). Sleep-disordered breathing and school performance in children. *Pediatrics*, 102:616–620.

Gozal D, Pope DW (2001). Snoring during early childhood and academic performance at ages thirteen to fourteen years. *Pediatrics*, 107:1394–1399.

Grandjean E et al. (1973). *A survey of aircraft noise in Switzerland.* United States Environmental Protection Agency Publications, Washington, United States, 500:1973–008.

Griefahn B et al. (2000). Physiological, subjective and behavioural responses during sleep to noise from rail and road traffic, *Noise & Health*, 3 (9):59–71.

Griefahn B et al. (2002). Fluglärmkriterien für ein Schutzkonzept bei wesentlichen Änderungen oder Neuanlagen von Flughäfen/Flugplätzen. *Zeitschrift für Lärmbekämpfung*, 49:171–181.

Grimwood C (1993). *Effects of environmental noise on people at home.* BRE information paper.

Grossi G et al. (2003). Physiological correlates of burnout among women. *Journal of Psychosomatic Research*, 55 (4):309–316.

Guazzi M et al. (1968). Effects of cardiacdenervation on blood pressure and heart rate during natural sleep in the cat. *Cardiovascular Research*, 2:265–270.

Guilleminault C et al. (1982a). Circadian rhythm disturbances and sleep disorders in shift workers (EEG Suppl. No. 36). In: Buser PA, Cobb WA, Okuma T, eds. *Kyoto Symposia*, Elsevier, Amsterdam, pp. 709–714.

Guilleminault C et al. (1982b). Children and nocturnal snoring. Evaluation of the effects of sleep related respiratory resistive load and daytime functioning. *European Journal of Pediatrics*, 139:165–171.

Guilleminault C et al. (1996). Recognition of sleep disordered breathing in children. *Pediatrics*, 98:871–882.

Haines MM et al. (2001a). Chronic aircraft noise exposure, stress responses, mental health and cognitive performance in school children. *Psychological Medicine*, 31:265–277.

Haines MM et al. (2001b). A follow-up study of the effects of chronic aircraft noise exposure on child stress responses and cognition. *International Journal of Epidemiology*, 30:839–845.

Hall M et al. (2000). Symptoms of stress and depression as correlates of sleep in primary insomnia. *Psychosomatic Medicine*, 62:227–230.

Halpern D (1995). *Mental health and the built environment.* Taylor and Francis, London.

Hamelin P (1987). Lorry drivers' time habits in work and their involvement in traffic accidents. *Ergonomics*, 30:1323–1333.

Hampton SM et al. (1996). Postprandial hormone and metabolic responses in simulated shift work. *Journal of Endocrinology*, 151:259–267.

Hansen J (2001). Light at night, shiftwork, and breast cancer risk. *Journal of the National Cancer Institute*, 93 (20):1513–1515.

Harrington JM (1978). *Shift work and health. A critical review of the literature.* HMSO, London.

Harrington JM (1994). Shift work and health – a critical review of the literature on working hours. *Annals of the Academy of Medicine, Singapore*, 23:699–705.

Harris W (1977). Fatigue, circadian rhythm and truck accidents. In: Mackie RR, ed. *Vigilance.* Plenum Press, New York, pp. 133–146.

Hartig T (2004). Restorative environments. In: Spielberger C, ed. *Encyclopedia of applied psychology.* Academic Press, London.

010236

Hatfield J, Soames Job RF (2001). Optimism bias about environmental degradation: the role of the range of impact of precautions. *Journal of Environmental Psychology*, 21 (1):17–30 (14).

Hatfield J et al. (2001). The influence of psychological factors on self-reported physiological effects of noise. *Noise & Health*, 3 (10):1–13.

Health Council of the Netherlands (1994). *Noise and health. Report by a committee of the Health Council of the Netherlands, The Hague.*

Health Council of the Netherlands (1999). *Public health impact of large airports. Report by a committee of the Health Council of the Netherlands, The Hague.*

Health Council of the Netherlands (2003). *Report of a workshop on sleep, health, and noise. Held in connection with ICBEN on 2 July 2003.* Gezondheidsraad, The Hague.

Health Council of the Netherlands (2004). *The influence of night-time noise on sleep and health.* The Hague, publication No. 2004/14E.

Hennekens CH, Buring JE (1987). *Epidemiology in medicine.* Little, Brown and Company, Boston/Toronto.

Henry JP (1992). Biological basis of the stress response. *Integrative Physiological and Behavioral Science*, 27:66–83.

Henry JP, Stephens PM (1977). *Stress, health and the social environment, a sociobiologic approach to medicine.* Springer-Verlag, New York.

Hense H-W, Herbold M, Honig K (1989). *Risikofaktor Lärm in Felderhebungen zu Herz-Kreislauferkrankungen. Umweltforschungsplan des Bundesministers für Umwelt, Naturschutz und Reaktorsicherheit.* Forschungsbericht Nr. 89-10501111, Umweltbundesamt, Berlin.

Herbold M, Hense H-W, Keil U (1989). Effects of road traffic noise on prevalence of hypertension in men: results of the Lübeck blood pressure study. *Sozial- und Präventivmedizin*, 34:19–23.

Hildebrandt G, Rohmert W, Rutenfranz J (1974). 12- and 24-hour rhythms in error frequency of locomotive drivers and the influence of tiredness. *International Journal of Chronobiology*, 2:175–180.

Hiramatsu K et al. (1997). A survey on health effects due to aircraft noise on residents living around Kadena airport in the Ryukyus. *Journal of Sound and Vibration*, 205:451–460.

Hiramatsu K et al. (2000). An analysis of the general health questionnaire survey around airports in terms of annoyance reaction. *Internoise 2000*, Inrets, Nice.

Horne J, ed. (1988). *Why we sleep.* Oxford University Press, Oxford.

Horne et al. (1994). Sleep related vehicle accidents. *British Medical Journal*, 310:565–567.

Hume K, Morley H, Thomas C (2003). *Using complaints as a measure of annoyance at airport operations.* Manchester Metropolitan University, Manchester.

Hume KI, Van F, Watson A (1998). A field study of age and gender differences in habitual adult sleep. *Journal of Sleep Research*, 7:85–94.

Hume KI, Van F, Watson A (2003). *Effects of aircraft noise on sleep: EEG-Based measurements.* Manchester Metropolitan University, Manchester.

Hygge S, Evans GW, Bullinger M (2002). A prospective study of some effects of aircraft noise on cognitive performance in schoolchildren. *Psychological Science*, 13:469–474.

Hyyppa MT, Kronholm E (1989). Quality of sleep and chronic illness. *Journal of Clinical Epidemiology*, 42:633–638.

IEH (1997). *Workshop on non-auditory health effects of noise. Report No. R10.* Institute for Environment and Health, Leicester.

Ingre M, Åkerstedt T (2004). Effect of accumulated night work during the working lifetime, on subjective health and sleep in monozygotic twins. *Journal of Sleep Research*, 13 (1):45–48.

Irwin M, Fortner M, Clark C (1995). Reduction of natural killer cell activity in primary insomnia and in major depression. *Sleep Research*, 24:256.

010237

Ishihara K (1999). The effect of 2-h sleep reduction by a delayed bedtime on daytime sleepiness in children. *Psychiatry and Clinical Neurosciences*, 53:113–115.

Ishihara K, Miyke S (1998). A longitudinal study of the development of daytime sleepiness in children. *Psychiatry and Clinical Neurosciences*, 52:178–181.

Ising H (1983). *Streßreaktionen und Gesundheitsrisiko bei Verkehrslärmbelastung*. Dietrich Reimer Verlag, Berlin.

Ising H, Braun C (2000). Acute and chronic endocrine effects of noise: review of the research conducted at the Institute for Water, Soil and Air Hygiene. *Noise & Health*, 2 (7):7–24.

Ising H, Ising M (2002). Chronic cortisol increase in the first half of the night caused by road traffic noise. *Noise & Health*, 4 (16):13–21.

Ising H, Kruppa B (2004). Health effects caused by noise. Evidence in the literature from the past 25 years. *Noise & Health*, 6:5–13.

Ising H et al. (1980). Health effects of traffic noise. *International Archives of Occupational and Environmental Health*, 47:179–190.

Ising H et al. (2004). Low frequency noise and stress: bronchitis and cortisol in children exposed chronically to traffic noise and exhaust fumes. *Noise & Health*, 6 (23):23–30.

Ito A et al. (1994). Health effects on the residents due to aircraft noise around Kadena US Airbase in the Ryukyus. *Internoise 94, Yokohama, Japan*, pp. 247–250.

Iwata O (1984). The relationship of noise sensitivity to health and personality. *Japanese Psychological Research*, 26:75–81.

Järup L et al. (2003). Hypertension and exposure to noise near airports – the Hyena project. *Epidemiology*, 14 (5):S78. 34.

Jasanoff S (1993). Relating risk assessment and risk management. Complete separation of the two processes is a misconception. *EPA Journal*, Jan/Feb/Mar 1993:35–37.

Jelinkova A (1988). Coping with noise in noise sensitive subjects. In: Berglund B, ed. *Noise as a public health problem. Noise 88, Vol. 3,* Stockholm, Swedish Council for Building Research, pp. 27–30.

Jenkins L, Tarnopolsky A, Hand D (1981). Psychiatric admissions and aircraft noise from London Airport: four-year, three-hospitals study. *Psychological Medicine*, 11 (4):765–782.

Jenkins LM et al. (1979). Comparison of three studies of aircraft noise and psychiatric hospital admissions conducted in the same area. *Psychological Medicine*, 9 (4):681–693.

Jones DM, Chapman AJ, Auburn TC (1981). Noise the environment: a social perspective. *Journal of Applied Psychology*, 1:43–59.

Jurriëns AA et al. (1983). An essay in European research collaboration: common results from the project on traffic noise and sleep in the home. *Proceedings of the 4th International Congress on Noise as a Public Health Problem, Turin*, pp. 929–937.

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Kageyama T et al. (1997). A population study on risk factors for insomnia among adult Japanese women: a possible effect of road traffic volume. *Sleep*, 20 (11):963–971.

Kahn A et al. (1989). Sleep problems in healthy preadolescents. *Pediatrics*, 84:542–546.

Kalogjera-Sackellares D, Cartwright RD (1997). Comparison of MMPI profiles in medically and psychologically based insomnias. *Psychiatry Research*, 70 (1):49–56.

Kamp I (van) (1990). *Coping with noise and its health consequences*. Styx and PP Publications, Groningen.

010238

Karacan I et al. (1976). Prevalence of sleep disturbances in a primarily urban Florida county. *Social Science and Medicine*, 10:239–244.

Karagodina IL et al. (1969). Effect of aircraft noise on the population near airport. *Hygiene and Sanitation*, 34:182–187.

Karsdorf G, Klappach H (1968). Einflüsse des Verkehrslärms auf Gesundheit und Leistung bei Oberschülern einer Großstadt. *Zeitschrift für die gesamte Hygiene*, 14:52–54.

Kawachi I et al. (1995). Prospective study of shift work and risk of coronary heart disease in women. *Circulation*, 92:3178–3182.

Kecklund Q (1996). *Sleep and alertness: effects of shift work, early rising, and the sleep environment* (dissertation). Statens Institute for Psykosocial Miljomedicin (IPM), 252:1–94.

Kecklund G, Åkerstedt T (1993). Sleepiness in long distance truck driving: an ambulatory EEG study of night driving. *Ergonomics*, 36:1007–1017.

Kecklund G, Åkerstedt T (1997). Objective components of individual differences in subjective sleep quality. *Journal of Sleep Research*, 6, 217–220.

Keefe FB, Johnson LC, Hunter EJ (1971). EEG and autonomic response pattern during waking and sleep stages. *Psychophysiology*, 8:198–212.

Keipert JA (1985). The harmful effects of noise in a children's ward. *Australian Paediatric Journal*, 21:101–103.

Keith SW et al. (2006). Putative contributors to the secular increase in obesity: exploring the roads less traveled. *International Journal of Obesity*, 30:1585–1594.

Kempen EEMM (van) et al. (2002). The association between noise exposure and blood pressure and ischaemic heart disease: a meta-analysis. *Environmental Health Perspectives*, 110:307–317.

Kim K et al. (2000). An epidemiological study of insomnia among the Japanese general population. *Sleep*, 23 (1):41–47.

Klein DE, Bruner H, Holtman H (1970). Circadian rhythm of pilot's efficiency, and effects of multiple time zone travel. *Aerospace Medicine*, 41:125–132.

Knipschild, P. (1977a). V. Medical effects of aircraft noise: community cardiovascular survey. *International Archives of Occupational and Environmental Health*, 40:185–190.

Knipschild P (1977b). VI. Medical effects of aircraft noise: general practice survey. *International Archives of Occupational and Environmental Health*, 40:191–196.

Knipschild P, Oudshoorn N (1977). VII. Medical effects of aircraft noise: drug survey. *International Archives of Occupational and Environmental Health*, 40:197–200.

Knipschild P, Sallé H (1979). Road traffic noise and cardiovascular disease. *International Archives of Occupational and Environmental Health*, 44:55–59.

Knipschild P, Meijer H, Sallé H (1984). Wegverkeerslawaai, psychische problematiek en bloeddruk. *Tijdschr Soc Gezondheidszorg*, 62:758–765.

Knol AB, Staatsen BAM (2005). *Trends in the environmental burden of disease in the Netherlands 1980–2020, RIVM report 500029001/2005*. Bilthoven, Netherlands.

Knutsson A (1989a). Relationships between serum triglycerides and g-Glutamyltransferase among shift and day workers. *Journal of Internal Medicine*, 226:337–339.

Knutsson A (1989b). Shift work and coronary heart disease. *Scandinavian Journal of Social Medicine*, 544:1–36.

Knutsson A, Åkerstedt T, Jonsson B (1988). Prevalence of risk factors for coronary artery disease among day and shift workers. *Scandinavian Journal of Work, Environment and Health*, 14:317–321.

Knutsson A et al. (1986). Increased risk of ischemic heart disease in shift workers. *Lancet*, 12 (2):89–92.

010239

Kogi K, Ohta T (1975). Incidence of near accidental drowsing in locomotive driving during a period of rotation. *Journal of Human Ergology*, 4:65–76.

Kokokusha D (1973). *Report of investigation of living environment around Osaka International Airport.* Aircraft Nuisance Prevention Association.

Koller M (1983). Health risks related to shift work. *International Archives of Occupational and Environmental Health*, 53:59–75.

Koller M, Kundi M, Cervinka R (1978). Field studies of shift work at an Austrian oil refinery. Health and psychosocial wellbeing of workers who drop out of shift work. *Ergonomics*, 21:835–847.

Kopferschmitt J et al. (1992). Sleep disorders and use of psychotropic drugs in 6-year-old children. *Revue d'Epidemiologie et de Santé Publique*, 40:467–471.

Kraenz S et al. (2004) Prevalence and stress factors of sleep disorders in children starting school. *Praxis der Kinderpsychologie und Kinderpsychiatrie*, 53, 1:3–18.

Kranendonk F, Gerretsen E, Luxemburg LCJ (van) (1993). *Akoestische kwaliteit van woningen versus de beleving van burengeluid.* TNO-BOUW, TUE-Bouwkunde; 93-CBO-R3185.

Kripke DF, Cook B, Lewis OF (1971). Sleep of night workers: EEG recordings. *Psychophysiology*, 7:377–384.

Kripke DF et al. (1979). Short and long sleep and sleeping pills. Is increased mortality associated? *Archives of General Psychiatry*, 36:103–116.

Kripke DF et al. (2002). Mortality associated with sleep duration and insomnia. *Archives of General Psychiatry*, 99:131–136.

Kristensen TS (1989). Cardiovascular diseases and the work environment. *Scandinavian Journal of Work, Environment, and Health*, 15:165–179.

Kryter KD (1990). Aircraft noise and social factors in psychiatric hospital admission rates: a re-examination of some data, *Psychological Medicine*, 20 (2):395–411.

Kryter K, Poza F (1980). Effects of noise on some autonomic system activities. *Journal of the Acoustical Society of America*, 2036–2044.

Kupperman M et al. (1995). Sleep problems and their correlates in a working population. *Journal of General Internal Medicine*, 10:25–32.

Kushnir T, Melamed S (1992). The Gulf War and its impact on burnout and well-being of working civilians. *Psychological Medicine*, 22:987–995.

Lambert J, Plouhinec M (1985). Day and night annoyance: a comparison. *Proceedings of Internoise 85*, pp. 941–944.

Lambert J, Vallet M (1994). *Study related to the preparation of a communication on a future EC noise policy.* LEN Report No.9420, Brussels.

Langdon FJ, Buller IB (1977). Road traffic noise and disturbance to sleep. *Journal of Sound and Vibration*, 50:13–28.

Langdon FJ, Buller IB, Scholes WE (1981). Noise from neighbours and the sound insulation of party walls in houses. *Journal of Sound and Vibration*, 79:205–288.

Lauber JK, Kayten PJ (1988). Sleepiness, circadian dysrhythmia, and fatigue in transportation system accidents. *Sleep*, 11:503–512.

Lavie P, Pillar G, Malhotra A (2002). *Sleep disorders: diagnosis, management and treatment. A handbook for clinicians.* Martin Dunitz, London.

Ledoux S, Choquet M, Manfredi R (1994). Self-reported use of drugs for sleep or distress among French adolescents. *Journal of Adolescent Health,* 15:495–502.

Leger D et al. (2000). Prevalence of insomnia in a survey of 12 778 adults in France. *Journal of Sleep Research*, 9:35–42.

010240

Leger D et al. (2002). Medical and socio-professional impact of insomnia, *Sleep*, 25 (6):625–629.

Leidelmeijer K, Marsman G (1997). *Geluid van buren. Horen, hinder en sociale normen.* RIGO, Amsterdam, 97/65.

Leineweber C et al. (2003). Poor sleep increases the prospective risk for recurrent events in middle-aged women with coronary disease. The Stockholm Female Coronary Risk Study. *Journal of Psychosomatic Research*, 54:121–127.

Lennernas M, Åkerstedt T, Hambraeus L (1994). Nocturnal eating and serum cholesterol of three-shift workers. *Scandinavian Journal of Work, Environment and Health*, 20:401–406.

Lercher P (1992). *Auswirkungen des Straßenverkehrs auf Lebensqualität und Gesundheit.* Transitverkehrs-Studie, Teil I, Amt der Tiroler Landesregierung, Landesbaudirektion, Landessanitätsabteilung, Innsbruck.

Lercher, P (1996). Environmental noise and health: an integrated research perspective. *Environment International*, 22:117–128.

Lercher P, Kofler WW (1993). Adaptive behaviour to road traffic noise, blood pressure and cholesterol. In: Vallet M, ed. *Noise and Man '93. Proceedings of the 6th International Congress on Noise as a Public Health Problem, Nice 1993*. Arcueil Cedex, Institut National de Recherche sur les Transport et leur Sécurité.

Lercher P, Kofler WW (1996). Behavioral and health responses associated with road traffic noise exposure along alpine through-traffic routes. *The Science of the Total Environment*, 189/190:85–89.

Lercher P et al. (2002). Ambient neighbourhood noise and children's mental health. *Occupational and Environmental Medicine*, 59 (6):380–386.

Lindström B (1992). Quality of life: a model for evaluating health for all. Conceptual considerations and policy implications. *Sozial- und Präventivmedizin*, 37:301–306.

Lipworth L et al. (2001). Comparison of surrogate with self-respondents regarding medical history and prior medication use. *International Journal of Epidemiology*, 30:303–308.

Loeb M (1986). *Noise and human efficiency.* John Wiley & Sons, Chichester.

Lukas JS (1972a). Awakening effects of simulated sonic booms and aircraft on men and women. *Journal of Sound and Vibration*, 20:457–466.

Lukas JS (1972b). Effects of aircraft noise on human sleep. *American Industrial Hygiene Association Journal*, 33:298–303.

Lundberg U (1999). Coping with stress: neuroendocrine reactions and implications for health. *Noise & Health*, 4:67–74.

Lundberg O, Manderbacka K (1996). Assessing reliability of a measure of self-rated health. *Scandinavian Journal of Social Medicine*, 24:218–224.

Macey PM et al. (2002). Brain morphology associated with obstructive sleep apnea. *American Journal of Respiratory and Critical Care Medicine*, 15 (166):1382–1387.

Malmström M, Sundquist J, Johansson S-E (1999). Neighborhood environment and self-reported health status: a multilevel analysis. *American Journal of Public Health*, 89:1181–1186.

Manninen O, Aro S (1979). Urinary catecholamines, blood pressure, serum cholesterol and blood glucose response to industrial noise exposure. *Arhiv za Higijenu Rada i Toksikologiju*, 30:713–718.

Marcus CL (2001). Sleep-disordered breathing in children. *American Journal of Respiratory and Critical Care Medicine*, 164:16–30.

Marcus CL, Greene MG, Carroll JL (1998). Blood pressure in children with obstructive sleep apnea. *American Journal of Respiratory and Critical Care Medicine*, 157:1098–1103.

Marcus CL et al. (1994). Determinants of growth in children with the obstructive sleep apnea syndrome. *Journal of Pediatrics*, 125:556–562.

010241

Marth E et al. (1988). Fluglärm: Veränderung biochemischer Parameter. *Zentralblatt für Bakteriologie, Mikrobiologie und Hygiene*, 185:498–508.

Maschke C (2003). Epidemiological research on stress caused by traffic noise and its effects on high blood pressure and psychic disturbances. In: Jong RD et al., eds. *ICBEN 2003. Proceedings of the 8th International Congress on Noise as a Public Health Problem, Rotterdam.* ISBN 90-807990-1-7. Foundation ICBEN, Schiedam.

Maschke C, Rupp T, Hecht K (2000). The influence of stressors on biochemical reactions – a review of present scientific findings with noise. *International Journal of Hygiene and Environmental Health*, 203:45–53.

Maschke C, Wolf U, Leitmann T (2003). *Epidemiological examinations of the influence of noise stress on the immune system and the emergence of arteriosclerosis.* Report 298 62 515 (in German, executive summary in English), Wabolu-hefte 01/03, Umweltbundesamt, Berlin.

Mathers CD et al (2003). *Global burden of disease in 2002 – data sources, methods and results.* WHO Discussion Paper No 54. (http://www3.who.int/whosis/discussion_papers/discussion_papers.cfm?path=evidence,discussion_papers&language=english, accessed 11 November 2006).

Mathur R, Douglas NJ (1995). Frequency of EEG arousals from nocturnal sleep in normal subjects. *Sleep*, 18:330–333.

Matschat K, Müller EA (1984). *Vergleich nationaler und internationaler Fluglärmbewertungsverfahren. Aufstellung von Näherungsbeziehungen zwischen den Bewertungsmaßen.* Umweltforschungsplan des Bundsministers des Innern, Forschungsbericht Nr. 81-105 01 307, Umweltbundesamt, Berlin.

Matsui T et al. (2001). Association between blood pressure and aircraft noise exposure around Kadena airfield in Okinawa. In: Boone R, ed. *Internoise 2001. Proceedings of the 2001 International Congress and Exhibition on Noise Control Engineering, The Hague, Vol. 3.* Nederlands Akoestisch Genootschap, Maastricht.

Matsui T et al. (2004). The Okinawa study: effects of chronic aircraft noise on blood pressure and some other physiological indices. *Journal of Sound and Vibration*, 277:469–470.

Matsumoto K (1978). Sleep patterns in hospital nurses due to shift work: an EEG study. *Waking and Sleeping*, 2:169–173.

Meecham WC, Smith HG (1977). Effects of jet aircraft noise on mental hospital admissions. *British Journal of Audiology*, 11 (3):81–85.

Meier-Ewert HK et al. (2004). Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. *Journal of the American College of Cardiology*, 43:678-683.

Melamed S, Kushnir T, Shirom A (1992). Burnout and risk factors for cardiovascular diseases. *Behavioral Medicine*, 18:53–60.

Melamed S et al. (1999). Chronic burnout, somatic arousal and elevated salivary cortisol levels. *Journal of Psychosomatic Research*, 46:591–598.

Mellinger GD, Balter MB, Uhlenhuth EH (1985). Insomnia and its treatment. Prevalence and correlates. *Archives of General Psychiatry*, 42 (3):225–232.

Miedema HME (2003). *Self-reported sleep disturbance caused by aircraft noise.* TNO Inro, Delft, 2004–15.

Miedema HME (2004). Self-reported sleep disturbance caused by aircraft noise. TNO Inro, Delft, 2005–15.

Miedema HME, Vos H (1998). Exposure-response relationships for transportation noise. *Journal of the Acoustical Society of America*, 104:3432–3445.

Miedema HME, Passchier-Vermeer W, Vos H (2003). *Elements for a position paper on night-time transportation noise and sleep disturbance.* TNO Inro, Delft, 2002-59.

Ministerie Verkeer en Waterstaat (2005). *Evaluatie Schipholbeleid Schiphol beleefd door omwonenden*, The Hague, ISBN 90 369 18 537.

010242

Mitler MM et al. (1988). Catastrophes, sleep and public policy: consensus report. *Sleep*, 11:100–109.

Mitler MM et al. (1997). The sleep of long-haul truck drivers. *The New England Journal of Medicine*, 337:755–761.

Miyakita et al. (1998). Is residential sound-proofing an effective measure in reducing the effects of aircraft noise? *Internoise 1998*, Noise Control Foundation.

Moline ML et al. (2003). Sleep in women across the life cycle from adulthood through menopause. *Sleep Medicine Reviews*, 7:155–177.

Morabia A (1991). On the origin of Hill's causal criteria. *Epidemiology*, 2:367–369.

Morin CM, Rodrigue S, Ivers H (2003). Role of stress, arousal, and coping skills in primary insomnia. *American Psychosomatic Society*, 65:259–267.

Morrell S, Taylor R, Lyle D (1997). A review of health effects of aircraft noise. *Australian and New Zealand Journal of Public Health*, 21:221–236.

Morrell S et al. (1998). Cross-sectional relationship between blood pressure of school children and aircraft noise. In: Carter N, Job RFS, eds. *Noise effects '98. Proceedings of the 7th International Congress on Noise as a Public Health Problem, Sydney 1998.*

Morrell S et al. (2000). Cross-sectional and longitudinal results of a follow-up examination of child blood pressure and aircraft noise. The inner Sydney child blood pressure study. *Internoise 2000, 29th International Congress and Exhibition on Noise Control Engineering, Nice.*

Mosso A (1886). *Periodische Athmung und Luxusathmung*. Arch. Physiol. Suppl, 37:116.

Müller-Wenk R (2002). *Attribution to road traffic of the impact of noise on health*. Environmental Series No 339, BUWAL, Berne (http://www.buwalshop.ch, accessed 11 November 2006).

Murray CHR et al. (1996). *The global burden of disease*. World Health Organization, Geneva.

Muzet A, Ehrhart J (1978). Amplitude des modifications cardiovasculaires provoquées par le bruit au cours du sommeil. *Coeur et médecine interne*, 17:49–56.

Muzet A, Ehrhart J (1980). Habituation of heart rate and finger pulse resonses to noise during sleep. In: Tobias JV, ed. *Noise as a public health problem*. ASHA report No.10, Rockville, Maryland, pp. 401–404.

Muzet A et al. (1973). Relationship between subjective and physiological assessments of noise-disturbed sleep. *Proceedings of the International Congress on Noise as a Public Health Problem, US Environmental Agency, Dubrovnik, Yugoslavia*, pp. 575–586.

Nagaya T et al. (2002). Markers of insulin resistance in day and shift workers aged 20–59 years. *International Archives of Occupational and Environmental Health*, 75:562–568.

Naitoh P, Muzet A, Lienhard JP (1975). Effects of noise and elevated temperature on sleepcycle. *Sleep Research*, 4:174.

NCSDR (2003). National Center on Sleep Disorders Research (http://www.nhlbi.nih.gov/health/prof/sleep/res_plan/index.html, accessed 3 March 2007).

Neus H, Boikat U (2000). Evaluation of traffic noise-related cardiovascular risk. *Noise & Health*, 2 (7):65–77.

Neus H et al. (1980). On the reaction of finger pulse amplitude to noise. *International Archives of Occupational and Environmental Health*, 47:9–19.

Neus H et al. (1983). Traffic noise and hypertension. The Bonn traffic noise study. In: Rossi G (ed.). *Proceedings of the 4th International Congress on Noise as a Public Health Problem, Turin 1983*. Edizioni Tecniche a cura del Centro Ricerche e Studi Amplifon, Milan.

Niemann H, Maschke C (2004). *Noise effects and morbidity, WHO LARES final report*. Berlin, Interdisciplinary Research Network "Noise and Health" (http://www.euro.who.int/document/NOH/WHO_Lares.pdf, accessed 18 November 2004).

010243

Nieminen P et al. (2002). Growth and biochemical markers of growth in children with snoring and obstructive sleep apnea. *Pediatrics*, 109:e55.

Nijland HA et al. (2003). Costs and benefits of noise abatement measures. *Transport Policy*, 10:131–140.

Nilsson P et al. (2001). Sleep disturbances in association with elevated pulse rate for the prediction of mortality – consequences of mental strain? *Journal of Internal Medicine*, 250:521–529.

Nilsson P et al. (2002). Incidence of diabetes in middle-aged men is related to resting heart rate and difficulties to fall asleep (abstract). *7th International Congress of Behavioural Medicine.*

North FM et al. (1996). Psychosocial work environment and sickness absence among British civil servants: the Whitehall II study. *American Journal of Public Health*, 86:332–340.

NTSB (1995). *Factors that affect fatigue in heavy truck accidents*. National Transportation Safety Board, NTSB/SS-95/0 L.

Nurminen T (1989). Shift work, fetal development and course of pregnancy. *Scandinavian Journal of Work, Environment, and Health*, 15:395–403.

O'Brien LM, Gozal D (2002). Behavioral and neurocognitive implications of snoring and obstructive sleep apnea in children: facts and theory. *Pediatric Respiratory Reviews*, 3:3–9.

Ohayon MM, Roth T (2003). Place of chronic insomnia in the course of depressive and anxiety disorders. *Journal of Psychiatric Research*, 37 (1):9–15.

Öhrström E (1989). Sleep disturbance, psychosocial and medical symptoms – a pilot survey among persons exposed to high levels of road traffic noise. *Journal of Sound and Vibration*, 133:117–128.

Öhrström E (1993). *Long-term effects in terms of psychosocial wellbeing, annoyance and sleep disturbance in areas exposed to high levels of road traffic noise.* Health Council of the Netherlands, The Hague, A93/02E.

Öhrström E, Bjorkman M, Rylander R (1988). Noise annoyance with regard to neurophysiological sensitivity, subjective noise sensitivity and personality variables. *Psychological Medicine*, 18 (3):605–613.

Olkin I (1995). Meta-analysis: reconciling the results of independent studies. *Statistics in Medicine*, 14:457–472.

Ollerhead JB (1994). *Report of a field study of aircraft noise and sleep disturbance. A study commissioned by the Department of Transport from the Civil Aviation Authority*, HMSO, London.

Ollerhead et al. (1992). *Report on a field study of aircraft noise and sleep disturbance.* Department of Transport, London.

Ortscheid J, Wende H (2000). *Fluglärmwirkungen.* Forschungsbericht (in German), Umweltbundesamt, Berlin.

Ounsted MK, Hendrick AM (1977). The first-born child: patterns of development. *Developmental Medicine and Child Neurology*, 19:446–453.

Owens JA, Rosen CL and Mindell JA (2003). Medication use in the treatment of pediatric insomnia: results of a survey of community-based pediatricians. *Pediatrics*, 111:628–635.

Owens-Stively J et al. (1997). Neuro-psychological and behavioral correlates of obstructive sleep apnea in children. *Sleep Research*, 26 (suppl. 1):452.

Parish JM, Shepard JW (1990). Cardiovascular effects of sleep disorders. *Chest*, 97:1220–1226.

Partinen M, Eskelinen L, Tuomi K (1984). Complaints of insomnia in different occupations. *Scandinavian Journal of Work, Environment and Health*, 10:467–469.

Passchier-Vermeer W (1993). *Noise and health*. Health Council of the Netherlands, The Hague.

Passchier-Vermeer W (2000). *Noise and health of children.* TNO report PG/VGZ/2000.042, Organization for Applied Scientific Research (TNO), Leiden, Netherlands.

010244

Passchier-Vermeer W (2001). Effects of noise. In: Bistrup ML, ed. *Health effects of noise on children and perception of the risk of noise*. National Institute of Public Health, Denmark, pp. 47–68.

Passchier-Vermeer W (2003a). *Night-time noise events and awakening*. TNO Inro report 2003-32, TNO Institute for Traffic and Transport, Delft.

Passchier-Vermeer W (2003b). Relationship between environmental noise and health. *Journal of Aviation Environmental Research*, 7 (suppl.):35–44.

Passchier-Vermeer W and Passchier WF (2000). Noise exposure and public health. *Environmental Health Perspectives*, 108 (suppl. 1):123–131.

Passchier-Vermeer W et al. (2002). *Sleep disturbance and aircraft noise exposure*. TNO-PG, Leiden, Report No. 2002.027.

Passchier-Vermeer W et al. (2004). *Motility and road and rail traffic noise*. TNO-Inro, Delft, TNO report No. 2004.

Patel SR et al. (2004). A prospective study of sleep duration and mortality risk in women. *Sleep*, 27:440–444.

Patois E, Valatz JL, Alperovitch A (1993). Prevalence of sleep and wakefulness disorders in high school students at the Academy of Lyon. *Revue d'Epidemiologie et de Santé Publique*, 41:383–388.

Patton GC et al. (2000). Adolescent depressive disorder: a population-based study of ICD-10 symptoms. *Australian and New Zealand Journal of Psychiatry*, 34 (5):741–747.

Pearsons K, Bennett R, Fidell S (1973). *Effects of cessation of late-night landings on behavioral awakening*. BNN Systems and Technologies Corporation, Canoga Park, Report No. 2439.

Peigneux P et al. (2001). *Sleeping brain, learning brain. The role of sleep for memory systems*. NeuroReport 2001:12:A111–A124.

Peiyoong L, Hiscock H, Wake M (2003). Outcomes of infant sleep problems: a longitudinal study of sleep, behavior, and maternal well-being. *Pediatrics*, 111:203–207.

Porter ND, Flindell IH, Berry BF (1998). *Health effect-based noise assessment methods: a review and feasibility study*. National Physical Laboratory, Teddington.

Poustka F, Eckermann P, Schmeck K (1992). Effect of aircraft noise and psychosocial stressors on mental disturbances of children and adolescents: an epidemiological study in Westphalia. In: Remschmidt H, Schmidt MH, eds. *Developmental psychopathology*, Hogrefe and Huber Publishers, Gottingen.

Prokop O, Prokop L (1955). Ermiidung und Einschlafen am Steuer. *Zentralblatt fur Verkehrsmedizin, Verkehrspsychologie und angrenzende Gebiete*, 1:19–30.

Pulles T, Biesiot W, Stewart R (1988). Adverse effects of environmental noise on health: an interdisciplinary approach. *Environmental International*, 16:437–445.

Rai RM et al. (1981). Biochemical effects of chronic exposure to noise in man. *International Archives of Occupational and Environmental Health*, 48:331–337.

Raw GJ, Oseland NA (1991) Subjective response to noise through party floors in conversion flats. *Applied Acoustics*, 32:215–231.

Rechtschaffen A, Bergmann BM (1965). Sleep deprivation in the rat by the disk-over-water method. *Behavioural Brain Research*, 69:55–63.

Rechtschaffen A, Kales A, eds. (1968). *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects*. Brain Information Service/Brain Research Institute, UCLA, Los Angeles.

Rechtschaffen A, Hauri P, Zeitlin M (1966). Auditory awakening thresholds in REM and NREM sleep stages. *Perceptual and Motor Skills*, 22:927–942.

Reyner LA (1995). *Sleep disturbance and daytime sleepiness in normal subjects*. Loughborough University of Technology, Loughborough.

010245

Reyner et al. (1995). Patterns of spontaneous and evoked body movements during sleep. *Sleep*, 18 (3):209–211.

RFV (2003). *Lan-tidssjukskrivna - egenskaper vid 2003 ars RFV-LS-undersokning.*

Rhodes SK et al. (1995). Neurocognitive deficits in morbidly obese children with obstructive sleep apnea. *Journal of Pediatrics*, 127:741–744.

Ribet C, Derriennic F (1999). Age, working conditions, and sleep disorders: a longitudinal analysis in the French cohort. *Enquête Santé, Travail et Vieillissement (ESTEV)*, 22:491–504.

Richards W, Ferdman RM. (2000). Prolonged morbidity due to delays in the diagnosis and treatment of obstructive sleep apnea in children. *Clinical Pediatrics*, 39:103–108.

Riemann D, Berger M, Voderholzer U (2001). Sleep and depression – results from psychobiological studies: an overview, *Biological Psychology*, 57 (1–3):67–103.

Roberts E, Roberts CR, Chen IG. (2002). Impact of insomnia on future functioning of adolescents. *Journal of Psychosomatic Research*, 53 (1):561– 569.

Rockhill B (2005). Commentary. The message is rarely simple: the j-curve and beyond. *International Journal of Epidemiology*, 34:44–45.

Romon M, Nuttens MC, Fievet C (1992). Increased triglyceride levels in shift workers. *American Journal of Medicine*, 93 (3):259–262.

Rose G (2005). Incubation period of coronary heart disease. *International Journal of Epidemiology,* 34:242–244.

Rose GA, Blackburn H (1968). *Cardiovascular survey methods*. 1st edition, World Health Organization, Geneva.

Rosenlund M et al. (2001).Increased prevalence of hypertension in a population exposed to aircraft noise. *Journal of Occupational and Environmental Medicine*, 58:769–773.

Roth T, Ancoli-Israel S (1999). Daytime consequences and correlates of insomnia in the United States: results of the 1991 National Sleep Foundation Survey, II. *Sleep*, 22 (suppl. 2):S354–S358.

Sadeh A, Gruber R, Raviv A (2000). Sleep patterns and sleep disruptions in school-age children. *Developmental Psychology*, 36:291–301.

Sadeh A, Gruber R, Raviv A (2002). Sleep, neurobehavioral functioning, and behavior problems in school-age children. *Child Development*, 73:405–417.

Sadeh A, Gruber R, Raviv A (2003). The effects of sleep restriction and extension on school-age children: what a difference an hour makes. *Child Development*, 74:444–455.

Sadeh A et al. (1995). Sleep and psychological characteristics of children on a psychiatric inpatient unit. *Journal of the American Academy of Child and Adolescent Psychiatry*, 34 (6):813–819.

Sateia M et al. (2000). Evaluation of chronic insomnia. *Sleep*, 23 (2):243–263.

Scharnberg T et al. (1982). *Beeinträchtigung des Nachtschlafs durch Lärm. Eine interdisziplinäre Feldstudie der Technischen Universität Berlin und der Physikalisch-Technischen Bundesanstalt Braunschweig,* Umweltbundesamt, Berlin, Forschungsbericht Nr. 82-1050-1207.

Schernhammer ES et al. (2003). Night-shift work and risk of colorectal cancer in the nurses' health study. *Journal of the National Cancer Institute*, 95:825–828.

Schlesselman JJ (1987). "Proof" of cause and effect in epidemiologic studies: criteria for judgment. *Preventive Medicine*, 16:195–210.

Schreckenberg D et al. (1999). An interdisciplinary study on railway and road traffic noise: annoyance differences. 137th ASA Meeting and 25th DAGA Conference. *Journal of the Acoustical Society of America*, 105 (2):1219.

Schulte W, Otten H (1993). Ergebnisse einer Tiefflugläarmstudie in der Bundesrepublik Deutschland: Extraaurale Langzeitwirkungen [Results of a low-altitude flight noise study in Germany: long-term extra-aural effects]. In: Ising H, Kruppa B, eds. *Lärm und Krankheit – Noise and disease.*

010246

Proceedings of the International Symposium, Berlin 1991. Gustav Fischer Verlag, Stuttgart.

Schulze B et al. (1983). Verkehrslärm und kardiovaskuläres Risiko – eine epidemiologische Studie. *Das Deutsche Gesundheitswesen*, 38:596–600.

Schwela DH (2000). The World Health Organization guidelines for environmental health. *Noise/News International*, March, 9–22.

Segawa K et al. (1987). Peptic ulcer is prevalent among shift workers. *Digestive Diseases and Sciences*, 32:449–453.

Selye H (1956). *The stress of life*. McGraw-Hill, New York.

Sforza E, Jouny C, Ibanez V. (2000). Cardiac activation during arousal in humans: further evidence for hierarchy in the arousal response. *Clinical Neurophysiology*, 111:1611–1619.

Smith A (2003). The concept of noise sensitivity: implications for noise control, *Noise & Health*, 5 (18):57–59.

Smith AP, Broadbent DE (1981). Noise and levels of processing, *Acta Psychologica*, 47:129.

Smith AP, Broadbent DE (1992). *Non-auditory effects of noise at work: a review of the literature*. HSE Contract Research Report No 30. HMSO, London.

Smith L, Folkard S, Poole CJM (1994). Increased injuries on night shift. *Lancet*, 344:1137–39.

Smith MJ, Colligan MJ, Tasto DL (1982). Health and safety consequnces of shift work in the food-processing industry. *Ergonomics*, 25:133–144.

Spiegel K, Leproult R, van Cauter E (1999). Impact of sleep debt on metabolic and endocrine function. *Lancet*, 354:1435–1439.

Spreng M (2000). Central nervous system activation by noise. *Noise & Health*, 2 (7):49–57.

Spreng M (2004). Noise induced nocturnal cortisol secretion and tolerable overhead flights. *Noise & Health*, 6 (22):35–47.

Stansfeld SA (1992*). Noise, noise sensitivity and psychiatric disorder: epidemiological and psychophysiological studies*. Psychological Medicine Monograph Supplement 22, Cambridge University Press, Cambridge.

Stansfeld S, Haines M, Brown B (2000). Noise and health in the urban environment. *Reviews on Environmental Health*, 15 (1–2):43–82.

Stansfeld SA et al. (1985). Sensitivity to noise in a community sample: I. Measurement of psychiatric disorder and personality, *Psychological Medicine*, 15 (2):243–254.

Stansfeld SA et al. (1993). Road traffic noise, noise sensitivity and psychological disorder. *Psychological Medicine*, 23 (4):977–985.

Stansfeld S et al. (1996). Road traffic noise and psychiatric disorder: prospective findings from the Caerphilly Study. *British Medical Journal*, 313 (7052):266–267.

Stansfeld SA et al. (2002). Longitudinal effects of noise, noise sensitivity and psychosocial factors on men's psychological distress. *Epidemiology*, 13 (4):S90.

Stansfeld SA et al. (2005). Aircraft and road traffic noise and children's cognition and health: exposure–effect relationships. *Lancet*, 365:1942–1949.

Stein MA et al.(2001). Sleep and behavior problems in school-aged children. *Pediatrics*, 107:766 (e).

Stouthard M et al. (1997). *Disability weights for diseases in the Netherlands*, Department of Public Health, Netherlands.

Stradling JR et al. (1990). Effect of adenotonsillectomy on nocturnal hypoxaemia, sleep disturbance and symptoms in snoring children. *Lancet*, 335:249–253.

Suda M, Saito M (1979). Coordinative regulation of feeding behavior and metabolism by a circadian timing system. In: Suda M, Hayaishi O, Nakagawa H, eds. *Biological rhythms and their central mechanism*. Elsevier/North-Hollan Biomedical, New York, pp. 263–271.

010247

Suter AH (1992). Noise sources and effects – a new look. *Sound and Vibration*, 25th anniversary issue:18–38.

Tamakoshi A, Ohno Y (2004). Self-reported sleep duration as a predictor of all-cause mortality: Results from the JACC study, Japan. *Sleep*, 27:51–54.

Tarnopolsky A, Morton-Williams J, Barker SM (1980). *Aircraft noise and prevalence of psychiatric disorders*. Research report. Social and Community Planning Research.

Tarnopolsky A, Watkins G, Hand DJ (1980). Aircraft noise and mental health: I. Prevalence of individual symptoms. *Psychological Medicine*, 10 (4):683–698.

Tarnopolsky A et al. (1978). The effect of aircraft noise on the mental health of a community sample: a pilot study. *Psychological Medicine*, 8 (2):219–233.

Tate RB et al. (1995). Tracking of blood pressure over a 40-year period in the University of Manitoba follow-up study, 1948–1988. *American Journal of Epidemiology*, 142:946–954.

Taylor PJ, Pocock SJ (1972). Mortality of shift and day workers 1956–68. *British Journal of Industrial Medicine*, 29:201–207.

Tenkanen L et al. (1997). Shift work occupation and coronary heart disease over 6 years of follow-up in the Helsinki Heart Study. *Scandinavian Journal of Work, Environment and Health*, 23:257–265.

Tepas DI et al. (1981). Polysomnographic correlates of shift worker performance in the laboratory. In: Reinberg A, Vieux N, Andlauer P, eds. *Night and shift work: biological and social aspects.* Pergamon Press, Oxford, pp. 179–186.

Terzano MG et al. (1990). Modifications of sleep structure induced by increasing levels of acoustic perturbation in normal subjects. *Electroencephalography and clinical neurophysiology*, 76:29–38.

Terzano MG et al. (1993). Assessment of noise-induced sleep fragility in two age ranges by means of polysomnographic microstructure. *Journal of Sound and Vibration*, 162:345–359.

Theorell T et al. (1998). Decision latitude, job strain, and myocardial infarction: A study of working men in Stockholm. *American Journal of Public Health*, 88:382–388.

Thiessen GJ (1988). Effect of traffic noise on the cyclical nature of sleep. *Journal of the Acoustical Society of America*, 84:1741–1743.

Thiis-Evensen E (1958). Shift work and health. *Industrial Medicine and Surgery*, 27:493–497.

Thomas DA et al. (1996). The effect of sleep deprivation on sleep states, breathing events, peripheral chemosensiveness and arousal propensity in healthy 3-month old infants. *European Respiratory Journal*, 9:932–938.

Thomas JR, Jones DM (1982). Individual differences in noise annoyance and the uncomfortable loudness level, *Journal of Sound and Vibration*, 82:289–304.

Thompson SJ (1997). Cardiocascular and fetal effects of noise. In: IEH, ed. *Workshop on non-auditory health effects of noise. Report No. R10.* Institute for Environment and Health, Leicester.

Thunström M (2002). Severe sleep problems in infancy associated with subsequent development of attention-deficit hyperactivity disorder at 5.5 years of age. *Acta Paediatrica*, 31:584–592.

Tilley AJ, Wilkinson RT, Drud M (1981). Night and day shifts compared in terms of the quality and quantity of sleep recorded in the home and performance measured at work: a pilot study. In: Reinberg A, Vieux N, Andlauer P, eds. *Night and shift work. Biological and social aspects.* Pergamon Press, Oxford, pp. 187–196.

Tilley AJ et al. (1982). The sleep and performance of shift workers. *Human Factors*, 24:624–641.

Torsvall L, Åkerstedt T (1987). Sleepiness on the job: continuously measured EEG changes in train drivers. *Electroencephalography and Clinical Neurophysiology*, 66:502 –511.

Torsvall L, Åkerstedt T (1988). Disturbed sleep while being on call. An EEG study of apprehension in ships' engineers. *Sleep*, 11:35–38.

010248

Torsvall L et al. (1989). Sleep on the night shift: 24-hour EEG monitoring of spontaneous sleep/wake behavior. *Psychophysiology*, 26:352–358.

Trott GE et al. (1995). Self-assessment of prescribing behavior of psychotropic drugs for children and adolescents. Results of a survey of established physicians. *Psychiatrische Praxis*, 22 (6):235–239.

Tüchsen F (1993). Working hours and ischaemic heart disease in Danish men. A 4-year cohort study of hospitalization. *International Journal of Epidemiology*, 22:215–221.

Ullrich S (1998). Lärmbelastung durch den Straßenverkehr. *Zeitschrift für Lärmbekämpfung*, 45:22–26.

Urponen H et al. (1988). Self-evaluations of factors promoting and disturbing sleep: an epidemiological survey in Finland. *Social Science and Medicine*, 26:443–450.

Utley WA (1985). Descriptors for ambient noise. In: Bundesanstalt für Arbeitsschutz, ed. *Internoise 85. Proceedings of the International Conference on Noise Control Engineering in Munich 1985*. Wirtschaftsverlag NW, Verlag für neue Wissenschaft GmbH, Bremerhaven.

Valent F, Brusaferro S, Barbone F (2001). A case-crossover study of sleep and childhood injury. *Pediatrics*, 107: 23.

Vallet M et al. (1983). Heart rate reactivity to aircraft noise after a long term exposure. In: Rossi G (ed.). *Noise as a public health problem.* Centro Ricerche E Studi Amplifon, Milan, pp. 965–971.

Verdun Di Cantogno L et al. (1976). Urban traffic noise cardiocirculatory activity and coronary risk factors. *Acta Otolaryngologica*, (suppl.) 339:55–63.

Vernet M (1979). Effect of train noise on sleep for people living in houses bordering the railway line. *Journal of Sound and Vibration*, 66 (3):483–492.

Vgontzas AN et al. (2000). Sleep apnea and daytime sleepiness and fatigue: relation to visceral obesity, insulin resistance, and hypercytokinemia. *Journal of Clinical Endocrinology and Metabolism*, 85:1151-1158

Vgontzas AN et al. (2001). Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implication. *Journal of Clinical Endocrinology and Metabolism*, 86:3787–3794.

Vollrath M, Wicki W, Angst J (1989). The Zurich study. VIII. Insomnia: association with depression, anxiety, somatic syndromes, and course of insomnia. *European Archives of Psychiatry and Neurological Sciences*, 239 (2):113–124.

Vos J (2003). On the relevance of shooting-noise-induced sleep disturbance to noise zoning, *ICBEN-proceedings 2003*, Schiedam.

Wagner U, Gais S, Born J (2001). Emotional memory formation is enhanced across sleep intervals with high amounts of rapid eye movement sleep. *Learning and Memory*, 8:112–119.

Walter SD (1998). Attributable risk in practice. *American Journal of Epidemiology*, 148:411–413.

Ware JE, Sherbourne DC (1992). The MOS 36-item short-form health survey (SF-36) – Conceptual framework and item selection. *Medical Care*, 30:473–483.

Wassmer E et al. (1999). The acceptability of sleep-deprived electroencephalograms. *Seizure*, 8:434–435.

Watkins G, Tarnopolsky A, Jenkins LM (1981). Aircraft noise and mental health: II. Use of medicines and health care services, *Psychological Medicine*, 11 (1):155–168.

Waye KP et al. (2003). Effects of night-time low frequency noise on the cortisol response to awakening and subjective sleep quality. *Life Sciences*, 72:863–875.

Weber A, Jaekel-Reinhard A (2000). Burnout syndrome: a disease of modern societies? *Occupational Medicine*, 50, 512–517.

010249

Weed DL (2000). Interpreting epidemiological evidence: how meta-analysis and causal inference methods are related. *International Journal of Epidemiology*, 29:387–390.

Weed DL, Hursting SD (1998). Biologic plausibility in causal inference: current method and practice. *American Journal of Epidemiology*, 147:415–425.

Weinstein ND (1978). Individual differences in reactions to noise: a longitudinal study in a college dormitory. *Journal of Applied Psychology*, 63 (4):458–466.

Weinstein ND (1980). Individual differences in critical tendencies and noise annoyance. *Journal of Sound and Vibration*, 68:241–248.

Weissbluth M et al. (1983). Signs of airway obstruction during sleep and behavioral, developmental and academic problems. *Journal of Developmental and Behavioral Pediatrics*, 4:119–121.

WHO *Guidelines for community noise*. World Health Organization, Geneva. 1999 (http://www.who.int/docstore/peh/noise/guidelines2.html, accessed March 2007)

WHO-ISH Guidelines Subcommittee (1999). World Health Organization, International Society of Hypertension guidelines for the management of hypertension. *Journal of Hypertension*, 17:151–183.

WHO Regional Office for Europe (2000). *Noise and health*. WHO Regional Office for Europe, Copenhagen.

Wilkinson R et al. (1989). Alertness of night nurses: two shift systems compared, *Ergonomics*, 32:281–292.

Williams RL, Karacan L, Hursch CJ (1974). *EEG of human sleep*. John Wiley & Sons, New York.

Willner P, Neiva J (1986). Brief exposure to uncontrollable but not to controllable noise biases the retrieval of information from memory. *British Journal of Clinical Psychology*, 25 (Pt 2):93–100.

Wing JK, Cooper JE, Sartorius N (1974). *The measurement and classification of psychiatric symptoms*. Cambridge University Press, London.

Winkleby MA, Ragland DR, Syme SL (1988). Self-reported stressors and hypertension: evidence of an inverse association. *American Journal of Epidemiology*, 127:124–133.

Wölke G et al. (1990). Verkehrslärm und kardiovaskuläres Risiko. *Forum Städte-Hygiene*, 41:306–308.

Wubben F, Busink J (2004). *Night restrictions at Amsterdam Schiphol. A comparison with other European mainports*. To70 Aviation & Environment, The Hague, Netherlands.

Yong L-C et al. (1993). Longitudinal study of blood pressure. Changes and determinants from adolescence to middle age. The Dormont High School follow-up study, 1957/1963 to 1989/1990. *American Journal of Epidemiology*, 138:973–983.

Yoshida T et al. (1997). Effects of road traffic noise on inhabitants of Tokyo. *Journal of Sound and Vibration*, 205:517–522.

Zamir G et al. (1998). Sleep fragmentation in children with juvenile rheumatoid arthritis. *Journal of Rheumatology*, 25:1191–1197.

Zepelin H, McDonald CS, Zammit GK (1984). Effects of age on auditory awakening thresholds. *Journal of Gerontology*, 39 (3):294–300.

Zorick FJ, Walsh JK (2000). Evaluation and management of insomnia: an overview. In: Kryger MH, Roth T, Dement WC, eds. *Principles and practice of sleep medicine*. W.B.Saunders, Philadelphia, pp. 615–623.

Zuckerman B, Stevenson J, Bailey V (1987). Sleep problems in early childhood: continuities, predictive factors, and behavioural correlates. *Pediatrics* 80:664–671.

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APPENDIX 1. GLOSSARY OF TERMS AND ACRONYMS

Term/acronym	Definition
Actimetry	The measurement of accelerations associated with the movement of an actimeter
ADHD	Attention-deficit hyperactivity disorder
Behavioural awakening	Awakening that is registered by the subject by means of a conscious action
BP	Blood pressure
CAP	Cyclic alternating patterns
EBD	Environmental burden of disease
END	Environmental Noise Directive (2002/49/EC)
EEG	Electroencephalogram, recording of electric activity in the brain
ECG	Electrocardiogram, recording of electric activity of the heart
EEG awakening	Transition from a state of sleep to a state of consciousness, as determined by a sleep EEG
Heart rate acceleration	A temporary rise in heart rate relative to the average heart rate assessed shortly before a noise event
HPA axis	Hypothalamus-pituitary-adrenal axis
ICSD	International Classification of Sleep Disorders
IHD	Ischaemic heart disease
Insomnia	Sleeping disorder consistent with an internationally accepted definition (see ICSD), which takes account of difficulty falling or staying asleep, the daytime implications and the duration of the problems
$L_{Aeq,T}$	Exposure to noise for the duration of a given time interval T (a 24-hour period, a night, a day, an evening) is expressed as an equivalent sound pressure level (measured in dB(A)) over the interval in question
L_{Amax}	Maximum outdoor sound pressure level associated with an individual noise event
L_{night}	Refers to the EU definition in Directive 2002/49/EC: equivalent outdoor sound pressure level associated with a particular type of noise source during night-time (at least 8 hours), calculated over a period of a year
Motility onset	The presence of movement in a short time interval, following an interval without movement
Mg	Magnesium
Motility	The presence of movement in a short time interval, as recorded on an actigram
OR	Odds ratio: the ratio of the odds of an event occurring in another group, or to a sample-based estimate of that ratio

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Term/acronym	Definition
OSAS	Obstructive sleep apnoea syndrome
OTC	Over-the-counter (medicines sold without prescription)
Polysomnography	The measurement during a subject's time in bed of his or her brain activity by means of EEG, EOG and EMG. The technique involves the use of electrodes to record electrical potentials in the brain
REM	Rapid eye movement (sleep phase)
RR	Relative risk: a ratio of the probability of the event occurring in the exposed group vs. the control (non-exposed) group
SEL	Sound exposure level: equivalent outdoor sound pressure level associated with an individual noise event, with the equivalent level standardized at one second
Sleep disturbance	Disturbance of sleep by night-time noise, as perceived by a subject and described in a questionnaire response or journal entry
Sleep EEG	Graph created using data from EEG scanning during a subject's time in bed, showing the various stages of sleep as a function of time
Sleep fragmentation	Within a sleep period, the frequency and duration of intervals of wakefulness recorded on a sleep EEG or intervals of motility recorded on an actigram
Sleep latency	The length of time taken to fall asleep, i.e. the interval between the point at which a person begins trying to go to sleep or allowing him/herself to go to sleep and sleep inception time
Sleep stage change	Change from a deeper stage of sleep to a less deep stage, as determined by a sleep EEG
SMR	Standardized mortality ratios
SROBD	Sleep related obstructive breathing disorders
SWS	Slow-wave sleep (sleep phase)
UARS	Upper airway resistance syndrome

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APPENDIX 2. RELATIONS BETWEEN L_{NIGHT} AND INSTANTANEOUS EFFECTS

STATEMENT 1

Let f be a function of SEL that gives the expected number of instantaneous effects caused by a single event. With a given L_{night} and a given number of events N, the expected number of times that an effect occurs in the night, n, is maximal if all events have equal SEL, provided that f°10lg is increasing but negatively accelerated.

STATEMENT 2

If

$$n_{max} = 10^{(L_{night}-SEL+70.2)/10} \cdot f(SEL),$$

has a maximum over SEL and f is the quadratic function $f(SEL) = a\ SEL^2 + b\ SEL + c$, then the maximum occurs irrespective of L_{night} at

$$SEL_0 = 4.34 - A \pm ((A - 4.34)^2 - (c/a) + 8.68A)^{1/2},$$

where A = b/(2a). (Only with + at the place of ± the value will come in the realistic range of SEL)

STATEMENT 3A

If the shape of the time pattern of the sound level has a block form, then $SEL = L_{Amax} + 10lg(T)$, where L_{Amax} is the maximum sound level (integrated over 1-s) and T is the duration of the noise event in seconds.

STATEMENT 3B

If the sound level increases with rate a (in dB(A)/s) and after time point $t = 0$ decreases with rate –a, then $SEL \approx L_{Amax} - 10lg(a) + 9.4$.

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APPENDIX 3. ANIMAL STUDIES ON STRESS AND MORTALITY

INTRODUCTION

Is noise a health risk or does it just annoy? This basic question needs to be carefully answered when establishing night noise guidelines. No one will deny that in the case of high noise levels there is a risk of inner ear damage, but what about the moderate levels of environmental noise? To approach this rather difficult question, all available methods must be combined.

1. In animal experiments it is possible to assess the complete causal chain from noise exposure via physiological reactions and biological risk factors to morbidity or even mortality. However, a quantitative application of the results to humans is not possible. Instead, the method is useful in studying the pathomechanisms qualitatively.
2. Experiments on humans are, for ethical reasons, restricted to the study of reversible physiological reactions. But as long as there is no proof that reactions to chronically repeated noise exposures are increasing the risk of specific diseases, the results of such physiological studies are not considered conclusive.

3. Epidemiological studies have the advantage of investigating health effects which are particularly caused by chronic noise exposure although there is no possibility to control all influencing factors. Additionally, epidemiological studies have to be based upon biologically evident hypotheses.

A hypothetical model of noise-induced health effects is shown in Fig. 4.3 in Chapter 4, section 4.5.2 of this report. This model is based on the results of noise experiments with animals and humans. With animal experiments, the whole causal chain from noise exposure to health outcome can be traced as a direct pathway starting with a chronic high level noise exposure which, via endocrine stress reactions, leads, for example, to microcirculatory defects and to manifest hypertension.

Physiological experiments on humans have shown that noise of a moderate level acts via an indirect pathway and has health outcomes similar to those caused by high noise exposures on the direct pathway. The indirect pathway starts with noise-induced disturbances of activities such as communication or sleep. Since we are dealing with night noise guidelines, noise-induced sleep disturbances and any resulting persistent health effects are of primary interest here.

In physiological studies with experimentally changed noise exposure, the increase of arousals and of hormone excretion was studied in sleeping people. If this model is correct then in the cause–effect chain the arousal ought to precede the endocrine reactions. This order was derived from the different reaction times of the effects. While arousals appear within 1 second after a noise stimulus, hormones like catecholamines take several minutes, and cortisol about 10 minutes to be increased. This observation, together with the fact that arousals are evoked by equal or lower noise levels than the corresponding endocrine reactions, confirms the correctness of the model and leads to an important conclusion: noise exposure which does not evoke arousals in sleeping people will not induce adverse health effects.

This conclusion is essential with regard to night noise guidelines. However, the answers to the basic question as to whether certain health risks are connected with

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environmental noise must be clarified by epidemiological studies based on noise experiments on both humans and animals.

TYPES OF ANIMAL STUDIES

Noise has often been used as a stressor in animal studies. Even Selye (1953), who introduced the psychophysiological stress concept, used noise stimuli in his animal studies. Most of the modern animal studies testing the pharmacological effects of drugs are carried out with and without various stressors. The typical noise exposure is to short and very intensive sounds. One such example is the study of Diao et al. (2003) who exposed guinea pigs to 4 kHz octave band noise at 115 dB for 5 hours. But these experiments are of little value regarding the noise exposure types in question.

The same is true for another type of animal study concerned with the prevention of noise-induced health effects in wild and domestic animals (for review of the former kind see Fletcher, 1983). One example for the latter kind is the study of Geverink et al. (1998) on stress responses of pigs to transport and lairage sounds.

Since the subject of the present paper is noise-induced health effects in humans, the review addresses only those studies in which animals are used as a model for humans.

The animal model for aural effects in humans has been established in great detail, so that even quantitative transference of results from animals to humans is possible. However, inner ear damage generally occurs at much higher noise levels than the environmental levels under discussion in this paper. Therefore interest focuses on animal models with respect to extra-aural noise effects.

LIMITING ASPECTS OF ANIMAL MODELS

Other than in studies on aural effects, the animal model does not allow quantitative comparisons in studies of extra-aural noise effects. It may, however, be used for the qualitative investigation of pathophysiological mechanisms following exposure to acute and very short sounds. But an animal model for long-term noise effects as caused by chronically repeated noise exposures needs careful planning. First it has to be ensured that stress reactions in both humans and animals when activated by noise exposure are qualitatively comparable. Secondly, the stress effects of chronic noise exposure have to be assessed in humans, and the animal models should be designed correspondingly. However, in the animal model, influences from cortical interconnections have to be excluded as a factor in these noise experiments. Naturally, one cannot expect to establish an animal model for indirect environmental noise effects which in humans may, for example, disturb activities such as verbal communication, which in turn may induce stress hormone increases.

STRESS HORMONES IN NOISE-EXPOSED ANIMALS

HABITUATION

In short-term experiments any kind of exposure to loud noise will cause acute increases of stress hormones. Long-term repeated noise exposure, however, will

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cause a certain habituation in the animal. Periodic repetitions of identical noise bursts lead to almost complete habituation. This was probably the main reason why Borg (1981) found no adverse health effects in rats exposed for their whole lives to periodic noise pulses. Therefore, random series of noise pulses are now applied in most long-term studies.

Selye (1974) had already stated that not all stages of a stress response are noxious, especially in the case of mild or brief exposures. Since environmental noise is a mild stressor, adverse health effects are only to be expected under the condition that repeated noise exposures induce long-term stress hormone changes. According to the Allostatic Load Model (McEwen, 1998), the normal response to an environmental stressor such as noise is the physiological activation of the endocrine system enabling the body to cope with the stressor and, after the stress situation is terminated, to shut off the allostatic response.

J.D. HENRY'S MODEL OF BEHAVIOURAL STRESS EFFECTS

On the basis of the available literature on stress effects in animals and humans, Henry (1992) developed a model with regard to different biological effects and health risks associated with different coping styles. He explains that the neuroendocrine response to various challenges and threats varies according to the type and degree of control a mammal can exert over it. This in turn is strongly determined by the animal's previous experience. In general, the sympathetic adrenomedullary system is preferentially activated when the animal displays an active response to escape from or deal with an environmental challenge. This is the fight/flight mode of stress response. The adrenocortical axis is preferentially activated as the subjects become immobile/passive when no control or threat of its loss is experienced. This is the conservation/withdrawal mode of response.

THE NOISE STRESS MODEL

On the basis of noise effect studies in animals and humans (for review see Ising and Braun, 2000), a noise stress model was developed. It describes a differentiation of prevailing "stress hormones" under noise exposure. Predominantly adrenaline – and to a lesser degree noradrenaline – are released from the adrenal medulla as the normal response to novel noise stimuli of moderate intensity. Following long-term noise exposures of moderate intensity habituation will alter the response mode and predominantly noradrenaline is released. As a response to extremely intensive noise, near the inner ear pain threshold, predominantly cortisol is released from the adrenal cortex induced by increased releases of adrenocorticotropic hormone (ACTH), especially in the case of unexpected noise.

The described differentiation will only be observed under special conditions. Unexpected exposure for three minutes to white noise at 75 dB leads, in dogs that are awake, to increased adrenal secretion of adrenaline and noradrenaline and – following a delayed increase in plasma ACTH – an increase in cortisol secretion (Engeland, Miller and Gann, 1990).

The cortisol response as described is valid for animals and humans in their active phases. During sleep, however, several studies in humans showed cortisol increases under exposure to traffic noise of moderate levels (Maschke, Arndt and Ising, 1995;

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Evans et al.2001; Ising and Ising, 2002; Ising et al., 2004). It was hypothesized that noise stimuli signalling a danger, for example the noise of an approaching lorry, will, during sleep, normally generate a defeat reaction, which includes the release of cortisol from the adrenal cortex. Appropriate studies with sleeping animals after conditioning them – for example with a specific noise stimulus followed by pain – should be carried out to test this hypothesis.

Rats were exposed for a period of 12 hours to low-altitude flight noise – reproduced electro-acoustically once per hour on average at stochastically fluctuating intervals (L_{Amax} 125 dB, 10 dB downtime: 1 s, L_{eq}: 89 dB) (Ising et al., 1991; Ising, 1993). Adrenaline and noradrenaline excretions tended to decrease, whereas plasma cortisol increased significantly. Although in rats corticosterone is secreted rather than cortisol, we will simplify this paper by using cortisol for rats all the same. In this experiment, as well as in all others of our group, normally four rats were kept in one stainless steel cage, which was set on a funnel to collect their urine.
These results show that exposure to noise levels approaching or exceeding the pain threshold of the inner ear leads to endocrine reactions qualitatively different from those induced by less intensive noise.

The different endocrine reactions to acute and chronic noise exposure were studied in rats by Gesi et al. (2002b). They were exposed either to a single (6-hour) session of loud (100 dB(A)) noise, or to the same noise stimulus repeatedly every day for 21 consecutive days. Exposure to noise for 6 hours on one day induced parallel increases in dopamine, noradrenaline and adrenaline concentrations in tissue samples of the adrenal medulla. After 21 days of noise exposure, noradrenaline concentration was significantly higher than in controls, and that of adrenaline decreased significantly. Cortisol was not assessed in this study.

In another subchronic noise experiment, rats were exposed to irregular white noise at 90 dB for 3 and 9 hours per day during 18 and 8 days respectively (van Raaij et al., 1997). In rats with 3 hours of exposure per day the blood concentrations of adrenaline, noradrenaline and cortisol did not differ from controls. Exposure for 9 hours per day, however, resulted in significantly increased concentrations of noradrenaline and cortisol. At the end of the experiment all animals were subjected to restraint stress and their endocrine reactions were assessed. The authors sum up their findings as follows: these results indicate that chronic noise exposure at mild intensities induces subtle but significant changes in hormonal regulation.

The results of another experiment with different levels of random white noise pulses during 45 minutes per hour, 12 hours per day for 8 days demonstrate that cortisol responses to subchronic mild noise exposure do not monotonously increase with the noise levels (Bijlsma et al., 2001). While in rats exposed to 95 dB pulses plasma cortisol concentrations were raised twofold against controls, the exposure to 105 dB pulses did not increase cortisol significantly.

The time dependency of cortisol increase in the blood of rats under exposure to white noise (100 dB, 6 hours per day for 21 days) was examined by Gesi et al. (2001). The authors found a progressive increase in cortisol which reached a plateau 9 days from the beginning of exposure.

In summing up the results of these studies we can reach the following conclusions.

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- Acute exposure to unexpected and novel noise of moderate intensities leads to activation of both the sympathetic adrenal-medullary system with increased secretion of adrenaline and noradrenaline, and the HPA axis with increased secretion of ACTH and of cortisol from the adrenal cortex.

- Under chronic exposure to unpredictable noise, adrenaline secretion is reduced to normal or subnormal values while noradrenaline and ACTH/cortisol concentrations remain increased.

- Extremely intensive unpredictable noise near the inner ear pain threshold triggers, in mammals that are awake, a defeat reaction with increases of ACTH/cortisol while the catecholamines adrenaline and noradrenaline remain normal or are slightly decreased.

- Chronic noise exposure at mild intensities will induce changes in hormonal regulation, if the individual threshold of allostasis is exceeded. A chronic allostatic load leads to subtle but significant changes in hormonal regulation, which are at present not fully understood.

EFFECTS OF PRENATAL NOISE EXPOSURE ON THE SENSITIVITY TO STRESS

Pregnant rats were subjected to noise and light stress, three times weekly on an unpredictable basis throughout gestation (Weinstock et al., 1998). Blood concentrations of adrenaline, noradrenaline and cortisol at rest and after footshock were assessed. At rest cortisol was significantly increased in offspring of stressed rats in comparison to controls while adrenaline and noradrenaline did not differ in either of the groups. After footshock, noradrenaline was significantly higher in offspring of stressed rats, showing that prenatal stress can induce long-term changes in the sensitivity of the sympathicoadrenal system to stress.

Pregnant monkeys were repeatedly exposed to unpredictable noise during days 90–145 after conception (Clarke et al., 1994). Blood concentration of ACTH and cortisol were measured in offspring of stressed and control monkeys at rest and under four progressively stressful conditions. Prenatally stressed offspring showed higher ACTH than controls in all four stressful conditions while cortisol did not change under stress. These results indicate that prenatal stress may have long-term effects on the HPA axis regulation.

EFFECTS OF NOISE EXPOSURE ON CORTISOL AND THE IMMUNE SYSTEM

The effect of acute noise stress on rats was studied by assessing blood concentrations of cortisol and total as well as differential leukocyte count (Archana and Namasivayam, 1999). A significant increase in cortisol and a significant decrease of total leukocyte counts were found.

Rats were exposed to "rock" music (80dB) for 24 hours (McCarthy, Quimet and Daun, 1992). In vitro stimulation of leukocyte subpopulations revealed several noise effects. Neutrophils and macrophages secreted significantly less superoxide anion and interleukin-1. Such effects may be detrimental to wound healing.

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Pregnant rats were from gestation day 15 to day 21 daily exposed to the noise of a fire alarm bell (L_{Amax} = 85–90 dB) delivered randomly for 1 hour (Sobrian et al., 1997). In developing offspring mitogen-specific alterations in lymphoproliferatic activity and reduced immunoglobulin G levels were found at postnatal day 21.
Aguas et al. (1999) exposed a special breed of mice to low frequency noise – a model of noise – for three months as described below (Castelo Branco et al., 2003). These mice spontaneously developed an autoimmune disease at 6 months of age. Chronic low frequency noise exposure accelerated the expression of the autoimmune disease and affected the immune system, which was associated with kidney lesions and increased mortality.

Embryotoxic effects
Geber (1973) exposed pregnant rats day and night for three weeks to constantly changing sound mixtures between 76 and 94 dB for 6 minutes per hour, day and night, and demonstrated embryotoxic effects, notably calcification defects in the embryos.

Pregnant rats on a moderately magnesium deficient diet were exposed to noise during their active phase from 20.00 to 08.00 for three weeks (stochastically applied white noise impulses L_{Amax}: 87 dB, L_{eq}: 77 dB, t: 1 s duration) (Günther et al. 1981).

As compared to controls on the same diet, there was no difference in bone mineralization. The only significant effect was an increased fetal resorption rate.

The noise was changing in Geber's experiment but the noise level was comparable to the noise impulses stochastically applied by Günther et al. (L_{Amax} = 87 dB). Since these impulses were more frequent, their stress effect was at least as strong as the noise exposure employed by Geber. Therefore the major factor that differentiated the two exposure types in causing a reduced mineralization of the rat skeletons (Geber, 1973) must have been the additional noise exposure during sleep.

Castelo Branco et al. (2003) studied Wistar rats born under low frequency noise exposure. The third octave level of the applied broadband noise was > 90 dB for frequencies between 50 and 500 Hz. The broadband level was 109 dB(lin). The exposure schedule was chosen as a model for occupational noise: 8 hours per day, 5 days per week, and weekends in silence. Third generation rats born in low frequency noise environments were observed showing teratogenic malformations including loss of segments.

Morphological alterations in the myocardium caused by acute noise
Gesi et al. (2002a) reviewed the literature and stated that in experimental animals undergoing noise exposure, subcellular myocardial changes have been reported, especially at mitochondrial level; in particular, after 6 hours of exposure only the atrium exhibited significant mitochondrial alterations, whereas after 12 hours as well as subchronic exposure both atrium and ventricle were damaged.

Exposure of rats to 100 dB(A) noise for 12 hours caused a significant increase of DNA damage accompanied by ultrastructural alterations and increased noradrenaline concentrations in the myocardium (Lenzi et al., 2003). In another paper this group described an increase in mitochondrial calcium (Ca) influx caused by the same noise exposure. They described Ca accumulation at myocardial subcellular level. Summing up their results they wrote that: moreover, the present results joined with previous evi-

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dence indicate that calcium accumulation is the final common pathway responsible for noise-induced myocardial morphological alterations (Gesi et al., 2000).

Connective tissue proliferation
Hauss, Schmitt and Müller (1971) described a proliferation of connective tissue in the myocardium of rats under acute exposure to noise.

On the basis of these results a noise exposure experiment was carried out of 5 weeks with day and night exposure to stochastically triggered bells (L_{Amax}: 108 dB, t (duration of one signal):1 s, L_{eq}: 91 dB) (Ising, Noack and Lunkenheiner, 1974). We confirmed the results of Hauss, Schmitt and Müller (1971) using an electron microscope to demonstrate fibrosis in the interstitial tissue of the myocardium. Additionally electron dense areas (visible as black spots) located within bundles of collagen in the myocardium were observed. According to Selye (1962), these dark areas were most probably caused by high concentrations of calcium (Ca) carbonate or calcium phosphate deposits. This suggestion is consistent with the results of Gesi et al. (2000).

After publication of these findings, a reservation was correctly voiced that, as the noise exposure had not left intervals for sleep, it was not certain whether the myocardial damage was provoked by the noise stress as such or by a noise-induced lack of sleep. For this reason, all subsequent experiments provided for noise-free intervals of 8 to 12 hours during the rats' inactive phases to enable them to sleep.

Rats were exposed for 28 weeks to a random series of white noise impulses from 16.00 to 08.00 daily with an 8 hour rest in their inactive phase (Ising et al., 1979). The third octave spectrum of the noise was flat between 5 and 25 kHz and had a third octave level of 88 dB (lin) (broadband L_{Amax} = 97 dB(lin). L_{eq} = 87 dB(lin)). The duration of noise impulses was 4 seconds and the noise to pause ratio 1:10 on average. There was a small but significant increase in hydroxyproline as indicator of collagen in the rats' left myocardium. Electron micrographs showed, similar to the earlier experiment, collagen bundles in the otherwise empty interstitial space but no indication of calcium deposits.

Respiratory effects
Castelo Branco et al. (2003) studied respiratory epithelia in Wistar rats born under low frequency noise exposure and further exposed for up to 5403 hours during more than 2.5 years. The third octave level of the applied broadband noise was > 90 dB for frequencies between 50 and 500 Hz. The broadband level was 109 dB(lin). The exposure schedule was chosen as a model for occupational noise: 8 hours per day, 5 days per week and weekends in silence. Rats were gestated and born under the described noise exposure with additional exposures from 145 to 5304 hours. Transmission electron micrographs of the tracheal epithelium of rats exposed for 2438 hours revealed a subepithelial layer of hyperplastic collagen bundles, several of them exhibiting a degenerative pattern. The results indicate an increased proliferation as well as degenerative processes of collagen.

Castelo Branco et al. (2003) observed sheared cilia in the respiratory epithelia of Wistar rats born under and further chronically exposed to low frequency noise. As interpretation of their findings they stated that both mechanical and biochemical events may be responsible for this pattern of trauma.

Electrolytes: Ca/Mg shift
Acute exposure of rats to the fast rising overflight noise of low flying fighter planes

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reaching levels of up to 125 dB(A)) (Ising et al., 1991; Ising, 1993) resulted not only in an increase of cortisol but also in a decrease of intracellular magnesium (Mg) and an increase of Mg excretion.
In guinea pigs, acute stress – due to 2 hours of noise exposure (95 dB white noise) or to overcrowding in the cage – caused significant increases of serum Mg and decreases of erythrocyte Mg (Ising et al., 1986).

For chronic noise experiments an additional stress factor had been sought which would act synergistically with unwanted noise, since in the above described noise experiment, half a year of exposure led to but relatively mild health effects (Ising et al., 1979). The justification for using two stressors derives from the fact that humans have to cope with a whole range of more or less synergistic stress factors and not with noise alone.

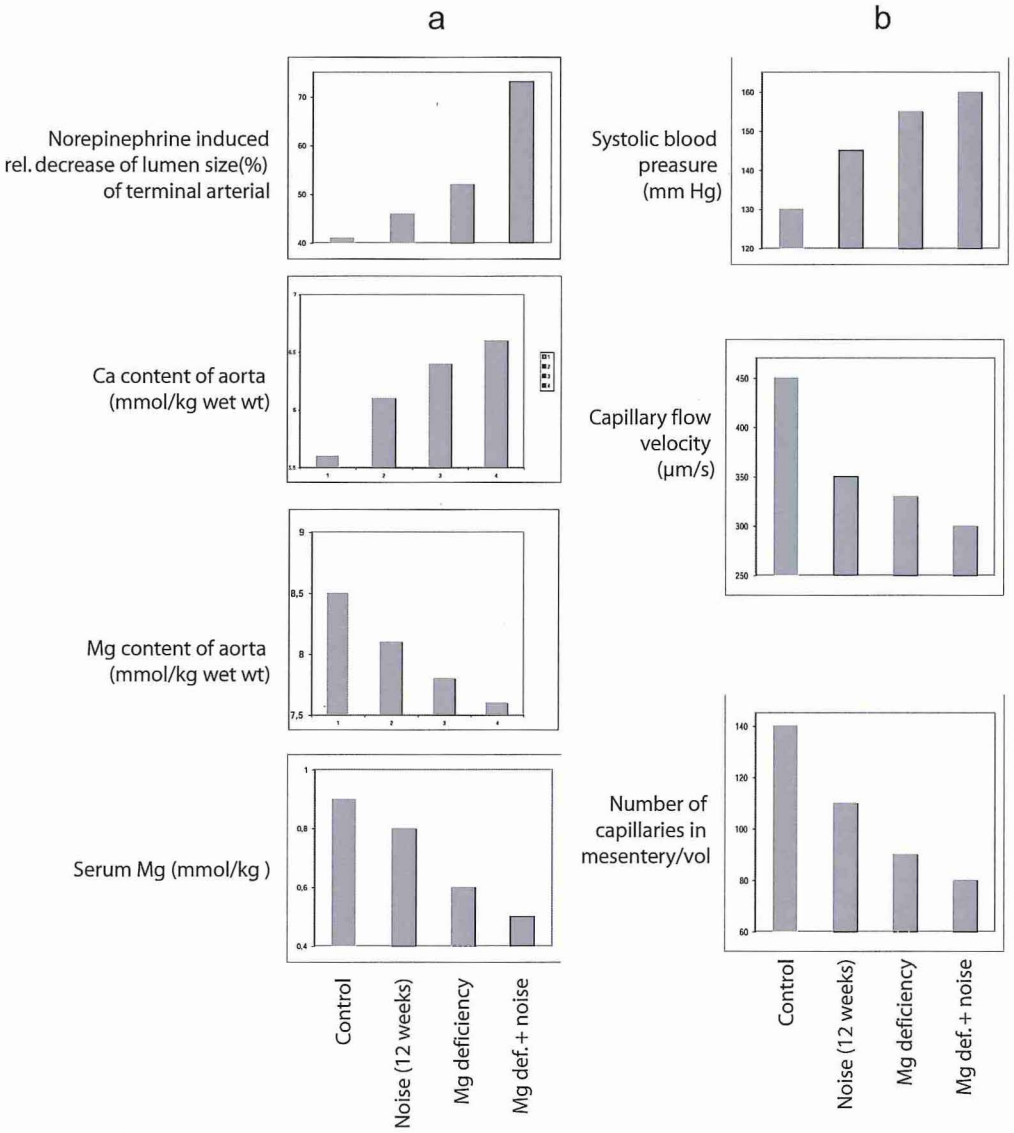
Organic damage as a result of chronic stress is likely to occur only under the condition that the overall exposure to stress exceeds a certain tolerance level during a relatively long period of time (Selye, 1974). For technical reasons, the two options available to supply a suitable additional stress factor were the cold or a magnesium (Mg) deficiency. Both factors, like habitual noise, cause an increased noradrenaline secretion. For practical reasons different degrees of a magnesium-deficient diet were selected as an additional stress factor. Noise exposure was provided by electroacoustically reproduced traffic noise of L_{Amax}: 86 dB, L_{eq}: 69 dB over 12 hours during the rats' active phase. For one group the noise level was slightly increased (L_{eq}: 75 dB). The experiment lasted 16 weeks (Günther, Ising and Merker, 1978). Magnesium deficiency combined with noise exposure led to dose-dependent increases in adrenaline and noradrenaline, which can be used to quantify the overall stress of the dietary treatment. As stress grew, the hydroxyproline (as an indicator of collagen) and calcium (Ca) content of the myocardium increased while the magnesium content decreased. Long-term stress therefore resulted in an intracellular Ca/Mg shift.

Altura et al. (1992) studied the relationship between microcirculation (measured several days after termination of noise exposure), hypertension and Ca/Mg shifts in vascular walls of noise-stressed rats on Mg deficient diets. Noise exposure during the first 8 weeks was set to an energy equivalent level of 85 dB(A) from 20.00 to 08.00. Noise impulses were randomly switched on at randomized peak levels of 80, 90 and 100 dB(A). During the final 4 weeks the equivalent noise level was elevated to 95 dB(A) and the daily exposure increased to 16 hours with an 8 hour rest during the animals' inactive phase. In aortic and port vein smooth muscle the Ca content increased with rising noise exposure, with decreasing Mg uptake, and with the combination of both together, while the Mg content decreased. Parallel to this the reactivity of terminal arterioles to noradrenaline was increased (Fig.1a).

Stress-induced Ca/Mg shifts in smooth muscle cells have the potential to increase the risk of hypertension and myocardial infarction (Ising, Havestadt and Neus, 1985). Stress increases the membrane permeability of catecholamine-sensitive cells, which in turn raises Ca influx into cells and liberates intracellular Mg. A depression of catecholamine-induced vasoconstriction by stress-dependent hypermagnesemia (excess serum Mg concentration) has been demonstrated experimentally. However, the benefit from this stress-depressing hypermagnesemia is obtained at the expense of increased renal Mg loss. In the long run, chronic stress combined with suboptimal Mg in diet will reduce the Mg release in acute stress situations, causing an increase of vasoconstriction and raising the risk of hypertension.

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Fig.1
Effects of 12 weeks noise exposure, Mg deficient diet and the combination of both in Wistar rats. (a) Ca/Mg shifts in vascular smooth muscle, Mg concentration in blood and reactivity of arterioles to noradrenaline. (b) Systolic BP, capillary blood flow velocity and numbers of capillaries/volume.

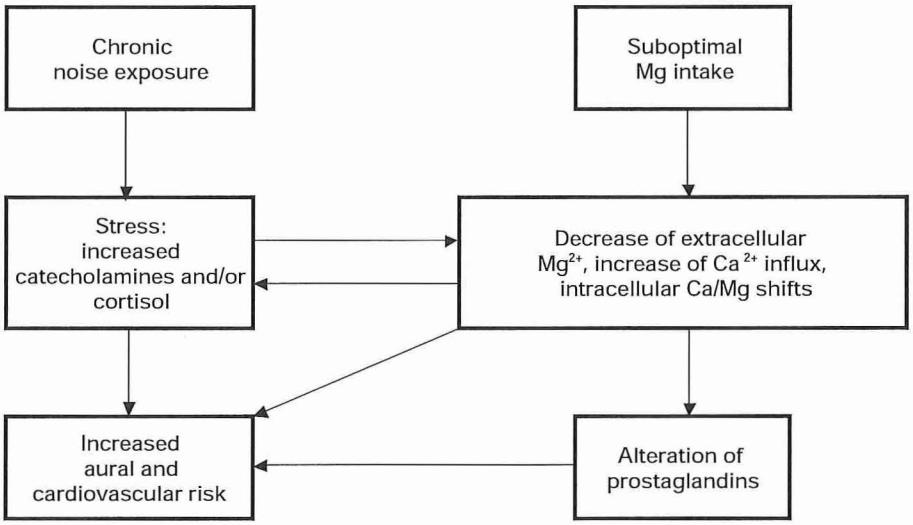


Source: Altura et al., 1992

Further analysis of the experimental results led to an interaction model between chronic stress and intracellular electrolyte shifts (Ising, 1981; Ising et al., 1986) (Fig.2). Chronic stress caused a loss of extracellular and intracellular Mg and an increase in intracellular Ca (Günther, Ising and Merker, 1978). A decrease of Mg was correlated with an increase in physiological noise sensitivity, that is, to more severe noradrenaline releases in animals and humans under noise exposure (Günther, Ising and Merker, 1978; Ising, Havestadt and Neus, 1985; Ising et al., 1986). There was a positive feedback mechanism between stress – caused by noise and other stressors – and intracellular Mg/Ca shifts, which may end in a circulus vitiosus and increase cardiovascular risks.

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Fig. 2
Interaction between stress and Ca/Mg shifts and its long term consequences



Hypertension

Rothlin, Cerletti and Emmenegger (1956) exposed rats for 1.5 years, day and night, to 90 dB "audiogenic stress" and observed a raising of systolic BP values from 120 mm Hg to about 150 mm Hg. He used a cross-breed of Albino rats and wild Norwegian rats since Albino rats did not develop hypertension under noise exposure. After termination of exposure the BP returned to normal.

Albino rats were exposed to noise during their whole lifespan (for review see Borg, 1981) to periodic noise impulses of 80 and 100 dB. This periodic exposure had no detrimental health effects, which can be understood on the basis of the work of Glass, Singer and Friedmann (1969). Unpredictable noise presentation was shown to cause lasting cortisol increases in rats in contrast to periodic exposure to 100 dB, which led to adaptation (De Boer, van der Gugten and Slangen, 1989). The unpredictability of a noise is a decisive precondition of long-term stress effects.

Exposure of primates to traffic noise for 10 hours per day during 9 months led to a significant BP increase, which persisted during 3 weeks after termination of exposure (Peterson et al., 1981). A replication of this experiment with a different species of primates failed to show an increase of their BP (Turkkan, Hienz and Harris, 1983).

In the above-mentioned experiment of Altura et al. (1992), exposure to unpredictable noise impulses led within 12 weeks to irreversible changes of microcirculation and an increase of systolic BP (Fig. 1b). The observed rarefication of capillaries in the mesentery can be interpreted as an indicator of accelerated ageing of the circulatory system.

Ageing and lifespan

The cortisol response and recovery after immobilization stress was compared in young and old rats. The results are demonstrated in Fig. 3 together with Sapolsky's Glucocorticoid Cascade Model (Sapolsky, Krey and McEwen, 1986). The stress response of young and old rats is more or less the same. However, while the young rats recover immediately after termination the old ones recover only in part.

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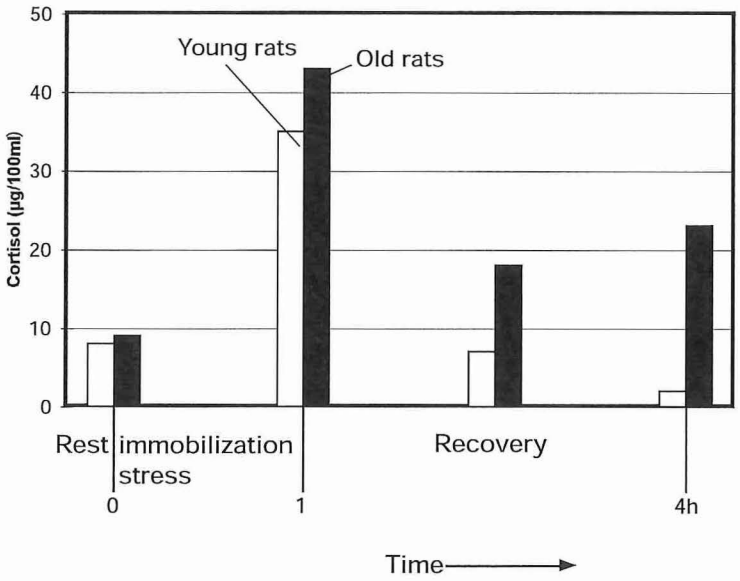
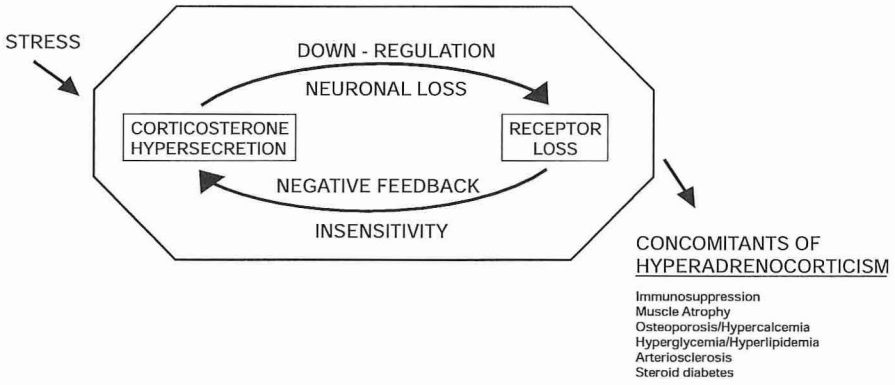


Fig. 3
Above: Recovery after stress (young and old rats). Below: Sapolsky´s Glucocorticoid Cascade Model



Therefore, acute stress leads, in old animals, to considerably prolonged cortisol increases. On the other hand, chronically repeated stress activates the HPA axis and can cause cortisol receptor losses even in younger animals, a process generally developing only in old age. Finally, chronic cortisol hypersecretion may occur along with follow-up health defects.

Aguas et al. (1999) exposed a special breed of mice to the above described model of occupational low frequency noise for three months. Chronic low frequency noise exposure accelerated the expression of the autoimmune disease and was associated with kidney lesions and increased mortality.

Chronic noise exposure of animals on a suboptimal Mg diet led to increases of connective tissue and calcium and decreases of Mg in the myocardium (Günther, Ising and Merker, 1978). These changes were correlated with noradrenaline changes. Since they are also correlated with normal ageing, the noise stress induced changes may be interpreted as accelerated ageing (Ising, Nawroth and Günther, 1981). Even the lifespan was reduced in rats on an Mg deficient diet, and was further dose-dependently reduced in combination with noise exposure (see Table 1).

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Table 1
Effects of noise exposure combined with dietary Mg-deficiency in rats

Treatment		Effect					
4 months	3 months	Urine		Myocardium			Death rate
Mg in diet	Noise	Noradrenaline	Adrenaline	Hydroxyproline	Ca	Mg	
	L_{eq}/L_{Amax}	(µg/g Cre)		(mg/g dry wt.)	(mg/g d.w.)	(mg/g d.w.)	
control	ambient	18 ± 4	12 ± 2	3.0 ± 0.1	3.0 ± 0.2	37.5 ± 0.8	0
suboptimal	ambient	23 ± 4	18 ± 2	3.0 ± 0.1	3.5 ± 0.5	38.0 ± 1.7	0
suboptimal	69/86 dB	37 ± 11	16 ± 2	3.0 ± 0.1	4.3 ± 0.2	37.9 ± 1.3	0
deficient	ambient	98 ± 17	20 ± 5	3.9 ± 0.1	6.2 ± 0.7	31.2 ± 1.4	38%
deficient	69/86 dB	129 ± 19	41 ± 10	4.6 ± 0.1	6.7 ± 0.6	29.8 ± 1.8	62%
deficient	75/86 dB	172 ± 26	60 ± 15	5.6 ± 0.9	8.0 ± 0.9	26.8 ± 0.8	75%

Adrenaline and noradrenaline excretion was measured during the fourth week of noise exposure; death rate is related to the 4-month period of Mg treatment; all other parameters were determined at the end of the experiment (mean values ± S.E.). Noise has the potential to cause stress reactions which are enhanced by suboptimal magnesium intake. Chronic noise-induced stress accelerates the ageing of the myocardium and thus increases the risk of myocardial infarction. The involved pathomechanisms include increases of catecholamines and/or cortisol under acute noise exposure and an interaction between endocrine reactions and intracellular Ca/Mg shifts.

WHAT CAN BE LEARNED FROM ANIMAL STUDIES ABOUT NOISE EFFECTS IN HUMANS?

The effects of low frequency noise – the "vibroacoustic disease" – were studied primarily in humans (for review see Castello Branco and Alves-Pereira, 2004).

In this context, the amygdalar contribution to conditioned fear learning, revealed for normal human subjects, has to be mentioned. Longer lasting activation of the HPA axis, especially abnormally increased or repeatedly elevated cortisol levels may lead to disturbances of the hormonal balance and even severe diseases in man (Spreng, 2000).

Catecholamines induce various detrimental effects on the heart (Ceremuzynski, 1981). Additionally, magnesium deficiency causes alterations of serum lipids (Weglicki et al., 1993), cytokines (Rayssiguier, 1990) and prostaglandines (Nigam, Averdunk and Günther, 1986), in particular an increase of thromboxan, which is released from thrombocytes (Neumann and Lang, 1995) and several other cell types and – in turn – thromboxan A2 can aggregate thrombocytes. All these alterations may increase the risk of myocardial infarction.
Beside these cardiovascular stress effects, chronically increased cortisol may induce

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neuronal degeneration and thus accelerate the ageing also of the brain (Sapolsky, Krey and McEwen, 1986), not only in rats but in humans as well (Sapolsky, 1994).

The importance of Ca/Mg shifts was confirmed by post mortem studies of hearts from victims of IHD (Elwood et al. 1980). The tissue samples were taken from areas of the myocardium not affected by the infarction and the results were stable after controlling for several confounders. The results are shown in Table 2. With normal ageing Ca increases and Mg decreases in the myocardium. This process is accelerated in myocardial infarction patients, which indicates an accelerated ageing of these peoples' heart muscle under the pathogenic influences that lead to myocardial infarction.

Table 2
Age dependency of myocardial Ca and Mg contents in ischaemic heart disease (IHD)

IHD deaths and non IHD deaths. (Mean Value ± SD, numbers in brackets)

	Group	Age < 45years	45–64 years	≥ 65 years
Ca [µg/g]	Non IHD	43 ± 15 (175)	50 ± 14 (281)	57 ± 22 (155)
	IHD	48 ± 10 (48)	53 ± 17 (389)	58 ± 21 (188)
Mg [µg/g]	Non IHD	183 ± 28	173 ± 34	178 ± 30
	IHD	170 ± 29	157 ± 30	156 ± 27
Ca/Mg	Non IHD	0.24	0.29	0.32
	IHD	0.28	0.34	0.37

Another factor which decreases Mg and increases Ca (Hofecker, Niedermüller and Skalicky, 1991) and collagen (Caspari, Gibson and Harris; 1976, Anversa et al., 1990; Gibbons, Beverly and Snyder, 1991) in the myocard is normal ageing (Ising, Nawroth and Günther, 1981). Therefore, it is plausible that the age-dependent decrease of Mg in hearts of IHD victims was about double of that in age-matched non-IHD deaths. This is therefore an indication that age- and stress-dependent electrolyte changes exist in humans and may be correlated with an increased risk of IHD.

Long-term experiments with Mg-deficient and noise-stressed rats showed that connective tissue and Ca in the myocardium increased with age while Mg decreased. Hence, stress caused by noise or cold is enhanced by suboptimal Mg intake and accelerates the ageing of the heart and decreases the lifespan (Heroux, Peter and Heggtveit, 1977; Ising, Nawroth and Günther, 1981; Günther, 1991).

Since coronary arteriosclerosis increases strongly with age (Lakatta, 1990) a biologically older heart is at a higher risk of IHD and of myocardial infarction. The interaction process described seems to be one of the pathomechanisms by which chronic noise stress increases the risk of myocardial infarction.

Several of the risk factors described in the literature to explain the correlation of work stress with myocardial infarction have been found to be increased under noise-induced stress as well, that is, increases of BP and total cholesterol.

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REFERENCES

Aguas AP et al. (1999). Acceleration of lupus erythematosus-like processes by low frequency noise in the hybrid NZB/W mouse model. *Aviation Space Environmental Medicine*, 70 (March suppl.):132–136.

Altura BM et al. (1992). Noise-induced hypertension and magnesium in rats: relationship to microcirculation and calcium. *Journal Applied Physics*:194–202.

Anversa P et al. (1990). Myocyte cell loss and myocyte cellular hyperplasia in the hypertrophied aging rat heart. *Circulation Research*, 67(4):871–885.

Archana R, Namasivayam A (1999). The effect of acute noise stress on neutrophil functions. *Indian Journal of Physiology and Pharmacology*, 43:491–495.

Bijlsma PB et al. (2001). Subchronic mild noise stress increases HRP permeability in rat small intestine in vitro. *Physiology & Behavior*, 73:43–49.

De Boer SF, van der Gugten J, Slangen JL (1989). Plasma catecholamine and corticosterone responses to predictable and unpredictable noise stress in rats. *Physiology & Behavior*, 45:789–795.

Borg E (1981). Physiological and pathogenic effects of sound. *Acta Oto-Laryngologica*, Supplement 381.

Caspari PG, Gibson K, Harris P (1976). Changes in myocardial collagen in normal development and after · blockade. Proc 6th Ann Meeting of the International Study Group for Research in Cardiac Metabolism. In: Harris P, Bing RJ, Fleckenstein A, eds. *Biochemistry and pharmacology of myocardial hypertrophy, hypoxia, and infarction*, 7:99–104.

Castelo Branco NAA, Alves-Pereira M (2004). Vibroacoustic disease. *Noise & Health*, 6, (23):3–20.

Castelo Branco NAA et al. (2003). SEM and TEM study of rat respiratory epithelia exposed to low frequency noise. In: Mendez-Vilas A, ed. *Science and Technology Education in Microscopy: An Overview*. Formatex, Badajoz, Spain, Vol.II, pp. 505–533.

Ceremuzynski L (1981). Hormonal and metabolic reactions evoked by acute myocardial infarction. *Circulation Research*, 48(6):767–776.

Clarke AS et al. (1994). Long-term effects of prenatal stress on HPA axis activity in juvenile rhesus monkeys. *Developmental Psychobiology*, 27(5):257–269.

Diao MF et al. (2003). Changes in antioxidant capacity of the guinea pig exposed to noise and the protective effect of alphalipoic acid against acoustic trauma. *Acta Physiologica Sinica*, 55(6):672–676.

Elwood PC et al. (1980). Magnesium and calcium in the myocardium: cause of death and area differences. *Lancet*, 2:720–722.

Engeland WC, Miller P, Gann DS (1990). Pituitary-adrenal and adrenomedullary responses to noise in awake dogs. *The American Journal of Physiology*, 258:672–677.

Evans G et al. (2001). Typical community noise exposure and stress in children. Journal of the Acoustical Society of America, 109(3):1023–1027:

Fletcher JL (1983). Effects of noise on wildlife: A review of relevant literature 1979–1983. In: Rossi G. *Noise as a public health problem*. 2:1153–1174, Centro Recherche E Studi Amplifon, Milan.

Geber WF (1973). Inhibition of fetal osteogenesis by maternal noise stress. *Federation Proceedings*, 32(11):2101–2104.

Gesi M et al. (2000). Ultrastructural localization of calcium deposits in rat myocardium after loud noise exposure. *Journal of Submicroscopic Cytology and Pathology*, 32(4):585–590.

Gesi M et al. (2001) Time-dependent changes in adrenal cortex ultrastructure and corticosterone levels after noise exposure in male rats. *European Journal of Morphology*, 39(3):129–135.

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Gesi M et al. (2002a). Morphological alterations induced by loud noise in the myocardium: the role of benzodiazepine receptors. *Microscopy Research and Technique*, 5(2):136–146.

Gesi M et al. (2002b). Brief and repeated noise exposure produces different morphological and biochemical effects in noradrenaline and adrenaline cells of adrenal medulla. *Journal of Anatomy*, 200:159–168.

Geverink NA et al. (1998). Responses of slaughter pigs to transport and lairage sounds. *Physiology & Behavior*, 63(4):667–673.

Gibbons GC III, Beverly PT, Snyder DL (1991). Myocardial fibrosis in ageing germ-free and conventional Lobund-Wistar rats: The protective effect of diet restriction. *Journal of Gerontology*, 46:B167–170.

Glass DC, Singer JE, Friedmann LN (1969). Psychic cost of adaptation to environmental stressor. *Journal of Personality and Social Psychology*, 12:200–210.

Günther T (1991). Magnesium deficiency, oxygen radicals and aging. *Magnesium Bulletin*, 13(3):78–81.

Günther T, Ising H, Merker HJ (1978). Elektrolyt- und Kollagengehalt im Rattenherzen bei chronischem Magnesium-Mangel und Streß. *Journal of Clinical Chemistry and Clinical Biochemistry*, 16:293–297.

Günther T et al. (1981). Embryotoxic effects of magnesium deficiency and stress on rats and mice. *Teratology*, 24:225–233.

Hauss W, Schmitt G, Müller U (1971). Über die Entstehung der Bindegewebsproliferation im Myocard nach Einwirkung verschiedener pathogener Reize. *Verhandlung der deutschen Gesellschaft für Medizin*, 77:1256–1263.

Henry JD (1992). Biological basis of the stress response. *Integrative Physiological and Behavioral Science*, 27:66–83.

Heroux O, Peter D, Heggtveit A (1977). Long-term effect of suboptimal dietary magnesium and calcium contents of organs, on cold tolerance and on lifespan, and its pathological consequences in rats. *Journal of Nutrition*, 107:1640–1652.

Hofecker G, Niedermueller H, Skalicky M (1991). Assessment of modifications of the rate of aging in the rat. *Archives of Gerontology and Geriatrics*, 12:273–289.

Ising H (1981). Interaction of noise-induced stress and Mg decrease. *Artery*, 9(3):205–211.

Ising H (1993). Extra-aural effects of chronic noise exposure in animals – a review. In: Ising H, Kruppa B, eds. *Noise and disease*, Gustav Fischer, Stuttgart, pp. 48–64.

Ising H, Braun C (2000). Acute and chronic endocrine effects of noise: review of the research conducted at the Institute for Water, Soil and Air Hygiene. *Noise & Health*, 7:7–24.

Ising H, Ising M (2002). Chronic cortisol increases in the first half of the night caused by road traffic noise. *Noise & Health*, 4(16):13–21.

Ising H, Havestadt C, Neus H (1985). *Health effects of electrolyte alterations in humans caused by noise stress*. InternoiseTagungsbericht, Schriftenreihe der Bundesanstalt fuer Arbeitsschutz, Dortmund; II, pp. 973–976.

Ising H, Nawroth T, Guenther T (1981). Accelerated aging of rats by Mg deficiency and noise stress. *Magnesium Bulletin* 3(2):142–145.

Ising H, Noack W, Lunkenheimer PP (1974). Histomorphologische Herzschäden nach Lärmeinwirkung. *Bundesgesundheitsblatt*, 16:234–238.

Ising H et al. (1979). Increase of collagen in the rat heart induced by noise. *Environment International*, 2:95–105.

Ising H et al. (1986). Stress-induced Ca/Mg shifts and vascular response in animals and men; comparison to electrolyte alterations in myocardial infarction patients. *Magnesium Bulletin*, 8:95–103.

Ising H et al. (1990). Medically relevant effects of noise from military low-altitude flights – results of an interdisciplinary pilot study. *Environment International*, 16:411–423.

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Ising H et al. (1991). *Gesundheitliche Wirkungen des Tiefffluglärms – Hauptstudie*. Umweltbundesamt, Berlin.

Ising H et al. (2004). Low frequency noise and stress: bronchitis and cortisol in children exposed chronically to traffic noise and exhaust fumes. *Noise & Health*, 6(23):21–28.

Lakatta EG (1990). The aging heart. In: Geokas MC (mod.): The aging process. *Annals of Internal Medicine*, 113:455–466.

Lenzi P et al. (2003). DNA damage associated with ultrastructural alterations in rat myocardium after loud noise exposure. *Environmental Health Perspectives*, 111(4):467–471.

Maschke C, Arndt D, Ising H (1995). Nächtlicher Fluglärm und Gesundheit: Ergebnisse von Labor- und Feldstudien. *Bundesgesundheitsblatt*, 38(4):130–137.

McCarthy DO, Quimet ME, Daun JM (1992). The effects of noise stress on leukocyte function in rats. *Research in Nursing & Health*, 15(2):131–137.

McEwen BS (1998). Protective and damaging effects of stress mediators – Allostasis and allostatic load. New England Journal of Medicine, 840:33–44.

Neumann S, Lang H (1995). Entzuendung. In: Greiling H, Gressner AM. *Lehrbuch der Klinischen Chemie und Pathochemie*. Schattauer Verlag, pp. 1257–1305.

Nigam S, Averdunk R, Günther T (1986). Alteration of prostanoid metabolism in rats with magnesium deficiency. *Prostaglandins Leukotrienes and Medicine*, 23:1–10.

Peterson EA et al. (1981). Noise raises blood pressure without impairing auditory sensitivity. *Science*, 111(4489):1451–1453.

Van Raaij MT et al. (1997). Hormonal status and the neuroendocrine response to a novel heterotypic stressor involving subchronic noise exposure. *Neuroendocrinology*, 65:200–209.

Rayssiguier Y (1990). Magnesium und lipid metabolism. In: Sigel H, Sigel A, eds. *Metal ions in biological systems*. Marcel Dekker INC, New York/Basel, Vol. 26, pp. 341–358.

Rothlin E, Cerletti A, Emmenegger H (1956). Experimental psycho-neurogenic hypertension and its treatment with hydrogenated ergot alkaloids (Hydergine). *Acta Medica Scandinavica*, 154 (suppl. 312):27–35.

Sapolsky RM (1994). *Why zebras don't get ulcers: A guide to stress, stress-related diseases, and coping*. WH Freeman, New York.

Sapolsky RM, Krey LC, McEwen BS (1986). The neuroendocrinology of stress and aging: the glucocorticoid cascade hypothesis. *Endocrine Reviews*, 7:284–306.

Selye H (1953). *The stress of life*. McGraw-Hill, New York.

Selye H (1962). *Calciphylaxis*. Chicago Press.

Selye H (1974). *Stress without distress*. Lippincott, Philadelphia, PA.

Sobrian SK et al. (1997) Gestational exposure to loud noise alters the development and postnatal responsiveness of humoral and cellular components of the immune system in offspring. *Environmental Research*, 73;227–241.

Spreng M (2000). Possible health effects of noise induced cortisol increase. *Noise & Health*, 7:59–63.

Turkkan HJS, Hienz RD, Harris AH (1983). The non-auditory effects of noise on the baboon. In: Rossi G. *Noise as a public health problem*. Vol.2, Centro Recherche e Studi Amplifon, Milan.

Weglicki WB et al. (1993). Modulation of cytokines and myocardial lesions by vitamine E and chloroquine in a Mg-deficient rat model. *American Journal of Physiology*, 264:C723–726.

Weinstock M et al. (1998). Effect of prenatal stress on plasma corticosterone and catecholamines in response to footshock in rats. *Physiology & Behavior*, 64(4):439–444.

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APPENDIX 4. NOISE AND SLEEP IN CHILDREN

FACTORS THAT MODIFY AUDITORY AROUSAL THRESHOLDS IN CHILDREN

By the time that most studies were conducted in infants, it had become progressively evident that arousal and awakening thresholds are influenced by a variety of factors. These significantly modify the response to ambient noise of sleeping infants. Some factors inhibit the arousal response, while others enhance the response.

PRENATAL AND PERINATAL FACTORS

Age of gestation
In 97 healthy infants, auditory awakening thresholds decreased significantly from the 44th to the 60th week after conception (Kahn, Picard and Blum, 1986). Awakening thresholds were defined as the infant opening their eyes and/or crying. Mean awakening thresholds dropped from 98.5+/-11 at the 44th week after conception to 83 dB(A) by the 60th week after conception.

Cigarette smoke
To evaluate the effects of cigarette smoke on polygraphic arousal thresholds, 26 newborns were studied with polygraphic recordings for one night: 13 were born to mothers who did not smoke, and 13 were born to mothers who smoked (over 9 cigarettes per day) (Franco et al., 1999). Another group of infants with a median postnatal age of 12 weeks were also studied: 21 born to non-smoking mothers and 21 born to smoking mothers. The auditory arousal thresholds of the infants of both age groups were measured with the use of auditory challenges of increasing intensity, administered during REM sleep. More intense auditory stimuli were needed to induce arousals in newborns (p=.002) and infants (p=.044) of smokers than in infants of non-smokers (mean value of 84+/-11 dB(A) for smokers and 81.6+/-20 for non-smokers). Behavioural awakening (infants opening their eyes and/or crying) occurred significantly less frequently in the newborns of smokers (p=.002) than of non-smokers.

It was concluded that newborn and infants born to smoking mothers had higher arousal thresholds to auditory challenges than those born to non-smoking mothers. From the present findings, it appeared that the impact of exposure to cigarette smoke occurred mainly before birth.

POSTNATAL FACTORS

The following postnatal factors modify arousal from sleep.

Sleep stage
In infants, auditory stimuli have generally indicated increased responses during active as compared with quiet sleep (Busby, Mercier and Pivik, 1994).

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Time of the night

In 31 infants, the arousal thresholds decreased across the night (mean value of 67+/-12.5 dB(A) in the first part of the night, for 51+/-3.5 in the third part of the night; p=.017) (Franco et al., 2001). Similar findings had been reported in adult subjects (Rechtschaffen, Hauri and Zeitlin, 1966).

Body position during sleep

To investigate whether prone or supine sleeping was associated with a different response threshold to environmental stimuli, 25 3-month-old healthy infants with a median age of 9 months were exposed to an auditory challenge while sleeping successively prone or supine (Franco et al., 1998). Three infants were excluded from the study because they awoke while their position was being changed. For the 22 infants included in the analysis, more intense auditory stimuli were needed to arouse the infants in the prone position (median of 70 db(A), range values 50 to more than 100 db(A)) than in the supine position (median of 60 db(A), range values 50–90 db(A)) (p=.011). Arousal thresholds were higher in the prone than in the supine position in 15 infants, unchanged in 4 infants and lower in the prone position in 3 infants (p=.007). It was concluded that infants show higher arousal thresholds to auditory challenges when sleeping in the prone position than when sleeping in the supine position. The findings could not readily be explained. The difference in arousal thresholds could be related to difference in chest wall mechanoreceptor responses, or differences in BP and/or central baroreceptors responses.

Ambient room temperature

Two groups of healthy infants with a median age of 11 weeks were recorded polygraphically during one night: 31 infants were studied at 24°C and 31 infants at 28°C. To determine their arousal thresholds, the infants were exposed to white noises of increasing intensities during REM and non-REM sleep (Franco et al., 2001). The arousal thresholds decreased across the night in the infants sleeping at 24°C (p= .017). The finding was not found for the infants sleeping at 28°C. When analysing the arousal responses according to time of the night, it was found that the auditory thresholds were significantly higher at 28°C (75+/-19 dB(A)) than at 24°C (51+/-3.5 dB(A)) between 03.00 and 06.00 (p=.003). These findings were only seen in REM sleep.

Sleeping with the head covered by bedclothes

To evaluate the influence of covering the face of sleeping infants with a bed sheet, 18 healthy infants with a median of 10.5 weeks (range 8–15 weeks) were recorded polygraphically for one night (Franco et al., 2002). They slept in their usual supine position. During sleep, a bed sheet was gently placed on their face for 60 minutes. With the face free or covered by the sheet, the infants were exposed to white noises of increasing intensities during REM and non-REM sleep. Compared to with their face free, during the periods when their faces were covered, the infants had increases in pericephalic ambient temperature (p<.001), increases in REM sleep (p=.035) and body movements (p=.011) and a decrease in non-REM sleep (p<.001). Respiratory frequency was increased in both REM (p=.001) and non-REM (p<.001) sleep. With their face covered, the infants had higher auditory arousal thresholds (mean of 76+/-23 dB(A)) than with their face free (mean of 58+/-14 dB(A)) (p=.006). The difference was seen in REM sleep only. A positive correlation was found between pericephalic temperature and arousal thresholds in REM sleep (r=.487; p=.003).

Short sleep deprivation

Following short sleep deprivation, a study reported that in infants there was no measurable change in arousal propensity by auditory stimuli (1 kHz pure tone, delivered in the midline of the cot, from 73 dB and increased in 3 dB steps to 100 dB)

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during quiet sleep (Thomas et al., 1996). Another study was undertaken to evaluate the influence of a brief period of sleep deprivation on sleep and arousal characteristics of healthy infants (Franco et al., submitted). Thirteen healthy infants with a median age of 8 weeks (range 7–18 weeks) were recorded polygraphically during a morning nap and an afternoon nap in a sleep laboratory. They were two hours sleep-deprived, either in the morning or in the afternoon before being allowed to fall asleep. Six infants were sleep-deprived before the morning nap and seven before the afternoon nap. During each nap, the infants were exposed to white noises of increasing intensities in REM sleep to determine their arousal thresholds. Following sleep deprivation, the infants tended to have less gross body movements during sleep ($p = .054$). They had a significant increase in obstructive sleep apnoeas ($p = .012$). The infants' auditory arousal thresholds were significantly higher following sleep-deprivation (mean of 76+/-13.5 dB(A)) than during normal sleep (mean of 56+/-8.4 dB(A)) ($p = .003$) and during REM sleep. It was concluded that short-term sleep deprivation in infants is associated with the development of obstructive sleep apnoeas and a significant increase in arousal thresholds.

Pacifiers and breastfeeding

Fifty-six healthy infants were studied polygraphically during one night: 36 infants used a pacifier regularly during sleep; 20 never used a pacifier (Franco et al., 2000). Thumb users or occasional pacifier users were not included in the study. The infants were recorded at a median age of 10 weeks (range 6–19 weeks). To evaluate their auditory arousal thresholds, the infants were exposed to white noise of increasing intensity during REM sleep. Polygraphic arousals occurred at significantly lower auditory stimuli in pacifier-users than in nonusers (mean of 60+/-11.6 with pacifiers, for 71+/-15.3 without pacifier; p=.010). Compared to non-users, pacifier-users were more frequently bottle-fed than breastfed (p=.036).

Among infants sleeping without a pacifier, breastfed infants had lower auditory thresholds than bottle-fed infants (mean of 67.7+-13.0 breastfed, for 77.7+-17.5 bottle-fed; p=.049). The question of how a pacifier contributes to protect the sleeping infant might be best explained by the observed loss of the pacifier early after sleep onset. This could contribute to disruption of the infant's sleep and favour arousals.

CONCLUSIONS

Various factors modify auditory arousal responses from sleep in healthy infants. Some inhibit arousals while others enhance the response. To evaluate the effect and dose-effect relationship on children therefore requires the careful determination of confounders that may bias studies and lead to conflicting results.

Additional confounders should be added to the list of factors that modify arousal thresholds. These include past experience with the stimulus (Rechtschaffen, Hauri and Zeitlin, 1966), or the presence of meaning in the noise as both of them are of critical importance in determining the persistence of physical reactions to the noise (McLean and Tarnopolsky, 1977). These are the reasons which lead most sleep/wake researchers to use white noises to stimulate the sleeping child.

Knowledge of these variables does little to clarify the physiological determinants of the awakening response, because knowledge of how such variables are related to possible physiological determinants is little better than that of the awakening response itself (Rechtschaffen, Hauri and Zeitlin, 1966).

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These findings however, underline the significant dose–response relationship between ambient noise and arousal or awakening from sleep in infants.

NOISE AND SLEEP FOR DIFFERENT STAGES OF DEVELOPMENT

THE FETUS

The human fetus spends most of its time in a state equivalent to sleep, similar to that recorded in newborn infants. The healthy fetus in utero was shown to react to external noises. This is the result of the development of the human cochlea and peripheral sensory end organs. These complete their normal development by 24 weeks of gestation. Sound is well transmitted into the uterine environment. Ultrasonographic observations of blink/startle responses to vibroacoustic stimulation are first elicited at 24–25 weeks of gestation, and are consistently present after 28 weeks, indicating maturation of the auditory pathways of the central nervous system (Committee on Environment Health of the American Academy of Pediatrics, 1997). The fetus reacts to 1–4 seconds of 100–130 dB of 1220–15000 Hz sound. The hearing threshold (the intensity at which one perceives sound) at 27–29 weeks of gestation is approximately 40 dB and decreases to a nearly adult level of 13.5 dB by 42 weeks of gestation, indicating continuing postnatal maturation of these pathways.

Teratogenic effects have been described in animals prenatally exposed to noise (Committee on Environment Health of the American Academy of Pediatrics, 1997). These were associated with higher levels of cortisol and corticotropin hormones in the exposed animals. No such effects could be demonstrated in humans, in whom studies on the relation between exposure to noises during gestation and shortened gestation or lower birth weights were inconclusive or conflicting. It is possible that in these studies, noise could be a marker of other risk factors (Committee on Environment Health of the American Academy of Pediatrics, 1997). In conclusion, most studies on the effects of noise on perinatal health have been criticized as being hampered by serious methodological limitations, both in terms of the measurement of exposure and outcome, and failure to control for other known determinants of the outcomes under investigation. The lack of properly controlled studies makes it difficult to draw conclusions about what effects ambient noise has on perinatal outcomes (Morrell, Taylor and Lyle, 1997).

NEWBORN INFANTS

A large number of investigations have been concerned with the responses of sleeping newborn infants to acoustic signals. Many of the studies arise from a large and general interest in child development as well as from a need for hearing tests for infants (Mills, 1975).

Infant incubators produce continuous noise levels of between 50 and 86 dB(lin) (American Academy of Pediatrics, 1974). Oxygen inlets produced an additional 2 dB (lin). Slamming of incubator doors and infant crying produced 90 to 100 dB(A)

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(American Academy of Pediatrics, 1974). It was shown that inside incubators, background noise level is about 50 dBA and can reach 120 dBA (Committee on Environment Health of the American Academy of Pediatrics, 1997). Much of the energy is located below 500 Hz, between 31 and 250 Hz (Mills, 1975).

Ambient noise appears to influence the quantity and quality of the sleep of newborns. Some newborns appear to be particularly responsive to ambient noises. Sleeping premature, anoxic, or brain-damaged infants detect intruding sounds better than sleeping healthy or term babies (Mills, 1975).

Newborn infants spend most of their time sleeping. Some studies have documented hearing loss in children cared for in intensive care units (Committee on Environment Health of the American Academy of Pediatrics, 1997). Noise and some ototoxic drugs act synergistically to produce pathological changes of the inner ears of experimental animals (neomycin, kanamycin, sodium salicylate). The relationship with the infant's clinical condition and associated treatments has, however, not yet been clearly defined. Infants exposed to sound levels of incubators are usually premature, on drugs and in very poor health. Moreover, the exposures are continuous. A weak infant could spend weeks sleeping in such a noise level without rest periods away from noise (Mills, 1975).

High noise levels may be associated with other types of responses. In young infants, sudden loud (of approximately 80 dB) environmental noise induced hypoxaemia.

Noise reduction was associated in neonates with increases in sleep time, in particular in quiet sleep (Committee on Environment Health of the American Academy of Pediatrics, 1997). It also resulted in fewer days of respiratory support and oxygen administration. Premature infants cared for with noise reduction had a better maturation of electroencephalograms.

A Committee on Environmental Health of the American Academy of Pediatrics (1997) concluded that high ambient noise in the neonatal intensive care unit (NICU) changed the behavioural and physiological responses of infants. For all the above observations and considerations, sound in infant intensive care units should be maintained at under 80 dB(A) (Graven, 2000). Among other recommendations, paediatricians were encouraged to monitor sound in the NICU, and within incubators, where a noise level greater than 45 dB is of concern.

INFANTS (1 MONTH TO 1 YEAR OLD)

Some studies of the effect of external noises on the sleep/wake reactions of infants were conducted in their natural home environment. The reactions of babies to aircraft noise were studied by means of electroplethysmography (PLG) and EEG (Ando and Hattori, 1977). The recordings were done in the morning, in the infants' sleeping rooms. The infants were exposed to recorded noise of a Boeing 727 at take-off. The noise was presented at 70, 80 and 90 dB(A) at peak level at the position of the babies' heads. The subjects who had not been awakened by exposure to aircraft noise were exposed to music (Beethoven's Ninth Symphony) at levels of 70, 80 and 90 dB(A). The frequency ranged between 100 Hz and 10 kHz. It was found that the babies whose mothers had moved to the area around the Osaka International Airport before conception (Group I; n=33) or during the first five months of pregnancy (Group II; n=17) showed little or no reaction to aircraft noise. In contrast,

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babies whose mothers had moved closer to the airport during the second half of the pregnancy or after birth (Group III; n=10 or IV; n=3) and the babies whose mothers lived in a quiet living area (Group V; n=8) reacted to the same auditory stimuli. The babies in groups I and II showed differential responses depending on whether the auditory stimuli were aircraft noise or music. Abnormal PLG and EEG were observed in the majority of babies living in an area where noise levels were over 95 dB(A). It was concluded that the difference in reactivity to aircraft noise may be ascribed to a prenatal difference in time of exposure to aircraft noise. The reactions diminished after the sixth month of life in groups I and II, and the ninth month in groups III to V. This phenomenon may be explained as habituation to aircraft noise after birth. However, in all groups, no habituation occurred for a noise level over 95 dB(A) (Ando and Hattori, 1977). This study was criticized, as the authors did not adjust for several important determinants of birth weight, such as prematurity and the mother's age, weight, smoking status or socioeconomic status (Morrell et al., 1997).

Noise levels may be constantly high in paediatric units. The mean noise levels measured in a centre of a surgical recovery room were 57.2 dB(A), while those measured at the patients' heads were 65.6 dB(A) (American Academy of Pediatrics, 1974). In a medical unit (6-bed wards containing 5 infants between 3 and 17 months) peak sound levels were recorded on the pillow of the cot for 12 min (Keipert, 1985). Infant crying produced 75–90 dB(A) and a beeper around 76–78 dB(A). Peak noise levels recorded at the nurses' station were about 78 dB(A) for telephone, 80 for infant crying, public address system, adult talking, and up to 90 dB(A) for child talking (Keipert, 1985).

In a study conducted on infants exposed to 50–80 dB(lin) in the range of 100–7000 Hz (American Academy of Pediatrics, 1974), a level of 70–75 dB(lin) for 3 minutes led to obvious disturbance or awakening in two thirds of the children. All infants awakened after 75 dB(lin) for 12 minutes.

In other studies conducted on the effects of awakening and arousal, it was shown that white noise intensity was significantly lower when it elicited polygraphically scored arousals than when it induced awakenings (Franco et al., 1998).

TODDLERS PREADOLESCENTS (8–12 YEARS OLD) ADOLESCENTS (13–18 YEARS OLD)

Developmental variations in auditory arousal thresholds during sleep were investigated in four groups of normal male subjects: children (n=6; 5–7 years old), preadolescents (n=10; 8–12 years old), adolescents (n=10; 13–16 years old), and young adults (n=10; 20–24 years old) (Busby, Mercier and Pivik, 1994). Arousal thresholds were determined during non-REM and REM sleep for tones (3-s, 1500 Hz pure tones delivered in an ascending series of increasing intensity, 5 dB increments beginning at 30 dB SPL (sound pressure level) re 0.0002 dynes/cm2 until awakening or maximum intensity of 120 dB) presented via earphone insert on a single night following two adaptation nights of undisturbed sleep. Age-related relationships were observed for both awakening frequency and stimulus intensity required to effect awakening, with awakenings occurring more frequently in response to lower stimulus intensities with increasing age. In children, 43.1% of stimuli induced awakenings, in preadolescents 54.8%, adolescents 72% and adults 100% ($X2=60.37$; $p<.001$). Partial arousals (brief EEG desynchronization and/or EMG activity with the subjects returning to sleep) occurred in 9.8% of children, 4.8% of preadoles-

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cents, 12.2% of adolescents, 0% of adults. Although stimulus intensities required for awakening were high and statistically equivalent across sleep stages in non-adults, higher intensity stimulus was required in stage 4 relative to stage 2 and REM sleep. Frequency of awakening increased with age, whereas stimulus intensities required to effect these awakenings decreased with age. These relationships were maintained for individual sleep stages. These results confirm previous observations of marked resistance to awakening during sleep in preadolescent children and suggest that processes underlying awakening from sleep undergo systematic modification during ontogenic development. The observed resistance to elicited awakening from sleep extending up to young adulthood implies the presence of an active, developmentally related process that maintains sleep (Busby, Mercier and Pivik, 1994).

In another study, children aged 5–7 years were shown to be 10–15 dB less sensitive to pure tones than adults aged 22–30 (Mills, 1975). Another report on male hyperactive and normal children aged 8–12 showed that these children were awakened with auditory stimulus intensity levels of up to 123 dB SPL, much higher than values reported for adults (range of 50–85 dB) (Busby, Mercier and Pivik, 1994).

In a study on four children (two males), aged 5–8 years old on the effects of simulated sonic booms (68 dB(A) near the subjects' ears), 94.1% of the subjects showed no change, 5.9% had shallower sleep, but none aroused or had behavioural awakening. In general, the frequency of arousal or behavioural awakenings and of sleep stage changes increased with age (up to 75 years) (Lukas, 1975).

In a prospective longitudinal investigation, which employed non-exposed control groups, effects of aircraft noise prior to and subsequent to inauguration of a new airport as well as effects of chronic noise and its reduction at an old airport (6–18 months after relocation), were studied in 326 children aged 9–13 years (Bullinger et al., 1999). The psychological health of children was investigated with a standardized quality of life scale as well as with a motivational measure. In addition, a self-report noise annoyance scale was used. In the children studied at the two airports over three time points, results showed a significant decrease of total quality of life 18 months after aircraft noise exposure as well as motivational deficits demonstrated by fewer attempts to solve insoluble puzzles in the new airport area. Parallel shifts in children's attributions for failure were also noted. At the old airport parallel impairments were present before the airport relocation but subsided thereafter (Bullinger et al., 1999).

In some studies, the effects of ambient noise on autonomic responses could be demonstrated in children. In children aged 6–12 years exposed to intermittent traffic noise during 4 nights (at a rate of 90 noises per hour; peak intensity of the noise, 45, 55 and 65 dB(A) varied semi-randomly) and 2 quiet nights. Heart rate was affected and relatively higher in noise during REM and stage 2 than during delta sleep (Muzet et al., 1980, in Abel, 1990).

CONCLUSIONS

Several studies on the extra-auditory effects of ambient noise on sleeping children were summarized in Table 1. In relation to ambient noise, specific changes were reported in both sleep quality and quantity. Some of the effects were shown to have a dose–response relationship. Several limitations to the present report should be discussed. Firstly, no one knows

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whether the inference that is often made that the effects of noise might develop with a longer exposure time (Abel, 1990) is correct. Serious cardiorespiratory or autonomic changes, such as increases in BP could only develop following a long time exposure starting from childhood. This, in fact, has never been documented, nor has the extent of variability between subjects due to difference in susceptibility. Secondly, there is no information to evaluate whether adaptation to ambient noise could limit the effects observed during short-term experiments. Thirdly, as the existing research data are applicable to generally healthy children, no one knows how the reported findings could be applied to ill children, children receiving medical treatment or very young premature infants. Finally, as most studies were conducted in laboratory controlled environments, no one knows the correlation between these studies and the effects of noise in the home. The multifactorial effects of the environment on sleep and arousal controls could be much more complex than expected. One might predict that, similarly as for adults, the effects of noise on the child's sleep and health are very complicated and depend upon the spectrum and level of the noise, temporal aspects of the noise, psychological responses to the noise and the nature of the evaluation technique. The complexity of the conditions related to sleep/wake controls was illustrated by the review of confounding factors affecting auditory arousal thresholds.

Despite these limitations, it can be concluded that, based on the evidence available, the extra-auditory effects of noise could be pervasive, affecting the children's physical and psychological well-being. Changes in sleep quantity and quality together with autonomic reactions are seen when a child is exposed to ambient noise during sleep. Ambient noise exerts a dose-effect relationship on changes in sleep/wake behaviours. These reflect modifications induced within the brain of the sleeping child. It remains, however, to be determined what pervasive effects long-term exposure to ambient noise has on the child's development, health and well-being. Evidence should also be defined to support an enforcement of strategies for noise reduction at the source as suggested by some studies. Noise-induced health effects on children, a clinical and public health concern, should be evaluated by further studies.

Table 1
Arousal and awakening in children a review of the literature

No.	dB(A)	% responses	Type of responses	Reference
1	80	70	Nenonates motor response	Steinschneider 1967
2	60	5	Neonates startle response	Gädeke et al.,1969
70	10			
80	20			
100	60			
3	60	7	Neonates startle response	Ashton 1967
65	10			
70	40			
4	80	70	Child awake	Semczuk 1967
5	100	70	Child awake	Busby, Mercier and Pivik 1994
6	100	76	Preadolescent awake	
7	100	86	Adolescent awake	
8	60	100	Adult	
9	120	72	Infant awake	Kahn, Picard and Blum, 1986
10	75	75	Infant awake	Gädeke et al., 1969

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REFERENCES

Abel SM (1990). The extra-auditory effects of noise and annoyance: an overview of research. *Journal of Otoryngology* 1:1–13.

American Academy of Pediatrics (1974). Committee on Environmental Hazards. Noise pollution: neonatal aspects. *Pediatrics*, 54:476–479.

Ando Y, Hattori H (1977). Effects of noise on sleep of babies. *Journal of the Acoustical Society of America*, 62:199–204.

Bullinger M et al. (1999). The psychological cost of aircraft noise for children. *Zentralblad Hygiene und Umweltmedizin*, 202:127–138.

Busby KA, Mercier L, Pivik RT (1994). Ontogenetic variations in auditory arousal threshold during sleep. *Psychophysiology*, 31:182–188.

Committee on Environment Health of the American Academy of Pediatrics (1997). Noise: a hazard for the fetus and newborn. *Pediatrics*, 100:724–727.

Franco P et al. (1998). Auditory arousal thresholds are higher when infants sleep in the prone position. *Journal of Pediatrics*, 132:240–243.

Franco P et al. (1999). Prenatal exposure to cigarettes is associated with decreased arousal propensity in infants. *Journal of Pediatrics*, 135:34–8.

Franco P et al. (2000). The influence of a pacifier on infants' arousals from sleep. *Journal of Pediatrics*, 136:775–779.

Franco P et al. (2001). Ambient temperature is associated with changes in infants' arousability form sleep. *Sleep*, 24:325–329.

Franco P et al. (2002). Decreased arousals in infants sleeping with the face covered by bedclothes. *Pediatrics*, 109:112–117.

Franco P. Decreased arousals in healthy infants following short-term sleep deprivation. *Sleep*, (submitted).

Gädeke R et al. (1969). The noise level in a childrens' hospital and the wake-up threshold in infants. *Acta Paediatrica Scandinavica*, 58:164–170.

Graven SN (2000). Sound and the developing infant in the NICU: conclusions and recommendations for care. *Journal of Perinatology*, 20:S88–93.

Kahn A, Picard E, Blum D (1986). Auditory arousal thresholds of normal and near-miss SIDS infants. *Developmental Medicine and Child Neurology*, 28:299–302.

Kahn A et al. (1989). Sleep problems in healthy preadolescents. *Pediatrics*, 84:542 – 546.

Keipert JA (1985). The harmful effects of noise in a children's ward. *Australian Paediatric Journal*, 21:101–103.

Lukas JS (1975). Noise and sleep: a literature review and a proposed criterion for assessing effect. *Journal of the Acoustical Society of America*, 58:1232–1242.

McLean EK, Tarnopolsky A (1977). Noise, discomfort and mental health. *Psychological Medicine*, 7:19–62.

Mills JH (1975). Noise and children: a review of literature. *Journal of the Acoustical Society of America*, 58:767–779.

Morrell S, Taylor R, Lyle D (1997). A review of health effects of aircraft noise. *Australian and New Zealand Journal of Public Health*, 21:221–236.

Muzet A et al. (1980). Habituation and age differences of cardiovascular responses to noise during sleep. In: *Sleep 1980, 5th European Congress of Sleep Research, Amsterdam*, pp 212–215 (quoted by Abel, 1980).

Rechtschaffen A, Hauri P, Zeitlin M (1966). Auditory awakening thresholds in REM and NREM sleep stages. *Perceptual and Motor Skills*, 22:927–942.

Semczuk B (1967). Studies on the influence of acoustic stimuli on respiratory movements. *Polish Medical Journal*, 7:1070–1100.

Thomas DA et al. (1996). The effect of sleep deprivation on sleep states, breathing events, peripheral chemosensiveness and arousal propensity in healthy 3 month old infants. *European Respiratory Journal*, 9:932–938.

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The WHO Regional Office for Europe

The World Health Organization (WHO) is a specialized agency of the United Nations created in 1948 with the primary responsibility for international health matters and public health. The WHO Regional Office for Europe is one of six regional offices throughout the world, each with its own programme geared to the particular health conditions of the countries it serves.

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Environmental noise is a threat to public health, having negative impacts on human health and wellbeing. This book reviews the health effects of night time noise exposure, examines dose effects relations, and presents interim and ultimate guideline values of night noise exposure. It offers guidance to policy-makers in reducing the health impacts of night noise, based on expert evaluation of scientific evidence in Europe.

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GUIDELINES

FOR

COMMUNITY NOISE

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This WHO document on the *Guidelines for Community Noise* is the outcome of the WHO- expert task force meeting held in London, United Kingdom, in April 1999. It bases on the document entitled "Community Noise" that was prepared for the World Health Organization and published in 1995 by the Stockholm University and Karolinska Institute.

World Health Organization, Geneva
Cluster of Sustainable Development and Healthy Environment (SDE)
Department of the Protection of the Human Environment (PHE)
Occupational and Environmental Health (OEH)

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Foreword

Noise has always been an important environmental problem for man. In ancient Rome, rules existed as to the noise emitted from the ironed wheels of wagons which battered the stones on the pavement, causing disruption of sleep and annoyance to the Romans. In Medieval Europe, horse carriages and horse back riding were not allowed during night time in certain cities to ensure a peaceful sleep for the inhabitants. However, the noise problems of the past are incomparable with those of modern society. An immense number of cars regularly cross our cities and the countryside. There are heavily laden lorries with diesel engines, badly silenced both for engine and exhaust noise, in cities and on highways day and night. Aircraft and trains add to the environmental noise scenario. In industry, machinery emits high noise levels and amusement centres and pleasure vehicles distract leisure time relaxation.

In comparison to other pollutants, the control of environmental noise has been hampered by insufficient knowledge of its effects on humans and of dose-response relationships as well as a lack of defined criteria. While it has been suggested that noise pollution is primarily a “luxury” problem for developed countries, one cannot ignore that the exposure is often higher in developing countries, due to bad planning and poor construction of buildings. The effects of the noise are just as widespread and the long term consequences for health are the same. In this perspective, practical action to limit and control the exposure to environmental noise are essential. Such action must be based upon proper scientific evaluation of available data on effects, and particularly dose-response relationships. The basis for this is the
process of risk assessment and risk management.

The extent of the noise problem is large. In the European Union countries about 40 % of the population are exposed to road traffic noise with an equivalent sound pressure level exceeding 55 dB(A) daytime and 20 % are exposed to levels exceeding 65 dB(A). Taking all exposure to transportation noise together about half of the European Union citizens are estimated to live in zones which do not ensure acoustical comfort to residents. More than 30 % are exposed at night to equivalent sound pressure levels exceeding 55 dB(A) which are disturbing to sleep. The noise pollution problem is also severe in cities of developing countries and caused mainly by traffic. Data collected alongside densely travelled roads were found to have equivalent sound pressure levels for 24 hours of 75 to 80 dB(A).

The scope of WHO’s effort to derive guidelines for community noise is to consolidate actual scientific knowledge on the health impacts of community noise and to provide guidance to environmental health authorities and professional trying to protect people from the harmful effects of noise in non-industrial environments. Guidance on the health effects of noise exposure of the population has already been given in an early publication of the series of Environmental Health Criteria. The health risk to humans from exposure to environmental noise was evaluated and guidelines values derived. The issue of noise control and health protection was briefly addressed.
At a WHO/EURO Task Force Meeting in Düsseldorf, Germany, in 1992, the health criteria and guideline values were revised and it was agreed upon updated guidelines in consensus. The essentials of the deliberations of the Task Force were published by Stockholm University and Karolinska Institute in 1995. In a recent Expert Task Force Meeting convened in April 1999 in London, United Kingdom, the Guidelines for Community Noise were extended to provide global coverage and applicability, and the issues of noise assessment and control were addressed in more detail. This document is the outcome of the consensus deliberations of the WHO Expert Task Force.

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Preface

Community noise (also called environmental noise, residential noise or domestic noise) is defined as noise emitted from all sources except noise at the industrial workplace. Main sources of community noise include road, rail and air traffic, industries, construction and public work, and the neighbourhood. The main indoor sources of noise are ventilation systems, office machines, home appliances and neighbours. Typical neighbourhood noise comes from premises and installations related to the catering trade (restaurant, cafeterias, discotheques, etc.); from live or recorded music; sport events including motor sports; playgrounds; car parks; and domestic animals such as barking dogs. Many countries have regulated community noise from road and rail traffic, construction machines and industrial plants by applying emission standards, and by regulating the acoustical properties of buildings. In contrast, few countries have regulations on community noise from the neighbourhood, probably due to the lack of methods to define and measure it, and to the difficulty of controlling it. In large cities throughout the world, the general population is increasingly exposed to community due to the sources mentioned above and the health effects of these exposures are considered to be a more and more important public health problem. Specific effects to be considered when setting community noise guidelines include: interference with communication; noise-induced hearing loss; sleep disturbance effects; cardiovascular and psycho-physiological effects; performance reduction effects; annoyance responses; and effects on social behaviour.

Since 1980, the World Health Organization (WHO) has addressed the problem of community noise. Health-based guidelines on community noise can serve as the basis for deriving noise standards within a framework of noise management. Key issues of noise management include abatement options; models for forecasting and for assessing source control action; setting noise emission standards for existing and planned sources; noise exposure assessment; and testing the compliance of noise exposure with noise immission standards. In 1992, the WHO Regional Office for Europe convened a task force meeting which set up guidelines for community noise. A preliminary publication of the Karolinska Institute, Stockholm, on behalf of WHO, appeared in 1995. This publication served as the basis for the globally applicable *Guidelines for Community Noise* presented in this document. An expert task force meeting was convened by WHO in March 1999 in London, United Kingdom, to finalize the guidelines.

The *Guidelines for Community Noise* have been prepared as a practical response to the need for action on community noise at the local level, as well as the need for improved legislation, management and guidance at the national and regional levels. WHO will be pleased to see that these guidelines are used widely. Continuing efforts will be made to improve its content and structure. It would be appreciated if the users of the *Guidelines* provide feedback from its use and their own experiences. Please send your comments and suggestions on the WHO *Guidelines for Community Noise – Guideline document* to the Department of the Protection of the Human Environment, Occupational and Environmental Health, World Health Organization, Geneva, Switzerland (Fax: +41 22-791 4123, e-mail: schwelad@who.int).

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Executive Summary

1. Introduction

Community noise (also called environmental noise, residential noise or domestic noise) is defined as noise emitted from all sources except noise at the industrial workplace. Main sources of community noise include road, rail and air traffic; industries; construction and public work; and the neighbourhood. The main indoor noise sources are ventilation systems, office machines, home appliances and neighbours.

In the European Union about 40% of the population is exposed to road traffic noise with an equivalent sound pressure level exceeding 55 dB(A) daytime, and 20% are exposed to levels exceeding 65 dB(A). When all transportation noise is considered, more than half of all European Union citizens is estimated to live in zones that do not ensure acoustical comfort to residents. At night, more than 30% are exposed to equivalent sound pressure levels exceeding 55 dB(A), which are disturbing to sleep. Noise pollution is also severe in cities of developing countries. It is caused mainly by traffic and alongside densely-travelled roads equivalent sound pressure levels for 24 hours can reach 75–80 dB(A).

In contrast to many other environmental problems, noise pollution continues to grow and it is accompanied by an increasing number of complaints from people exposed to the noise. The growth in noise pollution is unsustainable because it involves direct, as well as cumulative, adverse health effects. It also adversely affects future generations, and has socio-cultural, esthetic and economic effects.

2. Noise sources and measurement

Physically, there is no distinction between sound and noise. Sound is a sensory perception and the complex pattern of sound waves is labeled noise, music, speech etc. Noise is thus defined as unwanted sound.

Most environmental noises can be approximately described by several simple measures. All measures consider the frequency content of the sounds, the overall sound pressure levels and the variation of these levels with time. Sound pressure is a basic measure of the vibrations of air that make up sound. Because the range of sound pressures that human listeners can detect is very wide, these levels are measured on a logarithmic scale with units of decibels. Consequently, sound pressure levels cannot be added or averaged arithmetically. Also, the sound levels of most noises vary with time, and when sound pressure levels are calculated, the instantaneous pressure fluctuations must be integrated over some time interval.

Most environmental sounds are made up of a complex mix of many different frequencies. Frequency refers to the number of vibrations per second of the air in which the sound is propagating and it is measured in Hertz (Hz). The audible frequency range is normally considered to be 20–20 000 Hz for younger listeners with unimpaired hearing. However, our hearing systems are not equally sensitive to all sound frequencies, and to compensate for this various types of filters or frequency weighting have been used to determine the relative strengths of frequency components making up a particular environmental noise. The A-weighting is most commonly used and weights lower frequencies as less important than mid- and higher-frequencies. It is intended to approximate the frequency response of our hearing system.

The effect of a combination of noise events is related to the combined sound energy of those events (the equal energy principle). The sum of the total energy over some time period gives a level equivalent to the average sound energy over that period. Thus, LAeq,T is the energy average equivalent level of the A-weighted sound over a period T. LAeq,T should be used to measure continuing sounds, such as road traffic noise or types of more-or-less continuous industrial noises. However, when there are distinct events to the noise, as with aircraft or railway noise, measures of individual events such as the maximum

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noise level (LAmax), or the weighted sound exposure level (SEL), should also be obtained in addition to LAeq,T. Time-varying environmental sound levels have also been described in terms of percentile levels.

Currently, the recommended practice is to assume that the equal energy principle is approximately valid for most types of noise and that a simple LAeq,T measure will indicate the expected effects of the noise reasonably well. When the noise consists of a small number of discrete events, the A-weighted maximum level (LAmax) is a better indicator of the disturbance to sleep and other activities. In most cases, however, the A-weighted sound exposure level (SEL) provides a more consistent measure of single-noise events because it is based on integration over the complete noise event. In combining day and night LAeq,T values, night-time weightings are often added. Night-time weightings are intended to reflect the expected increased sensitivity to annoyance at night, but they do not protect people from sleep disturbance.

Where there are no clear reasons for using other measures, it is recommended that LAeq,T be used to evaluate more-or-less continuous environmental noises. Where the noise is principally composed of a small number of discrete events, the additional use of LAmax or SEL is recommended. There are definite limitations to these simple measures, but there are also many practical advantages, including economy and the benefits of a standardized approach.

3. Adverse health effects of noise

The health significance of noise pollution is given in chapter 3 of the *Guidelines* under separate headings according to the specific effects: noise-induced hearing impairment; interference with speech communication; disturbance of rest and sleep; psychophysiological, mental-health and performance effects; effects on residential behaviour and annoyance; and interference with intended activities. This chapter also considers vulnerable groups and the combined effects of mixed noise sources.

Hearing impairment is typically defined as an increase in the threshold of hearing. Hearing deficits may be accompanied by tinnitus (ringing in the ears). Noise-induced hearing impairment occurs predominantly in the higher frequency range of 3 000–6 000 Hz, with the largest effect at 4 000 Hz. But with increasing LAeq,8h and increasing exposure time, noise-induced hearing impairment occurs even at frequencies as low as 2 000 Hz. However, hearing impairment is not expected to occur at LAeq,8h levels of 75 dB(A) or below, even for prolonged occupational noise exposure.

Worldwide, noise-induced hearing impairment is the most prevalent irreversible occupational hazard and it is estimated that 120 million people worldwide have disabling hearing difficulties. In developing countries, not only occupational noise but also environmental noise is an increasing risk factor for hearing impairment. Hearing damage can also be caused by certain diseases, some industrial chemicals, ototoxic drugs, blows to the head, accidents and hereditary origins. Hearing deterioration is also associated with the ageing process itself (presbyacusis).

The extent of hearing impairment in populations exposed to occupational noise depends on the value of LAeq,8h, the number of noise-exposed years, and on individual susceptibility. Men and women are equally at risk for noise-induced hearing impairment. It is expected that environmental and leisure-time noise with a LAeq,24h of 70 dB(A) or below will not cause hearing impairment in the large majority of people, even after a lifetime exposure. For adults exposed to impulse noise at the workplace, the noise limit is set at peak sound pressure levels of 140 dB, and the same limit is assumed to be appropriate for environmental and leisure-time noise. In the case of children, however, taking into account their habits while playing with noisy toys, the peak sound pressure should never exceed 120 dB. For shooting noise with LAeq,24h levels greater than 80 dB(A), there may be an increased risk for noise-induced hearing impairment.

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The main social consequence of hearing impairment is the inability to understand speech in daily living conditions, and this is considered to be a severe social handicap. Even small values of hearing impairment (10 dB averaged over 2 000 and 4 000 Hz and over both ears) may adversely affect speech comprehension.

Speech intelligibility is adversely affected by noise. Most of the acoustical energy of speech is in the frequency range of 100–6 000 Hz, with the most important cue-bearing energy being between 300–3 000 Hz. Speech interference is basically a masking process, in which simultaneous interfering noise renders speech incapable of being understood. Environmental noise may also mask other acoustical signals that are important for daily life, such as door bells, telephone signals, alarm clocks, fire alarms and other warning signals, and music.

Speech intelligibility in everyday living conditions is influenced by speech level; speech pronunciation; talker-to-listener distance; sound level and other characteristics of the interfering noise; hearing acuity; and by the level of attention. Indoors, speech communication is also affected by the reverberation characteristics of the room. Reverberation times over 1 s produce loss in speech discrimination and make speech perception more difficult and straining. For full sentence intelligibility in listeners with normal hearing, the signal-to-noise ratio (i.e. the difference between the speech level and the sound level of the interfering noise) should be at least 15 dB(A). Since the sound pressure level of normal speech is about 50 dB(A), noise with sound levels of 35 dB(A) or more interferes with the intelligibility of speech in smaller rooms. For vulnerable groups even lower background levels are needed, and a reverberation time below 0.6 s is desirable for adequate speech intelligibility, even in a quiet environment.

The inability to understand speech results in a large number of personal handicaps and behavioural changes. Particularly vulnerable are the hearing impaired, the elderly, children in the process of language and reading acquisition, and individuals who are not familiar with the spoken language.

Sleep disturbance is a major effect of environmental noise. It may cause primary effects during sleep, and secondary effects that can be assessed the day after night-time noise exposure. Uninterrupted sleep is a prerequisite for good physiological and mental functioning, and the primary effects of sleep disturbance are: difficulty in falling asleep; awakenings and alterations of sleep stages or depth; increased blood pressure, heart rate and finger pulse amplitude; vasoconstriction; changes in respiration; cardiac arrhythmia; and increased body movements. The difference between the sound levels of a noise event and background sound levels, rather than the absolute noise level, may determine the reaction probability. The probability of being awakened increases with the number of noise events per night. The secondary, or after-effects, the following morning or day(s) are: reduced perceived sleep quality; increased fatigue; depressed mood or well-being; and decreased performance.

For a good night's sleep, the equivalent sound level should not exceed 30 dB(A) for continuous background noise, and individual noise events exceeding 45 dB(A) should be avoided. In setting limits for single night-time noise exposures, the intermittent character of the noise has to be taken into account. This can be achieved, for example, by measuring the number of noise events, as well as the difference between the maximum sound level and the background sound level. Special attention should also be given to: noise sources in an environment with low background sound levels; combinations of noise and vibrations; and to noise sources with low-frequency components.

Physiological Functions. In workers exposed to noise, and in people living near airports, industries and noisy streets, noise exposure may have a large temporary, as well as permanent, impact on physiological functions. After prolonged exposure, susceptible individuals in the general population may develop permanent effects, such as hypertension and ischaemic heart disease associated with exposure to high sound levels. The magnitude and duration of the effects are determined in part by individual characteristics, lifestyle behaviours and environmental conditions. Sounds also evoke reflex responses, particularly when they are unfamiliar and have a sudden onset.

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Workers exposed to high levels of industrial noise for 5–30 years may show increased blood pressure and an increased risk for hypertension. Cardiovascular effects have also been demonstrated after long-term exposure to air- and road-traffic with LAeq,24h values of 65–70 dB(A). Although the associations are weak, the effect is somewhat stronger for ischaemic heart disease than for hypertension. Still, these small risk increments are important because a large number of people are exposed.

Mental Illness. Environmental noise is not believed to cause mental illness directly, but it is assumed that it can accelerate and intensify the development of latent mental disorders. Exposure to high levels of occupational noise has been associated with development of neurosis, but the findings on environmental noise and mental-health effects are inconclusive. Nevertheless, studies on the use of drugs such as tranquillizers and sleeping pills, on psychiatric symptoms and on mental hospital admission rates, suggest that community noise may have adverse effects on mental health.

Performance. It has been shown, mainly in workers and children, that noise can adversely affect performance of cognitive tasks. Although noise-induced arousal may produce better performance in simple tasks in the short term, cognitive performance substantially deteriorates for more complex tasks. Reading, attention, problem solving and memorization are among the cognitive effects most strongly affected by noise. Noise can also act as a distracting stimulus and impulsive noise events may produce disruptive effects as a result of startle responses.

Noise exposure may also produce after-effects that negatively affect performance. In schools around airports, children chronically exposed to aircraft noise under-perform in proof reading, in persistence on challenging puzzles, in tests of reading acquisition and in motivational capabilities. It is crucial to recognize that some of the adaptation strategies to aircraft noise, and the effort necessary to maintain task performance, come at a price. Children from noisier areas have heightened sympathetic arousal, as indicated by increased stress hormone levels, and elevated resting blood pressure. Noise may also produce impairments and increase in errors at work, and some accidents may be an indicator of performance deficits.

Social and Behavioural Effects of Noise; Annoyance. Noise can produce a number of social and behavioural effects as well as annoyance. These effects are often complex, subtle and indirect and many effects are assumed to result from the interaction of a number of non-auditory variables. The effect of community noise on annoyance can be evaluated by questionnaires or by assessing the disturbance of specific activities. However, it should be recognized that equal levels of different traffic and industrial noises cause different magnitudes of annoyance. This is because annoyance in populations varies not only with the characteristics of the noise, including the noise source, but also depends to a large degree on many non-acoustical factors of a social, psychological, or economic nature. The correlation between noise exposure and general annoyance is much higher at group level than at individual level. Noise above 80 dB(A) may also reduce helping behaviour and increase aggressive behaviour. There is particular concern that high-level continuous noise exposures may increase the susceptibility of schoolchildren to feelings of helplessness.

Stronger reactions have been observed when noise is accompanied by vibrations and contains low-frequency components, or when the noise contains impulses, such as with shooting noise. Temporary, stronger reactions occur when the noise exposure increases over time, compared to a constant noise exposure. In most cases, LAeq,24h and L_{dn} are acceptable approximations of noise exposure related to annoyance. However, there is growing concern that all the component parameters should be individually assessed in noise exposure investigations, at least in the complex cases. There is no consensus on a model for total annoyance due to a combination of environmental noise sources.

Combined Effects on Health of Noise from Mixed Sources. Many acoustical environments consist of sounds from more than one source, i.e. there are mixed sources, and some combinations of effects are common. For example, noise may interfere with speech in the day and create sleep disturbance at night.

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These conditions certainly apply to residential areas heavily polluted with noise. Therefore, it is important that the total adverse health load of noise be considered over 24 hours, and that the precautionary principle for sustainable development be applied.

Vulnerable Subgroups. Vulnerable subgroups of the general population should be considered when recommending noise protection or noise regulations. The types of noise effects, specific environments and specific lifestyles are all factors that should be addressed for these subgroups. Examples of vulnerable subgroups are: people with particular diseases or medical problems (e.g. high blood pressure); people in hospitals or rehabilitating at home; people dealing with complex cognitive tasks; the blind; people with hearing impairment; fetuses, babies and young children; and the elderly in general. People with impaired hearing are the most adversely affected with respect to speech intelligibility. Even slight hearing impairments in the high-frequency sound range may cause problems with speech perception in a noisy environment. A majority of the population belongs to the subgroup that is vulnerable to speech interference.

4. Guideline values

In chapter 4, guideline values are given for specific health effects of noise and for specific environments.

Specific health effects.

Interference with Speech Perception. A majority of the population is susceptible to speech interference by noise and belongs to a vulnerable subgroup. Most sensitive are the elderly and persons with impaired hearing. Even slight hearing impairments in the high-frequency range may cause problems with speech perception in a noisy environment. From about 40 years of age, the ability of people to interpret difficult, spoken messages with low linguistic redundancy is impaired compared to people 20–30 years old. It has also been shown that high noise levels and long reverberation times have more adverse effects in children, who have not completed language acquisition, than in young adults.

When listening to complicated messages (at school, foreign languages, telephone conversation) the signal-to-noise ratio should be at least 15 dB with a voice level of 50 dB(A). This sound level corresponds on average to a casual voice level in both women and men at 1 m distance. Consequently, for clear speech perception the background noise level should not exceed 35 dB(A). In classrooms or conference rooms, where speech perception is of paramount importance, or for sensitive groups, background noise levels should be as low as possible. Reverberation times below 1 s are also necessary for good speech intelligibility in smaller rooms. For sensitive groups, such as the elderly, a reverberation time below 0.6 s is desirable for adequate speech intelligibility even in a quiet environment.

Hearing Impairment. Noise that gives rise to hearing impairment is by no means restricted to occupational situations. High noise levels can also occur in open air concerts, discotheques, motor sports, shooting ranges, in dwellings from loudspeakers, or from leisure activities. Other important sources of loud noise are headphones, as well as toys and fireworks which can emit impulse noise. The ISO standard 1999 gives a method for estimating noise-induced hearing impairment in populations exposed to all types of noise (continuous, intermittent, impulse) during working hours. However, the evidence strongly suggests that this method should also be used to calculate hearing impairment due to noise exposure from environmental and leisure time activities. The ISO standard 1999 implies that long-term exposure to LAeq,24h noise levels of up to 70 dB(A) will not result in hearing impairment. To avoid hearing loss from impulse noise exposure, peak sound pressures should never exceed 140 dB for adults, and 120 dB for children.

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Sleep Disturbance. Measurable effects of noise on sleep begin at LAeq levels of about 30 dB. However, the more intense the background noise, the more disturbing is its effect on sleep. Sensitive groups mainly include the elderly, shift workers, people with physical or mental disorders and other individuals who have difficulty sleeping.

Sleep disturbance from intermittent noise events increases with the maximum noise level. Even if the total equivalent noise level is fairly low, a small number of noise events with a high maximum sound pressure level will affect sleep. Therefore, to avoid sleep disturbance, guidelines for community noise should be expressed in terms of the equivalent sound level of the noise, as well as in terms of maximum noise levels and the number of noise events. It should be noted that low-frequency noise, for example, from ventilation systems, can disturb rest and sleep even at low sound pressure levels.

When noise is continuous, the equivalent sound pressure level should not exceed 30 dB(A) indoors, if negative effects on sleep are to be avoided. For noise with a large proportion of low-frequency sound a still lower guideline value is recommended. When the background noise is low, noise exceeding 45 dB LAmax should be limited, if possible, and for sensitive persons an even lower limit is preferred. Noise mitigation targeted to the first part of the night is believed to be an effective means for helping people fall asleep. It should be noted that the adverse effect of noise partly depends on the nature of the source. A special situation is for newborns in incubators, for which the noise can cause sleep disturbance and other health effects.

Reading Acquisition. Chronic exposure to noise during early childhood appears to impair reading acquisition and reduces motivational capabilities. Evidence indicates that the longer the exposure, the greater the damage. Of recent concern are the concomitant psychophysiological changes (blood pressure and stress hormone levels). There is insufficient information on these effects to set specific guideline values. It is clear, however, that daycare centres and schools should not be located near major noise sources, such as highways, airports, and industrial sites.

Annoyance. The capacity of a noise to induce annoyance depends upon its physical characteristics, including the sound pressure level, spectral characteristics and variations of these properties with time. During daytime, few people are highly annoyed at LAeq levels below 55 dB(A), and few are moderately annoyed at LAeq levels below 50 dB(A). Sound levels during the evening and night should be 5–10 dB lower than during the day. Noise with low-frequency components require lower guideline values. For intermittent noise, it is emphasized that it is necessary to take into account both the maximum sound pressure level and the number of noise events. Guidelines or noise abatement measures should also take into account residential outdoor activities.

Social Behaviour. The effects of environmental noise may be evaluated by assessing its interference with social behavior and other activities. For many community noises, interference with rest/recreation/watching television seem to be the most important effects. There is fairly consistent evidence that noise above 80 dB(A) causes reduced helping behavior, and that loud noise also increases aggressive behavior in individuals predisposed to aggressiveness. In schoolchildren, there is also concern that high levels of chronic noise contribute to feelings of helplessness. Guidelines on this issue, together with cardiovascular and mental effects, must await further research.

Specific environments.

A noise measure based only on energy summation and expressed as the conventional equivalent measure, LAeq, is not enough to characterize most noise environments. It is equally important to measure the maximum values of noise fluctuations, preferably combined with a measure of the number of noise events. If the noise includes a large proportion of low-frequency components, still lower values than the guideline values below will be needed. When prominent low-frequency components are present, noise

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measures based on A-weighting are inappropriate. The difference between dB(C) and dB(A) will give crude information about the presence of low-frequency components in noise, but if the difference is more than 10 dB, it is recommended that a frequency analysis of the noise be performed. It should be noted that a large proportion of low-frequency components in noise may increase considerably the adverse effects on health.

In Dwellings. The effects of noise in dwellings, typically, are sleep disturbance, annoyance and speech interference. For bedrooms the critical effect is sleep disturbance. Indoor guideline values for bedrooms are 30 dB LAeq for continuous noise and 45 dB LAmax for single sound events. Lower noise levels may be disturbing depending on the nature of the noise source. At night-time, outside sound levels about 1 metre from facades of living spaces should not exceed 45 dB LAeq, so that people may sleep with bedroom windows open. This value was obtained by assuming that the noise reduction from outside to inside with the window open is 15 dB. To enable casual conversation indoors during daytime, the sound level of interfering noise should not exceed 35 dB LAeq. The maximum sound pressure level should be measured with the sound pressure meter set at "Fast".

To protect the majority of people from being seriously annoyed during the daytime, the outdoor sound level from steady, continuous noise should not exceed 55 dB LAeq on balconies, terraces and in outdoor living areas. To protect the majority of people from being moderately annoyed during the daytime, the outdoor sound level should not exceed 50 dB LAeq. Where it is practical and feasible, the lower outdoor sound level should be considered the maximum desirable sound level for new development.

In Schools and Preschools. For schools, the critical effects of noise are speech interference, disturbance of information extraction (e.g. comprehension and reading acquisition), message communication and annoyance. To be able to hear and understand spoken messages in class rooms, the background sound level should not exceed 35 dB LAeq during teaching sessions. For hearing impaired children, a still lower sound level may be needed. The reverberation time in the classroom should be about 0.6 s, and preferably lower for hearing impaired children. For assembly halls and cafeterias in school buildings, the reverberation time should be less than 1 s. For outdoor playgrounds the sound level of the noise from external sources should not exceed 55 dB LAeq, the same value given for outdoor residential areas in daytime.

For preschools, the same critical effects and guideline values apply as for schools. In bedrooms in preschools during sleeping hours, the guideline values for bedrooms in dwellings should be used.

In Hospitals. For most spaces in hospitals, the critical effects are sleep disturbance, annoyance, and communication interference, including warning signals. The LAmax of sound events during the night should not exceed 40 dB(A) indoors. For ward rooms in hospitals, the guideline values indoors are 30dB LAeq, together with 40 dB LAmax during night. During the day and evening the guideline value indoors is 30 dB LAeq. The maximum level should be measured with the sound pressure instrument set at "Fast".

Since patients have less ability to cope with stress, the LAeq level should not exceed 35 dB in most rooms in which patients are being treated or observed. Attention should be given to the sound levels in intensive care units and operating theaters. Sound inside incubators may result in health problems for neonates, including sleep disturbance, and may also lead to hearing impairment. Guideline values for sound levels in incubators must await future research.

Ceremonies, Festivals and Entertainment Events. In many countries, there are regular ceremonies, festivals and entertainment events to celebrate life periods. Such events typically produce loud sounds, including music and impulsive sounds. There is widespread concern about the effect of loud music and impulsive sounds on young people who frequently attend concerts, discotheques, video arcades, cinemas, amusement parks and spectator events. At these events, the sound level typically exceeds 100 dB LAeq. Such noise exposure could lead to significant hearing impairment after frequent attendances.

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Noise exposure for employees of these venues should be controlled by established occupational standards; and at the very least, the same standards should apply to the patrons of these premises. Patrons should not be exposed to sound levels greater than 100 dB LAeq during a four-hour period more than four times per year. To avoid acute hearing impairment the LAmax should always be below 110 dB.

Headphones. To avoid hearing impairment from music played back in headphones, in both adults and children, the equivalent sound level over 24 hours should not exceed 70 dB(A). This implies that for a daily one hour exposure the LAeq level should not exceed 85 dB(A). To avoid acute hearing impairment LAmax should always be below 110 dB(A). The exposures are expressed in free-field equivalent sound level.

Toys, Fireworks and Firearms. To avoid acute mechanical damage to the inner ear from impulsive sounds from toys, fireworks and firearms, adults should never be exposed to more than 140 dB(lin) peak sound pressure level. To account for the vulnerability in children when playing, the peak sound pressure produced by toys should not exceed 120 dB(lin), measured close to the ears (100 mm). To avoid acute hearing impairment LAmax should always be below 110 dB(A).

Parkland and Conservation Areas. Existing large quiet outdoor areas should be preserved and the signal-to-noise ratio kept low.

Table 1 presents the WHO guideline values arranged according to specific environments and critical health effects. The guideline values consider all identified adverse health effects for the specific environment. An adverse effect of noise refers to any temporary or long-term impairment of physical, psychological or social functioning that is associated with noise exposure. Specific noise limits have been set for each health effect, using the lowest noise level that produces an adverse health effect (i.e. the critical health effect). Although the guideline values refer to sound levels impacting the most exposed receiver at the listed environments, they are applicable to the general population. The time base for LAeq for “daytime” and “night-time” is 12–16 hours and 8 hours, respectively. No time base is given for evenings, but typically the guideline value should be 5–10 dB lower than in the daytime. Other time bases are recommended for schools, preschools and playgrounds, depending on activity.

It is not enough to characterize the noise environment in terms of noise measures or indices based only on energy summation (e.g., LAeq), because different critical health effects require different descriptions. It is equally important to display the maximum values of the noise fluctuations, preferably combined with a measure of the number of noise events. A separate characterization of night-time noise exposures is also necessary. For indoor environments, reverberation time is also an important factor for things such as speech intelligibility. If the noise includes a large proportion of low-frequency components, still lower guideline values should be applied. Supplementary to the guideline values given in Table 1, precautions should be taken for vulnerable groups and for noise of certain character (e.g. low-frequency components, low background noise).

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Table 1: Guideline values for community noise in specific environments.

Specific environment	Critical health effect(s)	L_{Aeq} [dB(A)]	Time base [hours]	L_{Amax} fast [dB]
Outdoor living area	Serious annoyance, daytime and evening Moderate annoyance, daytime and evening	55 50	16 16	- -
Dwelling, indoors Inside bedrooms	Speech intelligibility & moderate annoyance, daytime & evening Sleep disturbance, night-time	35 30	16 8	 45
Outside bedrooms	Sleep disturbance, window open (outdoor values)	45	8	60
School class rooms & pre-schools, indoors	Speech intelligibility, disturbance of information extraction, message communication	35	during class	-
Pre-school bedrooms, indoor	Sleep disturbance	30	sleeping-time	45
School, playground outdoor	Annoyance (external source)	55	during play	-
Hospital, ward rooms, indoors	Sleep disturbance, night-time Sleep disturbance, daytime and evenings	30 30	8 16	40 -
Hospitals, treatment rooms, indoors	Interference with rest and recovery	#1		
Industrial, commercial shopping and traffic areas, indoors and outdoors	Hearing impairment	70	24	110
Ceremonies, festivals and entertainment events	Hearing impairment (patrons:<5 times/year)	100	4	110
Public addresses, indoors and outdoors	Hearing impairment	85	1	110
Music and other sounds through headphones/ earphones	Hearing impairment (free-field value)	85 #4	1	110
Impulse sounds from toys, fireworks and firearms	Hearing impairment (adults) Hearing impairment (children)	- -	- -	140 #2 120 #2
Outdoors in parkland and conservations areas	Disruption of tranquillity	#3		

#1: As low as possible.

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#2: Peak sound pressure (not LAF, max) measured 100 mm from the ear.
#3: Existing quiet outdoor areas should be preserved and the ratio of intruding noise to natural background sound should be kept low.
#4: Under headphones, adapted to free-field values.

5. Noise Management

Chapter 5 is devoted to noise management with discussions on: strategies and priorities in managing indoor noise levels; noise policies and legislation; the impact of environmental noise; and on the enforcement of regulatory standards.

The fundamental goals of noise management are to develop criteria for deriving safe noise exposure levels and to promote noise assessment and control as part of environmental health programmes. These basic goals should guide both international and national policies for noise management. The United Nation's Agenda 21 supports a number of environmental management principles on which government policies, including noise management policies, can be based: the principle of precaution; the "polluter pays" principle; and noise prevention. In all cases, noise should be reduced to the lowest level achievable in the particular situation. When there is a reasonable possibility that the public health will be endangered, even though scientific proof may be lacking, action should be taken to protect the public health, without awaiting the full scientific proof. The full costs associated with noise pollution (including monitoring, management, lowering levels and supervision) should be met by those responsible for the source of noise. Action should be taken where possible to reduce noise at the source.

A legal framework is needed to provide a context for noise management. National noise standards can usually be based on a consideration of international guidelines, such as these *Guidelines for Community Noise*, as well as national criteria documents, which consider dose-response relationships for the effects of noise on human health. National standards take into account the technological, social, economic and political factors within the country. A staged program of noise abatement should also be implemented to achieve the optimum health protection levels over the long term.

Other components of a noise management plan include: noise level monitoring; noise exposure mapping; exposure modeling; noise control approaches (such as mitigation and precautionary measures); and evaluation of control options. Many of the problems associated with high noise levels can be prevented at low cost, if governments develop and implement an integrated strategy for the indoor environment, in concert with all social and economic partners. Governments should establish a "National Plan for a Sustainable Noise Indoor Environment" that applies both to new construction as well as to existing buildings.

The actual priorities in rational noise management will differ for each country. Priority setting in noise management refers to prioritizing the health risks to be avoided and concentrating on the most important sources of noise. Different countries have adopted a range of approaches to noise control, using different policies and regulations. A number of these are outlined in chapter 5 and Appendix 2, as examples. It is evident that noise emission standards have proven insufficient and that the trends in noise pollution are unsustainable.

The concept of environmental an environmental noise impact analysis is central to the philosophy of managing environmental noise. Such an analysis should be required before implementing any project that would significantly increase the level of environmental noise in a community (typically, greater than a 5 dB increase). The analysis should include: a baseline description of the existing noise environment; the

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expected level of noise from the new source; an assessment of the adverse health effects; an estimation of the population at risk; the calculation of exposure-response relationships; an assessment of risks and their acceptability; and a cost-benefit analysis.

Noise management should:

1. Start monitoring human exposures to noise.
2. Have health control require mitigation of noise immissions, and not just of noise source emissions. The following should be taken into consideration:
    - specific environments such as schools, playgrounds, homes, hospitals.
    - environments with multiple noise sources, or which may amplify the effects of noise.
    - sensitive time periods such as evenings, nights and holidays.
    - groups at high risk, such as children and the hearing impaired.
3. Consider the noise consequences when planning transport systems and land use.
4. Introduce surveillance systems for noise-related adverse health effects.
5. Assess the effectiveness of noise policies in reducing adverse health effects and exposure, and in improving supportive "soundscapes".
6. Adopt these *Guidelines for Community Noise* as intermediary targets for improving human health.
7. Adopt precautionary actions for a sustainable development of the acoustical environments.

Conclusions and recommendations

In chapter 6 are discussed: the implementation of the guidelines; further WHO work on noise; and research needs are recommended.

Implementation. For implementation of the guidelines it is recommended that:

- Governments should protection the population from community noise and consider it an integral part of their policy of environmental protection.
- Governments should consider implementing action plans with short-term, medium-term and long-term objectives for reducing noise levels.
- Governments should adopt the *Health Guidelines for Community Noise* values as targets to be achieved in the long-term.
- Governments should include noise as an important public health issue in environmental impact assessments.
- Legislation should be put in place to allow for the reduction of sound levels.
- Existing legislation should be enforced.
- Municipalities should develop low noise implementation plans.
- Cost-effectiveness and cost-benefit analyses should be considered potential instruments for meaningful management decisions.
- Governments should support more policy-relevant research.

Future Work. The Expert Task Force worked out several suggestions for future work for the WHO in the field of community noise. WHO should:

- Provide leadership and technical direction in defining future noise research priorities.
- Organize workshops on how to apply the guidelines.

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- Provide leadership and coordinate international efforts to develop techniques for designing supportive sound environments (e.g. "soundscapes").
- Provide leadership for programs to assess the effectiveness of health-related noise policies and regulations.
- Provide leadership and technical direction for the development of sound methodologies for environmental and health impact plans.
- Encourage further investigation into using noise exposure as an indicator of environmental deterioration (e.g. black spots in cities).
- Provide leadership and technical support, and advise developing countries to facilitate development of noise policies and noise management.

Research and Development. A major step forward in raising the awareness of both the public and of decision makers is the recommendation to concentrate more research and development on variables which have monetary consequences. This means that research should consider not only dose-response relationships between sound levels, but also politically relevant variables, such as noise-induced social handicap; reduced productivity; decreased performance in learning; workplace and school absenteeism; increased drug use; and accidents.

In Appendices 1–6 are given: bibliographic references; examples of regional noise situations (African Region, American Region, Eastern Mediterranean Region, South East Asian Region, Western Pacific Region); a glossary; a list of acronyms; and a list of participants.

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Introduction

Community noise (also called environmental noise, residential noise or domestic noise) is defined as noise emitted from all sources, except noise at the industrial workplace. Main sources of community noise include road, rail and air traffic, industries, construction and public work, and the neighbourhood. Typical neighbourhood noise comes from premises and installations related to the catering trade (restaurant, cafeterias, discotheques, etc.); from live or recorded music; from sporting events including motor sports; from playgrounds and car parks; and from domestic animals such as barking dogs. The main indoor sources are ventilation systems, office machines, home appliances and neighbours. Although many countries have regulations on community noise from road, rail and air traffic, and from construction and industrial plants, few have regulations on neighbourhood noise. This is probably due to the lack of methods to define and measure it, and to the difficulty of controlling it. In developed countries, too, monitoring of compliance with, and enforcement of, noise regulations are weak for lower levels of urban noise that correspond to occupationally controlled levels (>85 dB LAeq,8h; Frank 1998). Recommended guideline values based on the health effects of noise, other than occupationally-induced effects, are often not taken into account.

The extent of the community noise problem is large. In the European Union about 40% of the population is exposed to road traffic noise with an equivalent sound pressure level exceeding 55 dBA daytime; and 20% is exposed to levels exceeding 65 dBA (Lambert & Vallet 19 1994). When all transportation noise is considered, about half of all European Union citizens live in zones that do not ensure acoustical comfort to residents. At night, it is estimated that more than 30% is exposed to equivalent sound pressure levels exceeding 55 dBA, which are disturbing to sleep. The noise pollution problem is also severe in the cities of developing countries and is caused mainly by traffic. Data collected alongside densely traveled roads were found to have equivalent sound pressure levels for 24 hours of 75–80 dBA (e.g. National Environment Board Thailand 19 1990; Mage & Walsh 19 1998).

(a) In contrast to many other environmental problems, noise pollution continues to grow, accompanied by an increasing number of complaints from affected individuals. Most people are typically exposed to several noise sources, with road traffic noise being a dominant source (OECD-ECMT 19 1995). Population growth, urbanization and to a large extent technological development are the main driving forces, and future enlargements of highway systems, international airports and railway systems will only increase the noise problem. Viewed globally, the growth in urban environmental noise pollution is unsustainable, because it involves not simply the direct and cumulative adverse effects on health. It also adversely affects future generations by degrading residential, social and learning environments, with corresponding economical losses (Berglund 1998). Thus, noise is not simply a local problem, but a global issue that affects everyone (Lang 1999; Sandberg 1999) and calls for precautionary action in any environmental planning situation.

The objective of the World Health Organization (WHO) is the attainment by all peoples of the highest possible level of health. As the first principle of the WHO Constitution the definition of ‘health’ is given as: “A state of complete physical, mental and social well-

being and not merely the absence of disease or infirmity". This broad definition of health embraces the concept of well-being and, thereby, renders noise impacts such as population annoyance, interference with communication, and impaired task performance as 'health' issues. In 1992, a WHO Task Force also identified the following specific health effects for the general population that may result from community noise: interference with communication; annoyance responses; effects on sleep, and on the cardiovascular and psychophysiological systems; effects on performance, productivity, and social behavior; and noise-induced hearing impairment (WHO 1993; Berglund & Lindvall 1995; *cf*. WHO 1980). Hearing damage is expected to result from both occupational and environmental noise, especially in developing countries, where compliance with noise regulation is known to be weak (Smith 1998).

Noise is likely to continue as a major issue well into the next century, both in developed and in developing countries. Therefore, strategic action is urgently required, including continued noise control at the source and in local areas. Most importantly, joint efforts among countries are necessary at a system level, in regard to the access and use of land, airspace and seawaters, and in regard to the various modes of transportation. Certainly, mankind would benefit from societal reorganization towards healthy transport. To understand noise we must understand the different types of noise and how we measure it, where noise comes from and the effects of noise on human beings. Furthermore, noise mitigation, including noise management, has to be actively introduced and in each case the policy implications have to be evaluated for efficiency.

This document is organized as follows. In Chapter 2 noise sources and measurement are discussed, including the basic aspects of source characteristics, sound propagation and transmission. In Chapter 3 the adverse health effects of noise are characterized. These include noise-induced hearing impairment, interference with speech communication, sleep disturbance, cardiovascular and physiological effects, mental health effects, performance effects, and annoyance reactions. This chapter is rounded out by a consideration of combined noise sources and their effects, and a discussion of vulnerable groups. In Chapter 4 the Guideline values are presented. Chapter 5 is devoted to noise management. Included are discussions of: strategies and priorities in the management of indoor noise levels; noise policies and legislation; environmental noise impact; and enforcement of regulatory standards. In Chapter 6 implementation of the WHO Guidelines is discussed, as well as future WHO work on noise and its research needs. In Appendices 1–6 are given: bibliographic references; examples of regional noise situations (African Region, American Region, Eastern Mediterranean Region, South East Asian Region, Western Pacific Region); a glossary; a list of acronyms; and a list of participants.

2. Noise sources and their measurement

2.1.Basic Aspects of Acoustical Measurements

Most environmental noises can be approximately described by one of several simple measures. They are all derived from overall sound pressure levels, the variation of these levels with time and the frequency of the sounds. Ford (1987) gives a more extensive review of various environmental noise measures. Technical definitions are found in the glossary in Appendix 3.

2.1.1. Sound pressure level

The sound pressure level is a measure of the air vibrations that make up sound. All measured sound pressures are referenced to a standard pressure that corresponds roughly to the threshold of hearing at 1 000 Hz. Thus, the sound pressure level indicates how much greater the measured sound is than this threshold of hearing. Because the human ear can detect a wide range of sound pressure levels (10–102 Pascal (Pa)), they are measured on a logarithmic scale with units of decibels (dB). A more technical definition of sound pressure level is found in the glossary.

The sound pressure levels of most noises vary with time. Consequently, in calculating some measures of noise, the instantaneous pressure fluctuations must be integrated over some time interval. To approximate the integration time of our hearing system, sound pressure meters have a standard *Fast* response time, which corresponds to a time constant of 0.125 s. Thus, all measurements of sound pressure levels and their variation over time should be made using the *Fast* response time, to provide sound pressure measurements more representative of human hearing. Sound pressure meters may also include a *Slow* response time with a time constant of 1 s, but its sole purpose is that one can more easily estimate the average value of rapidly fluctuating levels. Many modern meters can integrate sound pressures over specified periods and provide average values. It is not recommended that the *Slow* response time be used when integrating sound pressure meters are available.

Because sound pressure levels are measured on a logarithmic scale they cannot be added or averaged arithmetically. For example, adding two sounds of equal pressure levels results in a total pressure level that is only 3 dB greater than each individual sound pressure level. Consequently, when two sounds are combined the resulting sound pressure level will be significantly greater than the individual sound levels only if the two sounds have similar pressure levels. Details for combining sound pressure levels are given in Appendix 2.

2.1.2. Frequency and frequency weighting

The unit of frequency is the Hertz (Hz), and it refers to the number of vibrations per second of the air in which the sound is propagating. For tonal sounds, frequency is associated with the perception of pitch. For example, orchestras often tune to the frequency of 440 Hz. Most environmental sounds, however, are made up of a complex mix of many different frequencies. They may or may not have discrete frequency components superimposed on noise with a broad

frequency spectrum (i.e. sound with a broad range of frequencies). The audible frequency range is normally considered to range from 20–20 000 Hz. Below 20 Hz we hear individual sound pulses rather than recognizable tones. Hearing sensitivity to higher frequencies decreases with age and exposure to noise. Thus, 20 000 Hz represents an upper limit of audibility for younger listeners with unimpaired hearing.

Our hearing systems are not equally sensitive to all sound frequencies (ISO 1987a). Thus, not all frequencies are perceived as being equally loud at the same sound pressure level, and when calculating overall environmental noise ratings it is necessary to consider sounds at some frequencies as more important than those at other frequencies. Detailed frequency analyses are commonly performed with standard sets of octave or 1/3 octave bandwidth filters. Alternatively, Fast Fourier Transform techniques or other types of filters can be used to determine the relative strengths of the various frequency components making up a particular environmental noise.

Frequency weighting networks provide a simpler approach for weighting the importance of different frequency components in one single number rating. The A-weighting is most commonly used and is intended to approximate the frequency response of our hearing system. It weights lower frequencies as less important than mid- and higher-frequency sounds. C-weighting is also quite common and is a nearly flat frequency response with the extreme high and low frequencies attenuated. When no frequency analysis is possible, the difference between A-weighted and C-weighted levels gives an indication of the amount of low frequency content in the measured noise. When the sound has an obvious tonal content, a correction to account for the additional annoyance may be used (ISO 1987b).

2.1.3. Equivalent continuous sound pressure level

According to the equal energy principle, the effect of a combination of noise events is related to the combined sound energy of those events. Thus, measures such as the equivalent continuous sound pressure level (LAeq,T) sum up the total energy over some time period (T) and give a level equivalent to the average sound energy over that period. Such average levels are usually based on integration of A-weighted levels. Thus LAeq,T is the average energy equivalent level of the A-weighted sound over a period T.

2.1.4. Individual noise events

It is often desired to measure the maximum level (LAmax) of individual noise events. For cases such as the noise from a single passing vehicle, LAmax values should be measured using the *Fast* response time because it will give a good correlation with the integration of loudness by our hearing system. However, for very short-duration impulsive sounds it is often desirable to measure the instantaneous peak amplitude to assess potential hearing-damage risk. If actual instantaneous pressure cannot be determined, then a time-integrated ‘peak’ level with a time constant of no more than 0.05 ms should be used (ISO 1987b). Such peak readings are often made using the C- (or linear) frequency weightings.

Alternatively, discrete sound events can be evaluated in terms of their A-weighted sound exposure level (SEL, for defintion see appendix 5). The total amount of sound energy in a

particular event is assessed by the SEL. One can add up the SEL values of individual events to calculate a LAeq,T over some time period, T, of interest. In some cases the SEL may provide more consistent evaluations of individual noise events because they are derived from the complete history of the event and not just one maximum value. However, A-weighted SEL measurements have been shown to be inadequate for assessing the (perceived) loudness of complex impulsive sounds, such as those from large and small weapons (Berglund et al. 1986). In contrast, C-weighted SEL values have been found useful for rating impulsive sounds such as gun shots (Vos 1996; Buchta 1996; ISO 1987b).

2.1.5. Choice of noise measure

LAeq,T should be used to measure continuing sounds such as road traffic noise, many types of industrial noises and noise from ventilation systems in buildings. When there are distinct events to the noise such as with aircraft or railway noise, measures of the individual events should be obtained (using, for example, LAmax or SEL), in addition to LAeq,T measurements.

In the past, time-varying environmental sound levels have also been described in terms of percentile levels. These are derived from a statistical distribution of measured sound levels over some period. For example, L10 is the A-weighted level exceeded 10% of the time. L10 values have been widely used to measure road-traffic noise, but they are usually found to be highly correlated measures of the individual events, as are LAmax and SEL. L90 or L95 can be used as a measure of the general background sound pressure level that excludes the potentially confounding influence of particular local noise events.

2.1.6. Sound and noise

Physically, there is no distinction between sound and noise: sound is a sensory perception evoked by physiological processes in the auditory brain. The complex pattern of sound waves is perceptually classified as “Gestalts” and are labeled as noise, music, speech, etc. Consequently, it is not possible to define noise exclusively on the basis of the physical parameters of sound. Instead, it is common practice to define noise simply as unwanted sound. However, in some situations noise may adversely affect health in the form of acoustical energy.

2.2. Sources of Noise

This section describes various sources of noise that can affect a community. Namely, noise from industry, transportation, and from residential and leisure areas. It should be noted that equal values of LAeq,T for different sources do not always imply the same expected effect.

2.2.1. Industrial noise

Mechanized industry creates serious noise problems. It is responsible for intense noise indoors as well as outdoors. This noise is due to machinery of all kinds and often increases with the power of the machines. Sound generation mechanisms of machinery are reasonably well understood. The noise may contain predominantly low or high frequencies, tonal components,

be impulsive or have unpleasant and disruptive temporal sound patterns. Rotating and reciprocating machines generate sound that includes tonal components; and air-moving equipment tends also to generate noise with a wide frequency range. The high sound pressure levels are caused by components or gas flows that move at high speed (for example, fans, steam pressure relief valves), or by operations involving mechanical impacts (for example, stamping, riveting, road breaking). Machinery should preferably be silenced at the source.

Noise from fixed installations, such as factories or construction sites, heat pumps and ventilation systems on roofs, typically affect nearby communities. Reductions may be achieved by encouraging quieter equipment or by zoning of land into industrial and residential areas. Requirements for passive (sound insulating enclosures) and active noise control, or restriction of operation time, may also be effective.

2.2.2. Transportation noise

Transportation noise is the main source of environmental noise pollution, including road traffic, rail traffic and air traffic. As a general rule, larger and heavier vehicles emit more noise than smaller and lighter vehicles. Exceptions would include: helicopters and 2- and 3-wheeled road vehicles.

The noise of road vehicles is mainly generated from the engine and from frictional contact between the vehicle and the ground and air. In general, road-contact noise exceeds engine noise at speeds higher than 60 km/h. The physical principle responsible for generating noise from tire-road contact is less well understood. The sound pressure level from traffic can be predicted from the traffic flow rate, the speed of the vehicles, the proportion of heavy vehicles, and the nature of the road surface. Special problems can arise in areas where the traffic movements involve a change in engine speed and power, such as at traffic lights, hills, and intersecting roads; or where topography, meteorological conditions and low background levels are unfavourable (for example, mountain areas).

Railway noise depends primarily on the speed of the train, but variations are present depending upon the type of engine, wagons, and rails and their foundations, as well as the roughness of wheels and rails. Small radius curves in the track, such as may occur for urban trains, can lead to very high levels of high-frequency sound referred to as wheel squeal. Noise can be generated in stations because of running engines, whistles and loudspeakers, and in marshaling yards because of shunting operations. The introduction of high-speed trains has created special noise problems with sudden, but not impulsive, rises in noise. At speeds greater than 250 km/h, the proportion of high-frequency sound energy increases and the sound can be perceived as similar to that of overflying jet aircraft. Special problems can arise in areas close to tunnels, in valleys or in areas where the ground conditions help generate vibrations. The long-distance propagation of noise from high-speed trains will constitute a problem in the future if otherwise environment-friendly railway systems are expanded.

Aircraft operations generate substantial noise in the vicinity of both commercial and military airports. Aircraft takeoffs are known to produce intense noise, including vibration and rattle. The landings produce substantial noise in long low-altitude flight corridors. The noise is

produced by the landing gear and automatic power regulation, and also when reverse thrust is applied, all for safety reasons. In general, larger and heavier aircraft produce more noise than lighter aircraft. The main mechanism of noise generation in the early turbojet-powered aircraft was the turbulence created by the jet exhaust mixing with the surrounding air. This noise source has been significantly reduced in modern high by-pass ratio turbo-fan engines that surround the high-velocity jet exhaust with lower velocity airflow generated by the fan. The fan itself can be a significant noise source, particularly during landing and taxiing operations. Multi-bladed turbo-prop engines can produce relatively high levels of tonal noise. The sound pressure level from aircraft is, typically, predicted from the number of aircraft, the types of airplanes, their flight paths, the proportions of takeoffs and landings and the atmospheric conditions. Severe noise problems may arise at airports hosting many helicopters or smaller aircraft used for private business, flying training and leisure purposes. Special noise problems may also arise inside airplanes because of vibration. The noise emission from future superjets is unknown.

A sonic boom consists of a shock wave in the air, generated by an aircraft when it flies at a speed slightly greater than the local speed of sound. An aircraft in supersonic flight trails a sonic boom that can be heard up to 50 km on either side of its ground track, depending upon the flight altitude and the size of the aircraft (Warren 1972). A sonic boom can be heard as a loud double-boom sound. At high intensity it can damage property.

Noise from military airfields may present particular problems compared to civil airports (von Gierke & Harris 1987). For example, when used for night-time flying, for training interrupted landings and takeoffs (so-called touch-and-go), or for low-altitude flying. In certain instances, including wars, specific military activities introduce other intense noise pollution from heavy vehicles (tanks), helicopters, and small and large fire-arms.

2.2.3. Construction noise and building services noise

Building construction and excavation work can cause considerable noise emissions. A variety of sounds come from cranes, cement mixers, welding, hammering, boring and other work processes. Construction equipment is often poorly silenced and maintained, and building operations are sometimes carried out without considering the environmental noise consequences. Street services such as garbage disposal and street cleaning can also cause considerable disturbance if carried out at sensitive times of day. Ventilation and air conditioning plants and ducts, heat pumps, plumbing systems, and lifts (elevators), for example, can compromise the internal acoustical environment and upset nearby residents.

2.2.4. Domestic noise and noise from leisure activities

In residential areas, noise may stem from mechanical devices (e.g. heat pumps, ventilation systems and traffic), as well as voices, music and other kinds of sounds generated by neighbours (e.g. lawn movers, vacuum cleaners and other household equipment, music reproduction and noisy parties). Aberrant social behavior is a well-recognized noise problem in multifamily dwellings, as well as at sites for entertainment (e.g. sports and music events). Due to predominantly low-frequency components, noise from ventilation systems in residential buildings may also cause considerable concern even at low and moderate sound pressure levels.

The use of powered machines in leisure activities is increasing. For example, motor racing, off-road vehicles, motorboats, water skiing, snowmobiles etc., and these contribute significantly to loud noises in previously quiet areas. Shooting activities not only have considerable potential for disturbing nearby residents, but can also damage the hearing of those taking part. Even tennis playing, church bell ringing and other religious activities can lead to noise complaints.

Some types of indoor concerts and discotheques can produce extremely high sound pressure levels. Associated noise problems outdoors result from customers arriving and leaving. Outdoor concerts, fireworks and various types of festivals can also produce intense noise. The general problem of access to festivals and leisure activity sites often adds to road traffic noise problems. Severe hearing impairment may also arise from intense sound produced as music in headphones or from children's toys.

2.3. The Complexity of Noise and Its Practical Implications

2.3.1. The problem

One must consider many different characteristics to describe environmental noises completely. We can consider the sound pressure level of the noise and how this level varies over a variety of periods, ranging from minutes or seconds to seasonal variations over several months. Where sound pressure levels vary quite substantially and rapidly, such as in the case of low-level jet aircraft, one might also want to consider the rate of change of sound pressure levels (Berry 1995; Kerry et al. 1997). At the same time, the frequency content of each noise will also determine its effect on people, as will the number of events when there are relatively small numbers of discrete noisy events. Combinations of these characteristics determine how each type of environmental noise affects people. These effects may be annoyance, sleep disturbance, speech interference, increased stress, hearing impairment or other health-related effects.

Thus, in total there is a very complex multidimensional relationship between the various characteristics of the environmental noise and the effects it has on people. Unfortunately, we do not completely understand all of the complex links between noise characteristics and the resulting effects on people. Thus, current practice is to reduce the assessment of environmental noise to a small number of quite simple quantities that are known to be reasonably well related to the effects of noise on people (LAeq,T for continuing sounds and LAmax or SEL where there are a small number of distinct noise events). These simple measures have the distinct advantage that they are relatively easy and inexpensive to obtain and hence are more likely to be widely adopted. On the other hand, they may ignore some details of the noise characteristics that relate to particular types of effects on people.

2.3.2. Time variation

There is evidence that the pattern of noise variation with time relates to annoyance (Berglund et al. 1976). It has been suggested that the equal-energy principle is a simple concept for obtaining a measure representative of the annoyance of a number of noise events. For example, the LAeq,T of the noise from a busy road may be a good indicator of the annoyance this noise may

cause for nearby residents. However, such a measure may not be very useful for predicting the disturbance to sleep of a small number of very noisy aircraft fly-overs. The disturbance caused by small numbers of such discrete events is usually better related to maximum sound pressure levels and the number of events.

While using LAeq,T measures is the generally accepted approach, it is still important to appreciate the limitations and errors that may occur. For example, some years ago measures that assessed the variation of sound pressure levels with time were popular. Subsequently, these have been shown not to improve predictions of annoyance with road traffic noise (Bradley 1978). However, it is possible that time variations may contribute to explaining the very different amounts of annoyance caused by equal LAeq,T levels of road-traffic noise, train noise and aircraft noise (*cf*. Miedema & Vos 1998).

More regular variations of sound pressure levels with time have been found to increase the annoying aspects of the noise. For example, noises that vary periodically to create a throbbing or pulsing sensation can be more disturbing than continuous noise (Bradley 1994b). Research suggests that variations at about 4 per second are most disturbing (Zwicker 1989). Noises with very rapid onsets could also be more disturbing than indicated by their LAeq,T (Berry 1995; Kerry et al. 1997).

LAeq,T values can be calculated for various time periods and it is very important to specify this period. It is quite common to calculate LAeq,T values separately for day- and night-time periods. In combining day and night LAeq,T values it is usually assumed that people will be more sensitive to noise during the night-time period. A weighting is thus normally added to night-time LAeq,T values when calculating a combined measure for a 24 hour period. For example, day-night sound pressure measures commonly include a 10 dB night-time weighting. Other night-time weightings have been proposed, but it has been suggested that it is not possible to determine precisely an optimum value for night-time weightings from annoyance survey responses, because of the large variability in responses within groups of people (Fields 1986; see also Berglund & Lindvall 1995). Night-time weightings are intended to indicate the expected increased sensitivity to annoyance at night and do not protect people from sleep disturbance.

2.3.3. Frequency content and loudness

Noise can also be characterized by its frequency content. This can be assessed by various types of frequency analysis to determine the relative contributions of the frequency components to the total noise. The combined effects of the different frequencies on people, perceived as noise, can be approximated by simple frequency weightings. The A-weighting is now widely used to obtain an approximate, single-number rating of the combined effects of the various frequencies. The A-weighting response is a simplification of an equal-loudness contour. There is a family of these equal-loudness contours (ISO 1987a) that describe the frequency response of the hearing system for a wide range of frequencies and sound pressure levels. These equal-loudness contours can be used to determine the perceived loudness of a single frequency sound. More complicated procedures have been derived to estimate the perceived loudness of complex sounds (ISO 1975). These methods involve determining the level of the sound in critical bands and the mutual masking of these bands.

Many studies have compared the accuracy of predictions based on A-weighted levels with those based on other frequency weightings, as well as more complex measures such as loudness levels and perceived noise levels (see also Berglund & Lindvall 1995). The comparisons depend on the particular effect that is being predicted, but generally the correlation between the more complex measures and subjective scales are a little stronger. A-weighted measures have been particularly criticized as not being accurate indicators of the disturbing effects of noises with strong low-frequency components (Kjellberg et al. 1984; Persson & Björkman 1988; Broner & Leventhall 1993; Goldstein 1994). However, these differences in prediction accuracy are usually smaller than the variability of responses among groups of people (Fields 1986; see also Berglund & Lindvall 1995). Thus, in practical situations the limitations of A-weighted measures may not be so important.

In addition to equal-loudness contours, equal-noisiness contours have also been developed for calculating perceived noise levels (PNL) (Kryter 1959; Kryter 1994; see also section 2.7.2). Critics have pointed out that in addition to equal-loudness and equal-noisiness contours, we could have many other families of equal-sensation contours corresponding to other attributes of the noises (Molino 1974). There seems to be no limit to the possible complexity and number of such measures.

2.3.4. Influence of ambient noise level

A number of studies have suggested that the annoyance effect of a particular noise would depend on how much that noise exceeded the level of ambient noise. This has been shown to be true for noises that are relatively constant in level (Bradley 1993), but has not been consistently found for time-varying noises such as aircraft noise (Gjestland et al. 1990; Fields 1998). Because at some time during an aircraft fly-over the noise almost always exceeds the ambient level, responses to this type of noise are less likely to be influenced by the level of the ambient noise.

2.3.5. Types of noise

A number of studies have concluded that equal levels of different noise types lead to different annoyance (Hall et al. 1981; Griffiths 1983; Miedema 1993; Bradley 1994a; Miedema & Vos 1998). For example, equal LAeq,T levels of aircraft noise and road traffic noise will not lead to the same mean annoyance in groups of people exposed to these noises. This may indicate that the LAeq,T measure is not a completely satisfactory description of these noises and perhaps does not completely reflect the characteristics of these noises that lead to annoyance. Alternatively, the differences may be attributed to various other factors that are not part of the noise characteristics (e.g. Flindell & Stallen 1999). For example, it has been said that aircraft noise is more disturbing, because of the associated fear of aircraft crashing on people's homes (cf. Berglund & Lindvall 1995).

2.3.6. Individual differences

Finally, there is the problem of individual response differences. Different people will respond quite differently to the same noise stimulus (Job 1988). These individual differences can be

quite large and it is often most useful to consider the average response of groups of people exposed to the same sound pressure levels. In annoyance studies the percentage of highly annoyed individuals is usually considered, because it correlates better with measured sound pressure levels. Individual differences also exist for susceptibility to hearing impairment (e.g. Katz 1994).

2.3.7. Recommendations

In many cases we do not have specific, accurate measures of how annoying sound will be and must rely on the simpler quantities. As a result, current practice is to assume that the equal energy principle is approximately valid for most types of noise, and that a simple LAeq,T type measure will indicate reasonably well the expected effects of the noise. Where the noise consists of a small number of discrete events, the A-weighted maximum level (LAmax) will be a better indicator of the disturbance to sleep and other activities. However, in most cases the A-weighted sound exposure level (SEL) will provide a more consistent measure of such single-noise events, because it is based on an integration over the complete noise event.

2.4. Measurement Issues

2.4.1. Measurement objectives

The details of noise measurements must be planned to meet some relevant objective or purpose. Some typical objectives would include:

a. Investigating complaints.
b. Assessing the number of persons exposed.
c. Compliance with regulations.
d. Land use planning and environmental impact assessments.
e. Evaluation of remedial measures.
f. Calibration and validation of predictions.
g. Research surveys.
h. Trend monitoring.

The sampling procedure, measurement location, type of measurements and the choice of equipment should be in accord with the objective of the measurements.

2.4.2. Instrumentation

The most critical component of a sound pressure meter is the microphone, because it is difficult to produce microphones with the same precision as the other, electronic components of a pressure meter. In contrast, it is usually not difficult to produce the electronic components of a microphone with the desired sensitivity and frequency-response characteristics. Lower quality microphones will usually be less sensitive and so cannot measure very low sound pressure levels. They may also not be able to accurately measure very high sound pressure levels found closer to loud noise sources. Lower quality microphones will also have less well-defined frequency-response characteristics. Such lower quality microphones may be acceptable for survey type

measurements of overall A-weighted levels, but would not be preferred for more precise measurements, including detailed frequency analysis of the sounds.

Sound pressure meters will usually include both A- and C-weighting frequency-response curves. The uses of these frequency weightings were discussed above. They may also include a linear weighting. Linear weightings are not defined in standards and may in practice be limited by the response of the particular microphone being used. Instead of, or in addition to, frequency-response weightings, more complex sound pressure meters can also include sets of standard bandpass filters, to permit frequency analysis of sounds. For acoustical measurements, octave and one-third octave bandwidth filters are widely used with centre frequencies defined in standards (ISO 1975b).

The instantaneous sound pressures are integrated with some time constant to provide sound pressure levels. As mentioned above most meters will include both *Fast*- and *Slow*-response times. *Fast*-response corresponds to a time constant of 0.125 s and is intended to approximate the time constant of the human hearing system. *Slow*-response corresponds to a time constant of 1 s and is an old concept intended to make it easier to obtain an approximate average value of fluctuating levels from simple meter readings.

Standards (IEC 1979) classify sound pressure meters as type 1 or type 2. Type 2 meters are adequate for broad band A-weighted level measurements, where extreme precision is not required and where very low sound pressure levels are not to be measured. Type 1 meters are usually much more expensive and should be used where more precise results are needed, or in cases where frequency analysis is required.

Many modern sound pressure meters can integrate sound pressure levels over some specified time period, or may include very sophisticated digital processing capabilities. Integrating meters make it possible to directly obtain accurate measures of LAeq,T values over a user-specified time interval, T. By including small computers in some sound pressure meters, quite complex calculations can be performed on the measured levels and many such results can be stored for later read out. For example, some meters can determine the statistical distribution of sound pressure levels over some period, in addition to the simple LAeq,T value. Recently, hand-held meters that perform loudness calculations in real time have become available. Continuing rapid developments in instrumentation capabilities are to be expected.

2.4.3. Measurement locations

Where local regulations do not specify otherwise, measurements of environmental noise are usually best made close to the point of reception of the noise. For example, if there is concern about residents exposed to road traffic noise it is better to measure close to the location of the residents, rather than close to the road. If environmental noises are measured close to the source, one must then estimate the effect of sound propagation to the point of reception. Sound propagation can be quite complicated and estimates of sound pressure levels at some distance from the source will inevitably introduce further errors into the measured sound pressure levels. These errors can be avoided by measuring at locations close to the point of reception.

Measurement locations should normally be selected so that there is a clear view of the sound source and so that the propagation of the sound to the microphone is not shielded or blocked by structures that would reduce the incident sound pressure levels. For example, measurements of aircraft noise should be made on the side of the building directly exposed to the noise. The position of the measuring microphone relative to building façades or other sound-reflective surfaces is also important and will significantly influence measured sound pressure levels (ISO 1978). If the measuring microphone is located more than several meters from reflecting surfaces, it will provide an unbiased indication of the incident sound pressure level. At the other extreme, when a measuring microphone is mounted on a sound-reflecting surface, such as a building façade, sound pressure levels will be increased by 6 dB, because the direct and reflected sound will coincide. Some standards recommend a position 2 m from the façade and an associated 3 dB correction (ISO 1978; ASTM 1992). The effect of façade reflections must be accounted for to represent the true level of the incident sound. Thus, while locating the measuring microphone close to the point of reception is desirable, it leads to some other issues that must be considered to accurately interpret measurement results. Where exposures are measured indoors, it is necessary to measure at several positions to characterize the average sound pressure level in a room. In other situations, it may be necessary to measure at the position of the exposed person.

2.4.4. Sampling

Many environmental noises vary over time, such as for different times of day or from season to season. For example, road traffic noise may be considerably louder during some hours of the day but much quieter at night. Aircraft noise may vary with the season due to different numbers of aircraft operations. Although permanent noise monitoring systems are becoming common around large airports, it is usually not possible to measure sound pressure levels continuously over a long enough period of time to completely define the environmental noise exposure. In practice, measurements usually only sample some part of the total exposure. Such sampling will introduce uncertainties in the estimates of the total noise exposure.

Traffic noise studies have identified various sampling schemes that can introduce errors of 2-3 dB in estimates of daytime LAeq,T values and even larger errors in night-time sound pressure levels (Vaskor et al. 1979). These errors relate to the statistical distributions of sound pressure levels over time (Bradley et al. 1979). Thus, the sampling errors associated with road traffic noise may be quite different from those associated with other noise, because of the quite different variations of sound pressure levels over time. It is also difficult to give general estimates of sampling errors due to seasonal variations. When making environmental noise measurements it is important that the measurement sample is representative of all of the variations in the noise in question, including variations of the source and variations in sound propagation, such as due to varying atmospheric conditions.

2.4.5. Calibration and quality assurance

Sound pressure meters can be calibrated using small calibrated sound sources. These devices are placed on the measurement microphone and produce a known sound pressure level with a specified accuracy. Such calibrations should be made at least daily, and more often if there is

some possibility that handling of the sound pressure meter may have modified its sensitivity. It is also important to have a complete quality assurance plan. This should require annual calibration of all noise measuring equipment to traceable standards and should clearly specify correct measurement and operating procedures (ISO 1994).

2.5. Source Characteristics and Sound Propagation

To make a correct assessment of noise it is important to have some appreciation of the characteristics of environmental noise sources and of how sound propagates from them. One should consider the directionality of noise sources, the variability with time and the frequency content. If these are in some way unusual, the noise may be more disturbing than expected. The most common types of environmental noise sources are directional and include: road-traffic noise, aircraft noise, train noise, industrial noise and outdoor entertainment facilities (*cf*. section 2.2). All of these types of environmental noise are produced by multiple sources, which in many cases are moving. Thus, the characteristics of individual sources, as well as the characteristics of the combined sources, must be considered.

For example, we can consider the radiation of sound from individual vehicles, as well as from a line of vehicles on a particular road. Sound from an ideal point source (i.e. non-directional source) will spread out spherically and sound pressure levels would decrease 6 dB for each doubling of distance from the source. However, for a line of such sources, or for an integration over the complete pass-by of an individual moving source, the combined effect leads to sound that spreads cylindrically and to sound pressure levels that decrease at 3 dB per doubling of distance. Thus, there are distinct differences between the propagation of sound from an ideal point source and from moving sources. In practice one cannot adequately assess the noise from a fixed source with measurements at a single location; it is essential to measure in a number of directions from the source. If the single source is moving, it is necessary to measure over a complete pass-by, to account for sound variation with direction and time.

In most real situations this simple behaviour is considerably modified by reflections from the ground and from other nearby surfaces. One expects that when sound propagates over loose ground, such as grass, that some sound energy will be absorbed and sound pressure levels will actually decrease more rapidly with distance from the source. Although this is approximately true, the propagation of sound between sources and receivers close to the ground is much more complicated than this. The combination of direct and ground-reflected sound can combine in a complex manner which can lead to strong cancellations at some frequencies and not at others (Embleton & Piercy 1976). Even at quite short source-to-receiver distances, these complex interference effects can significantly modify the propagating sound. At larger distances (approximately 100 m or more), the propagation of sound will also be significantly affected by various atmospheric conditions. Temperature and wind gradients as well as atmospheric turbulence can have large effects on more distant sound pressure levels (Daigle et al. 1986). Temperature and wind gradients can cause propagating sound to curve either upwards or downwards, creating either areas of increased or decreased sound pressure levels at points quite distant from the source. Atmospheric turbulence can randomize sound so that the interference effects resulting from combinations of sound paths are reduced. Higher frequency sound is absorbed by air depending on the exact temperature and relative humidity of the air (Crocker &

Price 1975; Ford 1987). Because there are many complex effects, it is not usually possible to accurately predict sound pressure levels at large distances from a source.

Using barriers or screens to block the direct path from the source to the receiver can reduce the propagation of sound. The attenuating effects of the screen are limited by sound energy that diffracts or bends around the screen. Screens are more effective at higher frequencies and when placed either close to the sound source or the receiver; they are less effective when placed far from the receiver. Although higher screens are better, in practice it is difficult to achieve more than about a 10 dB reduction. There should be no gaps in the screen and it must have an adequate mass per unit area. A long building can be an effective screen, but gaps between buildings will reduce the sound attenuation.

In some cases, it may be desirable to estimate environmental sound pressure levels using mathematical models implemented as computer programmes (House 1987). Such computer programmes must first model the characteristics of the source and then estimate the propagation of the sound from the source to some receiver point. Although such prediction schemes have several advantages, there will be some uncertainty as to the accuracy of the predicted sound pressure levels. Such models are particularly useful for road traffic noise and aircraft noise, because it is possible to create data bases of information describing particular sources. For more varied types of noise, such as industrial noise, it would be necessary to first characterize the noise sources. The models then sum up the effects of multiple sources and calculate how the sound will propagate to receiver points. Techniques for estimating sound propagation are improving and the accuracy of these models is also expected to improve. These models can be particularly useful for estimating the combined effect of a large number of sources over an extended period of time. For example, aircraft noise prediction models are typically used to predict average yearly noise exposures, based on the combination of aircraft events over a complete year. Such models can be applied to predict sound pressure level contours around airports for these average yearly conditions. This is of course much less expensive than measuring at many locations over a complete one year-period. However, such models can be quite complex, and require skilled users and accurate data bases. Because environmental noise prediction models are still developing, it is advisable to confirm predictions with measurements.

2.6. Sound transmission Into and Within Buildings

Sources of environmental noise are usually located outdoors; for example, road traffic, aircraft or trains. However, people exposed to these noises are often indoors, inside their home or some other building. It is, therefore, important to understand how environmental noises are transmitted into buildings. Most of the same fundamentals discussed earlier apply to airborne sound propagation between homes in multifamily dwellings, via common walls and floors. However, within buildings we can also consider impact sound sources, such as footsteps, as well as airborne sounds.

The amount of incident sound that is transmitted through a building façade is measured in terms of the sound reduction index. The sound reduction index, or transmission loss, is defined as 10 times the logarithm of the ratio of incident-to-transmitted sound power, and it describes in decibels how much the incident sound is reduced on passing through a particular panel. This

index of constructions usually increases with the frequency of the incident sound and with the mass of the construction (Kremer 1950). Thus, heavier or more massive constructions tend to have higher sound reductions. When it is not possible to achieve the desired transmission loss by increasing the mass of a panel, increased sound reduction can be achieved by a double panel construction. The two layers should be isolated with respect to vibrations and there should be sound absorbing material in the cavity. Such double panel constructions can provide much greater sound reduction than a single panel. Because sound reduction is also greater at higher frequencies most problems occur at lower frequencies, where most environmental noise sources produce relatively high sound pressure levels.

The sound reduction of buildings can be measured in standard laboratory tests, where the test panel is constructed in an opening between two reverberant test chambers (ISO 1995; ASTM 1997). In these tests sound fields are quite diffuse in both test chambers and the sound reduction index is calculated as the difference between the average sound pressure levels in the two rooms, plus a correction involving the area of the test panel and the total sound absorption in the receiving room. The sound reduction of a complete building façade can also be measured in the field using either natural environmental noises or test signals from loudspeakers (ISO 1978; ASTM 1992). In either case the noise, as transmitted through the façade, must be greater in level than other sounds in the receiving room. For this outdoor-to-indoor sound propagation case, the measured sound reduction index will also depend on the angle of incidence of the outdoor sound, as well as the position of the outdoor measuring microphone relative to the building façade. Corrections of up to 6 dB must be made to the sound pressure level measured outdoors, to account for the effect of reflections from the façade (see also section 2.4.3).

The sound reduction of most real building façades is determined by a combination of several different elements. For example, a wall might include windows, doors or some other type of element. If the sound reduction index values of each element are known, the values for the combined construction can be calculated from the area-weighted sums of the sound energy transmitted through each separate element. Although parts of the building façade, such as massive wall constructions, can be very effective barriers to sound, the sound reduction index of the complete façade is often greatly reduced by less effective elements such as windows, doors or ventilation openings. Completely open windows as such would have a sound reduction index of 0 dB. If window openings makes up 10% of the area of a wall, the sound reduction index of the combined wall and open window could not exceed 10 dB. Thus it is not enough to specify effective sound reducing façade constructions, without also solving the problem of adequate ventilation that does not compromise the sound transmission reduction by the building façade.

Sound reduction index values are measured at different frequencies and from these, single number ratings are determined. Most common are the ISO weighted sound reduction index (ISO 1996) and the equivalent ASTM sound transmission class (ASTM 1994a). However, in their original form these single number ratings are only appropriate for typical indoor noises that usually do not have strong low frequency components. Thus, they are usually not appropriate single number ratings of the ability of a building façade to block typical environmental noises. More recent additions to the ISO procedure have included source spectrum corrections intended to correct approximately for other types of sources (ISO 1996). Alternatively, the ASTM-Outdoor-Indoor Transmission Class rating calculates the A-weighted level reduction to a

standard environmental noise source spectrum (ASTM 1994b). Within buildings the impact sound insulation index can be measured with a standard impact source and determined according to ISO and ASTM standards (ISO 1998; ASTM 1994c 1996)

2.7. More Specialized Noise Measures

2.7.1. Loudness and perceived noise levels

There are procedures to accurately rate the loudness of complex sounds (Zwicker 1960; Stevens 1972; ISO 1975a). These usually start from a 1/3 octave spectrum of the noise. The combination of the loudness contributions of each 1/3 octave band with estimates of mutual masking effects, leads to a single overall loudness rating in sones. A similar system for rating the noisiness of sounds has also been developed (Kryter 1994). Again a 1/3 octave spectrum of the noise is required and the 1/3 octave noise levels are compared with a set of equal-noisiness contours. The individual 1/3 octave band noisiness estimates are combined to give an overall perceived noise level (PNL) that is intended to accurately estimate subjective evaluations of the same sound. The PNL metric was initially developed to rate jet aircraft noise.

PNL values will vary with time, for example when an aircraft flies by a measuring point. The effective perceived noise level measure (EPNL) is derived from PNL values and is intended to provide a complete rating of an aircraft fly-over. EPNL values add both a duration correction and a tone correction to PNL values. The duration correction ensures that longer duration events are rated as more disturbing. Similarly, noise spectra that seem to have prominent tonal components are rated as more disturbing by the tone-correction procedure. There is some evidence that these tone corrections are not always successful in improving predictions of adverse responses to noise events (Scharf & Hellman 1980). EPNL values are used in the certification testing of new aircraft. These more precise measures ensure that the noise from new aircraft is rated as accurately as possible.

2.7.2. Aviation noise measures

There are many measures for evaluating the long-term average sound pressure levels from aircraft near airports (Ford 1987; House 1987). They include different frequency weightings, different summations of levels and numbers of events, as well as different time-of-day weightings. Most measures are based on either A-weighted or PNL-weighted sound pressure levels. Because of the many other large uncertainties in predicting community response to aircraft noise, there seems little justification for using the more complex PNL-weighted sound pressure levels and there is a trend to change to A-weighted measures.

Most aviation noise measures are based on an equal energy approach and hence they sum up the total energy of a number of aircraft fly-overs. However, some older measures were based on different combinations of the level of each event and the number of events. These types of measures are gradually being replaced by measures based on the equal energy hypothesis such as LAeq,T values. There is also a range of time-of-day weightings incorporated into current aircraft noise measures. Night-time weightings of 6–12 dB are currently in use. Some countries also include an intermediate evening weighting.

The day-night sound pressure level L_{dn} (von Gierke 1975; Ford 1987) is an LAeq,T based measure with a 10 dB night-time weighting. It is based on A-weighted sound pressure levels and the equal energy principle. The noise exposure forecast (NEF) (Bishop & Horonjeff 1967) is based on the EPNL values of individual aircraft events and includes a 12 dB night-time weighting. It sums multiple events on an equal energy basis. However, the Australian variation of the NEF measure has a 6 dB evening weighting and a 6 dB night-time weighting (Bullen & Hede 1983). The German airport noise equivalent level (LEQ(FLG)) is based on A-weighted levels, but does not follow the equal energy principle.

The weighted equivalent continuous perceived noise level (WECPNL) measure (Ford 1987) proposed by ICAO is based on the equal energy principle and maximum PNL values of aircraft fly-overs. However, in Japan an approximation to this measure is used and is based on maximum A-weighted levels. The noise and number index (NNI), formerly used in the United Kingdom, was derived from maximum PNL values but was not based on the equal energy principle. An approximation to the original version of the NNI has been used in Switzerland and is based on maximum A-weighted levels of aircraft fly-overs, but its use will soon be discontinued. Changes in these measures are slow because their use is often specified in national legislation. However, several countries have changed to measures that are based on the equal energy principle and A-weighted sound pressure levels.

2.7.3. Impulsive noise measures

Impulsive sounds, such as gun shots, hammer blows, explosions of fireworks or other blasts, are sounds that significantly exceed the background sound pressure level for a very short duration. Typically each impulse lasts less than one second. Measurements with the meter set to 'Fast' response (section 2.1.1) do not accurately represent impulsive sounds. Therefore the meter response time must be shorter to measure such impulse type sounds. C-weighted levels have been found useful for ratings of gun shots (ISO 1987). Currently no mathematical description exists which unequivocally defines impulsive sounds, nor is there a universally accepted procedure for rating the additional annoyance of impulsive sounds (HCN 1997). Future versions of ISO Standard 1996 (present standard in ISO 1987b) are planned to improve this situation.

2.7.4. Measures of speech intelligibility

The intelligibility of speech depends primarily on the speech-to-noise ratio. If the level of the speech sounds are 15 dB or more above the level of the ambient noise, the speech intelligibility at 1 m distance will be close to 100% (Houtgast 1981; Bradley 1986b). This can be most simply rated in terms of the speech-to-noise ratio of the A-weighted speech and noise levels. Alternatively, the speech intelligibility index (formerly the articulation index) can be used if octave or 1/3 octave band spectra of the speech and noise are available (ANSI 1997).

When indoors, speech intelligibility also depends on the acoustical properties of the space. The acoustical properties of spaces have for many years been rated in terms of reverberation times. The reverberation time is approximately the time it takes for a sound in a room to decrease to inaudibility after the source has been stopped. Optimum reverberation times for speech have

been specified as a function of the size of the room. In large rooms, such as lecture halls and theaters, a reverberation time for speech of about 1 s is recommended. In smaller rooms such as classrooms, the recommended value for speech is about 0.6 s (Bradley 1986b,c). More modern measures of room acoustics have been found to be better correlates of speech intelligibility, and some combine an assessment of both the speech/noise ratio and room acoustics (Bradley 1986a,c). The most widely known is the speech transmission index (STI) (Houtgast & Steeneken 1983), or the abbreviated version of this measure referred to as RASTI (Houtgast & Steeneken 1985; IEC 1988). In smaller rooms, such as school classrooms, the conventional approach of requiring adequately low ambient noise levels, as well as some optimum reverberation time, is probably adequate to ensure good speech intelligibility (Bradley 1986b). In larger rooms and other more specialized situations, use of the more modern measures may be helpful.

2.7.5. Indoor noise ratings

The simplest procedure for rating levels of indoor noise is to measure them in terms of integrated A-weighted sound pressure levels, as measured by LAeq,T. As discussed earlier, this approach has been criticized as not being the most accurate rating of the negative effects of various types of noises, and is thought to be particularly inadequate when there are strong low-frequency components. Several more complex rating schemes are available based on octave band measurements of indoor noises. In Europe the noise rating system (Burns 1968), and in North America the noise criterion (Beranek 1971), both include sets of equal-disturbance type contours. Measured octave band sound pressure levels are compared with these contours and an overall noise rating is determined. More recently, two new schemes have been proposed: the balanced noise criterion procedure (Beranek 1989) and the room criterion system (Blazier 1998). These schemes are based on a wider range of octave bands extending from 16–8 000 Hz. They provide both a numerical and a letter rating of the noise. The numerical part indicates the level of the central frequencies important for speech communication and the letter indicates whether the quality of the sound is predominantly low-, medium- or high-frequency in nature. Extensive comparisons of these room noise rating procedures have yet to be performed. Because the newer measures include a wider range of frequencies, they can better assess a wider range of noise problems.

2.8. Summary

Where there are no clear reasons for using other measures, it is recommended that LAeq,T be used to evaluate more-or-less continuous environmental noises. LAeq,T should also be used to assess ongoing noises that may be composed of individual events with randomly varying sound pressure levels. Where the noise is principally composed of a small number of discrete events the additional use of LAmax or SEL is recommended. As pointed out in this chapter, there are definite limitations to these simple measures, but there are also many practical advantages, including economy and the benefits of a standardized approach.

The sound pressure level measurements should include all variations over time to provide results that best represent the noise in question. This would include variations in both the source and in propagation of the noise from the source to the receiver. Measurements should normally be

made close to typical points of reception. The accuracy of the measurements and the details of the measurement procedure must be adapted to the type of noise and to other details of the noise exposure. Assessment of speech intelligibility, aviation noise or impulse noise may require the use of more specialized methods. Where the exposed people are indoors and noise measurements are made outdoors, the sound attenuating properties of the building façade must also be measured or estimated.

3. Adverse Health Effects Of Noise

3.1. Introduction

The perception of sounds in day-to-day life is of major importance for human well-being. Communication through speech, sounds from playing children, music, natural sounds in parklands, parks and gardens are all examples of sounds essential for satisfaction in every day life. Conversely, this document is related to the adverse effects of sound (noise). According to the International Programme on Chemical Safety (WHO 1994), an adverse effect of noise is defined as a change in the morphology and physiology of an organism that results in impairment of functional capacity, or an impairment of capacity to compensate for additional stress, or increases the susceptibility of an organism to the harmful effects of other environmental influences. This definition includes any temporary or long-term lowering of the physical, psychological or social functioning of humans or human organs. The health significance of noise pollution is given in this chapter under separate headings, according to the specific effects: noise-induced hearing impairment; interference with speech communication; disturbance of rest and sleep; psychophysiological, mental-health and performance effects; effects on residential behaviour and annoyance; as well as interference with intended activities. This chapter also considers vulnerable groups and the combined effects of sounds from different sources. Conclusions based on the details given in this chapter are given in Chapter 4 as they relate to guideline values.

3.2. Noise-Induced Hearing Impairment

Hearing impairment is typically defined as an increase in the threshold of hearing. It is assessed by threshold audiometry. Hearing handicap is the disadvantage imposed by hearing impairment sufficient to affect one's personal efficiency in the activities of daily living. It is usually expressed in terms of understanding conventional speech in common levels of background noise (ISO 1990). Worldwide, noise-induced hearing impairment is the most prevalent irreversible occupational hazard. In the developing countries, not only occupational noise, but also environmental noise is an increasing risk factor for hearing impairment. In 1995, at the World Health Assembly, it was estimated that there are 120 million persons with disabling hearing difficulties worldwide (Smith 1998). It has been shown that men and women are equally at risk of noise-induced hearing impairment (ISO 1990; Berglund & Lindvall 1995).

Apart from noise-induced hearing impairment, hearing damage in populations is also caused by certain diseases; some industrial chemicals; ototoxic drugs; blows to the head; accidents; and hereditary origins. Deterioration of hearing capability is also associated with the aging process *per se* (presbyacusis). Present knowledge of the physiological effects of noise on the auditory system is based primarily on laboratory studies on animals. After noise exposure, the first morphological changes are usually found in the inner and outer hair cells of the cochlea, where the stereocilia become fused and bent. After more prolonged exposure, the outer and inner hair cells related to transmission of high-frequency sounds are missing. See Berglund & Lindvall (1995) for further discussion.

The ISO Standard 1999 (ISO 1990) gives a method for calculating noise-induced hearing

impairment in populations exposed to all types of noise (continuous, intermittent, impulse) during working hours. Noise exposure is characterized by LAeq over 8 hours (LAeq,8h). In the Standard, the relationships between LAeq,8h and noise-induced hearing impairment are given for frequencies of 500–6 000 Hz, and for exposure times of up to 40 years. These relations show that noise-induced hearing impairment occurs predominantly in the high-frequency range of 3 000–6 000 Hz, the effect being largest at 4 000 Hz. With increasing LAeq,8h and increasing exposure time, noise-induced hearing impairment also occurs at 2 000 Hz. But at LAeq,8h levels of 75 dBA and lower, even prolonged occupational noise exposure will not result in noise-induced hearing impairment (ISO 1990). This value is equal to that specified in 1980 by the World Health Organization (WHO 1980a).

The ISO Standard 1999 (ISO 1990) specifies hearing impairment in statistical terms (median values, and percentile fractions between 0.05 and 0.95). The extent of noise-induced hearing impairment in populations exposed to occupational noise depends on the value of LAeq,8h and the number of years of noise exposure. However, for high LAeq,8h values, individual susceptibility seems to have a considerable effect on the rate of progression of hearing impairment. For daily exposures of 8–16 h, noise-induced hearing impairment can be reasonably well estimated from LAeq,8h extrapolated to the longer exposure times (Axelsson et al. 1986). In this adaptation of LAeq,8h for daily exposures other than 8 hours, the equal energy principle is assumed to be applicable. For example, the hearing impairment due to a 16 h daily exposure is equivalent to that at LAeq,8h plus 3 dB (LAeq,16h = LAeq,8h + $10*\log_{10}$ (16/8) = LAeq,8h + 3 dB. For a 24 h exposure, LAeq,24h = LAeq,8h + $10*\log_{10}$ (24/8) = LAeq,8h + 5 dB).

Since the calculation method specified in the ISO Standard 1999 (ISO 1990) is the only universally adopted method for estimating occupational noise-induced hearing impairment, attempts have been made to assess whether the method is also applicable to hearing impairment due to environmental noise, including leisure-time noise. There is ample evidence that shooting noise, with LAeq,24h values of up to 80 dB, induces the same hearing impairment as an equivalent occupational noise exposure (Smoorenburg 1998). Moreover, noise-induced hearing impairment studies from motorbikes are also in agreement with results from ISO Standard 1999 (ISO 1990). Hearing impairment in young adults and children 12 years and older has been assessed by LAeq on a 24 h time basis, for a variety of environmental and leisure-time exposure patterns (e.g. Passchier-Vermeer 1993; HCN 1994). These include pop music in discotheques and concerts (Babisch & Ising 1989; ISO 1990); pop music through headphones (Ising et al. 1994; Struwe et al. 1996; Passchier-Vermeer et al. 1998); music played by brass bands and symphony orchestras (van Hees 1992). The results are in agreement with values predicted by the ISO Standard 1999 method on the basis of adjusted time.

In the publications cited above, exposure to noise with known characteristics, such as duration and level, was related to hearing impairment. In addition to these publications, there is also an extensive literature showing hearing impairment in populations exposed to specific types of non-occupational noise, although these exposures are not well characterized. These noises originate from shooting, motorcycling, snowmobile driving, playing in arcades, listening to music at concerts and through headphones, using noisy toys, and fireworks (e.g. Brookhouser et al. 1992; see also Berglund & Lindvall 1995). Although the characteristics of these exposures are to a certain extent unknown, the details in the publications suggest that LAeq,24h values of these

exposures exceed 70 dB.

In contrast, epidemiological studies failed to show hearing damage in populations exposed to an LAeq,24h of less than 70 dB (Lindemann et al. 1987). The data imply that even a lifetime exposure to environmental and leisure-time noise with an LAeq,24h <70 dBA would not cause hearing impairment in the large majority of people (over 95%). Overall, the results of many studies strongly suggest that the method from ISO Standard 1999 can also be used to estimate hearing impairment due to environmental and leisure-time noise, in addition to estimating the effects of occupational noise exposure.

Although the evidence suggests that the calculation method from ISO Standard 1999 (ISO 1990) should also be accepted for environmental and leisure time noise exposures, large-scale epidemiological studies of the general population do not exist to support this proposition. Taking into account the limitations of the studies, care should be taken with respect to the following aspects:

a. Data from animal experiments indicate that children may be more vulnerable in acquiring noise-induced hearing impairment than adults.

b. At very high instantaneous sound pressure levels, mechanical damage to the ear may occur (Hanner & Axelsson 1988). Occupational limits are set at peak sound pressure levels of 140 dB (EU 1986a). For adults exposed to environmental and leisure-time noise, this same limit is assumed to be valid. In the case of children, however, taking into account their habits while playing with noisy toys, peak sound pressure levels should never exceed 120 dB.

c. For shooting noise with LAeq,24h over 80 dB, studies on temporary threshold shift suggest the possibility of an increased risk for noise-induced hearing impairment (Smoorenburg 1998).

d. Risk for noise-induced hearing impairment may increase when the noise exposure is combined with exposure to vibrations, the use of ototoxic drugs, or some chemicals (Fechter 1999). In these circumstances, long-term exposure to LAeq,24h of 70 dBA may induce small hearing impairments.

e. It is uncertain whether the relationships between hearing impairment and noise exposure given in ISO Standard 1999 (ISO 1990) are applicable for environmental sounds of short rise time. For example, in the case of military low-altitude flying areas (75–300 m above ground) LAmax values of 110–130 dB occur within seconds after the onset of the sound.

Usually noise-induced hearing impairment is accompanied by an abnormal loudness perception which is known as loudness recruitment (*cf*. Berglund & Lindvall 1995). With a considerable loss of auditory sensitivity, some sounds may be perceived as distorted (paracusis). Another sensory effect that results from noise exposure is tinnitus (ringing in the ears). Commonly, tinnitus is referred to as sounds that are emitted by the inner ear itself (physiological tinnitus).

Tinnitus is a common and often disturbing accompaniment of occupational hearing impairment (Vernon and Moller 1995) and has become a risk for teenagers attending pop concerts and discotheques (Hetu & Fortin 1995; Passchier-Vermeer et al. 1998; Axelsson & Prasher 1999). Noise-induced tinnitus may be temporary, lasting up to 24 hours after exposure, or may have a more permanent character, such as after prolonged occupational noise exposure. Sometimes tinnitus is due to the sound produced by the blood flow through structures in the ear.

The main social consequence of hearing impairment is an inability to understand speech in daily living conditions, which is considered a severe social handicap. Even small values of hearing impairment (10 dB averaged over 2 000 and 4 000 Hz, and over both ears) may have an effect on the understanding of speech. When the hearing impairment exceeds 30 dB (again averaged over 2 000 and 4 000 Hz and both ears) a social hearing handicap is noticeable (*cf*. Katz 1994; Berglund & Lindvall 1995).

In the past, hearing protection has mainly emphasized occupational noise exposures at high values of LAeq,8h, or situations with high impulsive sounds. The near-universal adoption of an LAeq,8h value of 85 dB (or lower) as the limit for unprotected occupational noise exposure, together with requirements for personal hearing protection, has made cases of severe unprotected exposures more rare. This is particularly true for developed countries. However, monitoring of compliance and enforcement action for sound pressure levels just over the limits may be weak, especially in non-industrial environments in developed countries (Franks 1998), as well as in occupational and urban environments in developing countries (Smith 1998). Nevertheless, regulations for occupational noise exposure exist almost worldwide and exposures to occupational noise are to a certain extent under control.

On the other hand, environmental noise exposures due to a number of noisy activities, especially those during leisure-time activities of children and young adults, have scarcely been regulated. Given both the increasing number of noisy activities and the increasing exposure duration, such as loud music in cars and the use of Walkmen and Discmen, regulatory activities in this field are to be encouraged. Dose-response data are lacking for the general population. However, judging from the limited data for study groups (teenagers, young adults and women), and the assumption that time of exposure can be equated with sound energy, the risk for hearing impairment would be negligible for LAeq,24h values of 70 dBA over a lifetime. To avoid hearing impairment, impulse noise exposures should never exceed 140 dB peak sound pressure in adults, and 120 dB peak sound pressure in children.

3.3. Interference with Speech Communication

Noise interference with speech comprehension results in a large number of personal disabilities, handicaps and behavioural changes. Problems with concentration, fatigue, uncertainty and lack of self-confidence, irritation, misunderstandings, decreased working capacity, problems in human relations, and a number of stress reactions have all been identified (Lazarus 1998). Particularly vulnerable to these types of effects are the hearing impaired, the elderly, children in the process of language and reading acquisition, and individuals who are not familiar with the spoken language (e.g., Lazarus 1998). Thus, vulnerable persons constitute a substantial proportion of a country's population.

Most of the acoustical energy of speech is in the frequency range 100–6 000 Hz, with the most important cue-bearing energy being between 300–3 000 Hz. Speech interference is basically a masking process in which simultaneous, interfering noise renders speech incapable of being understood. The higher the level of the masking noise, and the more energy it contains at the most important speech frequencies, the greater will be the percentage of speech sounds that become indiscernible to the listener. Environmental noise may also mask many other acoustical signals important for daily life, such as door bells, telephone signals, alarm clocks, fire alarms and other warning signals, and music (e.g., Edworthy & Adams 1996). The masking effect of interfering noise in speech discrimination is more pronounced for hearing-impaired persons than for persons with normal hearing, particularly if the interfering noise is composed of speech or babble.

As the sound pressure level of an interfering noise increases, people automatically raise their voice to overcome the masking effect upon speech (increase of vocal effort). This imposes an additional strain on the speaker. For example, in quiet surroundings, the speech level at 1 m distance averages 45–50 dBA, but is 30 dBA higher when shouting. However, even if the interfering noise is moderately loud, most of the sentences during ordinary conversation can still be understood fairly well. Nevertheless, the interpretation required for compensating the masking effect of the interfering sounds, and for comprehending what was said, imposes an additional strain on the listener. One contributing factor could be that speech spoken loudly is more difficult to understand than speech spoken softly, when compared at a constant speech-to-noise ratio (*cf*. Berglund & Lindvall 1995).

Speech levels vary between individuals because of factors such as gender and vocal effort. Moreover, outdoor speech levels decrease by about 6 dB for a doubling in the distance between talker and listener. Speech intelligibility in everyday living conditions is influenced by speech level, speech pronunciation, talker-to-listener distance, sound pressure levels, and to some extent other characteristics of interfering noise, as well as room characteristics (e.g. reverberation). Individual capabilities of the listener, such as hearing acuity and the level of attention of the listener, are also important for the intelligibility of speech. Speech communication is affected also by the reverberation characteristics of the room. For example, reverberation times greater than 1 s produce loss in speech discrimination. Longer reverberation times, especially when combined with high background interfering noise, make speech perception more difficult. Even in a quiet environment, a reverberation time below 0.6 s is desirable for adequate speech intelligibility by vulnerable groups. For example, for older hearing-handicapped persons, the optimal reverberation time for speech intelligibility is 0.3–0.5 s (Plomp 1986).

For complete sentence intelligibility in listeners with normal hearing, the signal-to-noise ratio (i.e. the difference between the speech level and the sound pressure level of the interfering noise) should be 15–18 dBA (Lazarus 1990). This implies that in smaller rooms, noise levels above 35 dBA interferes with the intelligibility of speech (Bradley 1985). Earlier recommendations suggested that sound pressure levels as high as 45 dBA would be acceptable (US EPA 1974). With raised voice (increased vocal effort) sentences may be 100% intelligible for noise levels of up to 55 dBA; and sentences spoken with straining vocal effort can be 100% intelligible with noise levels of about 65 dBA. For speech to be intelligible when listening to complicated

messages (at school, listening to foreign languages, telephone conversation), it is recommended that the signal-to-noise ratio should be at least 15 dBA. Thus, with a speech level of 50 dBA, (at 1 m distance this level corresponds to a casual speech level of both women and men), the sound pressure level of interfering noise should not exceed 35 dBA. For vulnerable groups even lower background levels are needed. If it is not possible to meet the strictest criteria for vulnerable persons in sensitive situations (e.g. in classrooms), one should strive for as low background levels as possible.

3.4. Sleep Disturbance

Uninterrupted sleep is known to be a prerequisite for good physiological and mental functioning of healthy persons (Hobson 1989); sleep disturbance, on the other hand, is considered to be a major environmental noise effect. It is estimated that 80-90% of the reported cases of sleep disturbance in noisy environments are for reasons other than noise originating outdoors. For example, sanitary needs; indoor noises from other occupants; worries; illness; and climate (e.g. Reyner & Horne 1995). Our understanding of the impact of noise exposure on sleep stems mainly from experimental research in controlled environments. Field studies conducted with people in their normal living situations are scarce. Most of the more recent field research on sleep disturbance has been conducted for aircraft noise (Fidell et al. 1994 1995a,b 1998; Horne et al. 1994 1995; Maschke et al. 1995 1996; Ollerhead et al. 1992; Passchier-Vermeer 1999). Other field studies have examined the effects of road traffic and railway noise (Griefahn et al. 1996 1998).

The primary sleep disturbance effects are: difficulty in falling asleep (increased sleep latency time); awakenings; and alterations of sleep stages or depth, especially a reduction in the proportion of REM-sleep (REM = rapid eye movement) (Hobson 1989). Other primary physiological effects can also be induced by noise during sleep, including increased blood pressure; increased heart rate; increased finger pulse amplitude; vasoconstriction; changes in respiration; cardiac arrhythmia; and an increase in body movements (cf. Berglund & Lindvall 1995). For each of these physiological effects, both the noise threshold and the noise-response relationships may be different. Different noises may also have different information content and this also could affect physiological threshold and noise-response relationships (Edworthy 1998).

Exposure to night-time noise also induces secondary effects, or so-called after effects. These are effects that can be measured the day following the night-time exposure, while the individual is awake. The secondary effects include reduced perceived sleep quality; increased fatigue; depressed mood or well-being; and decreased performance (Öhrström 1993a; Passchier-Vermeer 1993; Carter 1996; Pearsons et al. 1995; Pearsons 1998).

Long-term effects on psychosocial well-being have also been related to noise exposure during the night (Öhrström 1991). Noise annoyance during the night-time increased the total noise annoyance expressed by people in the following 24 h. Various studies have also shown that people living in areas exposed to night-time noise have an increased use of sedatives or sleeping pills. Other frequently reported behavioural effects of night-time noise include closed bedroom windows and use of personal hearing protection. Sensitive groups include the elderly, shift workers, persons especially vulnerable to physical or mental disorders and other individuals with

sleeping difficulties.

Questionnaire data indicate the importance of night-time noise on the perception of sleep quality. A recent Japanese investigation was conducted for 3 600 women (20–80 years old) living in eight roadside zones with different road traffic noise. The results showed that four measures of perceived sleep quality (difficulty in falling asleep; waking up during sleep; waking up too early; feelings of sleeplessness one or more days a week) correlated significantly with the average traffic volumes during night-time. An in-depth investigation of 19 insomnia cases and their matched controls (age,work) measured outdoor and indoor sound pressure levels during sleep (Kageyama et al. 1997). The study showed that road traffic noise in excess of 30 dB LAeq for nighttime induced sleep disturbance, consistent with the results of Öhrström (1993b).

Meta-analyses of field and laboratory studies have suggested that there is a relationship between the SEL for a single night-time noise event and the percentage of people awakened, or who showed sleep stage changes (e.g. Ollerhead et al. 1992; Passchier-Vermeer 1993; Finegold et al. 1994; Pearsons et al. 1995). All of these studies assumed that the number of awakenings per night for each SEL value is proportional to the number of night-time noise events. However, the results have been criticized for methodological reasons. For example, there were small groups of sleepers; too few original studies; and indoor exposure was estimated from outdoor sound pressure levels (NRC-CNRC 1994; Beersma & Altena 1995; Vallet 1998). The most important result of the meta-analyses is that there is a clear difference in the dose-response curves for laboratory and field studies, and that noise has a lower effect under real-life conditions (Pearsons et al. 1995; Pearsons 1998).

However, this result has been questioned, because the studies were not controlled for such things as the sound insulation of the buildings, and the number of bedrooms with closed windows. Also, only two indicators of sleep disturbance were considered (awakening and sleep stage changes). The meta-analyses thus neglected other important sleep disturbance effects (Öhrström 1993b; Carter et al. 1994a; Carter et al. 1994b; Carter 1996; Kuwano et al. 1998). For example, for road traffic noise, perceived sleep quality is related both to the time needed to fall asleep and the total sleep time (Öhrström & Björkman 1988). Individuals who are more sensitive to noise (as assessed by different questionnaires) report worse sleep quality both in field studies and in laboratory studies.

A further criticism of the meta-analyses is that laboratory experiments have shown that habituation to night-time noise events occurs, and that noise-induced awakening decreases with increasing number of sound exposures per night. This is in contrast to the assumption used in the meta-analyses, that the percentage of awakenings is linearly proportional to the number of night-time noise events. Studies have also shown that the frequency of noise-induced awakenings decreases for at least the first eight consecutive nights. So far, habituation has been shown for awakenings, but not for heart rate and after effects such as perceived sleep quality, mood and performance (Öhrström and Björkman 1988).

Other studies suggest that it is the difference in sound pressure levels between a noise event and background, rather than the absolute sound pressure level of the noise event, that determines the reaction probability. The time interval between two noise events also has an important influence

of the probability of obtaining a response (Griefahn 1977; *cf*. Berglund & Lindvall 1995). Another possible factor is the person's age, with older persons having an increased probability of awakening. However, one field study showed that noise-induced awakenings are independent of age (Reyner & Horne 1995).

For a good sleep, it is believed that indoor sound pressure levels should not exceed approximately 45 dB LAmax more than 10–15 times per night (Vallet & Vernet 1991), and most studies show an increase in the percentage of awakenings at SEL values of 55–60 dBA (Passchier-Vermeer 1993; Finegold et al. 1994; Pearsons et al. 1995). For intermittent events that approximate aircraft noise, with an effective duration of 10–30 s, SEL values of 55–60 dBA correspond to a LAmax value of 45 dB. Ten to 15 of these events during an eight-hour night-time implies an LAeq,8h of 20–25 dB. This is 5–10 dB below the LAeq,8h of 30 dB for continuous night-time noise exposure, and shows that the intermittent character of noise has to be taken into account when setting night-time limits for noise exposure. For example, this can be achieved by considering the number of noise events and the difference between the maximum sound pressure level and the background level of these events.

Special attention should also be given to the following considerations:

a. Noise sources in an environment with a low background noise level. For example, night-traffic in suburban residential areas.

b. Environments where a combination of noise and vibrations are produced. For example, railway noise, heavy duty vehicles.

c. Sources with low-frequency components. Disturbances may occur even though the sound pressure level during exposure is below 30 dBA.

If negative effects on sleep are to be avoided the equivalent sound pressure level should not exceed 30 dBA indoors for continuous noise. If the noise is not continuous, sleep disturbance correlates best with LAmax and effects have been observed at 45 dB or less. This is particularly true if the background level is low. Noise events exceeding 45 dBA should therefore be limited if possible. For sensitive people an even lower limit would be preferred. It should be noted that it should be possible to sleep with a bedroom window slightly open (a reduction from outside to inside of 15 dB). To prevent sleep disturbances, one should thus consider the equivalent sound pressure level and the number and level of sound events. Mitigation targeted to the first part of the night is believed to be effective for the ability to fall asleep.

3.5. Cardiovascular and Physiological Effects

Epidemiological and laboratory studies involving workers exposed to occupational noise, and general populations (including children) living in noisy areas around airports, industries and noisy streets, indicate that noise may have both temporary and permanent impacts on physiological functions in humans. It has been postulated that noise acts as an environmental stressor (for a review see Passchier-Vermeer 1993; Berglund & Lindvall 1995). Acute noise exposures activate the autonomic and hormonal systems, leading to temporary changes such as increased blood pressure, increased heart rate and vasoconstriction. After prolonged exposure, susceptible individuals in the general population may develop permanent effects, such as hypertension and ischaemic heart disease associated with exposures to high sound pressure levels (for a review see Passchier-Vermeer 1993; Berglund & Lindvall 1995). The magnitude and duration of the effects are determined in part by individual characteristics, lifestyle behaviours and environmental conditions. Sounds also evoke reflex responses, particularly when they are unfamiliar and have a sudden onset.

Laboratory experiments and field quasi-experiments show that if noise exposure is temporary, the physiological system usually returns - after the exposure terminates - to a normal (pre-exposure) state within a time in the range of the exposure duration. If the exposure is of sufficient intensity and unpredictability, cardiovascular and hormonal responses may appear, including increases in heart rate and peripheral vascular resistance; changes in blood pressure, blood viscosity and blood lipids; and shifts in electrolyte balance (Mg/Ca) and hormonal levels (epinephrine, norepinephrine, cortisol). The first four effects are of interest because of noise-related coronary heart disease (Ising & Günther 1997). Laboratory and clinical data suggest that noise may significantly elevate gastrointestinal motility in humans.

By far the greatest number of occupational and community noise studies have focused on the possibility that noise may be a risk factor for cardiovascular disease. Many studies in occupational settings have indicated that workers exposed to high levels of industrial noise for 5–30 years have increased blood pressure and statistically significant increases in risk for hypertension, compared to workers in control areas (Passchier-Vermeer 1993). In contrast, only a few studies on environmental noise have shown that populations living in noisy areas around airports and on noisy streets have an increased risk for hypertension. The overall evidence suggests a weak association between long-term environmental noise exposure and hypertension (HCN 1994; Berglund & Lindvall 1995; IEH 1997), and no dose-response relationships could be established.

Recently, an updated summary of available studies for ischaemic heart disease has been presented (Babisch 1998a; Babisch 1998b; Babisch et al. 1999; see also Thompson 1996). The studies reviewed include case-control and cross-sectional designs, as well as three longitudinal studies. However, it has not yet been possible to conduct the most advanced quantitative integrated analysis of the available studies. Relative risks and their confidence intervals could be estimated only for the classes of high noise levels (mostly >65 dBA during daytime) and low levels (mostly <55 dBA during daytime), rather than a range of exposure levels. For methodological reasons identified in the meta-analysis, a cautious interpretation of the results is warranted (Lercher et al. 1998).

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Prospective studies that controlled for confounding factors suggest an increase in ischaemic heart disease when the noise levels exceed 65–70 dB for LAeq (6–22). (For road traffic noise, the difference between LAeq (6-22h) and LAeq,24h usually is of the order of 1.5 dB). When orientation of the bedroom, window opening habits and years of exposure are taken into account, the risk of heart disease is slightly higher (Babisch et al. 1998; Babisch et al. 1999). However, disposition, behavioural and environmental factors were not sufficiently accounted for in the analyses carried out to date. In epidemiological studies the lowest level at which traffic noise had an effect on ischaemic heart disease was 70 dB for LAeq,24h (HCN 1994).

The overall conclusion is that cardiovascular effects are associated with long-term exposure to LAeq,24h values in the range of 65–70 dB or more, for both air- and road-traffic noise. However, the associations are weak and the effect is somewhat stronger for ischaemic heart disease than for hypertension. Nevertheless, such small risks are potentially important because a large number of persons are currently exposed to these noise levels, or are likely to be exposed in the future. Furthermore, only the average risk is considered and sensitive subgroups of the populations have not been sufficiently characterized. For example, a 10% increase in risk factors (a relative risk of 1.1) may imply an increase of up to 200 cases per 100 000 people at risk per year. Other observed psychophysiological effects, such as changes in stress hormones, magnesium levels, immunological indicators, and gastrointestinal disturbances are too inconsistent for conclusions to be drawn about the influence of noise pollution.

3.6. Mental Health Effects

Mental health is defined as the absence of identifiable psychiatric disorders according to current norms (Freeman 1984). Environmental noise is not believed to be a direct cause of mental illness, but it is assumed that it accelerates and intensifies the development of latent mental disorder. Studies on the adverse effects of environmental noise on mental health cover a variety of symptoms, including anxiety; emotional stress; nervous complaints; nausea; headaches; instability; argumentativeness; sexual impotency; changes in mood; increase in social conflicts, as well as general psychiatric disorders such as neurosis, psychosis and hysteria. Large-scale population studies have suggested associations between noise exposure and a variety of mental health indicators, such as single rating of well-being; standard psychological symptom profiles; the intake of psychotropic drugs; and consumption of tranquilizers and sleeping pills. Early studies showed a weak association between exposure to aircraft noise and psychiatric hospital admissions in the general population surrounding an airport (see also Berglund & Lindvall 1995). However, the studies have been criticized because of problems in selecting variables and in response bias (Halpern 1995).

Exposure to high levels of occupational noise has been associated with development of neurosis and irritability; and exposure to high levels of environmental noise with deteriorated mental health (Stansfeld 1992). However, the findings on environmental noise and mental health effects are inconclusive (HCN 1994; Berglund & Lindvall 1995; IEH 1997). The only longitudinal study in this field (Stansfeld et al. 1996) showed an association between the initial level of road traffic noise and minor psychiatric disorders, although the association for increased anxiety was weak and non-linear. It turned out that psychiatric disorders are associated with noise sensitivity,

rather than with noise exposure, and the association was found to disappear after adjustment for baseline trait anxiety. These and other results show the importance of taking vulnerable groups into account, because they may not be able to cope sufficiently with unwanted environmental noise (e.g. Stansfeld 1992). This is particularly true of children, the elderly and people with preexisting illnesses, especially depression (IEH 1997). Despite the weaknesses of the various studies, the possibility that community noise has adverse effects on mental health is suggested by studies on the use of medical drugs, such as tranquilizers and sleeping pills, on psychiatric symptoms and on mental hospital admission rates.

3.7. The Effects of Noise on Performance

It has been documented in both laboratory subjects and in workers exposed to occupational noise, that noise adversely affects cognitive task performance. In children, too, environmental noise impairs a number of cognitive and motivational parameters (Cohen et al. 1980; Evans & Lepore 1993; Evans 1998; Hygge et al. 1998; Haines et al. 1998). However, there are no published studies on whether environmental noise at home also impairs cognitive performance in adults. Accidents may also be an indicator of performance deficits. The few field studies on the effects of noise on performance and safety showed that noise may produce some task impairment and increase the number of errors in work, but the effects depend on the type of noise and the task being performed (Smith 1990).

Laboratory and workplace studies showed that noise can act as a distracting stimulus. Also, impulsive noise events (e.g. sonic booms) may produce disruptive effects as a result of startle responses. In the short term, noise-induced arousal may produce better performance of simple tasks, but cognitive performance deteriorates substantially for more complex tasks (i.e. tasks that require sustained attention to details or to multiple cues; or tasks that demand a large capacity of working memory, such as complex analytical processes). Some of the effects are related to loss in auditory comprehension and language acquisition, but others are not (Evans & Maxwell 1997). Among the cognitive effects, reading, attention, problem solving and memory are most strongly affected by noise. The observed effects on motivation, as measured by persistence with a difficult cognitive task, may either be independent or secondary to the aforementioned cognitive impairments.

Two types of memory deficits have been identified under experimental noise exposure: incidental memory and memory for materials that the observer was not explicitly instructed to focus on during a learning phase. For example, when presenting semantic information to subjects in the presence of noise, recall of the information content was unaffected, but the subjects were significantly less able to recall, for example, in which corner of the slide a word had been located. There is also some evidence that the lack of “helping behavior” that was noted under experimental noise exposure may be related to inattention to incidental cues (Berglund & Lindvall 1995). Subjects appear to process information faster in working memory during noisy performance conditions, but at a cost of available memory capacity. For example, in a running memory task, in which subjects were required to recall in sequence letters that they had just heard, subjects recalled recent items better under noisy conditions, but made more errors farther back into the list.

Experimental noise exposure consistently produces negative after-effects on performance (Glass & Singer 1972). Following exposure to aircraft noise, schoolchildren in the vicinity of Los Angeles airport were found to be deficient in proofreading, and in persistence with challenging puzzles (Cohen et al. 1980). The uncontrollability of noise, rather than the intensity of the noise, appears to be the most critical variable. The only prospective study on noise-exposed schoolchildren, designed around the move of the Munich airport (Hygge et al. 1996; Evans et al. 1998), confirmed the results of laboratory and workplace studies in adults, as well the results of the Los Angeles airport study with children (Cohen et al. 1980). An important finding was that some of the adaptation strategies for dealing with aircraft noise, such as tuning out or ignoring the noise, and the effort necessary to maintain task performance, come at a price. There is heightened sympathetic arousal, as indicated by increased levels of stress hormone, and elevation of resting blood pressure (Evans et al. 1995; Evans et al. 1998). Notably, in the airport studies reported above, the adverse effects were larger in children with lower school achievement.

For aircraft noise, it has been shown that chronic exposure during early childhood appears to impair reading acquisition and reduces motivational capabilities. Of recent concern are concomitant psychophysiological changes (blood pressure and stress hormone levels). Evidence indicates that the longer the exposure, the greater the damage. It seems clear that daycare centers and schools should not be located near major sources of noise, such as highways, airports and industrial sites.

3.8. Effects of Noise on Residential Behaviour and Annoyance

Noise annoyance is a global phenomenon. A definition of annoyance is “a feeling of displeasure associated with any agent or condition, known or believed by an individual or group to adversely affect them” (Lindvall & Radford 1973; Koelega 1987). However, apart from “annoyance”, people may feel a variety of negative emotions when exposed to community noise, and may report anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation, or exhaustion (Job 1993; Fields et al. 1997 1998). Thus, although the term annoyance does not cover all the negative reactions, it is used for convenience in this document.

Noise can produce a number of social and behavioural effects in residents, besides annoyance (for review see Berglund & Lindvall 1995). The social and behavioural effects are often complex, subtle and indirect. Many of the effects are assumed to be the result of interactions with a number of non-auditory variables. Social and behavioural effects include changes in overt everyday behaviour patterns (e.g. closing windows, not using balconies, turning TV and radio to louder levels, writing petitions, complaining to authorities); adverse changes in social behaviour (e.g. aggression, unfriendliness, disengagement, non-participation); adverse changes in social indicators (e.g. residential mobility, hospital admissions, drug consumption, accident rates); and changes in mood (e.g. less happy, more depressed).

Although changes in social behaviour, such as a reduction in helpfulness and increased aggressiveness, are associated with noise exposure, noise exposure alone is not believed to be sufficient to produce aggression. However, in combination with provocation or pre-existing anger or hostility, it may trigger aggression. It has also been suspected that people are less willing to help, both during exposure and for a period after exposure. Fairly consistent evidence

shows that noise above 80 dBA is associated with reduced helping behaviour and increased aggressive behaviour. Particularly, there is concern that high-level continuous noise exposures may contribute to the susceptibility of schoolchildren to feelings of helplessness (Evans & Lepore 1993)

The effects of community noise can be evaluated by assessing the extent of annoyance (low, moderate, high) among exposed individuals; or by assessing the disturbance of specific activities, such as reading, watching television and communication. The relationship between annoyance and activity disturbances is not necessarily direct and there are examples of situations where the extent of annoyance is low, despite a high level of activity disturbance. For aircraft noise, the most important effects are interference with rest, recreation and watching television. This is in contrast to road traffic noise, where sleep disturbance is the predominant effect (Berglund & Lindvall 1995).

A number of studies have shown that equal levels of traffic and industrial noises result in different magnitudes of annoyance (Hall et al. 1981; Griffiths 1983; Miedema 1993; Bradley 1994a; Miedema & Vos 1998). This has led to criticism (e.g. Kryter 1994; Bradley 1994a) of averaged dose-response curves determined by meta-analysis, which assumed that all traffic noises are the same (Fidell et al. 1991; Fields 1994a; Finegold et al. 1994). Schultz (1978) and Miedema & Vos (1998) have synthesized curves of annoyance associated with three types of traffic noise (road, air, railway). In these curves, the percentage of people highly or moderately annoyed was related to the day and night continuous equivalent sound level, L_{dn}. For each of the three types of traffic noise, the percentage of highly annoyed persons in a population started to increase at an L_{dn} value of 42 dBA, and the percentage of moderately annoyed persons at an L_{dn} value of 37 dBA (Miedema & Vos 1998). Aircraft noise produced a stronger annoyance response than road traffic, for the same L_{dn} exposure, consistent with earlier analyses (Kryter 1994; Bradley 1994a). However, caution should be exercised when interpreting synthesized data from different studies, since five major parameters should be randomly distributed for the analyses to be valid: personal, demographic, and lifestyle factors, as well as the duration of noise exposure and the population experience with noise (Kryter 1994).

Annoyance in populations exposed to environmental noise varies not only with the acoustical characteristics of the noise (source, exposure), but also with many non-acoustical factors of social, psychological, or economic nature (Fields 1993). These factors include fear associated with the noise source, conviction that the noise could be reduced by third parties, individual noise sensitivity, the degree to which an individual feels able to control the noise (coping strategies), and whether the noise originates from an important economic activity. Demographic variables such as age, sex and socioeconomic status, are less strongly associated with annoyance. The correlation between noise exposure and general annoyance is much higher at the group level than at the individual level, as might be expected. Data from 42 surveys showed that at the group level about 70% of the variance in annoyance is explained by noise exposure characteristics, whereas at the individual level it is typically about 20% (Job 1988).

When the type and amount of noise exposure is kept constant in the meta-analyses, differences between communities, regions and countries still exist (Fields 1990; Bradley 1996). This is well demonstrated by a comparison of the dose-response curve determined for road-traffic noise

(Miedema & Vos 1998) and that obtained in a survey along the North-South transportation route through the Austrian Alps (Lercher 1998b). The differences may be explained in terms of the influence of topography and meteorological factors on acoustical measures, as well as the low background noise level on the mountain slopes.

Stronger reactions have been observed when noise is accompanied by vibrations and contains low frequency components (Paulsen & Kastka 1995; Öhrström 1997; for review see Berglund et al. 1996), or when the noise contains impulses, such as shooting noise (Buchta 1996; Vos 1996; Smoorenburg 1998). Stronger, but temporary, reactions also occur when noise exposure is increased over time, in comparison to situations with constant noise exposure (e.g. HCN 1997; Klæboe et al. 1998). Conversely, for road traffic noise, the introduction of noise protection barriers in residential areas resulted in smaller reductions in annoyance than expected for a stationary situation (Kastka et al. 1995).

To obtain an indicator for annoyance, other methods of combining parameters of noise exposure have been extensively tested, in addition to metrics such as LAeq,24h and L_{dn}. When used for a set of community noises, these indicators correlate well both among themselves and with LAeq,24h or L_{dn} values (e.g. HCN 1997). Although LAeq,24h and L_{dn} are in most cases acceptable approximations, there is a growing concern that all the component parameters of the noise should be individually assessed in noise exposure investigations, at least in the complex cases (Berglund & Lindvall 1995).

3.9. The Effects of Combined Noise Sources

Many acoustical environments consist of sounds from more than one source. For these environments, health effects are associated with the total noise exposure, rather than with the noise from a single source (WHO 1980b). When considering hearing impairment, for example, the total noise exposure can be expressed in terms of LAeq,24h for the combined sources. For other adverse health effects, however, such a simple model most likely will not apply. It is possible that some disturbances (e.g. speech interference, sleep disturbance) may more easily be attributed to specific noises. In cases where one noise source clearly dominates, the magnitude of an effect may be assessed by taking into account the dominant source only (HCN 1997). Furthermore, at a policy level, there may be little need to identify the adverse effect of each specific noise, unless the responsibility for these effects is to be shared among several polluters (*cf*. The Polluter Pays Principle in Chapter 5, UNCED 1992).

There is no consensus on a model for assessing the total annoyance due to a combination of environmental noise sources. This is partly due to a lack of research into the temporal patterns of combined noises. The current approach for assessing the effects of “mixed noise sources” is limited to data on “total annoyance” transformed to mathematical principles or rules of thumb (Ronnebaum et al. 1996; Vos 1992; Miedema 1996; Berglund & Nilsson 1997). Models to assess the total annoyance of combinations of environmental noises may not be applicable to those health effects for which the mechanisms of noise interaction are unknown, and for which different cumulative or synergistic effects cannot be ruled out. When noise is combined with different types of environmental agents, such as vibrations, ototoxic chemicals, or chemical odours, again there is insufficient knowledge to accurately assess the combined effects on health

(Berglund & Lindvall 1995; HCN 1994; Miedema 1996; Zeichart 1998; Passchier-Vermeer & Zeichart 1998). Therefore, caution should be exercised when trying to predict the adverse health effects of combined factors in residential populations.

The evidence on low-frequency noise is sufficiently strong to warrant immediate concern. Various industrial sources emit continuous low-frequency noise (compressors, pumps, diesel engines, fans, public works); and large aircraft, heavy-duty vehicles and railway traffic produce intermittent low-frequency noise. Low-frequency noise may also produce vibrations and rattles as secondary effects. Health effects due to low-frequency components in noise are estimated to be more severe than for community noises in general (Berglund et al. 1996). Since A-weighting underestimates the sound pressure level of noise with low-frequency components, a better assessment of health effects would be to use C-weighting.

In residential populations heavy noise pollution will most certainly be associated with a combination of health effects. For example, cardiovascular disease, annoyance, speech interference at work and at home, and sleep disturbance. Therefore, it is important that the total adverse health load over 24 hours be considered and that the precautionary principle for sustainable development is applied in the management of health effects (see Chapter 5).

3.10. Vulnerable Groups

Protective standards are essentially derived from observations on the health effects of noise on "normal" or "average" populations. The participants of these investigations are selected from the general population and are usually adults. Sometimes, samples of participants are selected because of their easy availability. However, vulnerable groups of people are typically underrepresented. This group includes people with decreased personal abilities (old, ill, or depressed people); people with particular diseases or medical problems; people dealing with complex cognitive tasks, such as reading acquisition; people who are blind or who have hearing impairment; fetuses, babies and young children; and the elderly in general (Jansen 1987; AAP 1997). These people may be less able to cope with the impacts of noise exposure and be at greater risk for harmful effects.

Persons with impaired hearing are the most adversely affected with respect to speech intelligibility. Even slight hearing impairments in the high-frequency range may cause problems with speech perception in a noisy environment. From about 40 years of age, people typically demonstrate an impaired ability to understand difficult, spoken messages with low linguistic redundancy. Therefore, based on interference with speech perception, a majority of the population belongs to the vulnerable group.

Children have also been identified as vulnerable to noise exposure (see Agenda 21: UNCED 1992). The evidence on noise pollution and children's health is strong enough to warrant monitoring programmes at schools and preschools to protect children from the effects of noise. Follow up programmes to study the main health effects of noise on children, including effects on speech perception and reading acquisition, are also warranted in heavily noise polluted areas (Cohen et al. 1986; Evans et al. 1998).

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The issue of vulnerable subgroups in the general population should thus be considered when developing regulations or recommendations for the management of community noise. This consideration should take into account the types of effects (communication, recreation, annoyance, etc.), specific environments (*in utero*, incubator, home, school, workplace, public institutions, etc.) and specific lifestyles (listening to loud music through headphones, or at discotheques and festivals; motor cycling, etc.).

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4. Guideline Values

4.1. Introduction

The human ear and lower auditory system continuously receive stimuli from the world around us. However, this does not mean that all the acoustical inputs are necessarily disturbing or have harmful effects. This is because the auditory nerve provides activating impulses to the brain that enable us to regulate the vigilance and wakefulness necessary for optimal performance. On the other hand, there are scientific reports that a completely silent world can have harmful effects, because of sensory deprivation. Thus, both too little sound and too much sound can be harmful. For this reason, people should have the right to decide for themselves the quality of the acoustical environment they live in.

Exposure to noise from various sources is most commonly expressed as the average sound pressure level over a specific time period, such as 24 hours. This means that identical average sound levels for a given time period could be derived from either a large number of sound events with relatively low, almost inaudible levels, or from a few events with high sound levels. This technical concept does not fully agree with common experience on how environmental noise is experienced, or with the neurophysiological characteristics of the human receptor system.

Human perception of the environment through vision, hearing, touch, smell and taste is characterized by a good discrimination of stimulus intensity differences, and by a decaying response to a continuous stimulus (adaptation or habituation). Single sound events cannot be discriminated if the interval between events drops below a threshold value; if this occurs, the sound is interpreted as continuous. These characteristics are linked to survival, since new and different stimuli with low probability and high information value indicate warnings. Thus, when assessing the effects of environmental noise on people it is relevant to consider the importance of the background noise level, the number of events, and the noise exposure level independently.

Community noise studies have traditionally considered noise annoyance from single specific sources such as aircraft, road traffic or railways. In recent years, efforts have been made to compare the results from road traffic, aircraft and railway surveys. Data from a number of sources show that aircraft noise is more annoying than road traffic noise, which, in turn, is more annoying than railway noise. However, there is not a clear understanding of the mechanisms that create these differences. Some populations may also be at greater risk for the harmful effects of noise. Young children (especially during language acquisition), the blind, and perhaps fetuses are examples of such populations. There are no definite conclusions on this topic, but the reader should be alerted that guidelines in this report are developed for the population at large; guidelines for potentially more vulnerable groups are addressed only to a limited extent.

In the following, guideline values are summarized with regard to specific environments and effects. For each environment and situation, the guideline values take into consideration the identified health effects and are set, based on the lowest levels of noise that affect health (critical health effect). Guideline values typically correspond to the lowest effect level for general populations, such as those for indoor speech intelligibility. By contrast, guideline values for annoyance have been set at 50 or 55 dBA, representing daytime levels below which a majority of

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the adult population will be protected from becoming moderately or seriously annoyed, respectively.

In these *Guidelines for Community Noise* only guideline values are presented. These are essentially values for the onset of health effects from noise exposure. It would have been preferred to establish guidelines for exposure-response relationships. Such relationships would indicate the effects to be expected if standards were set above the WHO guideline values and would facilitate the setting of standards for sound pressure levels (noise immission standards). However, exposure-response relationships could not be established as the scientific literature is very limited. The best-studied exposure-response relationship is that between L_{dn} and annoyance (WHO 1995a; Berglund & Lindvall 1995; Miedema & Vos 1998). Even the most recent relationships between integrated noise levels and the percentage of highly or moderately annoyed people are still being scrutinized. The results of a forthcoming meta-analysis are expected to be published in the near future (Miedema, personal communication).

4.2. Specific Effects

4.2.1. Interference with communication

Noise tends to interfere with auditory communication, in which speech is a most important signal. However, it is also vital to be able to hear alarming and informative signals such as door bells, telephone signals, alarm clocks, fire alarms etc., as well as sounds and signals involved in occupational tasks. The effects of noise on speech discrimination have been studied extensively and deal with this problem in lexical terms (mostly words but also sentences). For communication distances beyond a few metres, speech interference starts at sound pressure levels below 50 dB for octave bands centered on the main speech frequencies at 500, 1 000 and 2 000 Hz. It is usually possible to express the relationship between noise levels and speech intelligibility in a single diagram, based on the following assumptions and empirical observations, and for speaker-to-listener distance of about 1 m:

a. Speech in relaxed conversation is 100% intelligible in background noise levels of about 35 dBA, and can be understood fairly well in background levels of 45 dBA.

b. Speech with more vocal effort can be understood when the background sound pressure level is about 65 dBA.

A majority of the population belongs to groups sensitive to interference with speech perception. Most sensitive are the elderly and persons with impaired hearing. Even slight hearing impairments in the high-frequency range may cause problems with speech perception in a noisy environment. From about 40 years of age, people demonstrate impaired ability to interpret difficult, spoken messages with low linguistic redundancy, when compared to people aged 20–30 years. It has also been shown that children, before language acquisition has been completed, have more adverse effects than young adults to high noise levels and long reverberation times.

For speech outdoors and for moderate distances, the sound level drops by approximately 6 dB for a doubling of the distance between speaker and listener. This relationship is also applicable to

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indoor conditions, but only up to a distance of about 2 m. Speech communication is affected also by the reverberation characteristics of the room, and reverberation times beyond 1 s can produce a loss in speech discrimination. A longer reverberation time combined with background noise makes speech perception still more difficult.

Speech signal perception is of paramount importance, for example, in classrooms or conference rooms. To ensure any speech communication, the signal-to-noise relationship should exceed zero dB. But when listening to complicated messages (at school, listening to foreign languages, telephone conversation) the signal-to-noise ratio should be at least 15 dB. With a voice level of 50 dBA (at 1 m distance this corresponds on average to a casual voice level in both women and men), the background level should not exceed 35 dBA. This means that in classrooms, for example, one should strive for as low background levels as possible. This is particularly true when listeners with impaired hearing are involved, for example, in homes for the elderly. Reverberation times below 1 s are necessary for good speech intelligibility in smaller rooms; and even in a quiet environment a reverberation time below 0.6 s is desirable for adequate speech intelligibility for sensitive groups.

4.2.2. Noise-induced hearing impairment

The ISO Standard 1999 (ISO 1990) gives a method of calculating noise-induced hearing impairment in populations exposed to all types of occupational noise (continuous, intermittent, impulse). However, noise-induced hearing impairment is by no means restricted to occupational situations alone. High noise levels can also occur in open-air concerts, discotheques, motor sports, shooting ranges, and from loudspeakers or other leisure activities in dwellings. Other loud noise sources, such as music played back in headphones and impulse noise from toys and fireworks, are also important. Evidence strongly suggests that the calculation method from ISO Standard 1999 for occupational noise (ISO 1990) should also be used for environmental and leisure time noise exposures. This implies that long term exposure to LAeq,24h of up to 70 dBA will not result in hearing impairment. However, given the limitations of the various underlying studies, care should be taken with respect to the following:

a. Data from animal experiments indicate that children may be more vulnerable in acquiring noise-induced hearing impairment than adults.

b. At very high instantaneous sound pressure levels mechanical damage to the ear may occur (Hanner & Axelsson 1988). Occupational limits are set at peak sound pressure levels of 140 dBA (EU 1986a). For adults, this same limit is assumed to be in order for exposure to environmental and leisure time noise. In the case of children, however, considering their habits while playing with noisy toys, peak sound pressure levels should never exceed 120 dBA.

c. For shooting noise with LAeq,24h over 80 dB, studies on temporary threshold shift suggest there is the possibility of an increased risk for noise-induced hearing impairment (Smoorenburg 1998).

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d. The risk for noise-induced hearing impairment increases when noise exposure is combined with vibrations, ototoxic drugs or chemicals (Fechter 1999). In these circumstances, long-term exposure to LAeq,24h of 70 dB may induce small hearing impairments.

e. It is uncertain whether the relationships in ISO Standard 1999 (ISO 1990) are applicable to environmental sounds having a short rise time. For example, in the case of military low-altitude flying areas (75–300 m above ground) LAmax values of 110–130 dB occur within seconds after onset of the sound.

In conclusion, dose-response data are lacking for the general population. However, judging from the limited data for study groups (teenagers, young adults and women), and on the assumption that time of exposure can be equated with sound energy, the risk for hearing impairment would be negligible for LAeq,24h values of 70 dB over a lifetime. To avoid hearing impairment, impulse noise exposures should never exceed a peak sound pressure of 140 dB peak in adults, and 120 dB in children.

4.2.3. Sleep disturbance effects

Electrophysiological and behavioral methods have demonstrated that both continuous and intermittent noise indoors lead to sleep disturbance. The more intense the background noise, the more disturbing is its effect on sleep. Measurable effects on sleep start at background noise levels of about 30 dB LAeq. Physiological effects include changes in the pattern of sleep stages, especially a reduction in the proportion of REM sleep. Subjective effects have also been identified, such as difficulty in falling asleep, perceived sleep quality, and adverse after-effects such as headache and tiredness. Sensitive groups mainly include elderly persons, shift workers and persons with physical or mental disorders.
Where noise is continuous, the equivalent sound pressure level should not exceed 30 dBA indoors, if negative effects on sleep are to be avoided. When the noise is composed of a large proportion of low-frequency sounds a still lower guideline value is recommended, because low-frequency noise (e.g. from ventilation systems) can disturb rest and sleep even at low sound pressure levels. It should be noted that the adverse effect of noise partly depends on the nature of the source. A special situation is for newborns in incubators, for which the noise can cause sleep disturbance and other health effects.

If the noise is not continuous, LAmax or SEL are used to indicate the probability of noise-induced awakenings. Effects have been observed at individual LAmax exposures of 45 dB or less. Consequently, it is important to limit the number of noise events with a LAmax exceeding 45 dB. Therefore, the guidelines should be based on a combination of values of 30 dB LAeq,8h and 45 dB LAmax. To protect sensitive persons, a still lower guideline value would be preferred when the background level is low. Sleep disturbance from intermittent noise events increases with the maximum noise level. Even if the total equivalent noise level is fairly low, a small number of noise events with a high maximum sound pressure level will affect sleep.

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Therefore, to avoid sleep disturbance, guidelines for community noise should be expressed in terms of equivalent sound pressure levels, as well as LAmax/SEL and the number of noise events. Measures reducing disturbance during the first part of the night are believed to be the most effective for reducing problems in falling asleep.

4.2.4. Cardiovascular and psychophysiological effects

Epidemiologial studies show that cardiovascular effects occur after long-term exposure to noise (aircraft and road traffic) with LAeq,24h values of 65–70 dB. However, the associations are weak. The association is somewhat stronger for ischaemic heart disease than for hypertension. Such small risks are important, however, because a large number of persons are currently exposed to these noise levels, or are likely to be exposed in the future. Other possible effects, such as changes in stress hormone levels and blood magnesium levels, and changes in the immune system and gastro-intestinal tract, are too inconsistent to draw conclusions. Thus, more research is required to estimate the long-term cardiovascular and psychophysiological risks due to noise. In view of the equivocal findings, no guideline values can be given.

4.2.5. Mental health effects

Studies that have examined the effects of noise on mental health are inconclusive and no guideline values can be given. However, in noisy areas, it has been observed that there is an increased use of prescription drugs such as tranquilizers and sleeping pills, and an increased frequency of psychiatric symptoms and mental hospital admissions. This strongly suggests that adverse mental health effects are associated with community noise.

4.2.6. Effects on performance

The effects of noise on task performance have mainly been studied in the laboratory and to some extent in work situations. But there have been few, if any, detailed studies on the effects of noise on human productivity in community situations. It is evident that when a task involves auditory signals of any kind, noise at an intensity sufficient to mask or interfere with the perception of these signals will also interfere with the performance of the task. A novel event, such as the start of an unfamiliar noise, will also cause distraction and interfere with many kinds of tasks. For example, impulsive noises such as sonic booms can produce disruptive effects as the result of startle responses; and these types of responses are more resistant to habituation.

Mental activities involving high load in working memory, such as sustained attention to multiple cues or complex analysis, are all directly sensitive to noise and performance suffers as a result. Some accidents may also be indicators of noise-related effects on performance. In addition to the direct effects on performance, noise also has consistent after-effects on cognitive performance with tasks such as proof-reading, and on persistence with challenging puzzles. In contrast, the performance of tasks involving either motor or monotonous activities is not always degraded by noise.

Chronic exposure to aircraft noise during early childhood appears to damage reading acquisition.

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Evidence indicates that the longer the exposure, the greater the damage. Although there is insufficient information on these effects to set specific guideline values, it is clear that day-care centres and schools should not be located near major noise sources, such as highways, airports and industrial sites.

4.2.7. Annoyance responses

The capacity of a noise to induce annoyance depends upon many of its physical characteristics, including its sound pressure level and spectral characteristics, as well as the variations of these properties over time. However, annoyance reactions are sensitive to many non-acoustical factors of social, psychological or economic nature, and there are also considerable differences in individual reactions to the same noise. Dose-response relations for different types of traffic noise (air, road and railway) clearly demonstrate that these noises can cause different annoyance effects at equal LAeq,24h values. And the same type of noise, such as that found in residential areas around airports, can also produce different annoyance responses in different countries.

The annoyance response to noise is affected by several factors, including the equivalent sound pressure level and the highest sound pressure level of the noise, the number of such events, and the time of day. Methods for combining these effects have been extensively studied. The results are not inconsistent with the simple, physically based equivalent energy theory, which is represented by the LAeq noise index.

Annoyance to community noise varies with the type of activity producing the noise. Speech communication, relaxation, listening to radio and TV are all examples of noise-producing activities. During the daytime, few people are seriously annoyed by activities with LAeq levels below 55 dB; or moderately annoyed with LAeq levels below 50 dB. Sound pressure levels during the evening and night should be 5–10 dB lower than during the day. Noise with low-frequency components require even lower levels. It is emphasized that for intermittent noise it is necessary to take into account the maximum sound pressure level as well as the number of noise events. Guidelines or noise abatement measures should also take into account residential outdoor activities.

4.2.8. Effects on social behaviour

The effects of environmental noise may be evaluated by assessing the extent to which it interferes with different activities. For many community noises, interference with rest, recreation and watching television seem to be the most important issues. However, there is evidence that noise has other effects on social behaviour: helping behaviour is reduced by noise in excess of 80 dBA; and loud noise increases aggressive behavior in individuals predisposed to aggressiveness. There is concern that schoolchildren exposed to high levels of chronic noise could be more susceptible to helplessness. Guidelines on these issues must await further research.

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4.3. Specific Environments

Noise measures based solely on LAeq values do not adequately characterize most noise environments and do not adequately assess the health impacts of noise on human well-being. It is also important to measure the maximum noise level and the number of noise events when deriving guideline values. If the noise includes a large proportion of low-frequency components, values even lower than the guideline values will be needed, because low-frequency components in noise may increase the adverse effects considerably. When prominent low-frequency components are present, measures based on A-weighting are inappropriate. However, the difference between dBC (or dBlin) and dBA will give crude information about the presence of low-frequency components in noise. If the difference is more than 10 dB, it is recommended that a frequency analysis of the noise be performed.

4.3.1. Dwellings

In dwellings, the critical effects of noise are on sleep, annoyance and speech interference. To avoid sleep disturbance, indoor guideline values for bedrooms are 30 dB LAeq for continuous noise and 45 dB LAmax for single sound events. Lower levels may be annoying, depending on the nature of the noise source. The maximum sound pressure level should be measured with the instrument set at "*Fast*".

To protect the majority of people from being seriously annoyed during the daytime, the sound pressure level on balconies, terraces and outdoor living areas should not exceed 55 dB LAeq for a steady, continuous noise. To protect the majority of people from being moderately annoyed during the daytime, the outdoor sound pressure level should not exceed 50 dB LAeq. These values are based on annoyance studies, but most countries in Europe have adopted 40 dB LAeq as the maximum allowable level for new developments (Gottlob 1995). Indeed, the lower value should be considered the maximum allowable sound pressure level for all new developments whenever feasible.

At night, sound pressure levels at the outside façades of the living spaces should not exceed 45 dB LAeq and 60 dB LAmax, so that people may sleep with bedroom windows open. These values have been obtained by assuming that the noise reduction from outside to inside with the window partly open is 15 dB.

4.3.2. Schools and preschools

For schools, the critical effects of noise are on speech interference, disturbance of information extraction (e.g. comprehension and reading acquisition), message communication and annoyance. To be able to hear and understand spoken messages in classrooms, the background sound pressure level should not exceed 35 dB LAeq during teaching sessions. For hearing impaired children, an even lower sound pressure level may be needed. The reverberation time in the classroom should be about 0.6 s, and preferably lower for hearing-impaired children. For assembly halls and cafeterias in school buildings, the reverberation time should be less than 1 s. For outdoor playgrounds, the sound pressure level of the noise from external sources should not exceed 55 dB LAeq, the same value given for outdoor residential areas in daytime.

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For preschools, the same critical effects and guideline values apply as for schools. In bedrooms in preschools during sleeping hours, the guideline values for bedrooms in dwellings should be used.

4.3.3. Hospitals

For most spaces in hospitals, the critical effects of noise are on sleep disturbance, annoyance and communication interference, including interference with warning signals. The LAmax of sound events during the night should not exceed 40 dB indoors. For wardrooms in hospitals, the guideline values indoors are 30 dB LAeq, together with 40 dB LAmax during the night. During the day and evening the guideline value indoors is 30 dB LAeq. The maximum level should be measured with the instrument set at *"Fast"*.

Since patients have less ability to cope with stress, the equivalent sound pressure level should not exceed 35 dB LAeq in most rooms in which patients are being treated or observed. Particular attention should be given to the sound pressure levels in intensive care units and operating theatres. Sound inside incubators may result in health problems, including sleep disturbance, and may lead to hearing impairment in neonates. Guideline values for sound pressure levels in incubators must await future research.

4.3.4. Ceremonies, festivals and entertainment events

In many countries, there are regular ceremonies, festivals and other entertainment to celebrate life events. Such events typically produce loud sounds including music and impulsive sounds. There is widespread concern about the effect of loud music and impulse sounds on young people who frequently attend concerts, discotheques, video arcades, cinemas, amusement parks and spectator events, etc. The sound pressure level is typically in excess of 100 dB LAeq. Such a noise exposure could lead to significant hearing impairment after frequent attendance.

Noise exposure for employees of these venues should be controlled by established occupational standards. As a minimum, the same standards should apply to the patrons of these premises. Patrons should not be exposed to sound pressure levels greater than 100 dB LAeq during a 4-h period, for at most four times per year. To avoid acute hearing impairment the LAmax should always be below 110 dB.

4.3.5. Sounds through headphones

To avoid hearing impairment in both adults and children from music and other sounds played back in headphones, the LAeq,24h should not exceed 70 dB. This implies that for a daily one-hour exposure the LAeq should not exceed 85 dB. The exposures are expressed in free-field equivalent sound pressure levels. To avoid acute hearing impairment, the LAmax should always be below 110 dB.

4.3.6. Impulsive sounds from toys, fireworks and firearms

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To avoid acute mechanical damage to the inner ear, adults should never be exposed to more than 140 dB peak sound pressure. To account for the vulnerability in children, the peak sound pressure level produced by toys should not surpass 120 dB, measured close to the ears (100 mm). To avoid acute hearing impairment, LAmax should always be below 110 dB.

4.3.7. Parkland and conservation areas

Existing large quiet outdoor areas should be preserved and the signal-to-noise ratio kept low.

4.4. WHO Guideline Values

The WHO guideline values in Table 4.1 are organized according to specific environments. When multiple adverse health effects are identified for a given environment, the guideline values are set at the level of the lowest adverse health effect (the critical health effect). An adverse health effect of noise refers to any temporary or long-term deterioration in physical, psychological or social functioning that is associated with noise exposure. The guideline values represent the sound pressure levels that affect the most exposed receiver in the listed environment.

The time base for LAeq for "daytime" and "night-time" is 16 h and 8 h, respectively. No separate time base is given for evenings alone, but typically, guideline value should be 5 –10 dB lower than for a 12 h daytime period. Other time bases are recommended for schools, preschools and playgrounds, depending on activity.

The available knowledge of the adverse effects of noise on health is sufficient to propose guideline values for community noise for the following:

a. Annoyance.
b. Speech intelligibility and communication interference.
c. Disturbance of information extraction.
d. Sleep disturbance.
e. Hearing impairment.

The different critical health effects are relevant to specific environments, and guideline values for community noise are proposed for each environment. These are:

a. Dwellings, including bedrooms and outdoor living areas.
b. Schools and preschools, including rooms for sleeping and outdoor playgrounds.
c. Hospitals, including ward and treatment rooms.
d. Industrial, commercial shopping and traffic areas, including public addresses, indoors and outdoors.
e. Ceremonies, festivals and entertainment events, indoors and outdoors.
f. Music and other sounds through headphones.
g. Impulse sounds from toys, fireworks and firearms.
h. Outdoors in parkland and conservation areas.

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It is not enough to characterize the noise environment in terms of noise measures or indices based only on energy summation (e.g. LAeq), because different critical health effects require different descriptions. Therefore, it is important to display the maximum values of the noise fluctuations, preferably combined with a measure of the number of noise events. A separate characterization of noise exposures during night-time would be required. For indoor environments, reverberation time is also an important factor. If the noise includes a large proportion of low frequency components, still lower guideline values should be applied.

Supplementary to the guideline values given in Table 4.1, precautionary recommendations are given in Section 4.2 and 4.3 for vulnerable groups, and for noise of a certain character (e.g. low-frequency components, low background noise), respectively. In Section 3.10, information is given regarding which critical effects and specific environments are considered relevant for vulnerable groups, and what precautionary noise protection would be needed in comparison to the general population.

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Table 4.1: Guideline values for community noise in specific environments.

Specific environment	Critical health effect(s)	LAeq [dB]	Time base [hours]	LAmax, fast [dB]
Outdoor living area	Serious annoyance, daytime and evening Moderate annoyance, daytime and evening	55 50	16 16	- -
Dwelling, indoors Inside bedrooms	Speech intelligibility and moderate annoyance, daytime and evening Sleep disturbance, night-time	35 30	16 8	 45
Outside bedrooms	Sleep disturbance, window open (outdoor values)	45	8	60
School class rooms and pre-schools, indoors	Speech intelligibility, disturbance of information extraction, message communication	35	during class	-
Pre-school Bedrooms, indoors	Sleep disturbance	30	sleeping -time	45
School, playground outdoor	Annoyance (external source)	55	during play	-
Hospital, ward rooms, indoors	Sleep disturbance, night-time Sleep disturbance, daytime and evenings	30 30	8 16	40 -
Hospitals, treatment rooms, indoors	Interference with rest and recovery	#1		
Industrial, commercial, shopping and traffic areas, indoors and Outdoors	Hearing impairment	70	24	110
Ceremonies, festivals and entertainment events	Hearing impairment (patrons:<5 times/year)	100	4	110
Public addresses, indoors and outdoors	Hearing impairment	85	1	110
Music through headphones/ Earphones	Hearing impairment (free-field value)	85 #4	1	110
Impulse sounds from toys, fireworks and firearms	Hearing impairment (adults) Hearing impairment (children)	- -	- -	140 #2 120 #2
Outdoors in parkland and conservation areas	Disruption of tranquillity	#3		

#1: as low as possible;
#2: peak sound pressure (not LAmax, fast), measured 100 mm from the ear;
#3: existing quiet outdoor areas should be preserved and the ratio of intruding noise to natural background sound should be kept low;
#4: under headphones, adapted to free-field values

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5. Noise Management

The goal of noise management is to maintain low noise exposures, such that human health and well-being are protected. The specific objectives of noise management are to develop criteria for the maximum safe noise exposure levels, and to promote noise assessment and control as part of environmental health programmes. This is not always achieved (Jansen 1998). The United Nations´ Agenda 21 (UNCED 1992), as well as the European Charter on Transport, Environment and Health (London Charter 1999), both support a number of environmental management principles on which government policies, including noise management policies, can be based. These include:

a. **The precautionary principle**. In all cases, noise should be reduced to the lowest level achievable in a particular situation. Where there is a reasonable possibility that public health will be damaged, action should be taken to protect public health without awaiting full scientific proof.

b. **The polluter pays principle**. The full costs associated with noise pollution (including monitoring, management, lowering levels and supervision) should be met by those responsible for the source of noise.

c. **The prevention principle**. Action should be taken where possible to reduce noise at the source. Land-use planning should be guided by an environmental health impact assessment that considers noise as well as other pollutants.

The government policy framework is the basis of noise management. Without an adequate policy framework and adequate legislation it is difficult to maintain an active or successful noise management programme. A policy framework refers to transport, energy, planning, development and environmental policies. The goals are more readily achieved if the interconnected government policies are compatible, and if issues which cross different areas of government policy are co-ordinated.

5.1. Stages in Noise Management

A legal framework is needed to provide a context for noise management (Finegold 1998; Hede 1998a). While there are many possible models, an example of one is given in Figure 5.1. This model depicts the six stages in the process for developing and implementing policies for community noise management. For each policy stage, there are groups of 'policy players' who ideally would participate in the process.

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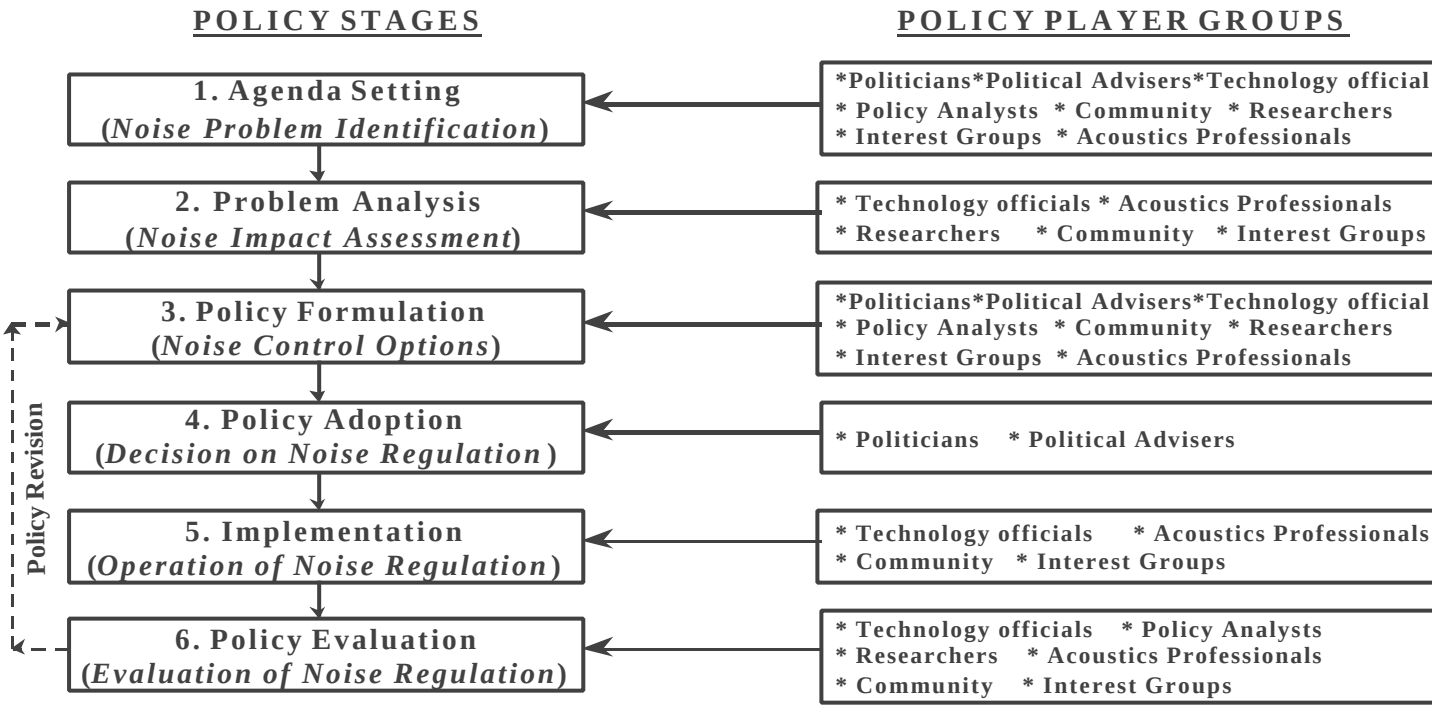


Figure 5.1. A model of the policy process for community noise management (Hede 1998a)

When goals and policies have been developed, the next stage is the development of the strategy or plan. Figure 5.2 summarizes the stages involved in the development of a noise management strategy. Specific abatement measures 19 are listed in Table 5.1.

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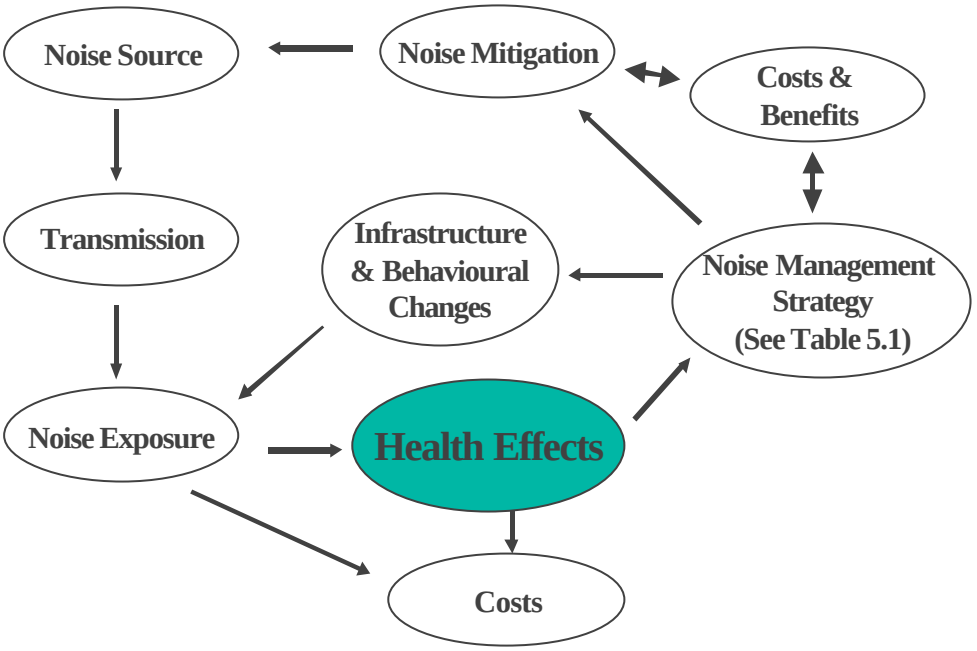


Figure 5.2. Stages involved in the development of a noise abatement strategy.

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Table 5.1. Recommended Noise Management Measures (following EEA 1995)

Legal measures	Examples
Control of noise emissions	Emission standards for road and off-road vehicles; emission standards for construction equipment; emission standards for plants; national regulations, EU Directives
Control of noise transmission	Regulations on sound-obstructive measures
Noise mapping and zoning around roads, airports, industries	Initiation of monitoring and modeling programmes
Control of noise immissions	Limits for exposure levels such as national immission standards; noise monitoring and modeling; regulations for complex noise situations; regulations for recreational noise
Speed limits	Residential areas; hospitals
Enforcement of regulations	Low Noise Implementation Plan
Minimum requirements for acoustical properties of buildings	Construction codes for sound insulation of building parts
Engineering Measures	
Emission reduction by source modification	Tyre profiles; low-noise road surfaces; changes in engine properties
New engine technology	Road vehicles; aircraft; construction machines
Transmission reduction	Enclosures around machinery; noise screens
Orientation of buildings	Design and structuring of tranquille uses; using buildings for screening purposes
Traffic management	Speed limits; guidance of traffic flow by electronic means
Passive protection	Ear plugs; ear muffs; insulation of dwellings; façade design
Implementation of land-use planning	Minimum distance between industrial, busy roads and residential areas; location of tranquillity areas; by-pass roads for heavy traffic; separating out incompatible functons
Education and information	
Raising public awareness	Informing the public on the health impacts of noise, enforcement action taken, noise levels, complaints
Monitoring and modeling of soundscapes	Publication of results
Sufficient number of noise experts	University or highschool curricula
Initiation of research and development	Funding of information generation according to scientific research needs
Initiation of behaviour changes	Speed reduction when driving; use of horns; use of loudspeakers for advertisements

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The process outlined in Figure 5.2 can start with the development of noise standards or guidelines. Ideally, it should also involve the identification and mapping of noise sources and exposed communities. Meteorological conditions and noise levels would also normally be monitored. These data can be used to validate the output of models that estimate noise levels. Noise standards and model outputs may be considered in devising noise control tactics aimed at achieving the noise standards. Before being enforced, current control tactics need to be revised, and if the standards are achieved they need continued enforcement. If the standards are not achieved after a reasonable period of time, the noise control tactics may need to be revised.

National noise standards can usually be based on a consideration of international guidelines, such as these Guidelines for Community Noise, as well as national criteria documents, which consider dose-response relations for the effects of noise on human health. National standards take into account the technological, social, economic, political and other factors specific for the country.

In many cases monitoring may show that noise levels are considerably higher than established guidelines. This may be particularly true in developing countries, and the question has to be raised as to whether national standards should reflect the optimum levels needed to protect human health, when this objective is unlikely to be achieved in the short- or medium-term with available resources. In some countries noise standards are set at levels that are realistically attainable under prevailing technological, social, economic and political conditions, even though they may not be fully consistent with the levels needed to protect human health. In such cases, a staged programme of noise abatement should be implemented to achieve the optimum health protection levels over the long term. Noise standards periodically change after reviews, as conditions in a country change over time, and with improved scientific understanding of the relationship between noise pollution and the health of the population. Noise level monitoring (Chapter 2) is used to assess whether noise levels at particular locations are in compliance with the standards selected.

5.2. Noise Exposure Mapping

A crucial component of a low-noise implementation plan is a reasonably quantitative knowledge of exposure (see Figure 5.2). Exposure should be mapped for all noise sources impacting a community; for example, road traffic, aircraft, railway, industry, construction, festivals and human activity in general. For some components of a noise exposure map or noise exposure inventory, accurate data may be available. In other cases, exposure can be calculated from the characteristics of the mechanical processes. While estimates of noise emissions are needed to develop exposure maps, measurements should be undertaken to confirm the veracity of the assumptions used in the estimates. Sample surveys may be used to provide an overall picture of the noise exposure. Such surveys would take account of all the relevant characteristics of the noise source. For example motor vehicle emissions may be estimated by calculations involving the types of vehicles, their number, their age and the characteristic properties of the road surface.

In developing countries, there is usually a lack of appropriate statistical information to produce noise exposure estimates. However, where action is needed to lower noise levels, the absence of comprehensive information should not prevent the development of provisional noise exposure estimates. Basic information about the exposed population, transport systems, industry and other

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relevant factors can be used to calculate provisional noise exposures. These can then be used to develop and implement interim noise management plans. The preliminary exposure estimates can be revised as more accurate information becomes available.

5.3. Noise Exposure Modeling

As indicated in Chapter 2 modeling is a powerful tool for the interpolation, prediction and optimization of control strategies. However, models need to be validated by monitoring data. A strength of models is that they enable examination and comparison of the consequences for noise exposure of the implementation of the various options for improving noise. However, the accuracy of the various models available depends on many factors, including the accuracy of the source emissions data and details of the topography (for which a geographical information system may be used). For transportation noise parameters such as the number, type and speed of vehicles, aircraft or trains, and the noise characteristics of each individual event must be known. An example of a model is the annoyance prediction model of the Government of the Netherlands (van den Berg 1996).

5.4. Noise Control Approaches

An integrated noise policy should include several control procedures: measures to limit the noise at the source, noise control within the sound transmission path, protection at the receiver's site, land-use planning, education and raising of public awareness. Ideally, countries should give priority to precautionary measures that prevent noise, but they must also implement measures to mitigate existing noise problems.

5.4.1. Mitigation measures

The most effective mitigation measure is to reduce noise emissions at the source. Therefore, regulations with noise level limits for the main noise sources should be introduced.

Road traffic noise. Limits on the noise emission of vehicles have been introduced in many countries (Sandberg 1995). Such limits, together with the relevant measuring methods, should also be introduced in other regions of the world. Besides these limits a special class of "low-noise trucks" has been introduced in Europe. These trucks follow state-of-the-art noise control and are widely used in Austria and Germany (Lang 1995). Their use is encouraged by economic incentives; for example, low-noise trucks are excepted from a night-time ban on certain routes, and their associated taxes are lower than for other trucks. In Europe, the maximum permissible noise levels range from 69 dBA for motor vehicles to 77 dBA for cars, and 83 dBA for heavy two-wheeled vehicles to 84 dBA for trucks. A number of European Directives give permissible sound levels for motor vehicles and motorcycles (EU 1970; EU 1978; EU 1996a; EU 1997). In addition to noise level limits for new vehicles (type test), noise emissions of vehicles already in use should be controlled regularly. Limits on the sound pressure levels for vehicles reduce the noise emission from the engines.

However, the main noise from traffic on highways is rolling noise. This may be reduced by quiet road surfaces (porous asphalt, "drain asphalt") or by selection of quiet tires. Road traffic

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noise may also be reduced by speed limits, provided the limits are enforced. For example, reducing the speed of trucks from 90 to 60 km/h on concrete roads would reduce the maximum sound pressure level by 5 dB, and the equivalent sound pressure level by 4 dB. Decreasing the speed of cars from 140 to 100 km/h would result in the same noise reduction (WHO 1995a). In the central parts of cities a speed limit of 30 km/h may be introduced. At 30 km/h cars produce maximum sound pressure levels that are 7 dB lower, and equivalent sound pressure levels that are 5 dB lower, than cars driving at 50 km/h.

Noise emission from road traffic may be further reduced by a night-time ban for all vehicles, or especially for heavy vehicles. Traffic management designed to ensure uniform traffic flow in towns also serves to reduce noise. "Low-noise behaviour" of drivers should be encouraged as well, by advocating defensive driving manners. In some countries, car drivers use their horns frequently, which results in noise with high peak levels. The unnecessary use of horns within cities should be forbidden, especially during night-time, and this rule should be enforced.

Railway noise and noise from trams. The main noise sources are the engine and the wheel-rail contact. Noise at the source can be reduced by well-maintained rails and wheels, and by the use of disc brakes. Sound pressure levels may vary by more than 10 dB, depending on the type of railway material. Replacement of steel wheels by rubber wheels could also reduce noise from railways and trams substantially. Other measures include innovations in engine and track technology (Moehler 1988; Öhrström & Skånberg 1996).

Aircraft noise. The noise emission of aircraft is limited by ICAO Annex 16, Chapter 2 and Chapter 3, which estimates maximum potential sound emissions under certification procedures (ICAO 1993). Aircraft following the norms of Chapter 3 represent the state-of-the-art of noise control of the 1970s. In many countries, non-certified aircraft (i.e. aircraft not fulfilling the ICAO requirements) are not permitted and Chapter 2 aircraft may not be registered again. After the year 2002 only Chapter 3 aircraft will be allowed to operate in many countries.

Similar legislation should be adopted in other countries. The use of low-noise aircraft may also be encouraged by setting noise-related charges (that is, landing charges that are related not only to aircraft weight and capacity, but also to noise emission). Examples of systems for noise-related financial charges are given in OECD 1991 (see also OECD-ECMT 1995). Night-time aircraft movements should be discouraged where they impact residential communities. Particular categories of aircraft (such as helicopters, rotorcraft and supersonic aircraft) pose additional problems that require appropriate controls. For subsonic airplanes two EU Directive give the permissible sound levels (EU 1980; EU 1989).

Machines and Equipment. Noise emission has to be considered a main property of all types of machines and equipment. Control measures include design, insulation, enclosure and maintenance.

Consumers should be encouraged to take noise emission into account when buying a product. Declaring the A-weighted sound power level of a product would assist the consumer in making this decision. The introduction of sound labeling is a major tool for reducing the noise emission of products on the market. For example, within the European Community, "permissible sound

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levels" and "sound power levels" have to be stated for several groups of machines; for example, lawn mowers, construction machines and household equipment (EU 1984a-f; EU 1986b,c). For other groups of machines sound level data have been compiled and are state-of-the-art with respect to noise control.

A second step would be the introduction of limits on the sound power levels for certain groups of machines, heating and ventilation systems (e.g. construction machines, household appliances). These limits may be set by law, in recommendations and by consumers, using state-of-the-art measurements. There have also been promising developments in the use of active noise control (involving noise cancellation techniques). These are to be encouraged.

Noise control within the sound transmission path. The installation of noise barriers can protect dwellings close to the traffic source. In several European countries noise barrier regulations have been established (WHO 1995b), but in practice they are often not adequately implemented. These regulations must define:

a. Measuring and calculation methods for deriving the equivalent sound pressure level of road or railway traffic, and schemes for determining the effectiveness of the barrier.

b. The sound pressure limits that are to be achieved by installing barriers.

c. The budgetary provisions.

d. The responsible authority.

Noise protection at the receiver's site. This approach is mainly used for existing situations. However, this approach must also be considered for new and, eventually, for old buildings in noisy areas. Residential buildings near main roads with heavy traffic, or near railway lines, may be provided with sound-proofed windows.

5.4.2. Precautionary measures

With careful planning, noise exposure can be avoided or reduced. A sufficient distance between residential areas and an airport will make noise exposure minimal, although the realization of such a situation is not always possible. Additional insulation of houses can help to reduce noise exposure from railroad and road traffic. For new buildings, standards or building codes should describe the positions of houses, as well as the ground plans of houses with respect to noise sources. The required sound insulation of the façades should also be described. Various countries have set standards for the maximum sound pressure levels in front of buildings and for the minimum sound insulation values required for façades.

Land use planning. Land use planning is one of the main tools for noise control and includes:

a. Calculation methods for predicting the noise impact caused by road traffic, railways,

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airports, industries and others.

b. Noise level limits for various zones and building types. The limits should be based on annoyance responses to noise.

c. Noise maps or noise inventories that show the existing noise situation. The construction of noise-sensitive buildings in noisy areas, or the construction of noisy buildings in quiet areas may thus be avoided.

Suggestions on how to use land use planning tools are given in several dedicated books (e.g. Miller & de Roo 1997). Different zones, such as quiet areas, hospitals, residential areas, commercial and industrial districts, can be characterized by the maximum equivalent sound pressure levels permissible in the zones. Examples of this approach can be found in OECD 1991 (also see OECD-ECMT 1995). More emphasis needs to be given to the design or retrofit of urban centres, with noise management as a priority (e.g. "soundscapes").

It is recommended that countries adopt the precautionary principle in their national noise policies. This principle should be applied to all noise situations where adverse noise effects are either expected or possible, even when the noise is below standard values.

Education and public awareness. Noise abatement policies can only be established if basic knowledge and background material is available, and the people and authorities are aware that noise is an environmental hazard that needs to be controlled. It is, therefore, necessary to include noise in school curricula and to establish scientific institutes to study acoustics and noise control. People working in such institutes should have the option of studying in other countries and exchanging information at international conferences. Dissemination of noise control information to the public is an issue for education and public awareness. Ideally, national and local advisory groups should be formed to promote the dissemination of information, to establish uniform methods of noise measurement and impact assessment, and to participate in the development and implementation of educational and public awareness programmes.

5.5. Evaluation of Control Options

Unless legal constraints in a country prescribe a particular option, the evaluation of control options must take into account technical, financial, social, health and environmental factors. The speed with which control options can be implemented, and their enforceability, must also be considered. Although considerable improvements in noise levels have been achieved in some developed countries, the financial costs have been high, and the resource demands of some of these approaches make them unsuitable for the poorer developing countries.

Technical factors. There needs to be confidence that the selected options are technically practical given the resources of the region. It must be possible to bring a selected option into operation, and maintain the expected level of performance in the long term, given the resources available. This may require regular staff training and other programmes, especially in developing countries.

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Financial factors. The selected options must be financially viable in the long term. This may require a comparative cost-benefit assessment of different options. These assessments must include not only the capital costs of bringing an option into operation, but also the costs of maintaining the expected level of performance in the long term.

Social factors. The costs and benefits of each option should be assessed for social equity, and the potential impact of an option on people's way of life, community structures and cultural traditions must be considered. Impacts may include disruption or displacement of residents, changes of land-use, and impacts on community, culture and recreation. Some impacts can be managed; in other cases, the impacts of an option can be mitigated by substitution of resources or uses.

Health and environmental factors. The costs and benefits of each option should be assessed for health and environmental factors. This may involve use of dose-response relations, or risk assessment techniques.

Effect-oriented and source-oriented principles. Noise control requirements in European countries are typically determined from the effects of noise on health and the environment (effect oriented) (e.g. Gottlob 1995; ten Wolde 1998). Increased noise emissions may be permitted if there would be no adverse health impacts, or if noise standards would not be exceeded. Action may be taken to reduce noise levels when it is shown that adverse health impacts will occur, or when noise levels exceed limits. Other countries base their noise management policies on the requirement for best available technology, or for best available techniques that do not entail excessive cost (source-oriented) (e.g. for aircraft noise, ICAO 1993; for road traffic noise, Sandberg 1995). Most developed countries apply a combination of both source-oriented and effect-oriented principles (EU 1996b; Jansen 1998; ten Wolde 1998).

5.6. Management of Indoor Noise

In modern societies, human beings spend most of their time in indoor environments. Pollution and degradation of the indoor environment cause illness, increased mortality, loss of productivity, and have major economic and social implications. Indoor noise problems are related to inadequate urban planning, design, operation and maintenance of buildings, and to the materials and equipment in buildings. Problems with indoor noise affect all types of buildings, including homes, schools, offices, health care facilities and other public and commercial buildings. The health effects of indoor noise include an increase in the rates of diseases and disturbances described in chapter 2. World-wide, the medical and social cost associated with these illnesses, and the related reduction in human productivity, can result in substantial economic losses.

Protection against noise generated within a building, or originating from outside the building, is a very complex problem. Soundproofing of ceilings, walls, doors and windows against airborne noise is important. Soundproofing of ceilings has to be sufficient to absorb sounds due to treading. Finally, noise emissions from the technological devices in the house must be sufficiently low. Governments should provide measurement protocols and data for use in reducing noise exposures in buildings. Governments should also be encouraged to support

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research on the relationship between noise levels inside buildings and health effects.

5.6.1. Government policy on indoor noise

Many of the problems associated with high noise levels can be prevented at low cost if governments develop and implement an integrated strategy for the indoor environment, in concert with all social and economic partners. Governments should establish a "National Plan for a Sustainable Indoor Noise Environment", that would apply to new construction as well as to existing buildings. Governments should set up a specific structure at an appropriate governmental level to achieve acceptable sound exposure levels within buildings. An example of existing documents that provide guidance and regulations, including strategies and management for the design of buildings, is given by Jansen & Gottlob (1996).

Guidance/education. Because our understanding of indoor noise is still developing, government activity should be focused on raising the awareness of various audiences. This education can take the form of providing general information, as well as providing technical guidance and training on how to minimize indoor noise levels. General information presented in the form of documents, videos, and other media can bring indoor noise issues to the attention of the general public and building professionals, including architects

Research support. Research is needed to develop technology for indoor noise diagnosis, mitigation and control. Efforts are also required to provide economical and practical alternatives for mitigation and control. Better means of measuring the effectiveness of absorption devices are needed; and diagnostic tools that are inexpensive and easy to use also need to be developed to help facility personnel. There is a particular need, too, for improving soundproofing methods, their implementation and for predicting the health effects of soundproofing techniques.

To provide accurate information for use in setting priorities for public health problems, governments should support problem assessment and surveys of indoor noise conditions. Building surveys are also necessary to provide baseline information about building characteristics and noise levels. When combined with occupant health surveys, these studies will help to establish the correlations between noise levels and adverse health effects. Surveys should be conducted to identify building types or vintages in which problems occur more frequently. The results of these studies will support effective risk reduction programmes. Epidemiological studies are also needed to aid in differentiating between noise-related symptoms and those due to other causes. Moreover, epidemiological studies are needed to assist in quantifying the extent of risk for indoor noise levels.

Economic research is needed to measure the costs of indoor noise control strategies to individuals, businesses and society. This includes developing methods for quantifying productivity loss and increased health costs due to noise, and for measuring the costs of various control strategies, including increased soundproofing and source control.

Development of standards and protocols. Efforts should be made to protect public health by setting reasonable noise exposure limits (immission standards) from known dose-response relationships. In cases where dose-response relationships have yet to be determined, but where

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health effects are generally recognized, exposure limits should be set conservatively and take into account risk, economic impact and feasibility. Efforts should also be made to incorporate noise-related specifications into building codes. Areas to target with building codes include ventilation design, building envelope design, site preparation, materials selection and commissioning. Standards and other regulations governing the use of sound proofing materials should also be developed.

Individuals involved in the diagnosis and mitigation of indoor noise problems should be trained in the multidisciplinary nature of the noise field. By instituting a series of credentials that recognize and highlight areas of expertise, consumers would be provided with the information to make informed choices when procuring indoor noise services. Companies which provide such services should be officially accredited. Guidelines or standards for sound emissions of air-conditioners, power generators and other building devices, would also provide useful information for manufacturers, architects, design engineers, building managers and others who play a role in selecting products used indoors.

5.6.2. Design considerations

Site investigation. Potential sites should be evaluated to determine whether they are prone to indoor noise problems. This evaluation should be consistent with national and local land use planning guidelines. Sites should be investigated to determine past uses and whether any sources of sound remain as a result. The potential for outdoor noise being carried to the site from adjacent areas, such as busy streets, should also be evaluated.

Building design. Buildings should be designed to be soundproof, to improve control over indoor noise. Soundproofing requires that outside noise be prevented from entering the building, and this should be estimated as part of the architectural and engineering design process. When soundproofing for outdoor noise, the total indoor noise load and the desired quality of the indoor space should be considered. Adequate soundproofing against outdoor noise is important in residential as well as commercial properties, and should be re-evaluated when interior spaces are rebuilt or renovated.

Indoor Spaces. The architectural layout should aim to reduce noise and provide a good sound quality to the space. This would include designing indoor spaces to have sufficiently short reverberation times. Designers and contractors should be encouraged to use sound-absorbing materials that lead to lower indoor noise levels, and materials with the best sound-absorbing properties should be specified. However, use of these materials should not be the only solution (Harris 1991). Possible conflicts with other environmental demands should also be identified; for example, the special demands by allergic people.

5.6.3. Indoor noise level control

Building maintenance personnel should be trained to understand the indoor noise aspects of their work, and be aware of how their work can directly impact the health and comfort of occupants. Many maintenance activities directly affect indoor noise levels, and some may indicate potential problems. Preventive maintenance is essential for the building systems to operate correctly and

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to provide suitable comfort conditions and low indoor noise levels. Detailed maintenance logs should be kept for all equipment. A schedule should be developed for routine equipment checks and calibration of control system components. Selection of low-noise domestic products should encouraged as far as is possible.

5.6.4. Resolving indoor noise problems

Addressing occupant complaints and symptoms. When complaints are received from occupants of a building, the cognizant authority should be responsive. The initial investigation into the cause of the complaint may be conducted by the in-house management staff, and they should continue an investigation as far as possible. If necessary, they should be responsible for hiring an outside consultant

Building diagnostic procedures. After receiving complaints related to indoor noise levels, facility personnel or consultants should attempt to identify the cause of the problem through an iterative process of information collection and hypothesis testing. To begin, a walkthrough inspection of the building, including the affected areas and the mechanical systems serving these spaces is required. A walkthrough can provide information on the soundproofing system of the building, the sound pathways and sound sources. Visual indicators of sound sources and soundproofing malfunctions should be evaluated first. Symptom logs and schedules of building activities may provide enough additional information to resolve the problem.

If a walkthrough alone does not provide a solution, measurements of sound pressure levels at various locations should be taken, and indoor and ambient levels of noise pollution should be compared. As part of the investigation, the absorption characteristics of walls and ceilings should be evaluated. Sophisticated sampling methods may be necessary to provide proof of a problem to the building owner or other responsible party. The results may be used to confirm a hypothesis or ascertain the source of the indoor noise problem. Whenever a problem is discovered during the investigation, a remedy to the situation should be attempted and a determination made of whether the complaint has been resolved.

In some cases, it should be recognized that difficulties in interpreting the sampling results may exist. The costs of certain types of testing should also be taken into account. Simple, cost-effective screening methods should be developed to make sampling a more attractive option for both investigators and clients. Finally, it must be remembered that several factors cause symptoms similar to those induced by noise pollution. Examples include air pollutants, ergonomics, lighting, vibration and psychosocial factors. Consequently, any investigation of noise complaints should also evaluate non-noise factors.

5.7. Priority Setting in Noise Management

Priorities in noise management will differ between countries, according to policy objectives, needs and capabilities. Priority setting in noise management refers to prioritizing health risks and concentrating on the most important sources of noise. For effective noise management, the goals, policies and noise control schemes have to be defined. Goals for noise management include eliminating noise, or reducing noise to acceptable levels, and avoiding the adverse health

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effects of noise on human health. Policies for noise management encompass laws and regulations for setting noise standards and for ensuring compliance. The amount of information to be included in low-noise implementation plans and the use of cost-benefit comparisons also fall within the purview of noise management policies. Techniques for noise control include source control, barriers in noise pathways and receiver protection. Adequate calculation models for noise propagation, as well as programmes for noise monitoring, are part of an overall noise control scheme.

As emphasized above, a framework for a political, regulatory and administrative approach is required to guarantee the consistent and transparent promulgation of noise standards. This ensures a sound and practical framework for risk-reducing measures and for the selection of abatement strategies.

5.7.1. Noise policy and legislation

Noise is both a local and a global problem. Governments in every country have a responsibility to set up policies and legislation for controlling community noise. There is a direct relationship between the level of development in a country and the degree of noise pollution impacting its people. As a society develops, it increases its level of urbanization and industrialization, and the extent of its transportation system. Each of these developments brings an increase in noise load. Without appropriate intervention the noise impact on communities will escalate (see Figure 5.3). If governments implement only weak noise policies and regulations, they will not be able to prevent a continuous increase in noise pollution and associated adverse health effects. Failure to enforce strong regulations is ineffective in combating noise as well.

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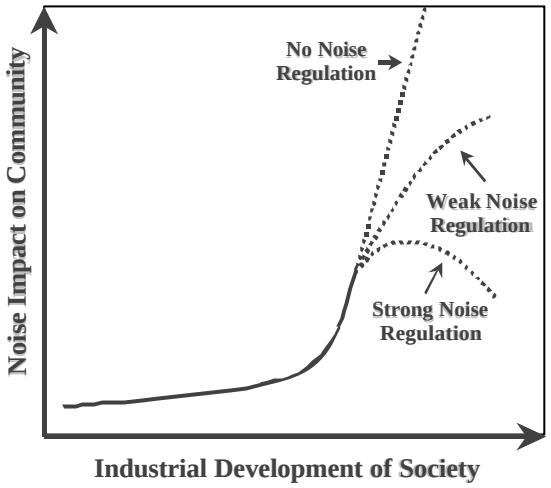


Figure 5.3. Relationship between noise regulation and impact with development (from Hede 1998b)

Policies for noise regulatory standards at the municipal, regional, national and supranational levels are usually determined by the legislatures. The regulatory standards adopted strongly depend on the risk management strategies of the legislatures, and can be influenced by sociopolitical considerations and/or international agreements. Although regulatory standards may be country specific, in general the following issues are taken into consideration:

a. Identification of the adverse public health effects that are to be avoided.

b. Identification of the population to be protected.

c. The type of parameters describing noise and the limit applicable to the parameters.

d. Applicable monitoring methodology and its quality assurance.

e. Enforcement procedures to achieve compliance with noise regulatory standards within a defined time frame.

f. Emission control measures and emission regulatory standards.

g. Immission standards (limits for sound pressure levels).

h. Identification of authorities responsible for enforcement.

i. Resource commitment.

Regulatory standards may be based solely on scientific and technical data showing the adverse effects of noise on public health. But other aspects are usually considered, either when setting standards or when designing appropriate noise abatement measures. These other aspects include the technological feasibility, costs of compliance, prevailing exposure levels, and the social,

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economic and cultural conditions. Several standards may be set. For example, effect-oriented regulatory standards may be set as a long-term goal, while less-stringent standards are adopted for the short term. As a consequence, noise regulatory standards differ widely from country to country (WHO 1995a; Gottlob 1995).

Noise regulatory standards can set the reference point for emission control and abatement policies at the national, regional or municipal levels, and can thus strongly influence the implementation of noise control policies. In many countries, exceeding regulatory standards is linked to an obligation to develop abatement action plans at the municipal, regional or national levels (low-noise implementation plans). Such plans have to address all relevant sources of noise pollution.

5.7.2. Examples of noise policies

Different countries have adopted a range of policies and regulations for noise control. A number of these are outlined in this section as examples.

Argentina. In Argentina, a national law recently limited the daily 8-h exposure to industrial noise to 80 dB, and it has had beneficial effects on hearing impairment and other hearing disorders among workers. In general, industry has responded by introducing constant controls on noise sources, combined with hearing tests and medical follow-ups for workers. Factory owners have recruited permanent health and safety engineers who control noise, supply advice on how to make further improvements, and routinely assess excessive noise levels. The engineers also provide education in personal protection and in the correct use of ear plugs, mufflers etc.

At the municipal level two types of noise have been considered. Unnecessary noise, which is forbidden; and excessive noise, which is defined for neighbourhood activities (zones), and for which both day and night-time maximum limits have been introduced. The results have been relatively successful in mitigating unwanted noise effects. At the provincial level, similar results have been accomplished for many cities in Argentina and Latin America.

Australia. In Australia, the responsibility for noise control is shared primarily by state and local governments. There are nationally-agreed regulatory standards for airport planning and new vehicle noise emissions. The Australian Noise Exposure Forecast (ANEF) index is used to describe how much aircraft noise is received at locations around an airport (DoTRS 1999). Around all airports, planning controls restrict the construction of dwellings within the 25 ANEF exposure contour and require sound insulation for those within 20 ANEF. Road traffic noise limits are set by state governments, but vary considerably in both the exposure metric and in maximum allowable levels. New vehicles are required to comply with stringent design rules for noise and air emissions. For example, new regulation in New South Wales adopts LAeq as the metric and sets noise limits of 60 dBA for daytime, and 55 dBA for night-time, along new roads. Local governments set regulations restricting noise emissions for household equipment, such as air conditioners, and the hours of use for noisy machines such as lawn mowers.

Europe. In Europe, noise legislation is not generally enforced. As a result, environmental noise

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levels are often higher than the legislated noise limits. Moreover, there is a gap between long-term political goals and what represents a "good acoustical environment". One reason for this gap is that noise pollution is most commonly regulated only for new land use or for the development of transportation systems, whereas enlargements at existing localities may be approved even though noise limits or guideline values are already surpassed (Gottlob 1995). A comprehensive overview of the noise situation in Europe is given in the Green Paper (EU 1996b), which was established to give noise abatement a higher priority in policy making. The Green Paper outlines a new framework for noise policy in Europe with the following options for future action:

a. Harmonizing the methods for assessing noise exposure, and encouraging the exchange of information among member states.

b. Establishing plans to reduce road traffic noise by applying newer technologies and fiscal instruments.

c. Paying more attention to railway noise in view of the future extension of rail networks.

d. Introducing more stringent regulation on air transport and using economic instruments to encourage compliance.

e. Simplifying the existing seven regulations on outdoor equipment by proposing a Framework Directive that covers a wider range of equipment, including construction machines and others.

Pakistan. In Pakistan, the Environmental Protection Agency is responsible for the control of air pollution nationwide. However, only recently have controls been enforced in Sindh in an attempt to raise public awareness and carry out administrative control on road vehicles producing noise (Zaidi, personal communication).

South Africa. In South Africa, noise control is three decades old. It began with codes of practice issued by the South African Bureau of Standards to address noise pollution in various sectors of the country (e.g. see SABS 1994 1996; and the contribution of Grond in Appendix 2). In 1989, the Environment Conservation Act made provision for the Minister of Environmental Affairs and Tourism to make regulations for noise, vibration and shock (DEAT 1989). These regulations were published in 1990 and local authorities could apply to the Minister to make them applicable in their areas. Later, the act was changed to make it obligatory for all authorities to apply the regulations. However, according to the new Constitution of South Africa of 1996, legislative responsibility for noise control rests exclusively with provincial and local authorities. The noise control regulations will apply to local authorities in South Africa as soon as they are published in the provinces. This will not only give local authorities the power to enforce the regulations, but also place an obligation on them to see that the regulations are enforced.

Thailand. In 1996, noise pollution regulations in Thailand stipulated that not more than 70 dBA LAeq,24h should be allowed in residential areas, and the maximum level of noise in industry

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should be no more than 85 dBA Leq 8h (Prasansuk 1997).

United States of America. Environmental noise was not addressed as a national policy issue in the USA until the implementation of the Noise Control Act of 1972. This congressional act directed the US Environmental Protection Agency to publish scientific information about noise exposure and its effects, and to identify acceptable levels of noise exposure under various conditions. The Noise Control Act was supposed to protect the public health and well-being with an adequate margin of safety. This was accomplished in 1974 with the publication of the US EPA "Levels Document" (US EPA 1974). It addressed issues such as the use of sound descriptions to describe sound exposure, the identification of the most important human effects resulting from noise exposure, and the specification of noise exposure criteria for various effects. Subsequent to the publication of the US EPA "Levels Document", guidelines for conducting environmental impact analysis were developed (Finegold et al. 1998). The day-night average sound level was thus established as the predominant sound descriptor for most environmental noise exposure.

It is evident from these examples that noise policies and regulations vary considerably across countries and regions. Moves towards global noise policies need to be encouraged to ensure that the world population gains the maximum health benefits from new developments in noise control.

5.7.3. Noise emission standards have proven to be inadequate

Much of the progress towards solving the noise pollution problem has come from advanced technology, which in turn has come about mainly as a result of governmental regulations (e.g. OECD-ECMT 1995). So far, however, the introduction of noise emission standards for vehicles has had limited impact on exposure to transportation noise, especially from aircraft and road traffic noise (Sandberg 1995). In part, this is because changes in human behaviour (of polluters, planners and citizens) have tended to offset some of the gains made. For example, mitigation efforts such as developing quieter vehicles, moving people to less noise-exposed areas, improving traffic systems and direct noise abatement and control (sound insulation, barriers etc.), have been counteracted by increases in the number of roads and highways built, by the number of traffic movements, and by higher driving speeds and the number of kilometers driven (OECD 1991; OECD-ECMT 1995).

Traffic planning and correction policies may diminish the number of people exposed to the very high community noise levels (>70 dB LAeq), but the number exposed to moderately high levels (55-65 dB LAeq) continues to increase in industrialized countries (Stanners & Bordeau 1995). In developing countries, exposure to excessive sound pressure levels (>85 dB LAeq), not only from occupational noise but also from urban, environmental noise, is the major avoidable cause of permanent hearing impairment (Smith 1998). Such sound pressure levels can also be reached by leisure activities at concerts, discotheques, motor sports and shooting ranges; by music played back in headphones; and by impulse noises from toys and fireworks.

A substantial growth in air transport is also expected in the future. Over the next 10 years large international airports may have to accommodate a doubling in passenger movements. General

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aviation noise at regional airports is also expected to increase (Large & House 1989). Although jet aircraft are expected to become less noisy due to regulation of noise emissions (ICAO 1993), the number of passengers is expected to increase. Increased air traffic movement between 1980 and 1990 is considered to be the main reason for the average 22% increase in the number of people exposed to noise above 67 dB LAeq at German airports (OECD 1993).

5.7.4. Unsustainable trends in noise pollution future policy planning

A number of trends are expected to increase environmental noise pollution, and are considered to be unsustainable in the long term. The OECD (1991) identified the following factors to be of increasing importance in the future:

a. The expanding use of increasingly powerful sources of noise.

b. The wider geographical dispersion of noise sources, together with greater individual mobility and spread of leisure activities.

c. The increasing invasion of noise, particularly into the early morning, evenings and weekends.

d. The increasing public expectations that are closely linked to increases in incomes and in education levels.

Apart from these, increased noise pollution is also linked to systemic changes in business practices (OECD-ECMT 1995). By accepting a just-in-time concept in transportation, products and components are stored in heavy-duty vehicles on roads, instead of in warehouses; and workers are recruited as temporary consultants just in time for the work, instead of as long-term employees.

In addition, the OECD (1991) report forecasts:

a. A strengthening of present noise abatement policies and their applications.

b. A further sharpening of emission standards.

c. A co-ordination of noise abatement measures and transport planning, to specifically reduce mobility.

d. A co-ordination of noise abatement measures with urban planning.

Planners need to know the likely effects of introducing a new noise source, or of increasing the level of an existing source, on the noise pollution in a community. Policy makers, when considering applications for new developmental projects, must take into account maximum levels, continuous equivalent sound pressure levels of both the background and the new noise source, the frequency of noise occurrence and the operating times of major noise sources.

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5.7.5. Analysis of the impact of environmental noise

The concept of an environmental noise impact analysis (ENIA) is central to the philosophy of managing environmental noise. An ENIA should be required before implementing any project that would significantly increase the level of environmental noise in a community (typically, greater than a 5dB increase). The first step in performing an ENIA is to develop a baseline description of the existing noise environment. Next, the expected level of noise from a new source is added to the baseline exposure level to produce the new overall noise level. If the new total noise level is expected to cause an unacceptable impact on human health, trade-off analyses should then be performed to assess the cost, technical feasibility and community acceptance of noise mitigation measures. It is strongly recommended that countries develop standardized procedures for performing ENIAs (Finegold et al. 1998; SABS 1998).

Assessment of adverse health effects. In setting noise standards (for example on the basis of these guidelines), the adverse health effects from which the population is to be protected need to be defined. Health effects range from hearing impairment to sleep disturbance, speech interference to annoyance. The distinction between adverse and non-adverse effects sometimes poses considerable difficulties. Even the elaborate definition of an adverse health effect given in Chapter 3 incorporates significant subjectivity and uncertainty. More serious noise effects, such as hearing impairment or permanent threshold shift, are generally accepted as adverse. Consideration of health effects that are both temporary and reversible, or that involve functional changes with uncertain clinical significance, requires a judgement on whether these less-serious effects should be considered when deriving guideline values. Judgements as to the adversity of health effects may differ between countries, because of factors such as cultural backgrounds and different levels of health status.

Estimation of the population at risk. The population at risk is that part of the population in a given country or community that is exposed to enhanced levels of noise. Each population has sensitive groups or subpopulations that are at higher risk of developing health effects due to noise exposure. Sensitive groups include individuals impaired by concurrent diseases or other physiological limitations and those with specific characteristics that makes them more vulnerable to noise (e.g. premature babies; see the contribution of Zaidi in Appendix 2). The sensitive groups in a population may vary across countries due to differences in medical care, nutritional status, lifestyle and demographic factors, prevailing genetic factors, and whether endemic or debilitating diseases are prevalent.

Calculation of exposure-response relationships. In developing standards, regulators should consider the degree of uncertainty in the exposure-response relationships provided in the noise guidelines. Differences in the population structure (age, health status), climate (temperature, humidity) and geography (altitude, environment) can influence the prevalence and severity of noise-related health effects. In consequence, modified exposure-response relationships may need to be applied when setting noise standards.

Assessment of risks and their acceptability. In the absence of distinct thresholds for the onset of health effects, regulators must determine what constitutes an acceptable health risk for the population and select an appropriate noise standard to protect public health. This is also true in

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cases where thresholds are present, but where it would not be feasible to adopt noise guidelines as standards because of economical and/or technical constraints. The acceptability of the risks involved, and hence the standards selected, will depend on several factors. These include the expected incidence and severity of the potential effects, the size of the population at risk, the perception of related risks, and the degree of scientific uncertainty that the effects will occur at any given noise level. For example, if it is suspected that a health effect is severe and the size of the population at risk is large, a more cautious approach would be appropriate than if the effect were less troubling, or if the population were smaller.

Again, the acceptability of risk may vary among countries because of differences in social norms, and the degree of adversity and risk perception by the general population and stakeholders. Risk acceptability is also influenced by how the risks associated with noise compare with risks from other pollution sources or human activities.

5.7.6. Cost-benefit analysis

In the derivation of noise standards from noise guidelines two different approaches for decision making can be applied. Decisions can be based purely on health, cultural and environmental consequences, with little weight to economic efficiency. This approach has the objective of reducing the risk of adverse noise effects to a socially acceptable level. The second approach is based on a formal cost-effectiveness, or cost-benefit analysis (CBA). The objective is to identify control actions that achieve the greatest net economic benefit, or are the most economically efficient. The development of noise standards should account for both extremes, and involve stakeholders and assure social equity to all the parties involved. It should also provide sufficient information to guarantee that stakeholders understand the scientific and economic consequences.

To determine the costs of control action, the abatement measures used to reduce emissions must be known. This is usually the case for direct measures at the source and these measures can be monetarized. Costs of action should include all costs of investment, operation and maintenance. It may not be possible to monetarize indirect measures, such as alternative traffic plans or change in behaviour of individuals.

The steps in a cost-benefit analysis include:

a. The identification and cost analysis of control action (such as emission abatement strategies and tactics).

b. An assessment of noise and population exposure, with and without the control action.

c. The identification of benefit categories, such as improved health and reduced property loss.

d. A comparison of the health effects, with and without control action.

e. A comparison of the estimated costs of control action with the benefits that accrue from such action.

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f. A sensitivity and uncertainty analysis.

Action taken to reduce one pollutant may increase or decrease the concentration of other pollutants. These additional effects should be considered, as well as pollutant interactions that may lead to double counting of costs or benefits, or to disregarding some costly but necessary action. Due to different levels of knowledge about the costs of control action and health effects, there is a tendency to overestimate the cost of control action and underestimate the benefits.

CBA is a highly interdisciplinary task. Appropriately applied, it is a legitimate and useful way of providing information for managers who must make decisions that impact health. CBA is also an appropriate tool for drawing the attention of politicians to the benefits of noise control. In any case, however, a CBA should be peer-reviewed and never be used as the sole and overriding determinant of decisions.

5.7.7. Review of standard setting

The setting of standards should involve stakeholders at all levels (industry, local authorities, non-governmental organizations and the general public), and should strive for social equity or fairness to all parties involved. It should also provide sufficient information to guarantee that the scientific and economic consequences of the proposed standards are clearly understood by the stakeholders. The earlier that stakeholders are involved, the more likely is their co-operation. Transparency in moving from noise guidelines to noise standards helps to increase public acceptance of necessary measures. Raising public awareness of noise-induced health effects (changing of risk perception) also leads to a better understanding of the issues involved (risk communication) and serves to obtain public support for necessary control action, such as reducing vehicle emissions. Noise standards should be regularly reviewed, and revised as new scientific evidence emerges.

5.7.8. Enforcement of noise standards: Low-noise implementation plans

The main objective of enforcing noise standards is to achieve compliance with the standards. The instrument used to achieve this goal is a Low-Noise Implementation Plan (LNIP). The outline of such a plan should be defined in the regulatory policies and should use the tactical instruments discussed above. A typical low-noise implementation plan includes:

a. A description of the area to be regulated.

b. An emissions inventory.

c. A monitored or simulated inventory of noise levels.

d. A comparison of the plan with emissions and noise standards or guidelines.

e. An inventory of the health effects.

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f. A causal analysis of the health effects and their attribution to individual sources.

g. An analysis of control measures and their costs.

h. An analysis of transportation and land-use planning.

i. Enforcement procedures.

j. An analysis of the effectiveness of the noise management procedures.

k. An analysis of resource commitment.

l. Projections for the future.

As the LNIP also addresses the effectiveness of noise control technologies and policies, it is very much in line with the Noise Control Assessment Programme (NCAP) proposed recently (Finegold et al. 1999).

5.8. Conclusions on Noise Management

Successful noise management should be based on the fundamental principles of precaution, the polluter pays and prevention. The noise abatement strategy typically starts with the development of noise standards or guidelines, and the identification, mapping and monitoring of noise sources and exposed communities. A powerful tool in developing and applying the control strategy is to make use of modeling. These models need to be validated by monitoring data. Noise parameters relevant to the important sources of noise must be known. Indoor noise exposures present specific and complex problems, but the general principles for noise management hold. The main means for noise control in buildings include careful site investigations, adequate building designs and building codes, effective means for addressing occupant complaints and symptoms, and building diagnostic procedures.

Noise control should include measures to limit the noise at the source, to control the sound transmission path, to protect the receiver's site, to plan land use, and to raise public awareness. With careful planning, exposure to noise can be avoided or reduced. Control options should take into account the technical, financial, social, health and environmental factors of concern. Cost-benefit relationships, as well as the cost-effectiveness of the control measures, must be considered in the context of the social and financial situation of each country. A framework for a political, regulatory and administrative approach is required for the consistent and transparent promulgation of noise standards. Examples are given for some countries, which may guide others in their development of noise policies.

Noise management should:

a. Start monitoring human exposures to noise.

b. Have health control require mitigation of noise emissions. The mitigation procedures

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should take into consideration specific environments such as schools, playgrounds, homes and hospitals; environments with multiple noise sources, or which may amplify the effects of noise; sensitive time periods, such as evenings, nights and holidays; and groups at high risk, such as children and the hearing impaired.

c. Consider noise consequences when making decisions on transport-system and land-use planning.

d. Introduce surveillance systems for noise-related adverse health effects.

e. Assess the effectiveness of noise policies in reducing noise exposure and related adverse health effects, and in improving supportive "soundscapes."

a. Adopt these Guidelines for Community Noise as long-term targets for improving human health.

g. Adopt precautionary actions for sustainable development of acoustical environments.

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6. Conclusions And Recommendations

6.1. Implementation of the Guidelines

The potential health effects of community noise include hearing impairment; startle and defense reactions; aural pain; ear discomfort speech interference; sleep disturbance; cardiovascular effects; performance reduction; and annoyance responses. These health effects, in turn, can lead to social handicap; reduced productivity; decreased performance in learning; absenteeism in the workplace and school; increased drug use; and accidents. In addition to health effects of community noise, other impacts are important such as loss of property value. In these guidelines the international literature on the health effects of community noise was reviewed and used to derive guideline values for community noise. Besides the health effects of noise, the issues of noise assessment and noise management were also addressed. Other issues considered were priority setting in noise management; quality assurance plans; and the cost-efficiency of control actions. The aim of the guidelines is to protect populations from the adverse health impacts of noise.

The following recommendations were considered appropriate:

a. Governments should consider the protection of populations from community noise as an integral part of their policy for environmental protection.

b. Governments should consider implementing action plans with short-term, medium-term and long-term objectives for reducing noise levels.

c. Governments should adopt the health guidelines for community noise as targets to be achieved in the long-term.

a. Governments should include noise as an important issue when assessing public health matters and support more research related to the health effects of noise exposure.

a. Legislation should be enacted to reduce sound pressure levels, and existing legislation should be enforced.

b. Municipalities should develop low-noise implementation plans.

c. Cost-effectiveness and cost-benefit analyses should be considered as potential instruments when making management decisions.

d. Governments should support more policy-relevant research into noise pollution (see section 6.3).

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6.2. Further WHO Work on Noise

The WHO Expert Task Force proposed several issues for future work in the field of community noise. These are:

a. The WHO should consider updating the guidelines on a regular basis.

b. The WHO should provide leadership and technical direction in defining future research priorities into noise.

c. The WHO should organize workshops on the application of the guidelines.

d. The WHO should provide leadership and co-ordinate international efforts to develop techniques for the design of supportive sound environments (e.g. 'soundscapes").

e. The WHO should provide leadership for programmes to assess the effectiveness of health-related noise policies and regulations.

f. The WHO should provide leadership and technical direction for the development of sound methodologies for EIAP and EHIAP.

g. The WHO should encourage further investigation into using noise exposure as an indicator of environmental deterioration, such as found in black spots in cities.

a. The WHO should provide leadership, technical support and advice to developing countries, to facilitate the development of noise policies and noise management.

6.3. Research Needs

In the publication entitled "Community Noise", examples of essential research and development needs were given (Berglund & Lindvall 1995). In part, the scientific community has already addressed these issues.

A major step forward in raising public awareness and that of decision makers is the recommendation of the present Expert Task Force to concentrate more **on variables which have monetary consequences. This means that research should consider the dose-response relationships between sound pressure levels and politically relevant variables, such as noise-induced social handicap, reduced productivity, decreased performance in learning, workplace and school absenteeism, increased drug use and accidents.**

There is also a need for continued efforts to understand community noise and its effects on the health of the world population. Below is a list of essential research needs in non-prioritized order. Research priorities may vary over time and by place and capabilities. The main goal in suggesting these research activities is to improve the scientific basis for policy-making and noise management. This will protect and improve the public health with regard to the effects of community noise pollution.

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Research related to measurement and monitoring systems for health effects

- Development of a global noise impact monitoring study. The study should be designed to obtain longitudinal data across countries on the health effects on communities of various types of environmental noise. A baseline survey could be undertaken in both developed and developing countries and monitoring surveys conducted every 3-5 years. Since a national map of noise exposure from all sources would be prohibitively expensive, periodic surveys of a representative sample of about 1000 people (using standard probability techniques) could be reliably generalized to the whole population of a country with an accuracy of plus-or-minus 3%. A small number of standard questions could be used across countries to obtain comparative data on the impact of all the main types of noise pollution.

- Development of continuous monitoring systems for direct health effects in critical locations.
- Development of standardized methods for low-cost assessment of local sound levels by measurement or model calculations.
- Development of instruments appropriate for local/regional surveys of people's perceptions of their noise/sound environments.

- Protocols for reliable measurements of high-frequency hearing (8000 Hz and above) and for evaluation of such measures as early biomarkers for hearing impairment/deficits.

Research related to combined noise sources and combined health effects

- Research into the combined health effects of traffic noise, with emphasis on the distribution of sound levels over time and over population sub-environments (time-activity pattern).
- Comprehensive studies on combined noise sources and their combinations of health effects in the 3 large areas of transport (road, rail and aircraft).
- Procedures for evaluating the various health effects of complex combined noise exposures over 24 hours on vulnerable groups and on the general population.
- Methods for assessing the total health effect from noise immission (and also other pollution) in sensitive areas (for example, airports, city centers and heavily-trafficked highways)

Research related to direct and/or long-term health effects (sensitive risk groups, sensitive areas and combined exposures)

- Identification of potential risk groups, including identification of sensitive individuals (such as people with particular health problems; people dealing with complex cognitive tasks; the blind; the hearing impaired; young children and the elderly), differences between sexes, discrimination of risk among age groups, and influence of transportation noise on pregnancy course and on fetal development.

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- Studies of dose-response relationships for various effects, and for continuous transportation noise at relatively low levels of exposure and low number of noise events per unit time (including traffic flow composition).
- Studies on the perception of control of noise exposure, genetic traits, coping strategies and noise annoyance as modifiers of the effects of noise on the cardiovascular system, and as causes of variability in individual responses to noise.
- Prospective longitudinal studies of transportation noise that examine physiological measures of health, including standardized health status inventory, blood pressure, neuro-endocrine and immune function.
- Knowledge on the health effects of low-frequency components in noise and vibration.
-

Research related to indirect or after-effects of noise exposure

- Field studies on the effects of exposure to specific sounds such as aircraft noise and loud music, including effects such as noise-induced temporary and permanent threshold shifts, speech perception and misperception, tinnitus and information retrieval.
- Studies on the influence of noise-induced sleep disturbance on health, work performance, accident risk and social life.
- Assessment of dose-response relationships between sound levels and politically relevant variables such as noise-induced social handicap, reduced productivity, decreased performance in learning, workplace and school absenteeism, increased drug use and accidents.
- Determination of the causal connection between noise and mental health effects, annoyance and (spontaneous) complaints in areas such as around large airports, heavy-trafficked highways, high-speed rail tracks and heavy vehicles transit routes. The connections could be examined by longitudinal studies, for example.
- Studies on the impact of traffic noise on recovery from noise-related stress, or from nervous system hyperactivity due to work and other noise exposures.

Research on the efficiency of noise abatement policies which are health based

- Determination of the accuracy and effectiveness of modern sound insulation (active noise absorption), especially in residential buildings, in reducing the long-term effects of noise on annoyance/sleep disturbance/speech intelligibility. This can be accomplished by studying sites that provide data on remedial activities and changes in behavioral patterns among occupants.
- Evaluation of environmental (area layout, architecture) and traffic planning (e.g. rerouting) interventions on annoyance, speech interference and sleep disturbance.
- Comparative studies to determine whether children and the hearing impaired have equitable access to healthier lives when compared with normal adults in noise-exposed areas.

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- Development of a methodology for the environmental health impact assessment of noise that is applicable in developing as well as developed countries.

Research into positive acoustical needs of the general population and vulnerable groups

- Development of techniques/protocols for the design of supportive acoustical environments for the general population and for vulnerable groups. The protocols should take into account time periods that are sensitive from physiological, psychological and socio-cultural perspectives.

- Studies to characterize good "restoration areas" which provide the possibility for rest without adverse noise load.

- Studies to assess the effectiveness of noise policies in maintaining and improving soundscapes and reducing human exposures.

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Appendix 1 : Bibliographical References

AAP 1997 Noise: A hazard for the fetus and newborn. American Academy of Pediatrics. Committee on Environmental Health. Pediatrics 100: 724-27.

Altena K 1987 Medische gevolgen van lawaai. Report GA-HR-03-01, Ministerie van Volkshuisvesting, Ruimtelijke Ordening en Milieu, Den Haag, Netherlands.

ANSI 1994 American National Standard Acoustical Terminology. ANSI S.1.-1994, American National Standards Institute, Inc., New York, NY, USA.

ANSI 1995 American National Standard Bioacoustical Terminology. ANSI S3.20-1995, American National Standards Institute, Inc., New York, NY, USA.

ANSI 1997 Methods for the Calculation of the Speech Intelligibility Index, American National Standard 3.5, American National Standard Institute, New York, NY, USA.

ASTM 1992 Standard Guide for Field Measurement of Airborne Sound Insulation of Building Facades and Facade Elements, ASTM E966, American Society For Testing and Materials, Philadelphia, West Conshohocken, PA, USA.

ASTM 1994a Classification of Rating Sound Insulation, ASTM E413, American Society For Testing and Materials, Philadelphia, PA, USA .

ASTM 1994b Standard Classification for Determination of Outdoor-Indoor Transmission Class, ASTM E1332, American Society For Testing and Materials, Philadelphia, PA, USA.

ASTM 1994c Standard Classification for Determination of Impact Insulation Class (IIC), ASTM E989, American Society For Testing and Materials, Philadelphia, PA, USA.

ASTM 1996 Standard Test Method for Laboratory Measurement of Impact Sound Transmission through Floor-ceiling Assemblies using the Tapping Machine, ASTM E492, American Society For Testing and Materials, Philadelphia, PA, USA.

ASTM 1997 Standard Test Method for Laboratory Measurement of Airborne Sound Transmission Loss of Building Partitions and Elements, ASTM E90, American Society For Testing and Materials, Philadelphia, PA, USA .

Axelsson A, Arvidson I, Jerson T 1986 Hearing in fishermen and coastguards. In R.J. Salvi, D. Henerson, R.P. Hamernik and V. Colletti (eds.), Basic and Applied Aspects of Noise-Induced Hearing Loss, pp. 513-20. Plenum Press, New York**.**

Axelsson A and Prasher DK 1999. Tinnitus: A warning signal to teenagers attending discotheques? Noise & Health 2: 1-2.

Babisch W 1998a Epidemiological studies on cardiovascular effects of traffic noise. In D. Prasher, D. and L. Luxon, L. (eds.) Advances in Noise Series, Vol. I: Biological Effects, pp. 312-327. Whurr Publishers, London, UK.

Babisch W 1998b Epidemiological studies of cardiovascular effects of traffic noise. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects ’98), Vol. 1, pp. 221-229. Noise Effects ’98 PTY Ltd., Sydney, Australia.

Babisch W and Ising H 1989 Zum Einfluss von Musik in Diskotheken auf die Hörfähigkeit von Jugendlichen. Sozial- und Präventivmedizin 34: 239-243.

Babisch W, Ising H, Gallacher JEJ, Sweetnam PM, Elwood PC 1998 The Caerphilly and Speedwell studies, 10 year follow up. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects ’98), Vol. 1, pp. 230-35. Noise Effects ’98 PTY Ltd., Sydney, Australia.

010376

Babisch W, Ising H, Gallacher JE, Sweetnam PM, Elwood PC 1999 Traffic noise and cardiovascular risk: The Caerphilly and Speedwell studies, third phase 10-year follow up. Archives of Environmental Health 54: 210-216.

Beersma DGM and Altena K 1995 Critical evaluation of the 1992 report of a field study of aircraft noise and sleep disturbance. Newsletter of the European Sleep Research Society 1: 5-7.

Bendat JS and Piersol AG 1971 Random Data: Analysis and Measurement Procedures. Wiley, New York, USA.

Beranek LL 1971 Noise and Vibration Control. McGraw Hill, New York, USA.

Beranek LL 1989 Balanced Noise Criterion (NCB) Curves. Journal of the Acoustical Society of America 86: 650-664.

Berglund B 1998 Community noise in a public health perspective In V.C. Goodwin and D.C. Stevenson (eds.) Inter Noise 98. Sound and Silence: Setting the Balance, Vol. 1, pp. 19-24. New Zealand Acoustical Society, Auckland, New Zealand.

Berglund B, Hassmén P, Job RFS 1996 Sources and effects of low-frequency noise. Journal of the Acoustical Society of America, 99: 2985-3002.

Berglund B and Lindvall T (Eds.) 1995 Community Noise. Document prepared for the World Health Organization. Archives of the Center for Sensory Research, 2: 1-195. A reprint of this document with corrections of language and references has been published in 1998. The 1995 document can be addressed on the Internet address www.who.int/peh/.

Berglund B and Nilsson ME 1997 Empirical issues concerning annoyance models for combined community noise. In F. Augustinovicz (ed.) Inter Noise 97. Help Quiet the World for a Higher Quality Life, Vol. 2, pp. 1053-58. Noise Control Foundation, Poughkeepsie, NY, USA.

Berglund B, Berglund U, Lindvall T 1976 Scaling loudness, noisiness and annoyance of community noise. Journal of the Acoustical Society of America 60: 1119-1125.

Berglund B, Berglund U, Lindberg S 1986 Loudness of impulse sound from different weapons. In R. Lutz (ed.) Inter Noise ’86, Vol. II, pp. 815-20. Noise Control Foundation, New York, USA..

Berry BF 1995 Towards a standardised descriptor of impulsive noise caused by low-altitude military aircraft”. In R.J. Bernhard and S. Bolton (ed.) Inter Noise ’95, Vol. 2, pp. 879-884. Noise Control Foundation, Poughkeepsie, NY, USA

Bishop DE and Horonjef RD 1967 Procedures for Developing Noise Exposure Forecast Areas for Aircraft Flight Operations. FAA Report DS-67-10 (August 1967), Washington DC, USA.

Blazier WE 1998 RC Mark II: A refined procedure for rating the noise of heating, ventilating, and air-conditioning (HVAC) systems in buildings. Noise Control Engineering Journal 45: 243-250.

Bradley JS 1978 Predictors of Adverse Human Responses to Traffic Noise. Proceedings of the ASTM Symposium on Community Noise, Kansas City, pp. 108-123.

Bradley JS 1985 Uniform derivation of optimum conditions for speech in rooms. National Research Council, Building Research Note, BRN 239, Ottawa, Canada.

Bradley JS 1986a Predictors of Speech Intelligibility in Rooms. Journal of the Acoustical Society of America 80: 837-845.

Bradley JS 1986b Speech Intelligibility Studies in Classrooms. Journal of the Acoustical Society of America 80: 846-854.

Bradley JS 1986c Optimum Conditions for Speech in Rooms. Proceedings of the 12th International Congress on the Acoustics, Paper E10-5, Toronto, Canada.

010377

Bradley JS 1993 Disturbance Caused by Residential Air Conditioner Noise. Journal of the Acoustical Society of America 93: 1978-1986.
Bradley JS 1994a On dose response curves of annoyance to aircraft noise. In S. Kuwano (Ed.), Inter-Noise 94. Noise Quantity and Quality, Vol 1, pp. 235-38. Noise Control Foundation, Poughkeepsie, NY, USA.
Bradley JS 1994b Annoyance Caused by Constant-Amplitude and Amplitude- Modulated Sounds Containing Rumble. Noise Control Engineering Journal 42: 203-208.
Bradley, J.S. 1996. Determining acceptable limits for aviation noise. In F.A. Hill and R. Lawrence (eds.) Inter Noise 96, Noise Control – The Next 25 Years, Book 5, pp. 2541-46. Institute of Acoustics, St Albans, UK.
Bradley JS, Vaskor JG, Dickinson SM 1979 The Effect of Skewness and Standard Deviation on Sampling Errors for Traffic Noise. Applied Acoustics 12: 397-409.
Broner N and Leventhall HG 1993 A Modified PNdB for Assessment of Low Frequency Noise. Journal of Sound and Vibration 73: 425-444.
Brookhouser PE, Worthington DW, Kelly WJ 1992 Noise-induced hearing loss in children. Laryngoscope 1992; 102: 645-655.
Brown AL 1994 Exposure of the Australian population to road traffic noise. Applied Acoustics, 43: 169-176.
Buchta E 1996 A field study on annoyance caused by shooting noise – Determination of the penalty for various weapon calibers. . In F.A. Hill and R. Lawrence (eds.) Inter Noise 96, Noise Control – The Next 25 Years, Book 5, pp. 2495-2500. Institute of Acoustics, St Albans, UK.
Bullen RB and Hede AJ 1983 Time of Day Corrections in Measures of Aircraft Noise Exposure. Journal of the Acoustical Society of America 73: 1624-1630.
Burns W 1968 Noise and Man. John Murray, London, UK.
Carter NL 1996 Transportation noise, sleep, and possible after-effects. Environment International 22: 105-116.
Carter NL and Hunyor SN 1991 A field study of traffic noise and cardiac arrythmia during sleep. In: Technical Papers: 4th Western Pacific Regional Acoustics Conference, pp. 165-172, Queensland Department of Environment and Heritage, Brisbane, Australia.
Carter NL, Hunyor SN, Crawford G, Kelly D, Smith AJ 1994a Environmental noise and sleep - a study of arousals, cardiac arrhytmia and urinary catecholamines. Sleep 17: 298-307.
Carter NL, Ingham P, Tran K, Huynor S 1994b A field study of the effects of traffic noise on heart rate and cardiac arrhythmia during sleep. Journal of Sound and Vibration, 169, 221-227.
CEC 1992a Towards Sustainability: A European Community Programme of Policy and Action in Relation to the Environment and Sustainable Development. COM (92) 23 Final – Vol. II. Office for Official Publications of the European Communities, Luxembourg.
CEC 1992b The Impact of Transport on the Environment: A Community Strategy for "Sustainable Mobility". COM (92) 46 Final. Office for Official Publications of the European Communities, Luxembourg..
CEC 1996. Future Noise Policy. European Commission Green Paper. COM(96) 540 Final Office for Official Publications of the European Communities, Luxembourg.
Chakrabarty D, Santra SC, Mukherjee, A 1997: Status of road traffic noise in Calcutta metropolis, India. Journal of the Acoustical Society of America 101: 943-949.
CMD (1997) Taber's Cyclopedic Medical Dictionary, edition 18, FA Davis Company, Philadelphia.

010378

Cohen S 1980 Aftereffects of stress on human performance and social behavior: A review of research and theory. Psychological Bulletin, 88: 82-108.

Cohen S, Evans GW, Krantz DS, Stokols D 1980 Physiological, motivational, and cognitive effects of aircraft noise on children. American Psychologist 35: 231-243.

Cohen S, Evans GW, Stokols D, Krantz DS 1986 Behavior, Health and Environmental Stress. Plenum Press, New York**.**

Crocker MJ and Price AJ 1975 Noise and Noise Control. Volume I, CRC Press, Cleveland USA.

Daigle GA, Embleton TFW, Piercy JE 1986 Propagation of Sound In the Presence of Gradients and Turbulence Close to the Ground. Journal of the Acoustical Society of America 79: 613-627.

DEAT 1989 Environment Conservation Act (Act No. 73 of 1989) Noise Control Regulations, Ministry of Environmental Affairs and Tourism, Pretoria, South Africa.

DeKoning HW 1987 Setting Environmental Standards. Guidelines for Decision-Making. World Health Organization, Geneva, Switzerland.

DIMD 1985 Dorland's illustrated medical dictionary, 26th edition, Saunders, Philadelphia [etc.], USA

DiMento JS 1981 Making usable information on environmental stressors: Opportunities for the research and policy communities. Journal of Social Issues 37:172-204.

DoTRS 1999 What is an ANEF? Commonwealth of Australia 1999, Department of Transport and Regional Services. Internet address: http://www.dotrs.gov.au/airports/avenv/noise/anef.htm

Edworthy J 1998 Warning people through noise. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 1, pp. 147-56. Noise Effects '98 PTY Ltd., Sydney, Australia.

Edworthy J and Adams AS 1996 Warning Design: A Research Perspective. Taylop and Francis, London.

EEA 1995 Europe's Environment. The Dobris Assessment. D. Stanners, P. Bourdeau (Eds.), European Environment Agency, Copenhagen, Denmark.

Embleton TFW and Piercy JE 1976 Outdoor Sound Propagation Over Ground of Finite Impedance. Journal of the Acoustical Society of America 59: 267-277.

EU 1970 Council Directive on the approximation of the laws of the Member States relating to the permissible sound level and the exhaust system of motor vehicles. Directive 70/157/EEC of 6 February 1970. Official Journal L 42 (23.2.1970), p. 16. Amended by 73/350/EEC of 7.11.1973; Official Journal L 321 (22.11.1973), p. 33. Amended by 77/212/EEC of 8.3.1977; Official Journal L 66 (12.3.1977), p. 33. Amended by 81/334/EEC of 13.4.1981; Official Journal L 131 (18.5.1981), p. 6. Amended by 84/424/EEC of 03.09.1984; Official Journal L 238 (06.09.84) p. 31. Amended by 87/354/EEC of 3/87; Official Journal L 192 (11.07.87) p. 43. Amended by 89/491/EEC of 3/89; Official Journal L 238 (15.08.89) p. 43. Amended by 92/97/EEC of 10.11.92; Official Journal L 371 (19.12.92) p. 1. Amended by 96/20/EEC of 27.03.1996; Official Journal L 92 (13.04.96) p. 23, Brussels, Belgium.

EU 1978 Council Directive on the approximation of the laws of the Member States on the permissible sound level and exhaust system of motorcycles. Directive 78/1015/EEC of 23 November 1978. Official Journal L 349 (13.12.1978), p. 21, Brussels, Belgium. (Directive 78/1015/EEC is superseded by 97/24/EC, see EU 1997).

010379

EU 1980 Council Directive on the limitation of noise emissions from subsonic airplanes. Directive 80/51/EEC of 20 December 1979. Official Journal L 18 (24.01.80) p. 26, Brussels, Belgium.

EU 1984a Council Directive on the approximation of the laws of the Member States relating to the permissible sound power level of compressors. Directive 84/533/EEC of 17 September 1984. Official Journal L 300 (19.11.1984), p. 123. Amended by 85/406/EEC of 11.7.1985. Official Journal L 233 (30.8.1985), p. 11, Brussels, Belgium.

EU 1984b Council Directive on the approximation of the laws of the Member States relating to the permissible sound power level of tower cranes. Directive 84/534/EEC of 17 September 1984. Official Journal L 300 (19.11.1984), p. 123. Amended by 87/405/EEC of 25.6.1987. Official Journal L 220 (8.8.1987) p. 60, Brussels, Belgium.

EU 1984c Council Directive on the approximation of the laws of the Member States relating to the permissible sound power level of welding generators. Directive 84/535/EEC of 17 September 1984. Official Journal L 300 (19.11.1984), p. 142. Amended by 85/407/EEC of 11.7.1985.Official Journal L 233 (30.8.1985), p. 16, Brussels, Belgium.

EU 1984d Council Directive on the approximation of the laws of the Member States relating to the permissible sound power level of power generators. Directive 84/536/EEC of 17 September 1984. Official Journal L 300 (19.11.1984), p. 149. Amended by 85/408/EEC of 11.7.1985. Official Journal L 233 (30.8.1985), p. 18, Brussels, Belgium.

EU 1984e Council Directive on the approximation of the laws of the Member States relating to the permissible sound power level of powered hand-held concrete-breakers and picks. Directive 84/537/EEC of 17 September 1984. Official Journal L 300 (19.11.1984), p. 156. Amended by 85/409/EEC of 11.7.1985. Official Journal L 233 (30.8.1985), p. 20, Brussels, Belgium.

EU 1984f Council Directive on the approximation of the laws of the Member States relating to the permissible sound power level of lawnmowers. Directive 84/538/EEC of 17 September 1984. Official Journal L 300 (19.11.1984), p. 171. 87. Amended by 87/ 252/EEC of 7.4.87. Official Journal L 117 (5.5.1987), p. 22. Amended by 88/80/EEC of 22.3.1988. Official Journal L 81 (26.3.1988), p. 69. Amended by 88/181/EEC of 22.3.1988. Official Journal L 81 (26.3.1988), p. 71, Brussels, Belgium.

EU 1986a Council Directive on the protection of workers from the risks related to exposure of noise at work. Directive 86/188/EEC of 12.5.1986. Official Journal L 137 (24.5.1986), p. 28, Brussels, Belgium.

EU 1986b Council Directive on the limitation of noise emitted by hydraulic excavators, rope-operated excavators, dozers, loaders and excavator-loaders. Directive 86/662/EEC of 22.12.1986 as amended by the Directive of 2.8.1989. Official Journal L 384 (31.12.1986), p. 1; L 253 (30.8.1989), p. 35, Brussels, Belgium.

EU 1986c Council Directive on airborne noise emitted by household appliances. Directive 86/594/EEC of 1.12.1986. Official Journal L 344 (6.12.1986), p. 24, Brussels, Belgium.

EU 1989 Council Directive on the limitation of noise emission from civil subsonic jet aeroplanes. Directive 89/629/EEC of 04 December1989. Official Journal L 363 (12.12.89) p. 27, Brussels, Belgium.

EU 1996a Council Directive to adapt in the light of technical progress Directive 70/157/EEC of 6 February 1970 on the approximation of the laws of the Member States regarding poermissible sound levels and exhaust systems of motor vehicles. Directive 96/20/EC of 27.03.1996. Official Journal L 92 (13.04.96) p. 23, Brussels, Belgium.

010380

EU 1996b b Future Noise Policy – European Commission Green Paper. Report COM (96) 540 final. Commission of the Euopean Communities, Brussels.
EU 1997 Directive on certain components and charractristics of two or three-wheel motor vehicles. Directive 97/24/EC of the European Parliament and of the Council of 17 June 1997. Official Journal L 226 1997, Brussels, Belgium.
Evans GW 1998 Motivational consequences of exposure to noise. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 1, pp. 311-320. Noise Effects '98 PTY Ltd., Sydney, Australia.
Evans GW and Lepore SJ 1993 Non-auditory effects of noise on children: A critical review. Children's Environments 10: 31-51.
Evans GW and Maxwell L 1997 Chronic noise exposure and reading deficits. Environment and Behavior 29: 638-656.
Evans GW, Hygge S, Bullinger M 1995 Chronic noise and psychological stress. Psychological Science 6: 333-338.
Evans GW, Bullinger M, Hygge S 1998 Chronic noise exposure and physiological response: A prospective study of children living under environmental stress. Psychological Science 9: 75-77.
Faiz A, Sinha, K, Wals, M and Valma, A 1990: Automotive air pollution: Issues and options for developing countries. WPS 492, World Bank, Washington, USA.
Falkemark G 1997 Trafikpolitiken, makten och miljön [Traffic politics, power and the environment]. I H.B. Andersson (Red.), Trafik och miljö [Traffic and the Environment]. Kommunikationsforskningsberedningen [Swedish Transport and Communications Research Board], Stockholm, Sweden.Fechter LS 1999 Mechanisms of ototoxicity by chemical contaminants: Prospects for intervention. Noise and Health, 2: 10-27.
Fidell S and Jones G 1975 Effects of cessation of late night on an airport community. Journal of Sound and Vibration, 42:411-427.
Fidell S, Barber, DS, Schultz TJ 1991 Updating a dosage-effect relationship for the prevalence of annoyance due to general transportation noise. Journal of the Acoustical Society of America 89: 221-233.
Fidell S, Pearsons K, Tabachnick B, Howe R, Silvati L, Barber DS 1995a Field study of noise-induced sleep disturbance. Journal of the Acoustical Society of America 98 (2 PART 1): 1025-1033.
Fidell S, Howe R, T Tabachnick BG, Pearsons KS, Sneddon MD 1995b Noise-induced sleep disturbance in residences near two civil airports. NASA Contractor Report 198252, NASA Langley Research Center, Hampton VA, USA.
Fidell S, Howe R, Tabachnick B, Pearsons K, Silvati L, Sneddon M, Fletcher E 1998 Field studies of habituation to change in night-time aircraft noise and of sleep motility measurement methods. BBN Report 8195, BNN Systems and Technologies Corporation, Canogan Park, California, USA.
Fidell S, Pearsons K, Howe R et al. 1994 Noise-induced sleep disturbance in residential settings. BNN Report 7932. BNN Systems and Technologies Corporation, Canoga Park, California, USA.
Fields JM 1986 Cumulative Airport Noise Exposure Metrics: An Assessment of Evidence for Time-of-Day Weightings. US FAA Report, DOT/FAA/EE-86/10, United States Federal Aviation Administration, Washington D.C., USA.

010381

Fields JM 1990 A quantitative summary of non-acoustical variables' effects on reactions to environmental noise. In: Noise Con 90, pp. 303-308. Noise Control Foundation, Poughkeepsie, NY, USA.

Fields JM 1993 Effects of personal and situational variables on noise annoyance in residential areas. Journal of the Acoustical Society of America 93: 2753-2763.

Fields JM 1994 A Review of an Updated Synthesis of Noise/Annoyance Relationships. NASA Contractor Report 194950, National Aeronautics and Space Administration, Langley Research Center, Hampton, VA, USA.

Fields JM 1998 Reactions to environmental noise in an ambient noise context in residential areas. Journal of the Acoustical Society of America 104: 2245-2260.

Fields JM, de Jong RG, Brown AL, Flindell IH, Gjestland T, Job RFS, Kurra S, Lercher P, Schuemer-Kors A, Vallet M, Yano M 1997 Guidelines for reporting core information from communicy noise reaction surveys. Journal of Sound and Vibration 206: 685-695.

Fields JM, de Jong RG, Flindell IA, Gjestland T, Job R.S, Kurra S, Schuemer-Kors,A, Lercher P, Vallet M, Yano T 1998 Recommendation for shared annoyance questions in noise annoyance surveys. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98.), Vol. 1, pp. 481-86. Noise Effects '98 PTY Ltd., Sydney, Australia.

Finegold LS, Harris CS, von Gierke HE 1994. Community annoyance and sleep disturbance: Updated criteria for assessing the impacts of general transportation noise on people. Noise Control Engineering Journal, 42: 25-30.

Finegold LS, von Gierke HE, McKinley RL, and Schomer PD 1998 Addressing the effectiveness of noise control regulations and policies In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 2, pp. 719-24. Noise Effects '98 PTY Ltd., Sydney, Australia.

Finegold LS, von Gierke HE, Schomer PD, Berry BF 1999 Proposal for monitoring worldwide noise exposure and assessing the effectiveness of noise exposure policies and noise control technologies. In: J. Cuschieri, S. Glegg, Yan Yong (eds.) Internoise 99 – The 1999 International Congress on Noise Control Engineering, 6-8 December 1999, Fort Lauderdale, Florida, USA, pp. 1917-1922.

Finke H-O 1980 Akustische Kennwerte für die Messung und Beurteilung von Gerauschimmissionen und deren Zusammenhang mit der subjektiven Gestörtheit. Dissertation of Technical University, Braunschweig, Germany.

Flindell IH and Stallen, PJM 1999 Non-acoustical factors in evnrionmental noise. Noise and Health 3: 11-16.

Florentine M, Buus S, Poulsen T 1996. Temporal integration of loudness as a function of level. Journal of the Acoustical Society of America, 99, 1633-44.

Ford RD 1987 Physical assessment of transportation noise. In P.M. Nelson (ed.) Transportation Noise Reference Book. Butterworth, London, UK.

Franks JH 1998 Preventing noise-induced hearing loss: A perspective view from the next millennium. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98.), Vol. 1, pp. 11-16. Noise Effects '98 PTY Ltd., Sydney, Australia..

Freeman K 1984 Mental Health and the Environment. Churchill Livingstone, London, UK.

French NR and Steinberg JC 1947 Factors governing the intelligibility of speech sounds. Journal of the Acoustical Society of America 19:90-119.

Gjestland T, Liasjo K, Granoien I, Fields JM 1990 Response to Noise Around Oslo Airport Fornebu. ELAB-RUNIT Report No. STF40 A90189, Trondheim, Norway.

010382

Globus G, Friedmann J, Cohen H, Pearsons SK, Fidell S 1974 The effects of aircraft noise on sleep electrophysiology as recorded in the home. In: Noise as a Public Health Problem. US Environmental Protection Agency, Report EPA 550/9-73.008, pp. 587-591, Washington DC, USA.

Goldstein M 1994 Low-frequency components in complex noise and their perceived loudness and annoyance. Arbete & Haelsa 1994: 2 (Work and Health; Scientific Journal of the National Institute of Occupational Health, Solna, Sweden).

Gottlob D 1995 Regulations for community noise. Noise/News International, December 1995, pp.223-236.

Griefahn B 1977 Long-term exposure to noise. Aspects of adaptation, habituation and compensation. Waking and Sleeping, 1:383-386.

Griefahn B 1990a Präventivmedizinische Vorschläge für den nächtlichen Schallschutz [Proposal for protection against noise during night from the point of view of preventive medicine]. Zeitschrift für Lärmbekämpfung 37: 7-14.

Griefahn B 1990b Sleeping in noisy environments: effects, limits and preventative measures. In: JA Horne (ed) Sleep 1990, pp. 391-93, Pontenagel Press, Bochum, Germany.

Griefahn B, Mehnert P, Moehler U, Schuemer-Kohrs A, Schuemer R 1996 Design of a field study on the effects of railway noise and road traffic noise. In F.A. Hill and R. Lawrence (eds.) Inter Noise 96. Noise Control – The Next 25 Years, Book 4, pp. 2183-88. Institute of Acoustics, St Albans, UK.

Griefahn B, Deppe C, Mehnert P, Moog R, Moehler U, Schuemer R 1998 What nighttimes are adequate to prevent noise effects on sleep? In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 2, pp. 445-450. Noise Effects '98 PTY Ltd., Sydney, Australia.

Griffiths ID 1983 Review of Community Response to Noise. In G. Rossi, (ed.), Noise as a Public Health Problem, Vol. 2, pp. 1031-1047. Milano, Italy: Centro Ricerche e Studi Amplifon.

Guski R 1987 Lärm–Wirkungen unerwünschter Geräusche. Huber, Bern, Switzerland.

Haines M, Stansfeld SA, Job RFS, Berglund B 1998 Chroinic aircraft noise exposure and child cognitive performance and stress. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 1, pp. 329-336. Noise Effects '98 PTY Ltd., Sydney, Australia.

Hall FL, Birnie SE, Taylor SM, Palmer JE 1981 Direct Comparison of Community Response to Road Traffic Noise and to Aircraft Noise. Journal of the Acoustical Society of America 70: 1690-1698.

Hall FL, Taylor SM, Birnie SE 1985 Activity interference and noise annoyance. Journal of Sound and Vibration 103: 237-252.

Halpern D 1995 Mental Health and the Built Environment. More than Bricks and Mortar? Taylor and Francis Ltd, London, UK.

Hanner P and Axelsson A 1988 Acute acoustic trauma. Scandinavian Audiology 17: 57-63.

Harris CM 1991 Handbook of Acoustical Measurements and Noise Control. Mc Graw Hill, New York, USA.

Hasan S and Beg MHA 1994 Noise induced hearing loss in industrial workers of Karachi. Pakistan Journal of Otolaryngology 10: 200-205.

Hasan S and Zaidi SH 1997 The prevalence of noise induced hearing loss in industrial (textile) workers of Karachi. Thesis, University of Karachi, Karachi, Pakistan.

010383

HCN 1994 Noise and Health. Publication No. 1994/15E, Health Council of the Netherlands, The Hague, Netherlands.
HCN 1997 Assessing noise exposure for public health purposes. Publication no. 1997/23E, Health Council of the Netherlands, Rijswijk, Netherlands.
Hede A 1998a Environmental noise regulation: A public policy perspective. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 2, pp. 687-96. Noise Effects '98 PTY Ltd., Sydney, Australia.
Hede, A 1998b: Towards a normative model of public policy for environmental noise, Acoustics Australia, 26, 95-100.
Hellström PA, Dengerink HA, Axelsson A 1992 Noise levels from toys and recreational articles for children and teenagers. British Journal of Audiology, 26: 267-270.
Hetu Rand Fortin M 1995 Potential risk of hearing damage associated with exposure to highly amplified music. Journal of the American Academy of Audiology 6: 378-386.
Hobson JA 1989 Sleep. Scientific American Library, W.H. Freeman and Co, New York, NY, USA.
Holmes G, Singh B.R. and Theodore L. 1993. Handbook of environmental management and technology. Wiley, New York.
Horne JA, Pankhurst FL, Reyner LA, Hume K, Diamond ID 1994 A field study of sleep disturbance: Effects of aircraft noise and other factors on 5,742 nights of actimetrically monitored sleep in a large subject sample. Sleep 17: 146-159.
House ME 1987 Measurement and Prediction of Aircraft Noise. In P.M. Nelson (ed.) Transportation Noise Reference Book, Butterworth, London.
Houtgast T 1981 The Effect of Ambient Noise on Speech Intelligibility in Classrooms. Applied Acoustics 14: 15-25.
Houtgast T and Steeneken, HJM. 1983. Experimental verification of the STI: A valid basis for setting indoor noise level criteria. In G. Rossi (ed.), Noise as a Public Health Problem. Vol. 1, pp. 477-487, Centro Ricerche e Studi Amplifon, Milano, Italy.
Houtgast T and Steeneken JM 1985 A Review of the MTF Concept in Room Acoustics and Its Use for Estimating Speech Intelligibility in Auditoria. Journal of the Acoustical Society of America 77: 1069-1077.
Humes LE, Dirks DD, Bell TS, Ahlstrom C, Kincaid GE 1986 Application of the Artic-ulation Index and the Speech Transmission Index to the recognition of speech by normal-hearing and hearing-impaired listeners. Journal of Speech Hearing Research, 29:447-462.
Hygge S, Evans GW, Bullinger M 1996 The Munich airport noise study: Cognitive effects on children from before to after the change over of airports. In F.A. Hill and R. Lawrence (eds.) Inter Noise 96. Noise Control – The Next 25 Years, Vol. 5, pp. 2189-92. Institute of Acoustics, St Albans, UK.
Hygge S, Jones DM, Smith AP 1998 Recent developments in noise and performance. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 1, pp. 321-28, Noise Effects '98 PTY Ltd., Sydney, Australia.
ICAO 1993 International Standards and Recommended Practices: Environmental Protection. Annex 16, Volume I: "Aircraft Noise". Annex 16 (Chapter 2 and 3) to the Convention on International Civil Aviation. International Civil Aviation Organization, [Air Navigation (Noise Certification) Order 1990 -Statutory Instrument 1514], Montreal, Canada.
IEC 1979 Sound Level Meters. IEC Publication 651, International Electrotechnical Commission, Geneva, Switzerland.

010384

IEC 1988 Sound system equipment, part 16: The objective rating of speech intelligibility in auditoria by the RASTI method. IEC Publication 268-/6, International Electrotechnical Commission, Geneva, Switzerland.

IEH 1997. The Non-Auditory Effects of Noise. IEH Report R10, Institute for Environment and Health, Leicester, UK.

Irle M 1975 Is aircraft noise harming people? In M. Deutsch and H.A. Hornstein (eds.), Applying Social Psychology, pp. 115-138, Erlbaum, Hillsdale, NJ. USA.

Ising H and Günther T 1997 Interaction between noise-induced stress and magnesium losses: relevance for long-term health effects. In F. Augustinovicz (ed.), Inter Noise 97. Help Quiet the World for a Higher Quality Life, Vol. 2, pp. 1099-1104. Noise Control Foundation, Poughkeepsie, NY, USA.

Ising H, Babisch W, Kruppa B 1998 Acute and chronic noise stress as cardiovascular risk factors. Federal Environmental Agency, Berlin, Germany.

Ising H, Babisch W, Gandert J, Scheuermann B 1988 Hörschäden bei jugendlichen Berufs-anfängern aufgrund von Freizeitlärm und Musik. Zeitschrift für Lärmbekämpfung, 35:35-41.

Ising H, Hanel J, Pilgramm M et al. 1994 Gehörschadensrisiko durch Musikhören mit Kopfhörern. Hals-Nasen-Ohren 1994; 42:764-8.

ISO 1975a Acoustics-Method for Calculating Loudness Level. ISO-532-1975 (E), International Organization for Standardization, Geneva, Switzerland.

ISO 1975b Acoustics-Preferred Frequencies for Measurements. International Standard ISO 266-1975 (E), International Organization for Standardization, Geneva, Switzerland

ISO 1978 Laboratory Measurement of Airborne Sound Insulation of Building Elements – Part V, Field Measurements of Airborne Sound Insulation of Façade Elements and Facades", International Standard ISO-140/V, International Organization for Standardization, Geneva, Switzerland.

ISO 1982 Acoustics – Description and measurement of environmental noise – Part 1: Basic quantities and procedures. International Standard ISO 1996-1, International Organization for Standardization, Geneva, Switzerland.

ISO 1987a Acoustics-Normal Equal Loudness Contours. International Standard ISO 226-1-1987 (E), International Organization for Standardization, Geneva, Switzerland.

ISO 1987b Acoustics – Description and measurement of environmental noise – Part 3: Application to noise limits. International Standard ISO 1996-3:1987(E), International Organization for Standardization, Geneva, Switzerland.

ISO 1990 Acoustics–Determination of occupational noise exposure and estimation of noise-induced hearing impairment. International Standard ISO 1999, International Organization for Standardization, Geneva, Switzerland.

ISO 1994 Quality systems -- Model for quality assurance in production, installation and servicing. International Standard ISO 9002, International Organization for Standardization, Geneva, Switzerland.

ISO 1995 Laboratory Measurement of Airborne Sound Insulation of Building Elements – Part III: Laboratory Measurements of Airborne Sound Insulation of Building Elements. International Standard ISO-140/III, International Organization for Standardization, Geneva, Switzerland .

ISO 1996a Acoustics – Rating of Sound Insulation in Buildings and of Building Elements – Part 1: Airborne Sound Insulation. International Standard ISO 717-1, International Organization for Standardization, Geneva, Switzerland.

010385

ISO 1996b Environmental management systems - specification with guidance for use. International Standard ISO 14001, International Organization for Standardization, Geneva, Switzerland.

ISO 1996c Environmental management systems - general guidelines on principles, systems and supporting techniques. International Standard ISO 14004, International Organization for Standardization, Geneva, Switzerland.

ISO 1998 Measurement of Sound Insulation In Buildings and of Building Elements – Part VI: Laboratory Measurements of impact sound insulation of floors. International Standard ISO-140/VI, International Organization for Standardization, Geneva, Switzerland.

Jansen G 1986 Zur "erheblichen Belästigung" und "Gefährdung" durch Lärm. Zeitschrift für Lärmbekämpfung 33:2-7.

Jansen G 1987 Verkehrslärmwirkungen bei besonderen Personengruppen. Zeitschrift für Lärmbekämpfung 34: 152-156.

Jansen G 1998 Health concepts and noise effects. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 2, pp. 697-702. Noise Effects '98 PTY Ltd., Sydney, Australia.

Jansen G and Gottlob D 1996 Requirements for the protection against outdoor noise in various countries with respect to standardization and regulations. In F.A. Hill and R. Lawrence (eds.) Inter Noise 96. Noise Control – The Next 25 Years, Book 6, pp. 3343-3346. Institute of Acoustics, St Albans, UK.

Jenkins LM, Tarnopolsky A, Hand DJ 1981 Psychiatric admissions and aircraft noise from London Airport: Four-year, three hospitals' study. Psychological Medicine 11: 765-782.

Job RFS 1988 Community response to noise: A review of factors influencing the relationship between noise exposure and reaction. Journal of the Acoustical Society of America 83: 991-1001.

Job RFS 1993 Psychological factors of community reaction to noise. In M. Vallet (ed.) Noise as a Public Health Problem, Vol. 3, pp. 48-59, INRETS - Institut National de REcherche sur les Transports et leur Sécurité, Arcueil, France.

Job RFS 1996 The influence of subjective reactions to noise on health effects of the noise. Environment International 22: 93-104.

Kabuto M and Suzuki S 1976 A trial measurement of individual noise exposure by time-zone in a day. Japanese Journal of Industrial Health, 18: 17-22.

Kabuto M, Ohtsuka R, Suzuki S 1978 Measurement and evaluation of individual noise exposure level of Tokyo metropolitan commuters in railroad cars above ground and in subways. Japanese Journal of Public Health, 2: 59-63.

Kageyama T, Kabuto M, Nitta H, Kurokawa Y, Taira K, Suzuki S, Takemoto T 1997 A populations study on risk factors for insomnia among adult Japanese women: A possible effect of road traffic volume. Sleep 20: 963-971.

Kastka J, Buchta E, Ritterstaedt U, Paulsen R, Mau U 1995 The long term effect of noise protection barriers on the annoyance response of residents. Journal of Sound and Vibration, 184: 823-852.

Katz J. (ed.) 1994 Handbook of Clinical Audiology. Fourth edition, Williams and Wilkins Baltimore, ML, USA.

Kerry G, Lomax C, and James DJ 1997 Assessment and Relevance of methods for calculating onset rate of low altitude flight noise. In F. Augustinovicz (ed.), Inter Noise 97. Help Quiet

010386

the World for a Higher Quality Life, vol. 3, pp. 1227-30. Noise Control Foundation, Poughkeepsie, NY, USA.
Kjellberg A, Goldstein M, and Gamberale F 1984 An Assessment of dBA for Predicting Loudness and Annoyance of Noise Containing Low Frequency Components. Journal of Low Frequency Noise and Vibration 3: 10-16.
Klæboe R, Kolbenstvedt M, Lercher P, and Solberg S 1998 Changes in noise reactions– Evidence for an area-effect? In V.C. Goodwin and D.C. Stevenson (eds.) Inter Noise 98. Sound and Silence: Setting the Balance, Vol. 2, pp. 1133-36. New Zealand Acoustical Society, Auckland, New Zealand.
Koelega HS (ed.) 1987 Environmental Annoyance: Characterization, Measurement, and Control. Elsevier, Amsterdam, Netherlands.
Kremer L 1950 Die Wissenschaftliche Grundlagen der Raumakustik. Band III, Hirzel
Verlag, Leipzig
Kryter KD 1959 Scaling Human Reaction to the Sound from Aircraft. Journal of the Acoustical Society of America 31: 1415-1429.
Kryter KD 1994 The Handbook of Noise and Hearing. Academic Press, San Diego, CA, USA.
Kutscheidt E 1989 Rechtsprobleme bei der Bewertung von Geräuschimmissionen. Neue Zeitschrift für Verwaltungsrecht 3:193-199.
Kuwano S, Namba S, and Mizunami T 1998 The effect of noise on the effort to sleep. In V.C. Goodwin and D.C. Stevenson (eds.) Inter Noise 98. Sound and Silence: Setting the Balance, Vol. 2, pp. 1083-1086. New Zealand Acoustical Society, Auckland, New Zealand.
Lara Sáenz A and Stephens RWB (eds.) 1986 Noise Pollution. Wiley, Chichester, UK.
Lambert J and Vallet M 1994 Study Related to the Preparation of a Communication on a Future EC Noise Policy. INRETS LRN Report No. 9420, INRETS - Institut National de REcherche sur les Transports et leur Sécurité, Bron, France.
Lang J 1986 Assessment of noise impact on the urban environment, A study on noise prediction models, Environmental Health Series 9, Regional Office for Europe, World Health Organization, Copenhagen, Denmark.
Lang J 1995 Noise abatement regulations and strategies. In: Noise - Selected presentations of an Informal Regional Consultation Meeting on Noise Pollution, 2-5 September 1991, Amman, Jordan, Eastern Mediterranean Regional Office, Regional Centre for Environmental Health Activities (CEHA), Amman, Jordan.
Lang WW 1999 Is noise policy a global issue, or is it a local issue? In: J. Cuschieri, S. Glegg, Yan Yong (eds.) Internoise 99 – The 1999 International Congress on Noise Control Engineering, 6-8 December 1999, Fort Lauderdale, Florida, USA, pp. 1939-1943.
Large JB and House ME 1989 The effect on aviation noise of the single European market. In G.C. Maling, Jr. (ed.), Inter-Noise 89, Vol. 1, pp. 695-700, Noise Control Foundation, Poughkeepsie, NY, USA.
Lazarus H 1990 New methods for describing and assessing direct speech communication under disturbing conditions. Environment Inernational 16: 373-392.
Lazarus H 1998 Noise and communication: The present state. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects ’98), Vol. 1, pp. 157-162. Noise Effects ’98 PTY Ltd., Sydney, Australia.
Lercher P 1996 Environmental noise and health: An integrated research perspective. Environment International 22: 117-129.

010387

Lercher P 1998a Context and coping as moderators of potential health effects in noise-exposed persons. In D. Prasher and L. Luxon, (Eds.), Advances in Noise Series. Vol. I: Biological Effects, pp. 328-335, Whurr Publishers, London, UK.

Lercher P 1998b Deviant dose-response curves for traffic noise in "sensitive areas"? In V.C. Goodwin and D.C. Stevenson (eds.) Inter Noise 98. Sound and Silence: Setting the Balance, Vol. 2, pp. 1141-1144. New Zealand Acoustical Society, Auckland, New Zealand.

Lercher P, Stansfeld SA, Thompson SJ 1998 Non-auditory health effects of noise: Review of the 1993-1998 period. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 1, pp. 213-220. Noise Effects '98 PTY Ltd., Sydney, Australia.

Lindemann HE, van der Klaauw MM, Platenburg-Gits FA 1987 Hearing acuity in male adolescents at the age of 17 – 23 years. Audiology 26: 65-78.

Lindvall T and Radford EP (eds.) 1973 Measurement of annoyance due to exposure to environmental factors. Environmental Research 6:1-36.

London Charter 1999 Charter on Transport, Environment and Health. Final version prepared following the Fourth Preparatory Meeting on "Transport, enviroment and Health, Vienna, 15-17 March 1999. EUR/ICP/EHCO 02 02 05/9 Rev.4 - 09009 – 16 June 1999. Regional Office for Europe, European Centre for Environment and Health, Rome Division, World Health Organization, Rome, Italy. Internet address: http://www.who.it/london_conference/teh.htm.

Lutman ME and Haggard MP (eds.) 1983 Hearing Science and Hearing Disorders. Academic Press, London, UK.

Mage, D and Walsh, M 1998: Case studies from cities around the world. In: Urban Traffic Pollution, eds. D. Schwela and O. Zali, E & Spon, London, UK.

Maschke C 1992 Der Einfluss von Nachtlärm auf den Schlafverlauf und die Katecholaminenausscheidung. Technische Universität, Berlin, Germany.

Maschke C, Arndt D, Ising H, et al. 1995 Nachtfluglärmwirkungen auf Anwohner. DFG-Forschungsbericht Gr. 452/8-2. Gustav Fischer Verlag, Stuttgart, Germany.

Miedema HME 1987 Annoyance from Combined Noise Sources. In H. S. Koelega (ed.), Environmental Annoyance: Characterization, Measurement and Control, pp. 313-320, Elsevier, Amsterdam, The Netherlands.

Miedema HME 1993 Response Functions for Environmental Noise. In M. Vallet (ed.) Noise as a Public Health Problem, Vol. 3, pp. 428-433, INRETS - Institut National de Recherche sur les Transports et leur Sécurité, Arcueil Cedex, France.

Miedema HME 1996 Quantification of Annoyance Caused by Environmental Noise and Odor. TNO, Leiden, Netherlands.

Miedema HME, Vos H 1998 Exposure response functions for transportation noise. Journal of the Acoustical Society of America 104: 3432-3445.

Miller JD 1978 General psychological and sociological effects of noise. In E.C. Carterette and M.P. Friedman (eds.), Handbook of Perception. Vol. VI: Hearing (pp. 641-676). Academic Press, New York NZ, USA.

Moehler U Community response to railway noise: A review of social surveys. Journal of Sound and Vibration 120: 321-332.

Molino JA 1974 Equal aversion levels for pure tones and 1/3 octave bands of noise. Journal of the Acoustical Society of America 55: 1285-1289.

National Environment Board of Thailand 1990 Air and noise pollution in Thailand 1989, Bangkok.

010388

NRC-CNRC 1994 NEF Validation Study. (2) Review of Aircraft Noise and its Effects. Contract Report A-1505.5 (Final). Institute for Research in Construction, Ottawa, Canada.
OECD 1991 Fighting Noise in the 1990's. Organisation for Economic Co-operation and Development, Paris, France.
OECD 1993 Environmental Data Compendium. Organisation for Economic Co-operation and Development, Paris, France.
OECD-ECMT. 1995. Urban Travel and Sustainable Development. European Conference of Ministers of Transport, Organisation for Economic Co-operation and Development, Paris, France.
Öhrström E 1993a Research on noise and sleep since 1988: Present state. In M. Vallet (ed.), Noise as a Public Health Problem, Vol. 3, pp. 331-338, INRETS - Institut National de REcherche sur les Transports et leur Sécurité, Arcueil, France.
Öhrström, E. 1993b. Effects of low levels of road traffic noise during night. . In M. Vallet (ed.), Noise as a Public Health Problem, Vol. 3, pp. 359-366, INRETS - Institut National de REcherche sur les Transports et leur Sécurité, Arcueil, France.
Öhrström E 1997 Effects of exposure to railway noise–A comparison between areas with and without vibration. Journal of Sound and Vibration 205, 555-560.
Öhrström E, Björkman M 1988 Effects of noise-disturbed sleep–A laboratory study on habituation and subjective noise sensitivity. Journal of Sound and Vibration 122: 277-290.
Öhrström E, Skånberg AB1996 A field survey on effects of exposure to noise and vibrations from railway traffic. Part I: Annoyance and activity disturbance effects. Journal of Sound and Vibration 193: 39-47.
Ollerhead JB, Jones CJ, Cadoux RE, Woodley A, Atkinson BJ, Horne JA, Pankhurst F, Reyner L, Hume KI, Van F, Watson A, Diamond I, Egger P, Holmes D, McKean J 1992 Report of a Field Study of Aircraft Noise and Sleep Disturbance. Department of Transport, London, UK.
Pankhurst FL and Horne JA 1994 The influence of bed partners on movement during sleep. Sleep 17: 308-315.
Passchier-Vermeer W 1991 Noise from toys and the hearing of children. NIPG-TNO Report 91.032, TNO Institute of Preventive Health Care, Leiden, Netherlands.
Passchier-Vermeer W 1993 Noise and Health. The Hague: Health Council of the Netherlands. [Publication No A93/02E, review prepared by TNO Institute of Preventive Health Care, Leiden]
Passchier-Vermeer W 1994 Sleep disturbance due to night-time aircraft noise. Leiden: TNO-PG Report 94.077, TNO Institute of Preventive Health Care, Leiden, Netherlands.
Passchier-Vermeer, W 1996. Effects of noise and health. Chapter 3 of a report on Noise and Health prepared by a committee of the Health Council of The Netherlands. Noise/News International: 137-150.
Passchier-Vermeer W and Rövekamp AJM 1985 Verband tussen gehoorschade en de sociale handicap door een verminderd hoorvermogen bij groepen personen die tijdens hun werk aan lawaai zijn geëxponeerd. In: Passchier-Vermeer W, et al. Preventie gehoorschade door lawaai.Voordrachten ter gelegenheid van het 10-jarig jubileum van NVBA, pp.185-202. NIPG-TNO Institute of Preventive Health Care, Leiden, Netherlands.
Passchier-Vermeer W and Zeichart K Vibrations in the living environment. TNO-PG Report 98.030, TNO Institute of Preventive Health Care, Leiden, Netherlands.
Passchier-Vermeer W, Vos H, Steenbekkers JHM 1998 Popmusic through headphones and hearing loss. Report 98.036, TNO Institute of Preventive Health Care, Leiden, Netherlands.

010389

Paulsen R and Kastka J 1995 Effects of combined noise and vibration on annoyance. Journal of Sound and Vibration 181: 295-314.
Pearsons KS 1998 Awakening and motility effects of aircraft noise. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 2, pp. 427-32. Noise Effects '98 PTY Ltd., Sydney, Australia.
Pearsons KS, Barber DS, Tabachnick BG, Fidell S 1995 Predicting noise-induced sleep disturbance. Journal of the Acoustic Society of America 97: 331-338.
Pearsons KS 1996 Recent field studies in the United States involving the disturbance of sleep from aircraft noise. In F.A. Hill and R. Lawrence (eds.) Inter Noise 96. Noise Control – The Next 25 Years, Book 5, pp. 2271-2276. Institute of Acoustics, St. Albans, UK.
Persson K and Björkman M 1988 Annoyance due to Low Frequency Noise and the Use of the dBA Scale. Journal of Sound and Vibration 127: 491-497.
Plomp R 1986 A signal to noise ratio model for the speech-reception threshold of the hearing impaired. Journal of Speech and Hearing Research 29: 146-152.
Prasanchuk S (1997) Noise pollution. Its effect on health and hearing – Thailand as a case study. Presentation at the PDH Informal Consultation on the Prevention of Noise-induced Hearing Loss, 28-30 October 1997, WHO/PDH/98.5. World Health Organization, Geneva.
Razi MS, Jafer S, Hillier V, Zaidi SH, Newton V 1995 Causes of bilateral hearing loss in school children. Pakistan Journal of Otolaryngology 11: 68-86.
Reyner LA and Horne JA 1995 Gender- and age-related differences in sleep determined by home-recorded sleep logs and actimetry from 400 adults. Sleep 18: 127-134.
Ronnebaum T Schulte-Fortkamp B Weber R 1996 Synergetic effects of noise from different sources: a literature study. In F.A. Hill and R. Lawrence (eds.), Inter Noise 96. Noise Control – – The Next 25 Years, Vol. 6, pp. 2241-2246. Institute of Acoustics, St. Albans, UK.
SABS 1994 Code of practice for the measurement and rating of environmental noise with respect to annoyance and speech communication (third revision). South African Bureau of Standards publication No. 0103, Pretoria, South Africa.
SABS 1996 Code of practice for calculating and predicting road traffic noise. South African Bureau of Standards publication No. 0210, Pretoria, South Africa.
SABS 1998 Code of practice for methods for environmental noise impact assessments. South African Bureau of Standards publication No. 0328, Pretoria, South Africa.
Salter L 1988 Mandated Science: Science and Scientists in the Making of Standards. Kluwer, Dordrecht, Netherlands.
Sandberg U (ed.) 1995 The effects of regulations on road vehicle noise. Report by the International Institute of Noise Control Engineering Working Party, Noise News International 6: 85-113.
Sandberg U 1999 Abatement of traffic, vehicle and tire/road noise – the global perspective. In: J. Cuschieri, S. Glegg, Yan Yong (eds.) Internoise 99 – The 1999 International Congress on Noise Control Engineering, 6-8 December 1999, Fort Lauderdale, Florida, USA, pp. 37-42.
Sargent JW and Fothergill LC 1993 The noise climate around our homes. Information Paper 21/93, Building Research Establishment, Garston, UK.
Scharf B 1970 Critical bands. In J.V.Tobias (ed.), Foundations of Modern Auditory Theory, Vol. 1, pp. 157-202. Academic Press, New York, NY, USA.
Scharf B and Hellman R 1980 How Best to Predict Human Response to Noise on the Basis of Acoustic Variables. In J.V. Tobias, G. Jansen andW.D. Ward (eds.) Noise as a Public Health Problem, ASHA Report no. 10, pp. 475-487, Rockville, ML, USA.

010390

Schulz TJ 1978 Synthesis of Social Surveys on noise annoyance. Journal of the Acoustical Society of America, 64: 377-405.
Schwela D 1998. WHO Guidelines on Community Noise. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 2, pp. 475-480. Noise Effects '98 PTY Ltd., Sydney, Australia.
Smith AW 1998 The World Health Organisation and the prevention of deafness and hearing impairment caused by noise. Noise and Health 1: 6-12.
Smith AP 1990 Noise, performance efficiency and safety. International Archive of Occupational and Environmental Health 62: 1-5.
Smoorenburg GF 1998 Effects of impulse noise on man. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98), Vol. 1, pp. 1-10, Noise Effects '98 PTY Ltd., Sydney, Australia.
Smoorenburg GF and Goldstein-Brouwers WG 1986 Spreaakverstaan in relatie tot het toonaudiogram bij slechthorendheid ten gevolge van lawaai. IZF-TNO Report 1986 C-17, Soesterberg, Netherlands.
Stanners D and Bordeau P (Eds.) 1995 Europe's Environment the Dobris Assessment. Noise and radiation, pp.359-374, European Environment Agency, Copenhagen, Denmark.
Stansfeld SA 1992 Noise, noise sensitivity and psychiatric disorder: Epidemiological and psychological studies. Psychological Mededicine, Monograph Suppl. 22, Cambridge University Press, Cambridge, UK.
Stansfeld SA, Gallacher J, Babisch W, Shipley S 1996. Road traffic noise and psychiatric disorder: prospective findings from the Caerphilly Study. British Medical Journal 313: 266-267.
Stevens SS 1972 Perceived level of noise by Mark VII and decibels (E). Journal of the Acoustical Society of America 51: 575-601.
Struwe F, Jansen G, Schwarze S, Schwenzer C, Nitzsche M 1996 Untersuchung von Hörgewohn-heiten und möglichen Gehörrisiken durch Schalleinwirkungen in der Freizeit unter beson-derer Berücksichtigung des Walkman-Hörens. Düsseldorf: Institut für Arbeitsmedizin der Heinrich-Heine-Universität. In B. Babissch , G. Bambach, H. Ising, B. Kruppa, P. Plath, E. Rebentisch, F. Struwe Gehörgefährdung durch laute Musik und Freizeitlärm, pp. 44 - 154. Institut für Wasser-, Boden- und Lufthygiene des Umweltbundesamtes, Berlin, Germany.
Tafalla RJ and Evans GW 1997 Noise, physiology and human performance: The potential role of effort. Journal of Occupational Health Psychology 2: 148-155.
Tempest W (ed.) 1985 The Noise Handbook. Academic Press, London, UK.
ten Wolde T 1998 European Commission's future noise policy. In V.C. Goodwin and D.C. Stevenson (eds.) Inter Noise 98. Sound and Silence: Setting the Balance, Vol. 3, pp. 1719-1722. New Zealand Acoustical Society, Auckland, New Zealand.
Thiessen GJ 1978 Disturbance oif sleep by noise. Journal of the Acoustical Society of America, 64: 216-222.
Thompson SJ 1996 Non-auditory health effects of noise: an updated review. In F.A Hill and R. Lawrence (eds.) Inter Noise 96. Noise Control – The Next 25 Years, Vol. 4, pp. 2177-2182. Institute of Acoustics, St. Albans, UK.
UNCED 1992 Agenda 21. United Nations Conference on Environment and Development, Conches, Switzerland.

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US EPA. 1974. Information on levels of environmental noise requisite to protect public health and welfare with an adequate margin of safety. Report EPA 550/9-74.004, US Environmental Protection Agency, Washington DC, USA.
Vallet M 1996 Charactéristiques et indicateurs de la gene due au bruit des avions. Synthèse. INRETS LRN Report No. 29, Institut National de REcherche sur les Transports et leur Sécurité, Arcueil, France.
Vallet M 1998 Sleep disturbance by noise: Recent orientations. In N.L. Carter and R.F.S. Job (eds.) Noise as a Public Health Problem (Noise Effects '98),, Vol. 2, pp. 421-26, Noise Effects '98 PTY Ltd., Sydney, Australia.
Vallet M and Vernet I 1991 Night aircraft noise index and sleep research results. In A. Lawrence (ed.), Inter-Noise 91. The Cost of Noise, Vol. 1, pp. 207-210. Noise Control Foundation, Poughkeepsie, NY, USA.
Vallet M, Gagneux J-M, Clairet JM, Laurens JF, Letisserand D 1983 Heart rate reactivity to aircraft noise after a long-term exposure. In G. Rossi (ed.), Noise as a Public Health Problem, Vol. 2, pp. 965-971, Centro Richerche e Studi Amplifon, Milano, Italy.
van den Berg M 1996 Noise management in the Netherlands. Noise and Vibration, September 1996, 11-12.
van Hees O 1992 Gehoorafwijkingen bij beroepsmusici. Thesis, GUA, Amsterdam, Netherlands.
Vaskor JG, Dickinson SM, Bradley JS 1979 Effect of Sampling on the Statistical Descriptors of Traffic Noise. Applied Acoustics 12: 111-124.
Vernon JA, Moller AR (Eds.) 1995 Mechanisms of Tinnitus. Allyn and Bacon, Needham Heights, Ma, USA.
von Gierke HE 1975 Noise – How Much is Too Much? Noise Control Engineering Journal 5: 24-34.
von Gierke HE and Harris CS 1987 Annoyance Response to Military Flight Operations and the Development of Standard Criteria for Community Annoyance. In H.S. Koelega (ed.), Environmental Annoyance, Characterization, Measurement and Control, pp. 257-269, Elsevier, Amsterdam, The Netherlands.
Vos, J 1992 Annoyance caused by simultaneous impulse, road-traffic and aircraft sounds: a quantitative model. Journal of the Acoustical Society of America 91: 3330-45.
Vos J 1996 Annoyance caused by impulse sounds produced by small, medium-large, and large firearms. In F.A Hill and R. Lawrence (eds.) Inter-Noise 96. Noise Control – The Next 25 Years, Book 5, pp. 2231-36. Institute of Acoustics, St. Albans, UK.
Warren CHE 1972 The sonic boom exposure effects 1.2: The sonic boom–generation and propagation. Journal of Sound and Vibration 20: 485-497.
WCED 1987 Our Common Future. World Commission on Environment and Development The Brundtland Commission, Oxford University Press, Oxford UK.
Westman JC and Walters JR 1981 Noise and stress. A comprehensive approach. Environmental Health Perspectives 41: 291-309.
WHO 1980a Noise. Environmental Health Criteria Document No. 12. World Health Organization, Geneva, Switzerland.
WHO 1980b. Glossary on Air Pollution. WHO Regional Office of Europe, Series No. 9, World Health Organization, Copenhagen, Denmark.
WHO 1993 The Environmental Health Criteria Document on Community Noise. Report on the Task Force Meeting, Düsseldorf, Germany, 24-28 November 1992. WHO Regional Office for Europe, Report EUR/HFA Target 24, World Health Organization, Copenhagen, Denmark.

010392

WHO 1994 Assessing human health risks of chemicals: Derivation of guidance values for health-based exposure limits. Environmental Health Criteria No. 170, World Health Organization, Geneva, Switzerland.

WHO 1995a Concern for Europe's Tomorrow – Health and the Environment in the WHO European Region. WHO European Centre for Environment and Health. Wissenschaftliche Verlagsgesellschaft, Stuttgart, Germany.

WHO 1995b Noise - Selected presentations of an Informal Regional Consultation Meeting on Noise Pollution, 2-5 September 1991, Amman, Jordan, Eastern Mediterranean Regional Office, Regional Centre for Environmental Health Activities (CEHA), Amman, Jordan.

WHO 1998 Air Management Information System. AMIS CD ROM, 2nd edition. World Health Organization, Geneva, Switzerland.

Zaidi SH 1998 Current status and future trends of deafness and hearing impairment in the Tropics. Pakistan Journal of Otolaryngology 14: 38-45.

Zaidi SH 1999 An epidemiological study of hearing impairment in the Himalayan territories of Pakistan. Hearing International Newsletter 8: 1-4.

Zeichart K 1998 Kombintorische Wirkungen von Bahnlärm und Bahnerschütterungen. Zeitschrift für Lärmbekämpfung 45: 7-16.

Zwicker E 1960 Ein Verfahren zur Berechnung der Lautstärke. Acustica 10: 304-308.

Zwicker E 1989 On the dependence of unbiased annoyance on loudness. In G. Maling Jr. (ed.) Inter Noise 89, Vol. 2, pp. 809-14. Noise Control Foundation, Poughkeepsie, NY, USA. .

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Appendix 2 : Examples Of Regional Noise Situations

REGION OF THE AMERICAS

Latin America (Guillermo Fuchs, Argentina).

As more and more cities in Latin America surpass the 20 million inhabitants mark, the noise pollution situation will continue to deteriorate. Most noise pollution in Latin American cities comes from traffic, industry, domestic situations and from the community. Traffic is the main source of outdoor noise in most big cities. The increase in automobile engine power and lack of adequate silencing results in LAeq street levels >70 dB, above acceptable limits. Vehicle noise has strong low-frequency peaks at ~13 Hz, and at driving speeds of 100 Km/h noise levels can exceed 100 dB. The low-frequency (LF) noise is aerodynamic in origin produced, for example, by driving with the car windows open. Little can be done to mitigate these low-frequency noises, except to drive with all the windows closed. Noise exposure due to leisure activities such as carting, motor racing and Walkman use is also growing at a fast rate. Walkman use in the street not only contributes to temporary threshold shifts (TTS) in hearing, but also endangers the user because they may not hear warning signals Construction sites, pavement repairs and advertisements also contribute to street noise, and noise levels of 85–100 dB are common.

The Centro de Investigaciones Acústicas y Luminotécnicas (CIAL) in Córdoba, Argentina has investigated noise pollution in both the field and in the laboratory. The most noticeable effect of excessive urban noise is hearing impairment, but other psychophysiological effects also result. For example, tinnitus resulting from sudden or continuous noise bursts, can produce a TTS of 20–30 dB, and prolonged exposures can result in permanent threshold shifts (PTS). By analyzing sound spectra down to a few Hertz, and at levels of up to 120 dB, discrete frequencies and bands of infrasound were found which damage hearing. With LF sounds at levels of 120 dB, TTS resulted after brief exposure, and PTS after only 30 min of exposure. The effects of noise on hearing can be especially detrimental to children in schools located downtown. Field studies in Córdoba city schools located near streets with high traffic density showed that speech intelligibility was dramatically degraded in classrooms that did not meet international acoustical standards. This is a particularly worrying problem for the younger students, who are in the process of language acquisition, and interferes with their learning process.

In general, community noise in Latin America remains above accepted limits. Particularly at night, sleep and rest are affected by transient noise signals from electronically amplified sounds, music and propaganda. Field research was carried out in four zones of Buenos Aires, to determine the effects of urban noise on the well-being, health and activities of the inhabitants. The effects of confounding variables were taken into consideration. It was concluded that night-time noise levels in downtown Buenos Aires were barely lower than daytime levels. The results showed that sleep, concentration, communication and well-being were affected in most people when noise levels exceeded those permitted by international laws. The reactions of the inhabitants to protect themselves from the effects of noise varied, and included changing rooms, closing windows and complaining to authorities.

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Individual responses to noise also vary, and depend on factors such as social, educational and economic levels, individual sensibility, attitudes towards noise, satisfaction with home or neighborhood, and cognitive and affective parameters. For example, at CIAL, two pilot studies were carried out with a group of adolescents to determine the influence of environmental conditions on the perception of noise. When music was played at very high sound levels (with sound peaks of 119 dBA) in a discotheque, judged to be a pleasant environment, the subjects showed less TTS than when exposed to the same music in the laboratory, which was considered to be an unpleasant environment.

At the municipal level Argentinean Ordinances consider two types of noises: unnecessary and excessive. Unnecessary noises are forbidden. Excessive noises are classified according to neighboring activities and are limited by maximum levels allowed for daytime (7 am to 10 pm) and night-time (10 pm to 7 am). This regulation has been relatively successful, but control has to be continuous. Similar actions have been prescribed at the provincial level in many cities of Argentina and Latin America. Control efforts aimed at reducing noise levels from individual vehicles are showing reasonably good improvements. However, many efforts of municipal authorities to mitigate noise pollution have failed because of economic, political and other pressures. For example, although noise control for automobiles has shown some improvement, efforts have been counteracted by the growth in the number and power of automobiles.

CIAL has designed both static and dynamic tests that can be used to set annual noise control limits. For roads and freeways where permitted speeds are above 80 Km/h, CIAL has also designed barriers which protect buildings lining the freeways. Considerable improvements have been obtained using these barriers with noise reductions of over 20 dB at buildings fronts. The most common types of barrier are concrete slabs or wooden structures, made translucent or covered with vegetation. Planted vegetation does not act as an efficient noise shield for freeway noise, except in cases of thick forest strips. In several cities, CIAL also designed ring roads to avoid heavy traffic along sensitive areas such as hospitals, schools and laboratories.

Efforts have not been successful in reducing the noise pollution from popular sports such as carting, motorboating and motocross, where noise levels can exceed 100 dB. In part, this is because individuals do not believe these activities can result in hearing impairment or have other detrimental effects, in spite of the scientific evidence. Argentinean and other Latin American authorities also have not been successful in reducing the sound levels from music centres, such as discotheques, where sound levels can exceed 100 dB between 11 pm and 6 am. However, public protest is increasing and municipal authorities have been applying some control. For instance, in big cities, discotheque owners and others are beginning to seek advice on how to isolate their businesses from apartment buildings and residential areas. Some improvements have been observed, but accepted limits have not yet been generally attained.

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United States of America (Larry Finegold)

Noise Exposure.

In the United States, there have only been a few major attempts to describe broad environmental noise exposures. Early estimates for the average daily exposure of various population groups were reported in the U.S. Environmental Protection Agency's *Levels Document* (US EPA 1974), but these were only partially verified by subsequent large-scale measurements. Another EPA publication the same year provided estimates of the national population distribution as a function of outdoor noise level, and established population density as the primary predictor of a community's noise exposure (Galloway et al. 1974). Methodological issues that need be considered when measuring community noise, including both temporal and geographic sampling techniques, have been addressed by Eldred (1975). This paper also provided early quantitative estimates of noise exposure at a variety of sites, from an isolated spot on the North rim of the Grand Canyon to a spot in downtown Harlem in New York City. Another nationwide survey focused on exposure to everyday urban noises, rather than the more traditional approach of measuring exposure to high-level transportation noise from aircraft, traffic and rail (Fidell 1978). This study included noise exposure and human response data from over 2 000 participants at 24 sites.

A comprehensive report, *Noise In America: The Extent of the Problem,* included estimates of occupational noise exposure in the US in standard industrial classification categories (Bolt, Beranek & Newman, Inc. 1981). A more recent paper reviewed the long-term trends of noise exposure in the US and its impact over a 30-year time span, starting in the early 1970's. The focus was primarily on motor vehicle and aircraft noise, and the prediction was for steadily decreasing population-weighted day-night sound exposure (Eldred 1988). However, it remains to be seen whether the technological improvements in noise emission, such as changing from Chapter 2 to Chapter 3 aircraft, will be offset in the long run by the larger carriers and increased operations levels that are forecast for all transportation modes. Although never implemented in its entirety, a comprehensive plan for measuring community environmental noise and associated human responses was proposed over 25 years ago in the US (Sutherland et al. 1973).

Environmental Noise Policy in the United States

One of the first major breakthroughs in developing an environmental noise policy in the United States occurred in 1969 with the adoption of the National Environmental Policy Act (NEPA). This Congressional Act mandated that the environmental effects of any major development project be assessed if federal funds were involved in the project. Through the Noise Control Act (NCA) of 1972, the U.S. Congress directed the US Environmental Protection Agency (EPA) to publish scientific information about the kind and extent of all identifiable effects of different qualities and quantities of noise. The US EPA was also requested to define acceptable noise levels under various conditions that would protect the public health and welfare with an adequate margin of safety. To accomplish this objective, the 1974 US EPA *Levels Document* formally introduced prescribed noise descriptors and prescribed levels of environmental noise exposure. Along with its companion document, *Guidelines for Preparing Environmental Impact Statements on Noise*, which was published by the U.S. National Research Council in 1977, the

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Levels Document has been the mainstay of U.S. environmental noise policy for nearly a quarter of a century. These documents were supplemented by additional Public Laws, Presidential Executive Orders, and many-tiered noise exposure guidelines, regulations, and Standards. Important examples include *Guidelines for Considering Noise in Land Use Planning and Control*, published in 1980 by the US Federal Interagency Committee on Urban Noise; and *Guidelines for Noise Impact Analysis*, published in 1982 by the US EPA.

One of the distinctive features of the US EPA *Levels Document* is that it does not establish regulatory goals. This is because the noise exposure levels identified in this document were determined by a negotiated scientific consensus and were chosen without concern for their economic and technological feasibility; they also included an additional margin of safety. For these reasons, an A-weighted Day-Night Average Sound Level (DNL) of 55 dB was selected in the *Levels Document* as that required to totally protect against outdoor activity interference and annoyance. Land use planning guidelines developed since its publication allow for an outdoor DNL exposure in non-sensitive areas of up to 65 dB before sound insulation or other noise mitigation measures must be implemented. Thus, separation of short-, medium- and long-term goals allow noise-exposure goals to be established that are based on human effects research data, yet still allow for the financial and technological constraints within which all countries must work.

The US EPA's Office of Noise Abatement and Control (ONAC) provided a considerable amount of impetus to the development of environmental noise policies for about a decade in the US. During this time, several major US federal agencies, including the US EPA, the Department of Transportation, the Federal Aviation Administration, the Department of Housing and Urban Development, the National Aeronautics and Space Administration, the Department of Defense, and the Federal Interagency Committee on Noise have all published important documents addressing environmental noise and its effects on people. Lack of funding, however, has made the EPA ONAC largely ineffective in the past decade. A new bill, the *Quiet Communities Act* has recently been introduced in the U.S. Congress to re-enact and fund this office (House of Representatives Bill, H.R. 536). However, the passage of this bill is uncertain, because noise in the US, as in Europe, has not received the attention that other environmental issues have, such as air and water quality.

In the USA there is growing debate over whether to continue to rely on the use of DNL (and the A-Weighted Equivalent Continuous Sound Pressure Level upon which DNL is based) as the primary environmental noise exposure metric, or whether to supplement it with other noise descriptors. Because a growing number of researchers believe that "Sound Exposure" is more understandable to the public, the American National Standards Institute has prepared a new Standard, which allows the equivalent use of either DNL or Sound Exposure (ANSI 1996). The primary purpose of this new standard, however, is to provide a methodology for modeling the Combined or Total Noise Environment, by making numerical adjustments to the exposure levels from various noise sources before assessing their predicted impacts on people. A companion standard (ANSI 1998) links DNL and Sound Exposure with the current USA land use planning table. The latter is currently being updated by a team of people from various federal government agencies and when completed should improve the capabilities of environmental and community land-use planners. These documents will complement the newly revised ANSI standard on

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acoustical terminology (ANSI 1994).

To summarize progress in noise control made in the USA in the nearly 25 years since the initial national environmental noise policy documents were written, the Acoustical Society of America held a special session in Washington, D.C. in 1995. The papers presented in this special session were then published as a collaborative effort between the Acoustical Society of America and the Institute of Noise Control Engineering (von Gierke & Johnson 1996). This document is available from the Acoustical Society of America, as are a wide range of standards related to various environmental noise and bioacoustics topics from the ANSI.

A document from the European Union is now also available, which includes guidelines for addressing noise in environmental assessments (EU 1996). Policy documents from organizations such as ISO, CEN, and ICAO have shown that international cooperation is quite possible in the environmental noise arena. The ISO document, entitled *Acoustics - Description and Measurement of Environmental Noise* (ISO 1996), and other international standards have already proven themselves to be invaluable in moving towards the development of a harmonized environmental noise policy. The best way to move forward in developing a harmonized environmental noise policy is to take a look at the various national policies that have already been adopted in many countries, including those both from the European member states and from the USA, and to decide what improvements need to be made to the existing policy documents. A solid understanding of the progress that has already been achieved around the world would obviously provide the foundation for the development of future noise policies.

Implementation Concepts and Tools

Development of appropriate policies, regulations, and standards, particularly in the noise measurement and impact assessment areas, is a necessary foundation for implementing effective noise abatement policies and noise control programs. A well-trained cadre of environmental planners will be needed in the future to perform land-use planning and environmental impact analysis. These professionals will require both a new generation of standardized noise propagation models to deal with the Total Noise Environment, as well as sophisticated computer-based impact analysis and land-use planning tools.

A more thorough description of the current noise environment in major cities, suburbs, and rural areas is needed to support the noise policy development process. A new generation of noise measurement and monitoring systems, along with standards related to their use, are already providing considerable improvement in our ability to accurately describe complex noise environments. Finally, both active and passive noise control technologies, and other noise mitigation techniques, are rapidly becoming available for addressing local noise problems. Combined with a strong public awareness and education program, land-use planning and noise abatement efforts certainly have the potential to provide us with an environment with acceptable levels of noise exposure.

References

ANSI 1994 American National Samerican Standard Acoustical Terminology. American National Standard S1. American National Standards Institute, New York, NY, USA.

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ANSI 1996)Quantities and Procedures for Description and Measurement of Environmental Sound - Part 4: Assessment and Prediction of Long-Term Community Response. American National Standard S12.9-Part 4, American National Standard Institute, New York, NY, USA.
ANSI 1998 Quantities and Procedures for Description and Measurement of Environmental Sound - Part 5: Sound Level Descriptors for Determination of Compatible Land Use. American National Standard S12.9-Part 5, American National Standard Institute, New York, NY, USA.
Bolt, Beranek, Newman, Inc. (BBN) 1981 Noise in America: The extent of the problem. Cambridge, MA, USA.
Eldred km 1975 Assessment of community noise. Noise Control Engineering Journal 3: 88-95.
Eldred km 1988 Noise at the year 2000. In Proceedings of the Fifth International Congress on Noise as a Public Health Problem (B. Berglund et al., eds.). Stockholm: Swedish Council for Building Research.
Fidell S (1978) Nationwide urban noise survey. Journal of the Acoustical Society of America 64: 198-206.
Galloway W, Eldred K, and Simpson M 1974 Population distribution of the United States as a function of outdoor noise. US Environmental Protection Agency Report No. 550/9-74-009. Washington, D.C., USA.
Schori JR, McGatha EA (1973) A real-world assessment of noise exposure. Sound and Vibration 12: 24-30.
Sutherland LC, Braden, MH, and Colman R 1973 A programme for the measurement of environmental noise in the community and its associated human response. Vols. I and II. Report No. DOT-TST-74-5, Washington, D.C.: Department of Transportation, Office of Noise Abatement.
US EPA 1974 Information on levels of environmental noise requisite to protect public health and welfare with an adequate margin of safety. EPA/ONAC Report 550/9-74-004, U.S. Environmental Protection Agency, Washington, D.C., USA.
von Gierke HE, Johnson LC 1996 Noise Control - Where Do We Stand Today? Noise Control Engineering Journal.

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AFRICAN REGION

South Africa (Etienne Grond, South Africa)

Introduction

Cultural and developmental levels diverge greatly in South Africa, and the country can be divided into a first world sector, a developing sector and a third world sector. This contributes to huge variations in both the awareness of noise pollution and in population exposure to noise pollution. Noise-related health problems will in all probability show the same large variations.

Legal requirements

Noise control in South Africa has a history dating back about three decades. Noise control began with codes of practice issued by the South African Bureau of Standards (SABS) to address noise pollution in different sectors. Since then, Section 25 of the Environment Conservation Act (Act 73 of 1989) made provision for the Minister of Environmental Affairs and Tourism to regulate noise, vibration and shock at the national level. These regulations were published in 1990 and local authorities could apply to the Minister to make them applicable in their areas of jurisdiction. However, a number of the bigger local authorities did not apply for the regulations since they already had by-laws in place, which they felt were sufficient. By the middle of 1992 only 29 local authorities had applied the regulations and so the act was changed to make it obligatory for all authorities to apply the regulations. However, by the time the regulations were ready to be published, the new Constitution of South Africa came into effect and this listed noise control as an exclusive legislative competence of provincial and local authorities. This meant that the national government could not publish the regulations. However, provincial governments have agreed to publish the regulations in their respective areas. The regulations will apply to all local authorities as soon as they are published in the provinces, and will give local authorities both the power and the obligation to enforce the regulations.

The Department of Environmental Affairs and Tourism also published regulations during 1997 to make Environmental Impact Assessments mandatory for most new developments, as well as for changes in existing developments. This means that any impact that a development might have on its surrounding environment must be evaluated and, where necessary, the impact must be mitigated to acceptable levels. The noise control regulations also state that a local authority may declare a “controlled area,” which is an area where the average noise level exceeds 65 dBA over a period of 24 h period. This means that educational and residential buildings, hospitals and churches may not be situated within such areas.

Occupational noise exposure is regulated by the Department of Manpower, under the Occupational Health and Safety Act (Act 85 of 1993). These regulations states that workers may not be exposed to noise levels of higher than 85 dBA and that those exposed to such levels must make use of equipment to protect their hearing. The problem, however, is that most workers tend not to make use of the provided equipment, either because the equipment is not comfortable, or because they are not aware of the risks high noise levels pose to their hearing. A further problem is that small industries often do not supply the workers with the necessary

equipment, or supply inferior equipment that is less costly.

Codes of practice

The codes of practice issued by the SABS were for the most part replaced by IEC (International Electrotechnical Commission) standards and adopted as SABS ISO codes of practice. They are still being used in South Africa and are regularly updated. A relevant list can be found in the references. The SABS has also published a number of recommended practices (ARP). These include the ARP 020: "Sound impact investigations for integrated environmental management" that is currently being upgraded to a code of practice. Such codes of practice can be referred to as requirements in legislation and will be known as SABS 0328: "Methods for environmental noise impact assessments." The codes of practice published in South Africa cover hearing protection; measurement of noise; occupational noise; environmental noise; airplane noise; and building acoustics, etc.

Courses

Local authorities responsible for applying regulations published by the Department of Environmental Affairs and Tourism must employ a noise control officer who has at least three years tertiary education in engineering, physical sciences or health sciences, and who is registered with a professional council. Alternatively, a consultant with similar training may be employed. Most of the universities in South Africa provide the relevant training, with at least part of the training in acoustics. Universities and technical colleges also provide a number of special acoustics courses. Over the last couple of years awareness of environmental conservation has expanded dramatically within the academic community, and most universities and colleges now have degree courses in environmental management. At the very least, these courses include a six-month module in acoustics, and usually also include training in basic mathematics; the physics of sound; sound measuring methodologies; and noise pollution.

Community awareness and exposure to noise pollution

This topic should be discussed with respect to three separate population sectors: the first-world sector (developed), the developing sector and the third-world sector (rural).

Developed sector

This sector of the population is more-or-less as developed as their European and American counterparts. They have been exposed to noise pollution for a considerable time and, for the most part, are aware of the health consequences of high noise levels. People in this group are also aware of the existence of legal measures by which noise pollution can be addressed. Not surprisingly, most of the complaints and legal action regarding noise pollution are received from this group. Information about noise-related health problems is very limited, but because this group is highly aware of the risks posed by high noise levels, future studies will probably show that people in this category have the fewest health problems. The majority of people in this group are less exposed to high noise levels at work, and they live in more affluent neighborhoods with large plots and separating walls. Their houses tend to be built with materials that are noise

reducing. They also live further away from major noise-producing activities, such as highways, airports and large industries.

Developing sector

This sector of the population has the greatest exposure to high noise levels, both at home and in the workplace. Overall, they are relatively poor and cannot afford to live in quiet areas, or afford large plots or solid building materials. A large component of this sector resides in squatter communities where building are made of any material available, from plastic to corrugated sheets and wood. The buildings are right next to each other and there is almost no noise attenuation between residencies.

People in this category usually live close to major access routes into the cities, because they make use of public transportation and taxis to get to their places of work. Often, too, they live close to their places of work, which are usually big industries with relatively high levels of noise pollution. These people usually work in high noise areas, and because of their lack of awareness of the effects of high noise levels, often do not make use of available hearing protection equipment. Because of a lack of funds, these people also cannot get out of high noise areas and go to recreational areas for relaxation and lower noise levels. Not much information is available on the adverse health problems in this sector. However, workers in this sector should undergo regular medical examinations and the results can be obtained from the industries involved.

Rural sector

As the name suggests, people in this sector live in rural surroundings and for the most part are not subjected to noise levels that could be detrimental to their health. However, they are almost totally unaware of the risks posed by high noise levels. Some of these people work on farms and work with machinery that emits relatively high noise levels, but because of their lack of awareness they do not make use of hearing protection equipment. One advantage they do have is that they return to homes in quiet surroundings and their hearing has a chance to recover. To date, no studies have been carried out to determine the state of their hearing and it would be impossible to state that they have no health problems related to high noise levels.

References

Environment Conservation Act (Act No. 73 of 1989) Noise Control Regulations, Ministry of Environmental Affairs and Tourism, Pretoria, South Africa.

Relevant SABS codes of practice:

SABS 083-1996. The measurement and assessment of occupational noise for hearing conservation purposes.

SABS 0103-1994. The measurement and rating of environmental noise with respect to annoyance and speech communication (third revision).

SABS 0115-1974. The measurement of noise and the determination of disturbance from aeroplanes for certification purposes.

SABS 0117-1974. The determination and limitation of disturbance around an aerodrome due to noise from aeroplanes (Amendment no 1 - 1984).

SABS 0205-1986. The measurement of noise emitted by motor vehicles in motion.

SABS 0210-1996. Calculating and predicting road traffic noise (Amendment no 1 - 1997).

SABS 0211-1987. The measurement of noise inside motor vehicles.

SABS 0218/1-1988. Acoustical properties of buildings Part 1: The grading of buildings according to their airborne-sound insulation properties (under revision).

SABS 0218/2-1988. Acoustical properties of buildings Part 2: The assessment of building plans and buildings with respect to their acoustical properties (under revision).

SABS 0234-1991. Determination of sound power levels of multi-source industrial plants for the evaluation of the sound pressure levels in the environment - Engineering method (Incorporating ISO 8297:1994).

ARP 020-1992. Sound impact investigations for integrated environmental management. (To be superseded and replaced by SABS 0328: Methods for environmental noise impact assessments).

EASTERN MEDITERRANEAN REGION (Shabih H. Zaidi)

Scope

In the Eastern Mediterranean region some countries have highly developed industries, while others have none. In other cases, the agricultural economy is inseparably mixed with high-technology industries, such as the oil industry, which can be seen in nearly the whole of the Arabian Peninsula. Other examples of where agriculture and industry are intertwined can be seen in Pakistan, Jordan and Egypt. The main focus of this paper is community noise, but because industry is so widely distributed, some discussion of industrial noise is inevitable. The scope of this paper is to document the available scientific data on community noise in the WHO Regional Office of the Eastern Mediterranean (EMRO) region, including preventive strategies, legislation, compensation and future trends.

Sources of Noise Pollution

Sources of noise pollution in the Eastern Mediterranean region include noise from transportation, social and religious activities, building and civil works, roadside workshops, mechanical floor shops and others. During civil works and building booms, noise levels in all countries of the Eastern Mediterranean region could easily reach 85dBA during the daytime over an 8 h work period. In Pakistan, unprotected construction work goes on at all times of the day and night and uses outdated machinery; and the noise is compounded by workers shouting. On a typical building site noise levels reach 90–100 dBA.

In Karachi, the main artery for daily commuters is a long road that terminates at the harbor. In the densest area of this road there are a hundred small and large mechanical workshops, garages, metal sheet workers, dent removers, painters, welders and repair shops, all of which create a variety of noises. In the middle of this area at the Tibet Centre the LAeq,8h is 90dBA (Zaidi 1989). A similar picture is seen elsewhere in cities like Lahore, Peshawar, etc. Fortunately, the same is not true for other newly built cities in the EMRO region, such as Dubai, or Tripoli, where strict rules separate industrial zones from residential areas.

A special noise problem is Karachi harbour. This port serves the whole of Pakistan as well as Afghanistan and several Asian states, such as Kyrgyzstan, Kazakhstan and Uzbekistan. The noise level at the main wharf of Karachi Port ranges between 90–110 dBA on any given day. Other special sources of noise are the Eastern Mediterranean airports, and indeed most of the airports in the Middle East. Most northbound air traffic originates in Pakistan, Dubai, Sharjah etc. and flights usually depart after midnight so as to arrive in Europe during the daytime. A study is currently underway in Karachi to identify the damage caused by these nocturnal flights to those living under the flight path (SH Zaidi, GH Shaikh & AN Zaidi, personal communication).

Sadly, violence has become part of Eastern culture and is a significant source of noise pollution. Wars generate a lot of noise, and although noise-induced hearing loss is a secondary issue compared with the killing, after the wars many people are hearing impaired. This has been seen following conflicts in Balochistan, Peshawar and Afghanistan, where perforated ear drums,

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profound hearing loss and stress-related psychosomatic illnesses are common in the refugee camps. The noise levels during a recent mass demonstration in Karachi, which included the firing of automatic weapons, reached 120 dBA at a distance of 50 m from the scene.

The Effects of Noise on Health

There is good evidence that environmental noise causes a range of health effects, including hearing loss, annoyance, cardiovascular changes, sleep disturbance and psychological effects. Although the health effects of noise pollution have not been documented for the entire EMRO region, data are available for Pakistan and can be used to illustrate the general problem. In this report, noise exposure is mainly expressed as LAeq,24h values.

Noise-induced hearing loss (NIHL).

It is believed that exposure to environmental noise in the EMRO countries is directly related to the living habits, economic prosperity and outdoor habits of people. It has been estimated that no more than 5% of the people are exposed to environmental sound levels in excess of 65dBA over a 24-h period. Similarly, for indoor noise, it is believed that the average family is not exposed to sound levels in excess of 70 dBA over a 24-h period. However, it is difficult to generalize for all countries in the EMRO region, because of ancient living styles and different cultural practices, such as taking siestas between 13:00–16:00 and stopping work at 20:00.

Exposure to noise while travelling to schools, offices or workplaces may vary tremendously between cities in the region. In Karachi, for example, traffic flow is undisciplined, erratic and irrational, with LAeq,8h values of 80–85 dBA. In Riyadh, by contrast, traffic flow is orderly with LAeq levels of 70 dBA during a normal working day. In Karachi, noise levels show significant diurnal variation, reaching levels in excess of 140 dB during the peak rush hour at around 5.00 p.m. (Zaidi 1989). At the Tibet Centre, located at a busy downtown junction, noise levels were 60–70 dB at 9 am, but reached levels in excess of 140 dB between 5-7 p.m. A study conducted on a day that transportation workers went on strike established that road traffic is the most significant source of noise pollution in this city: in the absence of buses, rickshaws, trucks and other public vehicles the LAeq level declined from 90dB to 75dB (Zaidi 1990). Motor engines, horns, loud music on public buses and rickshaws generate at least 65% of the noise in Karachi (Zaidi 1997; Shams 1997). Rickshaws can produce noise levels of 100–110 dBA and do not have silencers. On festive occasions, such as national holidays or political rallies, motorbikes running at high speeds along the Clifton beach in Karachi easily make noise exceeding 120 dBA. (Zaidi 1996).

Another study conducted at 14 different sites in Karachi showed that, in 11 of the sites, the average noise level ranged between 79–80 dB (Bosan & Zaidi 1995). The maximum noise levels at all these sites exceeded 100 dB. Speech interference, measured by the Preferred Speech Interference Level and the Articulation Index, was significant (Shaikh & Rizvi 1990). The study results indicated that two people facing each other at a distance of 1.2 m would have to shout to be intelligible; and the Articulation Indexes demonstrated that communication was unsatisfactory. Of perhaps greater concern are the results of a survey of 587 males between the ages of 17 and 45 years old, who worked as shopkeepers, vehicle drivers, builders and office

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assistants. Audiograms showed that 14.6% of the subjects had significant hearing impairment at 3 000–4 000 Hertz (Hasan et al., 2000).

Noise pollution from leisure activities can vary from country to country in the EMRO region. The Panthans in northern Pakistan, for example, like to shoot in the air on festive occasions, such as weddings, without using any noise protection devices. A minimum of 1 000 shots are fired on such occasions; and at a traditional tribal dance called the 'Khattak" the noise level recorded during a particularly enthralling performance in a sports arena was 120dBA. The hunting of wild boar is a common sport in the hinterlands of Sindh. With the rifle shots and the noise made by the beaters, noise levels can easily reach 110 –120 dBA. In some EMRO countries, the younger crowd has taken up the Western habit of listening to Pop music for many hours. Discos and floorshows are confined to a few countries, such as Egypt. Open-air concerts are usually held in stadiums. The noise level recorded at a particularly popular concert was 130 dBA at a distance of 20 m from the stage and 35 m from the amplifiers.

In a study of road traffic at 25 different sites in Peshawar, the third most populous city in Pakistan, 90 traffic constables were taken as cohorts to investigate the extent of NIHL. Of these, 50 did not have any previous history of noise exposure and were taken as controls. Detailed evaluation and audiological investigations established that constables exposed to a noise level of 90 dBA for 8 hours every day suffered from NIHL. Compared to the control subjects, the constables had significant hearing impairment at 3 000 Hz, measured by Pure Tone Audiometry (Akhter 1996).

A similar study of traffic constables in Karachi showed that 82.8% of the constables suffered from NIHL (Itrat & Zaidi 1999). The study also showed that 33.3% of rickshaw drivers, and 56.9% of shopkeepers who worked in noisy bazaars, had hearing impairment. If these findings can be extrapolated to the total populations, there are 1 566 traffic constables (out of a total of 1 890 constables), and 4 067 rickshaw drivers (out of a total of 12 202 drivers) who suffer from NIHL. As has been reported by other researchers, the study also found evidence of acclimatization in the subjects: following an initial, rapid decline, hearing loss stabilized after prolonged noise exposure.

***Annoyance*.**

The citizens of Karachi commonly complain that noise causes irritability and stress. The main sources have been identified as traffic noise, industrial noise and noise generated by human activity. Unfortunately no data are available for the level of annoyance caused by noise exposure in the EMRO region. From limited research around the world, it can be estimated that 35–40% of employees in office buildings are seriously annoyed by noise at sound levels in excess of 55–60 dBA. In countries such as Pakistan, Iran, Jordan and Egypt that level is often seen in most offices. Annoyance is a non-tangible entity and cannot be quantified scientifically. It is a human reaction and perhaps its parameters could include irritability, apprehension, fear, anger, frustration, uneasiness, apathy, chaos and confusion. If such are the parameters, then on a scale of 0–10, with 10 being the greatest annoyance, many EMRO countries could easily score 6 or higher.

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Effects of noise on sleep and the cardiovascular system.

In the Eastern Mediterranean region no specific data are available on the effects of noise on sleep or the cardiovascular system. However, factory workers, traffic constables, rickshaw drivers and shopkeepers frequently complain about fatigue, irritability and headaches; and one of the most common causes of poor performance in offices is sleep disturbance. The rising incidence of tinnitus in cities like Karachi is also related to noise exposure, and tinnitus itself can lead to sleep deprivation. Although the effects of noise on the cardiovascular system have been well documented for other countries (Berglund & Lindvall 1995), data are lacking for the EMRO region. However, the prevalence of cardiovascular diseases are on the rise in the EMRO countries, particularly hypertension. While most of the increase in these diseases is due to a rich diet and lack of exercise, the relationship between noise and cardiovascular changes is worth investigating.

The risk to unborn babies and newborns.

Although evidence from other countries indicates that noise may damage the hearing of a fetus, there are no data from the EMRO countries to confirm this. With newborn babies, however, noise from incubators is a major cause of hearing loss in the EMRO region, particularly as 20–27% of them are born underweight (Razi et al. 1995). Once exposed to noise in an incubator, the chances of hearing impairment rapidly rises compared with cohorts in developed countries. Several other factors have also been identified as causing deafness and hearing impairment in newborns in the Eastern Mediterranean region (Zaidi 1998; Zakzouk et al. 1994). They are:

a. Discharge from the ears.

b. Communicable infections.

c. Ototoxicity.

d. Noise.

e. Consanguinity.

f. Iodine deficiency.

Noise Control

Although noise control legislation exists in several EMRO countries, it is seldom enforced, particularly in Pakistan and some neighboring countries. Noise control begins with education, public awareness and the appropriate use of media in highlighting the effects of noise. In Calcutta, for instance, public orientation and mass media mobilization have produced tangible results, and this can easily be done in other countries. Three strategies have been devised for noise control, all of which are practicable in EMRO region countries. They are control at the source, control along the path and control at the receiving end.

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There are many ways noise can be controlled at the source. For example, most of the equipment and machinery used in EMRO countries is imported from the West. Noise control could begin by importing quieter machinery, built with newer materials like ceramics or frictionless parts. And at the local level, the timely replacement of parts and proper maintenance of the machines should be carried out. Vehicles like the rickshaw should be banned, or at least be compelled to maintain their silencers, and all vehicles must be put to a road worthiness test periodically. This already occurs in some EMRO countries, but not all. Horns, hooters, music players and other noise making factors must also be controlled. The use of amplifiers and public address systems should also be banned, and social, leisure and religious activities should be restricted to specific places and times.

Along the sound path, barriers can be used to control noise. There are three kinds of barriers available, namely, space absorbers made out of porous material, resonant absorbers and panel absorbers. Architects, for example, use hollow blocks of porous material. The air gaps between building walls not only keep the buildings cool in hot weather, but also reduce the effects of noise. Ceilings and roofs are often treated with absorbent material. In large factories, architects use corrugated sheets and prefabricated material, which are helpful in reducing noise levels. In Pakistan, some people use clay pots in closely ranked positions on rooftops to reduce the effect of heat as well as noise. For civic works and buildings, special enclosures, barriers and vibration controlling devices should be used. Public halls, such as cinemas, mosques and meeting places should have their walls and floors carpeted, and covered with hangings, mats etc. An effective material is jute, which is grown in many countries, mainly Bangladesh, and it is quite economical. Some of the old highways and most of the busy expressways need natural noise barriers, such as earth banks, trees and plants.

References.

Akhter NH 1996 Noise induced hearing loss in traffic police constables of Peshawar. Journal of College of Physicians and Surgeons Pakistan 6: 265-268.

Berglund B and Lindvall T 1995 Community Noise. Archives of the Centre for Sensory Research, volume 2, issue 1, pp. 56,57. Stockholm University and Karolinska Institute, Stockholm.

Bosan Altaf, Zaidi SH Nobel Tracy 1995 The problem of noise, Pakistan Journal of Otolaryngology 11, 128-131.

Hasan S, Zaidi AN, Tahira S, Zaidi SH 2000 Audiological profile of NIHLin IMPACT Ear Camps. Pakistan Journal of Oto-Rhino-Laryngology, in print.

Itrat J, Zaidi SH 1999 Deafness in Pakistan, Pakistan Journal of Otolaryngology 15: 78-83.

Razi MS, Jaffer S, Hillier V, Zaidi SH, Newton V 1995 Causes of bilateral hearing loss in school children. Pakistan Journal of Otolaryngology 11: 68-86.

Shams ZI 1997 The hazards of noise pollution. Daily Dawn, 30th September 1997.

Shaikh GH, Rizvi SSH 1987 Road transport in Karachi city. Pakistan Journal of Science and research, 30: 197.

The environmental protection agency Sindh. The study of noise pollution in Karachi. Executive Report. Karachi Pakistan. ACE (pvt.) Ltd. Karachi.

Zaidi SH 1989 Noise level and the sources of noise pollution in Karachi. Journal of Pakistan Medical Association, 39: 62-65.

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Zaidi SH 1990 Noise level in Karachi on a transporter's strike day. Journal of Pakistan Medical Association, 40: 299-300.
Zaidi SH 1996 Noise Ototoxicity. Pakistan Journal of Otolaryngology, 12: 194.
Zaidi SH 1997 Noise induced hearing loss in Pakistan. Hearing International, 6: 12-13.
Zaidi SH 1998 The causes of deafness and Hearing Impairment in the Tropics: Keynote speech delivered at the Centennial Congress of Liverpool School of Tropical Medicine in Sept 1998. Pakistan Journal of Otolaryngology 14: 102-107.
Zakzouk SM, Bafaqeh SA, Al-Mahmmod H, Essa A 1994 Demographic factors and hearing impairment in Saudi Arabian Children., Journal of Laryngology and Otology 108: 294-298.

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SOUTH-EAST ASIAN REGION. (Sudhakar B. Ogale)

Introduction

The ability to hear sound is a sensory function vital for human survival and communication. However, not all sounds are wanted. Unwanted sounds, for which the term "noise" is normally used, often originate from human activities such as road traffic, rail traffic, aircraft, discos, electric power generators, festivals, firecrackers and toys. In general, however, data on noise pollution in South east Asian countries are not available. For example, there are no comprehensive statistical data regarding the incidence and etiology of hearing impairment. Consequently, it is difficult to estimate the exact percentage of the population affected by community noise.

Excessive noise is the major contributor to many stress conditions. It reduces resistance to illness by decreasing the efficiency of the immune system, and is the direct cause of some gastrointestinal problems. Noise also increases the use of drugs, disturbs sleep and increases proneness to accidents. An increased incidence of mental illness and hospital admissions, increases in absenteeism from work and lethargy from sleep disturbance all result from noise pollution and cause considerable loss of industrial production.

Noise Exposure in India

India is rapidly becoming industrialized and more mechanized, which directly affects noise levels. However, no general population study regarding the magnitude of the noise problem in India has been performed.

Road Traffic Noise

Exposure. A study by the Indian Institute of Road Traffic (IRT) reported that Delhi was the noisiest city in India, followed by Calcutta and Bombay (IRT 1996; Santra & Chakrabarty 1996). The survey examined whether road-traffic noise affected people with respect to annoyance, sleep disturbance, interference with communication and hearing impairment. It showed that 35% of the population in four major cities have bilateral sensory neural hearing loss at noise emission levels above 82 dBA. This is of particular concern in light of a second study, showing that LAeq,24h levels at 24 kerbside locations in Calcutta were 80–92 dBA (Chakrabarty et al. 1997) The mean noise emission levels of four different vehicle categories are presented in Table A2.1.

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Table A2.1: Mean noise emission levels of vehicles

Type of vehicle	Mean sound pressure level
2 wheelers (motor cycle)	82 dBA
3 wheelers (auto rickshaw)	87 dBA
Motor car (taxi, private cars)	85 dBA
Heavy vehicles (trucks)	92 dBA

Control Measures. Only recently has noise pollution been considered an offence in India, under the Environmental (Protection) Act 1986. Several measures are being taken to reduce traffic-noise exposure. These include:

a. Planting trees, shrubs and hedges along roadsides.

b. Mandatory, periodic vehicle inspections by road traffic control.

c. Reintroduction of silent zones, such as around schools, nursing homes and hospitals that face main roads.

d. Regulation of traffic discipline, and a ban on the use of pressure horns.

e. Enforcement of exhaust noise standards.

f. Mandating that silencers be effective in three-wheeled vehicles.

g. The use and construction of bypass roads for heavy vehicles.

h. Limiting night-time access of heavy vehicles to roads in residential neighbourhoods

i. Installation of sound-proof windows.

j. Proper planning of new towns and buildings.

Air Traffic Noise

Many airports were originally built at some distance from the towns they served. But due to growing populations and the lack of space, buildings are now commonly constructed alongside airports in India.

Exposure. A survey revealed that aircraft produced a high level of noise during take-off, with sound pressure levels of 97–109 dBA for the Airbus, and 109 dBA for Boeing aircraft (SB Ogale, unpublished observations). During landing, the aircraft produced a sound pressure level of 108 dBA. Although exposure to aircraft noise is considered to be less of a problem than exposure to traffic noise, the effects of air-traffic noise are similar to those of road traffic, and include palpitations and frequent awakenings at night.

Control measures. The use of ear muffs must be made obligatory at the airport. This can reduce noise exposure to a safe level. An air-traffic control act should also enforce the use and introduction of low-noise aircraft, and mandate fewer night-time flights.

Rail Traffic Noise

Very little attention has been paid to the problems of railway noise.

Exposure. In Bombay, where the majority of residential buildings are situated on either side of railway tracks, residents are more prone to suffer from acoustic trauma. More than 14% of the population in Bombay suffer from sleep disturbances during night, due to high-speed trains and their whistling. A study on surface railways (SB Ogale, unpublished observations) revealed that platform noise was 71–73 dBA in the morning and 78–83 dBA in the evening. The noise from loudspeakers mounted in the platform was 87–90 dBA. At a distance of 1 m from the engine, the whistle noise was 105–108 dBA for a train with an electric engine, up to 110 dBA for a train with diesel engine and 118 dBA for steam engine trains. Vacuum brakes produced noise levels as high as 95 dBA. This suggests that unprotected railway staff on platforms are at risk of permanent noise induced hearing loss.

Festival noise

Festival noise in India was first surveyed in Bombay in late 1970, during the Ganpati festival period. A similar study (Santra et al. 1996) was conducted soon after in Calcutta at the Durga Pooja festival during evening hours (18:00–22:00). The music from loudspeakers produces sound pressure levels of more than 112 dBA. During the festival period the residents experienced a noisy environment for 8–10 h at a stretch, with noise level of 85–95 dBA. This level is above the 80 dBA limit set by WHO for industrial workers exposed to noise for a maximum period of 8 hours.

Control measures. In a religious country, it is politically difficult to restrict religious music, even in the interests of public health. A ban on all music from loudspeakers after 22:00 would decrease the sound pressure levels to below the permissible legal limit. A preventive programme is advocated to measure noise levels with sound level metres.

Fire crackers and toy weapons noise

Exposure. A study conducted by Gupta & Vishvakarma (1989) at the time of Deepawali, an Indian festival of fireworks, determined the auditory status of 600 volunteers from various age groups, before and after exposure to firecrackers. The study also measured the acoustical output of representative samples of toy weapons and firecrackers, and the noise intensity level at critical spectator points. The average sound level at a distance of 3 m from the noise source was 150 dBA, exceeding the 130 dBA level at which adults are at risk for hearing damage. On average, 2.5% of the people surveyed during Deepawali had persistent sensory neural hearing loss of 30 dBA, with those in the 9–15 year old age group being most affected.

Control Measures. A judicious approach in the manufacture and use of toy weapons and firecrackers is encouraged, in addition to legal restraints. Fireworks should be more a display of light, rather than sound.

Generator Noise

Diesel generators are often used in India to produce electric power. Big generators produce sound pressure levels exceeding 96 dBA (SB Ogale, unpublished observations).

Conclusions

No comprehensive statistical data are available for community noise in India, however, the main sources of environmental noise are road traffic, air traffic, rail traffic, festivals, firecrackers and diesel generators. The adverse effects of noise are difficult to quantify, since tolerance to noise levels and to different types of noise varies considerably between people. Noise intensity also varies significantly from place to place. It should also be noted that noise data from different countries are often not obtained by the same method, and in general models have been used which are based on data from a limited number of locations. Noise control measures could be taken at several levels, including building design, legal measures, and educating the people on the health dangers of community noise. In India, what is needed now is noise control legislation and its strict enforcement, if a friendly, low-noise environment is to be maintained.

Noise Exposure in Indonesia

According to a report by the WHO, the noise exposure and control situation in Indonesia is as follows (Dickinson 1993).

Exposure. No nationwide data are available for Indonesia. However, during the last three decades there has been rapid growth in transportation, industry and tourism in Indonesia.

Control Measures. With the large majority of people having little income, protection of the physical environment has not been a first-order priority. The following recommendations have been made with respect to community noise (Dickinson 1993):

a. The cities of Indonesia have relatively large populations and each provincial government will need the staff and equipment to monitor and manage the environment.

b. Sound level meters with noise analysis computer programmes should be purchased.

c. Training courses and adequate equipment should be provided.

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d. Noise management planning for airports should be promoted.

e. Reduction measures should be taken for road-traffic noise.

Noise Exposure in Bangladesh

Exposure. In Bangladesh no authentic statistical data on the effects of community noise on deafness or hearing impairment are available (Amin 1995).

Control Measures. Governments have meager resources, a vast population to contend with and high illiteracy rates; consequently, priorities are with fighting hunger, malnutrition, diseases and various man-made and natural calamities. The governments are unable to give the necessary attention towards the prevention, early detection and management of noise disabilities in the country. Close cooperation is needed between the national and international organizations, to exchange ideas, skills and knowledge (Amin 1995).

Noise Exposure in Thailand

Exposure. Noise from traffic, construction, and from factories and industry has become a big problem in the Bangkok area. The National Environmental Board of Thailand was set up two decades ago and has been active in studying the pollution problems in Thailand. Indeed, a committee on noise pollution control was set up to study the noise pollution in Bangkok area and its surroundings. Although regulations and recommendations were made for controlling various sources of noise, the problem was not solved due to a lack of public awareness, the difficulty of proving that noise had adverse effects on health and hearing, and the difficulty of getting access to control noise. A general survey revealed that 21.4% of the Bangkok population is suffering from sensory neural hearing loss (Prasanchuk 1997). Noise sources included street noise, traffic noise, industrial noise and leisure noise.

Control Measures. In 1996, regulations for noise pollution control set LAeq,24h levels at 70 dBA for residential areas, and less than 50 dBA to avoid annoyance. The National Committee on Noise Pollution Control has been asked to study the health effects of noise in the Bangkok area and its surroundings, and determine whether these regulations are realistic and feasible.

References.

Amin 1995 Community Noise. Proceedings of the International Symposium on Deafness and Hearing Impairment in Developing Countries, Manchester, 6-8 July 1995.
Chakrabarty D, Santra SC, Mukherjee A, Roy B, Das P 1997 Status of road traffic noise in Calcutta metropolis, India. Journal of the Acoustical Society of America 101: 943-949
Dickinson P (1993) Noise assessment and management in Indonesia. Report SEA/EH 461, Regional Office for South-East Asia, World Health Organization, New Delhi, India.
Gupta D, Vishvakarma SK. 1989 Toy weapons and fire crackers. Laryngoscope 99: 330-334.

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IRT 1996 Indian Council of Medical Research Bulletin Vol. 26, No. 8, August 1996, New Delhi.
Prasanchuk S (1997) Noise pollution. Its effect on health and hearing – Thailand as a case study. Presentation at the PDH Informal Consultation on the Prevention of Noise-induced Hearing Loss, 28-30 October 1997, WHO/PDH/98.5. World Health Organization, Geneva, Switzerland.
Santra SC, Chakrabarty D 1996 Need for rationalization of noise standards in Indian perspectives. Presented at the First National Workshop on Development and Use of Environmental Reference Materials, 14-16 February 1996, Central Pollution Control Board, New Delhi.
Santra SC, Chakrabarty D, Mukherjee A, Roy B 1996 Noise status of Calcutta metropolis. A resume. Man and Environment, Annual Report, pp. 19-22.

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WESTERN PACIFIC REGION.

In this section, information on noise pollution and control will be given for three countries in the Western Pacific Region, namely Australia, the People's Republic of China and Japan. From a noise pollution point of view China may be viewed as a developing country, whereas Japan and Australia, with their high level of industrialization, represent developed countries.

Australia (Andrew Hede & Michinori Kabuto)

***Exposure*.** Australia has a population of 18 million with the majority living in cities that have experienced increasing noise pollution from a number of sources. The single most serious source of noise is road traffic, although in major cities such as Sydney, Melbourne and Perth, large communities are exposed to aircraft noise as well. Other important sources of noise pollution are railway noise and neighbourhood noise (including barking dogs, lawn mowers and garbage collection). A particular problem in Australia is that the climate encourages most residents to live with open windows, and few houses have effective noise insulation.

A study of road-traffic noise was conducted at 264 sites in 11 urban centres with populations in excess of 100 000 people (Brown et al. 1994). Noise was measured one metre from the façade of the most exposed windows and at window height. From the results, it was estimated that over 9% of the Australian population is exposed to LA10,18h levels of 68 dB or greater, and 19% of the population is exposed to noise levels of 63 dB or greater. In terms of LAeq values for daytimes, noise exposure in Australia is worse than in the Netherlands, but better than in Germany, France, Switzerland or Japan.

Control. In the mid-1990's, when a third runway was built at Sydney Airport, the government funded noise insulation of high-exposed dwellings. Increasingly, too, major cities are using noise barriers along freeways adjacent to residential communities. In most states barriers are mandatory for new freeways and for new residential developments along existing freeways and major motorways. There has been considerable testing of noise barriers by state agencies, to develop designs and materials that are cost effective.

Brown AL et al. (1994) Exposure of the Australian Population in Road Traffic Noise. Applied Acoustics 43: 169-176.
OECD (1991) Fighting Noise in the 1990's. Organization for Economic Cooperation and Development, Paris, France.

China (Chen Ming)

Introduction

Urban noise pollution has become a contemporary world problem. Urban noise influences people's living, learning and working. People exposed to noise feel disagreeable and cannot concentrate on work. Rest and sleep are also disturbed. People exposed to high-intensity noise

do not hear alarm signals and cannot communicate with each other. This can result in injury and, indeed, with the modernization of China, construction accidents related to noise are increasing. According to statistics for several cities in China, including Beijing, Shanghai, Tientsin and Fuzhou, the proportion of total accidents that were noise related was 29.7% in 1979, 34.6% in 1980, 44.8% in 1981 and 50% in 1990. It is therefore very important to control noise pollution in China.

Long-term exposure to urban environmental noise can lead to temporary hearing loss (assessed by temporary threshold shift), permanent hearing loss (assessed by permanent threshold shift) or deafness. Microscopy studies have shown that in people exposed to noise for long periods, hair cells, nerve fibers and ganglion cells were absent in the cochleae, especially in the basal turns. The primary lesion is in the 8–10 mm region of the cochlea, which is responsible for detecting sound at a frequency of 4 000 Hz. People chronically exposed to noise may first complain about tinnitus and, later on, about hearing loss. This is especially true for patients who have bilateral hearing loss at 4 000 Hz, but who have relatively good hearing other frequencies. Non-auditory symptoms of noise include effects on the nervous system, cardiovascular system and blood system. These symptoms were rarely observed in China in the past, but today more and more people complain about hearing damage and non-auditory physiological effects.

Urban environmental noise has thus become a common concern of all members of society. A key to resolving the complex noise issue lies in the effective control of urban noise sources. Control measures include reducing noise at its source, changing noise transmission pathways, building design, community planning and the use of personal hearing protection.

Urban environmental noise sources can be divided into industrial noise, traffic noise, building architecture noise and community district noise sources. Only the last three types are of concern here.

Traffic Noise

There are four sources of traffic noise: road traffic, railway transport, civil aviation and water transport; of these, road traffic is the main source of urban noise. The sound emission levels of heavy-duty trucks are 82–92 dBA and 90–100 dBA for electric horns; air horns are even worse, with sound emission levels of 105–110 dBA. Most urban noise from automobiles is in the 70–75 dB range, and it has been estimated that 27% of all complaints are about traffic noise. When a commercial jet takes off, speech communication is interrupted for up to 1 km on both sides of the runway, but people as far away as 4 km are disturbed in their sleep and rest. If a supersonic passenger plane flies at an altitude of 1 500 m, its sound pressure waves can be heard on the ground in a 30–50 km radius.

Building Noise

As a result of urban development in China, construction noise has become an increasingly serious problem. It is estimated that 80% of the houses in Fuzhou were built in the past 20 years. According to statistics, the noise from ramming in posts and supports is about 88 dB and the noise from bulldozers and excavators is about 91 dB, 10 m from the equipment. About 98% of

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industrial noise is in the 80–105 dB range, and it is estimated that 20% of all noise complaints is about industrial noise.

Community Noise

The main sources of community noise include street noise, noise from electronic equipment (air conditioners, refrigerators, washing machines, televisions), music, clocks, gongs and drums. Trumpets, gongs, drums and firecrackers, in particular, seriously disturb normal life and lead to annoyance complaints.

In conclusion, urban noise pollution in China is serious and is getting worse. To control noise pollution, China has promulgated standard sound values for environmental noise. These are summarized in table A2.2.

Table A2.2: LAeq standard values in dB for environmental noise in urban areas.

Applied area	day	night
Special residential quarters[1]	45	35
Residential and cultural education area[2]	50	40
Type 1 mixed area[3]	55	45
Type 2 mixed area[4] or commercial area	60	50
Industrial area	65	55
Arterial roads[5]	70	55

1 Special residential quarters: quiet residential area
2 Residential and cultural education area: residential quarters, cultural, educational offices
3 Type 1 mixed area: mixture of commercial area and residential quarters
4 Type 2 mixed area: mixture of industrial area, commercial area, residential quarters and others
5 Roads with traffic volume of more than 100 cars per hour

The peak sound levels for frequent noises emitted during the night-time are not allowed to exceed standard values by more than 10 dBA. Single, sudden noises during the night-time are not allowed to exceed standard values by more than 15dBA.

References

Liu Tian Gi et al.: Noise pollution and control. Environmental protection. Chemical industrial publishing company 1996, 6. First publication P137-154.
National environmental protection leader group. GB 3096-82. Urban environmental noise

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standard. The national standard of the People's Republic of China. 1982, 8, 1.
Feng Gen Quan: The main result of field investigation for industrial continuous noise. Foreign Medicine. Otorhinolaryngology. 1978, 2:12.
Beijing Medical Quarantine Station: The investigation analysis of the noise hazard condition in nine provinces and cities. The noise work investigation cooperation group in nine provinces and cities (internal material). 1982.
Cheng Ming: The diagnostic criteria of occupational deafness and experience of hearing conservation in China. Noise as public health problem. New advance in noise research, part 1. Swedish Council for building research. Stockholm, Sweden 1990, pp. 137-44.

Japan (Michinori Kabuto)

Environmental Quality Standards

Noise standards for both general and roadside areas were set in Japan in 1967, through the "Basic Law for Environmental Pollution." This law was updated in September 1999. Each standard is classified according to the type of land use and the time of day. In ordinary residential areas, the night-time standard is 45 dB LAeq, but in areas that require even lower noise exposure, such as hospitals, this is lowered to 40 dB LAeq. In contrast, the daytime levels for commercial and industrial areas is as high as 60 dBA. Standards for roadside areas are 70 dB LAeq for daytime and 65 dB LAeq for nighttime. Between 1973–1997 noise standards for aircraft noise, super-express train noise and conventional railway train noise were also implemented. Standards for aircraft noise were set in terms of the weighted equivalent continuous perceived noise level (WECPNL). For residential areas, the WECPNL standard is 70 dBA, and is 75 dBA for areas where it is necessary to maintain a normal daily life.

For super-express trains, the Environmental Agency required noise levels to be below 75 dBA in densely populated residential areas, such as along the Tokaido and Sanyo Shinkansen lines, as well as in increasingly populated areas, such as along the Tohoku and Joetsu Shinkansen lines. The standards were to be met by 1990, but by 1991 this level had been achieved at only 76% of the measuring sites on average. Noise countermeasures included the installation of new types of sound-proof walls, and laying ballast mats along densely populated stretches of the four Shinkansen lines. Noise and vibration problems can also result from conventional trains, such as occurred with the opening of the Tsugaru Strait and Seto Ohashi railway lines in 1988. Various measures have since been taken to address the problems.

Complaints About Community Noise.

In Japan, complaints to local governments about environmental problems have been summarized annually and reported by Japan Environmental Agency. Thirty-seven percent of all complaints was due to factory (machinery) noise; 22% to construction noise; 3% to road traffic noise; 4% to air traffic noise; 0.8% to rail traffic noise; 9% to night-time business; 6% to other commercial activities; 2.5% to loudspeaker announcements; 9% to domestic noise; and 8% was due to miscellaneous complaints.

Sources of Noise Exposure and their Effects

Road-traffic noise. The number of automobiles in Japan has increased from 20 million in 1971 to 70 million in 1994, a 3.5-fold increase. One-third of this increase was due to heavy-duty vehicles. Since 1994, out of a total of 1 150 000 km of roads in Japan, only 29 930 km have been designed according to noise regulations. According to 1998 estimates by the Environmental Agency, 58% of all roads passed through residential areas. Daytime noise limits were exceeded in 92% of all cases, and night-time limits were exceeded in 87% of all cases. The study also estimated that 0.5 million houses within 10 m of the roads were exposed to excessive traffic noise. In a recent lawsuit, the Japanese Supreme Court ruled that people should be compensated when exposed to night-time noise levels exceeding 65 dB Laeq. This would apply to people living alongside 2 000 km of roads in Japan.

A recent epidemiological study examined insomnia in 3 600 women living in eight different roadside areas exposed to night-time traffic. Insomnia was defined as one or more of the following symptoms: difficulty in falling asleep; waking up during sleep; waking up too early; and feelings of sleeplessness one or more days a week over a period of at least a month. The data were adjusted for confounding variables, such as age, medical care, whether the subjects had young children to care for, and sleep apnea symptoms. The results showed that the odds ratio for insomnia was significantly correlated with the average night-time traffic volume for each of the eight areas and suggested that insomnia could be attributed solely to night-time road traffic.

From the most noisy areas in the above study 19 insomnia cases were selected for a further in-depth examination. The insomnia cases were matched in age and work with 19 control subjects. Indoor and outdoor sound levels during sleep were measured simultaneously at 0.6 s intervals. For residences facing roads with average night-time traffic volume of 6 000 vehicles per hour, the highest sound levels observed were 78–93 dBA. The odds ratios for insomnia in each of the quartiles for LAmax,1min; L50,1min; L10,1min and LAeq,1min generally showed a linear trend and ranged between 1 (lowest quartile) and 6–7 (highest quartile). It was concluded that insomnia was likely to result when night-time indoor LAeq, 1min sound levels exceeded 30 dBA.

Air-traffic noise At the larger Japanese airports (Osaka, Tokyo, Fukuoka), jet airplanes have rapidly increased in number and have caused serious complaints and lawsuits from those living nearby. Complaints about jet-fighter noise are also common from residents living in the vicinity of several U.S. airbases located in Japan. In the case of Kadena and Futemma airbases on Okinawa, a recent study by the Okinawa Prefecture Government suggested that hearing loss, child misbehaviour and low birth-weight babies were possible health effects of the noise associated with these bases (RSCANIH 1997). Using measurements taken in 1968 during the Vietnam War, it was estimated that the WECPNL was 99–108 dBA at the Kadena village fire station. Similar WECPNL estimates of 105 dBA were also obtained for Yara (Kadena-cho) and Sunabe (Chatan-cho) bases. These levels correspond to a LAeq,24h value of 83 dB, and are of serious concern in light of recommendations by the Japan Association of Industrial Health that occupational noise exposure levels should not exceed 85 dB for an 8-h work day if hearing loss is to be avoided.

Audiogrammes of subjects living in areas surrounding Kadena airport indicated that they had progressive hearing loss at higher frequencies. Eight subjects had hearing impairment in the 3–6 kHz range, which strongly suggested that the hearing loss was due to excessive noise exposure. Since the examiners confirmed the subjects had not been exposed to repeated intense noise at their residences or workplaces, the most likely cause of their hearing loss was the intense aircraft noise during take-offs, landings and tune-ups at Kadena airport.

The effects of noise were examined in children from nursery schools and kindergartens in towns surrounding Kadena airport. The children were scored with respect to seven variables: cold symptoms, emotional instability, discontentment-anxiety, headache-stomachache, passivity, eating problems and urination problems. Confounding factors, such as sex, age, birth order, the number of parents living together, the mother's age when the child was born, reaction to noise and the extent of noise exposure, were taken into account. The results showed that children exposed to noise had significantly more problems with respect to their behaviour, physical condition, character and reaction to noise, when compared to a control group of children that had not been exposed to airport noise. This was especially true of for children exposed to a WECPNL of 75 or more. Thus, small children acquire both physical and mental disorders from chronic exposure to aircraft noise.

Chronic exposure to aircraft noise also affects the birth-weight of children. The birth-weights of infants were analyzed using records from 1974 to 1993 in the Okinawa Prefecture. Confounding factors such as the mother's age, whether there were single or multiple embryos, the child's sex, and the legitimacy of the child were considered. The results showed that 9.1 % of all infants born in Kadena-cho, located closest to Kadena airport, had low birth-weights. This was significantly higher than the 7.6 % rate seen in other municipalities around Kadena and Futemma airfields, and much higher than the 7 % rate in cities, towns and villages on other parts of Okinawa Island.

Rail-traffic noise. Commuter trains and subway cars expose Tokyo office workers to much higher noise levels than do other daily activities (Kabuto & Suzuki 1976). Exposure to indoor noise may vary according to railway line or season (there are more open windows in good weather), but the levels range from 65–85 dBA. In general, these values exceeded the LAeq,24h level of 70 dBA for auditory protection (US EPA 1974).

Neighbourhood noise. Neighbourhood noise, including noise from late-night business operations, noise caused by loudspeaker announcements, and noise from everyday activities, have accounted for approximately 39% of all complaints about noise in recent years. At present, noise controls for late-night business operations have been enforced by ordinances in 39 cities and prefectures, and in 42 cities for loudspeaker announcements.

References

Japan Environmental Agency Government of Japan (1994): Quality of the Environment in Japan 1994, Global Environment Department, Environment Agency, Tokyo.

Kabuto M and Suzuki S (1976) A trial measurement of individual noise exposure by time zone in a day. Japanese Journal of Industrial Health, 18:17-22.

Kabuto M, Ohtsuka R, Suzuki S (1978) Measurement and evaluation of individual noise exposure level of Tokyo metropolitan commuters and railroad cars above ground and in subways. Japanese Journal of Public Health 2: 59-63.

Kageyama, T., Kabuto, M. Nitta H, Kurokawa Y, Taira K, Suzuki S, Takemoto T (1974): A population study on risk factors for insomnia among adult Japanese women: A possible effects of road traffic volume. Sleep 20: 963-971.

RSCAINH (1997) Summary of the Interim Report of the Field Study on Public Health around U.S. Bases in Okinawa, Research Study Committee of Aircraft Noise Influences to Health, Okinawa Prefecture Government, Okinawa Public Health Association.

US EPA (1974) information on levels of environmental noise requisite to protect public health and welfare with an adequate margin of safety. Rep. No. 550/9-74.004.

Appendix 3 : Glossary

Acoustic	Pertaning to sound or to the sense of hearing (CMD 1997)
Acoustic dispersion	Change of speed of sound with frequency (ANSI 1994)
Acoustic trauma	Injury to hearing by noise, especially loud noise (CMD 1997)
Adverse effect	(of noise:) A change in morphology and physiology of an organism which results in impairment of functional capacity or impairment of capacity to compensate for additional stress or increase in susceptibility to the harmful effects of other environmental influences. This definition includes any temporary or long term lowering of physical, psychological or social functioning of humans or human organs (WHO 1994)
Annoyance	A feeling of displeasure associated with any agent or condition known or believed by an individual or a group to be adversely affecting them" (Lindvall and Radford 1973; Koelega 1987). Any sound that is perceived as irritating or a nuisance (ANSI 1995)
Anxiety	A feeling of apprehension, uncertainty, and fear without apparent stimulus, and associated with physiological changes (tachycardia, sweating, tremor, etc.) (DIMD 1985). A vaguer feeling of apprehension, worry, uneasiness, or dread, the source of which is often nonspecific or unknown to the individual (CMD 1997).
Audiometry	Testing of the hearing sense (CMD 1997). Measurement of hearing, including aspects other than hearing sensitivity (ANSI 1995)
Auditory	Pertaining to the sense of hearing (CMD 1997)
Auditory threshold	Minimum audible sound perceived (CMD 1997)
A-weighting	A frequency dependent correction that is applied to a measured or calculated sound of moderate intensity to mimick the varying sensitivity of the ear to sound for different frequencies

Ambient noise	All-encompassing sound at a given place, usually a composite of sounds from many sources near and far (ANSI 1994)
Articulation index	Numerical value indicating the proportion of an average speech signal that is understandable to an individual (ANSI 1995)
Bel	Unit of level when the base of the logarithm is ten, and the quantities concerned are proportional to power; unit symbol B (ANSI 1994)
Cardiovascular	Pertaining to the heart and blood vessels (DIMD 1985)
Cochlea	A winding cone-shaped tube forming a portion of the inner ear. It contains the receptor for hearing (CMD 1997)
Cognitive	Being aware with perception, reasoning, judgement, intuition, and memory (CMD 1997)
Community noise	Noise emitted from all noise sources except noise at the industrial workplace (WHO 1995a)
Cortisol	A glucocortical hormone of the outer layer of the adrenal gland (CMD 1997)
Critical health effect	Health effect with lowest effect level
C-weighting	A frequency dependent correction that is applied to a measured or calculated sound of high intensity to mimick the varying sensitivity of the ear to sound for different frequencies
dB	Decibel, one-tenth of a bel
dBA	A-weighted frequency spectrum in dB, see A-weighting
dBC	C-weighted frequency spectrum in dB, see C-weighting
dBlin	Unweighted frequency spectrum in dB
Decibel	Unit of level when the base of the logarithm is the tenth root of ten, and the quantities concerned are proportional to power; unit symbol dB (ANSI 1994)

Ear plug	Hearing protector that is inserted into the ear canal (ANSI 1994)
Ear muff	Hearing protector worn over the pinna (external part) of an ear (ANSI 1994)
Effective perceived noise level	Level of the time integral of the antilogarithm of one tenth of tone-corrected perceived noise level over the duration of an aircraft fly-over, the reference duration being 10 s (ANSI 1994)
Emission	(of sounds). Sounds generated from all types of sources
Epinephrine	A hormone secreted by the adrenal medulla (inner or central portion of an organ) in response to stimulation of the sympathetic nervous system (CMD 1997)
Equal energy principle	Hypothesis that states that the total effect of sound is proportional to the total amount of sound energy received by the ear, irrespective of the distribution of that energy in time
Equivalent sound pressure level	Ten times the logarithm to the base ten of the ratio of the time-mean-square instantaneous sound pressure, during a stated time interval T, to the square of the standard reference sound pressure (ANSI 1994)
Exposure-response curve	Graphical representation of exposure-response relationship
Exposure-response relationship	(With respect to noise:) Relationship between specified sound levels and health impacts
Frequency	For a function periodic in time, the reciprocal of the period (ANSI 1994)
Frequency-weighting	A frequency dependent correction that is applied to a measured or calculated sound (ANSI 1994)
Gastro-intestinal	Pertaining to the stomach and intestines (CMD 1997)
Hearing impairment, hearing loss	A decreased ability to perceive sounds as compared which what the individual or examiner would regard as normal (CMD 1997)
Hearing threshold	For a given listener and specified signal, the minimum (a) sound pressure level or (b) force level that is capable of

	evoking an auditory sensation in a specified function of trials (ANSI 1994)
Hertz	Unit of frequency, the number of times a phenomenon repeats itself in a unit of time; abbreviated to Hz
Hysteria	A mental disorder, usually temporary, presenting somatic (pertaining to the body) symptoms, stimulating almost any type of physical disease. Symptoms include emotional instability, various sensory disturbations, and a marked craving for sympathy (CMD 1997)
Immission	Sounds impacting on the human ear.
Impulsive sound	Sound consisting of one or more very brief and rapid increases in sound pressure
Incubator	An enclosed crib, in which the temperature and humidity may be regulated, for care of premature babies (CMD 1997)
Isolation, insulation	(With respect to sound:) Between two rooms in a specified frequency band, difference between the space-time average sound presssure levels in the two enclosed spaces when one or more sound sources operates in one of the rooms (ANSI 1994). (With respect to vibrations:) Reduction in the capacity of a system to respond to excitation, attained by use of resilient support (ANSI 1994).
Ischaemic Heart Disease	Heart disease due to a local and temporary deficiency of blood supply due to obstruction of the circulation to a part (CMD 1997)
Loudness level	Of a sound, the median sound pressure level in a specified number of trials of a free progressive wave having a frequency of 1000 Hz that is judged equally loud as the unknown sound when presented to listeners with normal hearing who are facing the source; unit phon (ANSI 1994)
Level	Logarithm of the ratio of a quantity to a reference quantity of the same kind; unit Bel (ANSI 1994)
Maximum sound level	Greatest fast (125 milliseconds) A-weighted sound level, within a stated time interval (ANSI 1994)

Mental Health	The absence of identifiable psychiatric disorder according to current norms (Freeman 1984). In noise research, mental health covers a variety of symptoms, ranging from anxiety, emotional stress, nervous complaints, nausea, headaches, instability, argumentativeness, sexual impotency, changes in general mood and anxiety, and social conflicts, to more general psychiatric categories like neurosis, phychosis and hysteria (Berglund and Lindvall 1995).
Morphological	Pertaining to the science of structure and form of organisms without regard to function (CMD 1997)
Nausea	An unpleasant sensation usually preceding vomiting (CMD 1997)
Neurosis	An emotional disorder due to unresolved conflicts, anxiety being its chief characteristic (DIMD 1985)
Noise	Undesired sound. By extension, noise is any unwarranted disturbance within a useful frequency band, such as undesired electric waves in a transmission channel or device (ANSI 1994).
Noise induced temporary threshold shift	Temporary hearing impairment occurring as a result of noise exposure, often phrased temporary threshold shift (adapted from ANSI 1994)
Noise induced permanent threshold shift	Permanent hearing impairment occurring as a result of noise exposure, often phrased permanent threshold shift (adapted from ANSI 1994)
Noise level	Level of undesired sound
Norepinephrine	A hormone produced by the adrenal medulla (inner or central portion of an organ), similar in chemical and pharmacological properties to epinephrine, but chiefly a vasoconstrictor with little effect on cardiac output (CMD 1997)
Oscillation	Variation, usually with time, of the magnitude of a quantity with respect to a specified reference when the magnitude is alternately greater and smaller than the reference (ANSI 1994)

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Ototoxic	Having a detrimental effect on the organs of hearing (CMD 1997)
Paracusis	Any abnormality or disorder of the sense of hearing (CMD 1997)
Pascal	Unit of pressure, equal to one newton per square meter, abbreviated to Pa
Peak sound pressure	Greatest absolute instantaneous sound pressure within a specified time interval (ANSI 1994)
Peak sound pressure level	Level of peak sound pressure with stated frequency weighting, within a specified time interval (ANSI 1994)
Perceived noise level	Frequency-weighted sound pressure level obtained by a stated procedure that combines the sound pressure levels in the 24 one-third octave bands with midband frequencies from 50 Hz to 10 kHz (ANSI 1994)
Permanent threshold shift, permanent hearing loss	Permanent increase in the auditory threshold for an ear (adapted from ANSI 1995) (see also: noise induced permanent threshold shift)
Presbyacusia, presbycusis	The progressive loss of hearing ability due to the normal aging process (CMD 1997)
Psychiatric disorders	Mental disorders
Psychosis	Mental disturbance of a magnitude that there is a personality disintegration and loss of contact with reality (CMD 1997)
Psychotropic drug	A drug that affects psychic function, behaviour or experience (CMD 1997)
Reverberation time	Of an enclosure, for a stated frequency or frequency band, time that would be required for the level of time-mean-square sound pressure in the enclosure to decrease by 60 dB, after the source has been stopped (ANSI 1994)
Sensorineural	Of or pertaining to a sensory nerve; pertaining to or affecting a sensory mechanism and/or a sensory nerve (DIMD 1985)

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Signal	Information to be conveyed over a communication system (ANSI 1994)
Signal-to-noise ratio	Ratio of a measure of a signal to the same measure of the noise (ANSI 1995) (see also: noise –in its extended meaning)
Silencer	Duct designed to reduce the level of sound; the sound-reducing mechanisms may be either absorptive or reactive, or a combination (ANSI 1994)
Sound absorption	Change in sound energy into some other form, usually heat, in passing through a medium or on striking a surface (ANSI 1994)
Sound energy	Total energy in a given part of a medium minus the energy that would exist at that same part with no sound waves present (ANSI 1994)
Sound exposure	Time integral of squared, instantaneous frequency-weighted sound pressure over a stated time interval or event (ANSI 1994)
Sound exposure level	Ten times the logarithm to the base ten of the ratio of a given time integral of squared, instantaneous A-weighted sound pressure, over a stated time interval or event, to the product of the squared reference sound pressure of 20 micropascals and reference duration of one second (ANSI 1994)
Sound intensity	Average rate of sound energy transmitted in a specified direction at a point through a unit area normal to this direction at the point considered (ANSI 1994)
Sound level meter	Device to be used to measure sound pressure level with a standardized frequency weighting and indicated exponential time weighting for measurements of sound level, or without time weighting for measurement of time-average sound pressure level or sound exposure level (ANSI 1994)
Sound pressure	Root-mean-square instantaneous sound pressure at a point, during a given time interval (ANSI 1994), where the *instantaneous* sound pressure is the total instantaneous pressure in that point minus the static pressure (ANSI 1994)

Sound pressure level	Ten times the logarithm to the base ten of the ratio of the time-mean-square pressure of a sound, in a stated frequency band, to the square of the reference sound pressure in gases of 20 Pa (ANSI 1994)
Sound reduction index	Single-number rating of airborne sound insulation of a partition (ANSI 1994)
Sound transmission class	Single-number rating of airborne sound insulation of a building partition (ANSI 1994)
Speech interference level	One-fourth of the the sum of the band sound pressure levels for octave-bands with nominal midband frequencies of 500, 100, 2000 and 4000 Hz (ANSI 1994)
Speech intelligibility	That property which allows units of speech to be identified (ANSI 1995)
Speech perception	Psychological process that relates a sensation caused by a spoken message to a listener's knowledge of speech and language (ANSI 1995)
Speech comprehension	(a) Highest level of speech perception. (b) Knowledge or understanding of a verbal statement (ANSI 1995)
Speech transmission index	Physical methgod for measuring the quality of speech-transmission channels accounting for nonlinear distortions as well as distortions of time (ANSI 1995)
Stereocilia	Nonmotile protoplasmic projections from free surfaces on the hair cells of the receptors of the inner ear (CMD 1997)
Stress	The sum of the biological reactions to any adverse stimulus, physical, mental or emotional, internal or external, that tends to disturb the organism's homeostasis (DIMD 1985)
Temporary threshold shift, temporary hearing loss	Temporary increase in the auditory threshold for an ear caused by exposure to high-intensity acoustic stimuli (adapted from ANSI 1995) (see also: noise induced temporary threshold shift).
Tinnitus	A subjective ringing or tinkling sound in the ear (CMD 1997). Otological condition in which sound is perceived by

	a person without an external auditory stimulation. The sound may be a whistling, ringing, buzzing, or cricket type sounds, but auditory hallucinations of voices are excluded (ANSI 1995).
Vibration	Oscillation of a parameter that defines the motion of a mechanical system (ANSI 1994)

For references see Appendix A.

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Appendix 4 : Acronyms

AAP	American Academy of Pediatrics
AI	Articulation Index
AMIS	Air Management Information System (WHO, Healthy Cities)
ANEF	Australian Noise Exposure Forecast
ANSI	American National Standard Institute, Washington DC, USA
ASCII	American Standard Code for Information Interchange
ASHA	American Speech-Language-Hearing Association, Rockville, MD, USA
ASTM	American Society for Testing and Materials, West Conshohocken, PA, USA
CEN	Comité Européen de Normalisation, Brussels, Belgium (European Committee for Standardization)
CFR	Code of Federal Regulations (United States)
CIAL	Centro de Investigaciones Acústicas y Luminotécnicas, Córdoba, Argentina (Centre of acoustical and light-technical investigations)
CMD	Cyclopedic Medical Dictionary
CNRC	Conseil National de Recherches du Canada (National Research Council)
COPD	Chronic Obstructive Pulmonary Disease
CSD	Commission for Sustainable Development
CSIRO	Commonwealth Scientific and Industrial Research Organization
CVS	Cardiovascular System
DNL	Day-Night Average Sound Level (United States)
EC DG	European Commission Directorate General
ECE	Economic Commission for Europe
ECMT	European Conference of Ministers of Transport
EHIAP	Environmental Health Impact Assessment Plan
EIAP	Environmental Impact Assessment Plan
EMRO	WHO Regional Office of the Eastern Mediterranean
ENIA	Environmental Noise Impact Analysis
EPNL	Effective Perceived Noise Level measure
EU	European Union
FAA	Federal Aviation Administration (United States)
FFT	Fast Fourier Transform technique
GIS	Geographic Information System
Hz	Hertz, the unit of frequency
ICAO	International Civil Aviation Organization
ICBEN	International Commission on the Biological Effects of Noise
IEC	International Electrotechnical Commission
ILO	International Labour Office, Geneva, Switzerland
INCE	Institute of Noise Control Engineering of the United States of America
INRETS	Institut National de REcherche sur les Transports et leur Sécurité, Arcueil, France (National Research Institute for Transport and their Safety)
ISO	International Standards Organization
I-INCE	International Institute of Noise Control Engineering
L10	10 percentile of sound pressure level

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L50	Median sound pressure level
L90	90-percentile of sound pressure level
LA	Latin America
LAeq,T	A-weighted equivalent sound pressure level for period T
LAmax	Maximum A-weighted sound pressure level in a stated interval
L_{dn}	Day and night continuous equivalent sound pressure level
Leq,T	Equivalent sound pressure level for period T
LEQ(FLG)	Descriptor used for aircraft noise (Germany)
LNIP	Low Noise Implementation Plan
Lp	Sound pressure level
MTF	Modulation Transfer Function
NASA	National Aeronautics and Space Administration (United States)
NC	Noise Criterion
NCA	Noise Control Act (United States)
NCB	Balanced Noise Criterion procedure system
NEF	Noise Exposure Forecast
NEPA	National Environmental Policy Act (United States)
NGO	Non Governmental Organization
NIHL	Noise Induced Hearing Loss
NIPTS	Noise Induced Permanent Threshold Shift
NITTS	Noise Induced Temporary Threshold Shift
NNI	Noise and Number Index
NR	Noise Rating
NRC	National Research Council (United States, Canada)
OECD	Organisation for Economic Co-operation and Development, Paris, France.
ONAC	Office of Noise Abatement and Control of the US EPA
OSHA	Occupational Safety and Health Administration
Pa	Pascal, the unit of pressure
PAHO	Pan American Health Organization
PHE	Department for Protection of the Human Environment, WHO, Geneva
PNL	Perceived Noise Level
PSIL	Preferred Speech Interference Level
PTS	Permanent Threshold Shift
RASTI	Rapid Speech Transmission Index
RC	Room Criterion
SABS	South African Bureau of Standards
SEL	Sound Exposure Level
STC	Sound Transmission Class
STI	Speech Transmission Index
TTS	Temporary Threshold Shift
UK	United Kingdom
UN	United Nations
UNCED	United Nations Conference on Environment and Development (Rio de Janeiro, June 1992)
UNDP	United Nations Development Programme
UNECE	United Nations Economic Commission for Europe

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UNEP	United Nations Environment Programme
UNESCO	United Nations Educational, Scientific and Cultural Organization
US EPA	United States Environmental Protection Agency
USA	United States of America
WCED	World Commission on Environment and Development (Brundtland Commission)
WECPNL	Weighted Equivalent Continuous Perceived Noise Level
WHO	World Health Organization
WWF	World Wildlife Fund

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Appendix 5 : Equations and other technical information

Basic acoustical measures

Sound Pressure Level

The time-varying sound pressure will completely define a sound in a given location. The sound pressure range is wide within which human listeners can receive (10^{-5} - 10^{2} N/m^2). Therefore, it is practical to measure sound pressure level on a logarithmic scale. Sound intensity level is defined as 10 times the logarithm (to the base 10) of the ratio of the sound intensity of a target sound to the sound intensity of another (reference) sound. Sound intensity is proportional to the squared sound pressure because the static mass density of the sound medium as well as the speed of sound in this medium are invariant. The sound pressure level (L_p) of a sound may be expressed as a function of sound pressure (p) and is, thus, possible to measure:

$$L_p = 10 \log_{10} (p/p_{ref})^2$$

For the purpose of measuring sound pressure level in a comparative way, the reference pressure, p_{ref}, has an internationally agreed value of $2 \cdot 10^{-5}$ N/m^2 (earlier 20 μPa). Sound pressure level is then expressed in decibel (dB) relative to this reference sound.

Sound Pressure Level of Combined Sounds

Whereas sound intensities or energies or pressures are additive, non-correlated time-varying sound pressure levels have first to be expressed as mean square pressure, then added, and then transferred to a sound pressure value again. For example, if two sound sources are combined, each of a sound pressure level of 80 dB, then the sound pressure level of the resulting combined sound will become 83 dB:

$$L_p = 10 * \log_{10} (10^8 + 10^8) = 10 * \log_{10} (2 * 10^8) = 10 * (\log_{10} 2 + \log_{10} 10^8) = 10 * (0.3 + 8) = 83$$

It is only sounds with similar sound pressure levels that when combined will result in a significant increase in sound pressure level relative to the louder sound. In the example given above, a doubling of the sound energy from two sources will only result in a 3-dB increase in sound pressure level. For two sound sources that emit non-correlated time-varying sound pressures, this represents the maximum increase possible. The sound pressure level outcome, resulting from combining two sound pressure levels in dB, is displayed in Figure A.5.1.

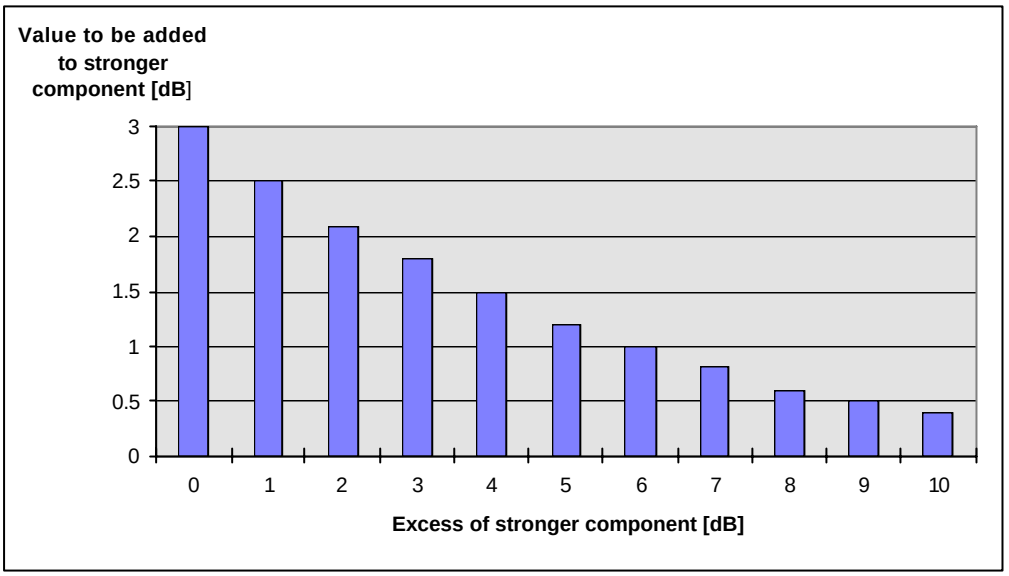


Figure A.5.1: Estimate of combined sound levels

Equivalent Continuous Sound Pressure Level

Average sound pressure level is determined for a time period of interest, T, which may be an interval in seconds, minutes, or hours. This gives a dB-value in Leq that stands for equivalent continuous sound pressure level or simply sound level. It is derived from the following mathematical expression in which A-weighting has been applied:

.

$$LAeq,T = 10 \log_{10}\{(1/T) \int_0^T 10^{Lp(t)/10} dt\} \text{ [dBA]}$$

Because the integral is a measure of the total sound energy during the period T, this process is often called “energy averaging”. For similar reasons, the integral term representing the total sound energy may be interpreted as a measure of the total noise dose. Thus, Leq is the level of that steady sound which, over the same interval of time as the fluctuating sound of interest, has the same mean square sound pressure, usually applied as an A-frequency weighting. The interval of time must be stated.

Sound exposure level

Individual noise events can be described in terms of their sound exposure level (SEL). SEL is defined as the constant sound level over a period of 1 s that would have the same amount of energy as the complete noise event (Ford 1987). For a single noise event occurring over a time interval T, the relationship between SEL and LAeq,T is,

$$SEL = LAeq,T + 10 \log_{10} (T/T_0)$$

In this equation T_0 is 1 s.

Day and night continuous sound pressure level

There are different definition in different countries. One definition is (von Gierke 1975; Ford 1987):

L_{dn} = LAeq,16h + LAeq,8h – 10 dBA

Where LAeq,16h is the day equivalent sound pressure level and LAeq,8h is the night equivalent sound pressure level.

Sound Transmission into and within buildings

An approximate relationship between sound reduction index (R), the frequency (f), the mass per unit area of the panel (m) in kg/m^2, and the angle of incidence (θ) is given by

$$R(\theta) = 20 \log\{f\, m\, \mathrm{COS}(\theta)\} - 42.4, \text{ (dB)}$$

This relationship indicates that the sound reduction index will increase with the mass of a panel and with the frequency of the sound as well as varying with the angle of incidence of the sound. It is valid for limp materials but is a good approximation to the behaviour of many real building materials at lower frequencies.

The sound reduction index versus frequency characteristics are usually complicated by a coincidence dip which occurs around the frequency where the wavelength of the incident sound is the same as the wavelength of bending waves in the building façade material. The frequency at which the coincidence dip occurs is influenced by the stiffness of the panel material. Thicker, and hence stiffer materials, will have coincidence dips that are lower in frequency than less stiff materials. Figure A.5.2 plots measured sound reduction index values versus frequency for 4 mm thick glass and illustrates the coincidence dip for this glass at a frequency centered just above 3 kHz.

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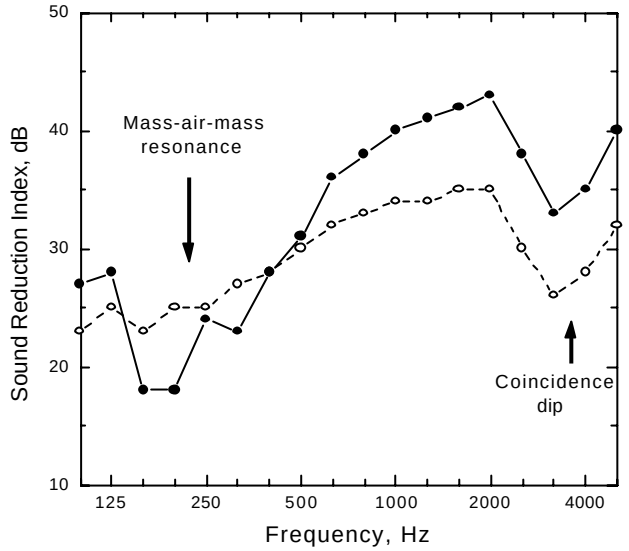


Figure A.5.2: Sound reduction index versus frequency for single and double layers of 4 mm glass (air separation 13 mm).

As also illustrated in Figure A.5.2 for two layers of 4 mm glass, the low frequency sound reduction can be severely limited by the mass-air-mass resonance. This resonance is due to the combination of the masses of the two layers and the stiffness of the enclosed air space. As the Figure A.5.2 example shows, this resonance can often dramatically reduce the low frequency sound reduction of common double window constructions.

The sound reduction of various building constructions can be calculated as the difference between the average sound levels in the two rooms ($L_1 - L_2$) plus a correction involving the area of the test panel (S) in m^2 and the total sound absorption (A) in m^2 in the receiving room,

$$R = L_1 - L_2 + 10 \log\{S/A\} \text{ [dB].}$$

For outdoor-to-indoor sound propagation, the measured sound reduction index will also depend on the angle of incidence of the outdoor sound as well as the position of the outdoor measuring microphone relative to the building façade,

$$R = L_1 - L_2 + 10 \log\{4S\ COS(\theta)/A\} + k \text{ [dB].}$$

When the outdoor incident sound level L_1 is measured with the outdoor microphone positioned against the external façade surface, measured incident sound pressures will be 6 dB higher due to pressure doubling. This occurs because the incident sound and reflected sound arrive at the microphone at the same time. If the external microphone is located 2 m from the façade, there will not be exact pressure doubling but an approximate doubling of the measured sound energy corresponding to a 3 dB increase in sound level. The table below indicates the appropriate values of k to be used in the above equation, depending on the location of the outdoor microphone, to account for sound reflected from the façade.

k = 0, dB	L_1 does not include reflected sound.
k = -3, dB	L_1 measured 2 m from façade and includes reflected energy.
k = -6, dB	L_1 measured at the façade surface and includes pressure doubling effect.

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Appendix 6 : Participant list of THE WHO Expert Task Force meeting on Guidelines For Community Noise, 26-30 April 1999, MARC, London, UK

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Meeting January 14 1965

The Environment and Disease: Association or Causation?

by Sir Austin Bradford Hill CBE DSC FRCP(hon) FRS
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Amongst the objects of this newly-founded Section of Occupational Medicine are firstly 'to provide a means, not readily afforded elsewhere, whereby physicians and surgeons with a special knowledge of the relationship between sickness and injury and conditions of work may discuss their problems, not only with each other, but also with colleagues in other fields, by holding joint meetings with other Sections of the Society'; and, secondly, 'to make available information about the physical, chemical and psychological hazards of occupation, and in particular about those that are rare or not easily recognized'.

At this first meeting of the Section and before, with however laudable intentions, we set about instructing our colleagues in other fields, it will be proper to consider a problem fundamental to our own. How in the first place do we detect these relationships between sickness, injury and conditions of work? How do we determine what are physical, chemical and psychological hazards of occupation, and in particular those that are rare and not easily recognized?

There are, of course, instances in which we can reasonably answer these questions from the general body of medical knowledge. A particular, and perhaps extreme, physical environment cannot fail to be harmful; a particular chemical is known to be toxic to man and therefore suspect on the factory floor. Sometimes, alternatively, we may be able to consider what *might* a particular environment do to man, and then see whether such consequences are indeed to be found. But more often than not we have no such guidance, no such means of proceeding; more often than not we are dependent upon our observation and enumeration of defined events for which we then seek antecedents. In other words we see that the event B is associated with the environmental feature A, that, to take a specific example, some form of respiratory illness is associated with a dust in the environment. In what circumstances can we pass from this observed *association* to a verdict of *causation*? Upon what basis should we proceed to do so?

President's Address

I have no wish, nor the skill, to embark upon a philosophical discussion of the meaning of 'causation'. The 'cause' of illness may be immediate and direct, it may be remote and indirect underlying the observed association. But with the aims of occupational, and almost synonymously preventive, medicine in mind the decisive question is whether the frequency of the undesirable event B will be influenced by a change in the environmental feature A. *How* such a change exerts that influence may call for a great deal of research. However, before deducing 'causation' and taking action we shall not invariably have to sit around awaiting the results of that research. The whole chain may have to be unravelled or a few links may suffice. It will depend upon circumstances.

Disregarding then any such problem in semantics we have this situation. Our observations reveal an association between two variables, perfectly clear-cut and beyond what we would care to attribute to the play of chance. What aspects of that association should we especially consider before deciding that the most likely interpretation of it is causation?

(1) *Strength*. First upon my list I would put the strength of the association. To take a very old example, by comparing the occupations of patients with scrotal cancer with the occupations of patients presenting with other diseases, Percival Pott could reach a correct conclusion because of the *enormous* increase of scrotal cancer in the chimney sweeps. 'Even as late as the second decade of the twentieth century', writes Richard Doll (1964), 'the mortality of chimney sweeps from scrotal cancer was some 200 times that of workers who were not specially exposed to tar or mineral oils and in the eighteenth century the relative difference is likely to have been much greater.'

To take a more modern and more general example upon which I have now reflected for over fifteen years, prospective inquiries into smoking have shown that the death rate from cancer of the lung in cigarette smokers is nine to ten times the rate in non-smokers and the rate in heavy cigarette smokers is twenty to thirty times

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as great. On the other hand the death rate from coronary thrombosis in smokers is no more than twice, possibly less, the death rate in non-smokers. Though there is good evidence to support causation it is surely much easier in this case to think of some features of life that may go hand-in-hand with smoking – features that might conceivably be the real underlying cause or, at the least, an important contributor, whether it be lack of exercise, nature of diet or other factors. But to explain the pronounced excess in cancer of the lung in any other environmental terms requires some feature of life so intimately linked with cigarette smoking and with the amount of smoking that such a feature should be easily detectable. If we cannot detect it or reasonably infer a specific one, then in such circumstances I think we are reasonably entitled to reject the vague contention of the armchair critic 'you can't prove it, there *may* be such a feature'.

Certainly in this situation I would reject the argument sometimes advanced that what matters is the absolute difference between the death rates of our various groups and not the ratio of one to other. That depends upon what we want to know. If we want to know how many extra deaths from cancer of the lung will take place through smoking (i.e. presuming causation), then obviously we must use the absolute differences between the death rates – 0·07 per 1,000 per year in non-smoking doctors, 0·57 in those smoking 1–14 cigarettes daily, 1·39 for 15–24 cigarettes daily and 2·27 for 25 or more daily. But it does not follow here, or in more specifically occupational problems, that this best measure of the effect upon mortality is also the best measure in relation to ætiology. In this respect the ratios of 8, 20 and 32 to 1 are far more informative. It does not, of course, follow that the differences revealed by ratios are of any practical importance. Maybe they are, maybe they are not; but that is another point altogether.

We may recall John Snow's classic analysis of the opening weeks of the cholera epidemic of 1854 (Snow 1855). The death rate that he recorded in the customers supplied with the grossly polluted water of the Southwark and Vauxhall Company was in truth quite low – 71 deaths in each 10,000 houses. What stands out vividly is the fact that the small rate is 14 times the figure of 5 deaths per 10,000 houses supplied with the sewage-free water of the rival Lambeth Company.

In thus putting emphasis upon the strength of an association we must, nevertheless, look at the obverse of the coin. We must not be too ready to dismiss a cause-and-effect hypothesis merely on the grounds that the observed association appears to be slight. There are many occasions in medicine when this is in truth so. Relatively few persons harbouring the meningococcus fall sick of meningococcal meningitis. Relatively few persons occupationally exposed to rat's urine contract Weil's disease.

(2) *Consistency:* Next on my list of features to be specially considered I would place the *consistency* of the observed association. Has it been repeatedly observed by different persons, in different places, circumstances and times?

This requirement may be of special importance for those rare hazards singled out in the Section's terms of reference. With many alert minds at work in industry today many an environmental association may be thrown up. Some of them on the customary tests of statistical significance will appear to be unlikely to be due to chance. Nevertheless whether chance is the explanation or whether a true hazard has been revealed may sometimes be answered only by a repetition of the circumstances and the observations.

Returning to my more general example, the Advisory Committee to the Surgeon-General of the United States Public Health Service found the association of smoking with cancer of the lung in 29 retrospective and 7 prospective inquiries (US Department of Health, Education & Welfare 1964). The lesson here is that broadly the same answer has been reached in quite a wide variety of situations and techniques. In other words we can justifiably infer that the association is not due to some constant error or fallacy that permeates every inquiry. And we have indeed to be on our guard against that.

Take, for instance, an example given by Heady (1958). Patients admitted to hospital for operation for peptic ulcer are questioned about recent domestic anxieties or crises that may have precipitated the acute illness. As controls, patients admitted for operation for a simple hernia are similarly quizzed. But, as Heady points out, the two groups may not be *in pari materia*. If your wife ran off with the lodger last week you still have to take your perforated ulcer to hospital without delay. But with a hernia you might prefer to stay at home for a while – to mourn (or celebrate) the event. No number of exact repetitions would remove or necessarily reveal that fallacy.

We have, therefore, the somewhat paradoxical position that the different results of a different inquiry certainly cannot be held to refute the

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original evidence; yet the same results from precisely the same form of inquiry will not invariably greatly strengthen the original evidence. I would myself put a good deal of weight upon similar results reached in quite different ways, e.g. prospectively and retrospectively.

Once again looking at the obverse of the coin there will be occasions when repetition is absent or impossible and yet we should not hesitate to draw conclusions. The experience of the nickel refiners of South Wales is an outstanding example. I quote from the Alfred Watson Memorial Lecture that I gave in 1962 to the Institute of Actuaries:

'The population at risk, workers and pensioners, numbered about one thousand. During the ten years 1929 to 1938, sixteen of them had died from cancer of the lung, eleven of them had died from cancer of the nasal sinuses. At the age specific death rates of England and Wales at that time, one might have anticipated one death from cancer of the lung (to compare with the 16), and a fraction of a death from cancer of the nose (to compare with the 11). In all other bodily sites cancer had appeared on the death certificate 11 times and one would have expected it to do so 10–11 times. There had been 67 deaths from all other causes of mortality and over the ten years' period 72 would have been expected at the national death rates. Finally division of the population at risk in relation to their jobs showed that the excess of cancer of the lung and nose had fallen wholly upon the workers employed in the chemical processes.

'More recently my colleague, Dr Richard Doll, has brought this story a stage further. In the nine years 1948 to 1956 there had been, he found, 48 deaths from cancer of the lung and 13 deaths from cancer of the nose. He assessed the numbers expected at normal rates of mortality as, respectively 10 and 0·1.

'In 1923, long before any special hazard had been recognized, certain changes in the refinery took place. No case of cancer of the nose has been observed in any man who first entered the works after that year, and in these men there has been no excess of cancer of the lung. In other words, the excess in both sites is uniquely a feature in men who entered the refinery in, roughly, the first 23 years of the present century.

'No causal agent of these neoplasms has been identified. Until recently no animal experimentation had given any clue or any support to this wholly statistical evidence. Yet I wonder if any of us would hesitate to accept it as proof of a grave industrial hazard?' (Hill 1962).

In relation to my present discussion I know of no parallel investigation. We have (or certainly had) to make up our minds on a unique event; and there is no difficulty in doing so.

(3) *Specificity:* One reason, needless to say, is the specificity of the association, the third characteristic which invariably we must consider. If, as here, the association is limited to specific workers and to particular sites and types of disease and there is no association between the work and other modes of dying, then clearly that is a strong argument in favour of causation.

We must not, however, over-emphasize the importance of the characteristic. Even in my present example there is a cause and effect relationship with two different sites of cancer – the lung and the nose. Milk as a carrier of infection and, in that sense, the cause of disease can produce such a disparate galaxy as scarlet fever, diphtheria, tuberculosis, undulant fever, sore throat, dysentery and typhoid fever. Before the discovery of the underlying factor, the bacterial origin of disease, harm would have been done by pushing too firmly the need for specificity as a necessary feature before convicting the dairy.

Coming to modern times the prospective investigations of smoking and cancer of the lung have been criticized for not showing specificity – in other words the death rate of smokers is higher than the death rate of non-smokers from many causes of death (though in fact the results of Doll & Hill, 1964, do not show that). But here surely one must return to my first characteristic, the strength of the association. If other causes of death are raised 10, 20 or even 50% in smokers whereas cancer of the lung is raised 900–1,000% we have specificity – a specificity in the magnitude of the association.

We must also keep in mind that diseases may have more than one cause. It has always been possible to acquire a cancer of the scrotum without sweeping chimneys or taking to mule-spinning in Lancashire. One-to-one relationships are not frequent. Indeed I believe that multi-causation is generally more likely than single causation though possibly if we knew all the answers we might get back to a single factor.

In short, if specificity exists we may be able to draw conclusions without hesitation; if it is not apparent, we are not thereby necessarily left sitting irresolutely on the fence.

(4) *Temporality:* My fourth characteristic is the temporal relationship of the association – which is the cart and which the horse? This is a question which might be particularly relevant with diseases of slow development. Does a particular diet lead to disease or do the early stages of the disease lead to those peculiar dietetic habits? Does a

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particular occupation or occupational environment promote infection by the tubercle bacillus or are the men and women who select that kind of work more liable to contract tuberculosis whatever the environment – or, indeed, have they already contracted it? This temporal problem may not arise often but it certainly needs to be remembered, particularly with selective factors at work in industry.

(5) *Biological gradient:* Fifthly, if the association is one which can reveal a biological gradient, or dose-response curve, then we should look most carefully for such evidence. For instance, the fact that the death rate from cancer of the lung rises linearly with the number of cigarettes smoked daily, adds a very great deal to the simpler evidence that cigarette smokers have a higher death rate than non-smokers. That comparison would be weakened, though not necessarily destroyed, if it depended upon, say, a much heavier death rate in light smokers and a lower rate in heavier smokers. We should then need to envisage some much more complex relationship to satisfy the cause-and-effect hypothesis. The clear dose-response curve admits of a simple explanation and obviously puts the case in a clearer light.

The same would clearly be true of an alleged dust hazard in industry. The dustier the environment the greater the incidence of disease we would expect to see. Often the difficulty is to secure some satisfactory quantitative measure of the environment which will permit us to explore this dose-response. But we should invariably seek it.

(6) *Plausibility:* It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day.

To quote again from my Alfred Watson Memorial Lecture (Hill 1962), there was

'. . . no biological knowledge to support (or to refute) Pott's observation in the 18th century of the excess of cancer in chimney sweeps. It was lack of biological knowledge in the 19th that led a prize essayist writing on the value and the fallacy of statistics to conclude, amongst other "absurd" associations, that "it could be no more ridiculous for the stranger who passed the night in the steerage of an emigrant ship to ascribe the typhus, which he there contracted, to the vermin with which bodies of the sick might be infected". And coming to nearer times, in the 20th century there was no biological knowledge to support the evidence against rubella.'

In short, the association we observe may be one new to science or medicine and we must not dismiss it too light-heartedly as just too odd. As Sherlock Holmes advised Dr Watson, 'when you have eliminated the impossible, whatever remains, *however improbable*, must be the truth.'

(7) *Coherence:* On the other hand the cause-and-effect interpretation of our data should not seriously conflict with the generally known facts of the natural history and biology of the disease – in the expression of the Advisory Committee to the Surgeon-General it should have coherence.

Thus in the discussion of lung cancer the Committee finds its association with cigarette smoking coherent with the temporal rise that has taken place in the two variables over the last generation and with the sex difference in mortality – features that might well apply in an occupational problem. The known urban/rural ratio of lung cancer mortality does not detract from coherence, nor the restriction of the effect to the lung.

Personally, I regard as greatly contributing to coherence the histopathological evidence from the bronchial epithelium of smokers and the isolation from cigarette smoke of factors carcinogenic for the skin of laboratory animals. Nevertheless, while such laboratory evidence can enormously strengthen the hypothesis and, indeed, may determine the actual causative agent, the lack of such evidence cannot nullify the epidemiological observations in man. Arsenic can undoubtedly cause cancer of the skin in man but it has never been possible to demonstrate such an effect on any other animal. In a wider field John Snow's epidemiological observations on the conveyance of cholera by the water from the Broad Street pump would have been put almost beyond dispute if Robert Koch had been then around to isolate the vibrio from the baby's nappies, the well itself and the gentleman in delicate health from Brighton. Yet the fact that Koch's work was to be awaited another thirty years did not really weaken the epidemiological case though it made it more difficult to establish against the criticisms of the day – both just and unjust.

(8) *Experiment:* Occasionally it is possible to appeal to experimental, or semi-experimental, evidence. For example, because of an observed association some preventive action is taken. Does it in fact prevent? The dust in the workshop is reduced, lubricating oils are changed, persons stop smoking cigarettes. Is the frequency of the associated events affected? Here the strongest

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support for the causation hypothesis may be revealed.

(9) *Analogy:* In some circumstances it would be fair to judge by analogy. With the effects of thalidomide and rubella before us we would surely be ready to accept slighter but similar evidence with another drug or another viral disease in pregnancy.

Here then are nine different viewpoints from all of which we should study association before we cry causation. What I do not believe – and this has been suggested – is that we can usefully lay down some hard-and-fast rules of evidence that *must* be obeyed before we accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question – is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?

Tests of Significance

No formal tests of significance can answer those questions. Such tests can, and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the 'proof' of our hypothesis.

Nearly forty years ago, amongst the studies of occupational health that I made for the Industrial Health Research Board of the Medical Research Council was one that concerned the workers in the cotton-spinning mills of Lancashire (Hill 1930). The question that I had to answer, by the use of the National Health Insurance records of that time, was this: Do the workers in the card-room of the spinning mill, who tend the machines that clean the raw cotton, have a sickness experience in any way different from that of other operatives in the same mills who are relatively unexposed to the dust and fibre that were features of the cardroom? The answer was an unqualified 'Yes'. From age 30 to age 60 the cardroom workers suffered over three times as much from respiratory causes of illness whereas from non-respiratory causes their experience was not different from that of the other workers. This pronounced difference with the respiratory causes was derived not from abnormally long periods of sickness but rather from an excessive number of repeated absences from work of the cardroom workers.

All this has rightly passed into the limbo of forgotten things. What interests me today is this: My results were set out for men and women separately and for half a dozen age groups in 36 tables. So there were plenty of sums. Yet I cannot find that anywhere I thought it necessary to use a test of significance. The evidence was so clear-cut, the differences between the groups were mainly so large, the contrast between respiratory and non-respiratory causes of illness so specific, that no formal tests could really contribute anything of value to the argument. So why use them?

Would we think or act that way today? I rather doubt it. Between the two world wars there was a strong case for emphasizing to the clinician and other research workers the importance of not overlooking the effects of the play of chance upon their data. Perhaps too often generalities were based upon two men and a laboratory dog while the treatment of choice was deduced from a difference between two bedfuls of patients and might easily have no true meaning. It was therefore a useful corrective for statisticians to stress, and to teach the need for, tests of significance merely to serve as guides to caution before drawing a conclusion, before inflating the particular to the general.

I wonder whether the pendulum has not swung too far – not only with the attentive pupils but even with the statisticians themselves. To decline to draw conclusions without standard errors can surely be just as silly? Fortunately I believe we have not yet gone so far as our friends in the USA where, I am told, some editors of journals will return an article because tests of significance have not been applied. Yet there are innumerable situations in which they are totally unnecessary – because the difference is grotesquely obvious, because it is negligible, or because, whether it be formally significant or not, it is too small to be of any practical importance. What is worse the glitter of the t table diverts attention from the inadequacies of the fare. Only a tithe, and an unknown tithe, of the factory personnel volunteer for some procedure or interview, 20% of patients treated in some particular way are lost to sight, 30% of a randomly-drawn sample are never contacted. The sample may, indeed, be akin to that of the man who, according to Swift, 'had a mind to sell his house and carried a piece of brick in his pocket, which he showed as a pattern to encourage purchasers'. The writer, the editor and the reader are unmoved. The magic formulæ are there.

Of course I exaggerate. Yet too often I suspect we waste a deal of time, we grasp the shadow and

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lose the substance, we weaken our capacity to interpret data and to take reasonable decisions whatever the value of P. And far too often we deduce 'no difference' from 'no significant difference'. Like fire, the χ^2 test is an excellent servant and a bad master.

The Case for Action

Finally, in passing from association to causation I believe in 'real life' we shall have to consider what flows from that decision. On scientific grounds we should do no such thing. The evidence is there to be judged on its merits and the judgment (in that sense) should be utterly independent of what hangs upon it – or who hangs because of it. But in another and more practical sense we may surely ask what is involved in our decision. In occupational medicine our object is usually to take action. If this be operative cause and that be deleterious effect, then we shall wish to intervene to abolish or reduce death or disease.

While that is a commendable ambition it almost inevitably leads us to introduce differential standards before we convict. Thus on relatively slight evidence we might decide to restrict the use of a drug for early-morning sickness in pregnant women. If we are wrong in deducing causation from association no great harm will be done. The good lady and the pharmaceutical industry will doubtless survive.

On fair evidence we might take action on what appears to be an occupational hazard, e.g. we might change from a probably carcinogenic oil to a non-carcinogenic oil in a limited environment and without too much injustice if we are wrong. But we should need very strong evidence before we made people burn a fuel in their homes that they do not like or stop smoking the cigarettes and eating the fats and sugar that they do like. In asking for very strong evidence I would, however, repeat emphatically that this does not imply crossing every 't', and swords with every critic, before we act.

All scientific work is incomplete – whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time.

Who knows, asked Robert Browning, but the world may end tonight? True, but on available evidence most of us make ready to commute on the 8.30 next day.

REFERENCES

Doll R (1964) In: Medical Surveys and Clinical Trials. Ed. L J Witts. 2nd ed. London; p 333

Doll R & Hill A B (1964) *Brit. med. J.* i, 1399, 1460

Heady J A (1958) *Med. World, Lond.* **89**, 305

Hill A B
(1930) Sickness amongst Operatives in Lancashire Spinning Mills. Industrial Health Research Board Report No. 59. HMSO, London
(1962) *J. Inst. Actu.* **88**, 178

Snow J (1855) On the Mode of Communication of Cholera. 2nd ed. London (Reprinted 1936, New York)

US Department of Health, Education & Welfare (1964) Smoking and Health. Public Health Service Publication No. 1103. Washington

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