Note: These pages are in manuscript format, pending publication of the book.

PREFACE

I wrote this report because I saw a medical problem that few clinicians were paying attention to or seemed to understand. Dr. Amanda Harry in the UK led the way in recognizing the cluster of symptoms people experience around wind turbines.[1] I, myself, began encountering the problem in numerous e-mails and telephone calls, beginning in 2004, shortly after wind developers turned up in my community and my husband and I began investigating industrial wind turbines. The uniformity of the complaints became quickly apparent, as did the potential for a relationship with the neurologic complex of *migraine, motion sickness, vertigo, noise and visual and gastrointestinal sensitivity, and anxiety* that has proven to be such a useful construct in medical practice.

A breakthrough came in early 2006, when I interviewed a couple who were about to move out of their home because of their own and their children's symptoms. The interview supported the relationship between turbine-associated symptoms and migraine/motion sensitivity, while introducing me to the curious phenomenon of vibration or pulsation felt in the chest. It was this element that piqued the interest of the National Academy of Sciences in its 2007 report to Congress, *Environmental Impacts of Wind-Energy Projects*. The authors wanted to learn more about this effect of low frequency noise.[2]

This study is my answer to their question.

As I have sought to understand these complaints, I have benefited from new research allowing us to better understand neurologic phenomena like spatial memory loss or fear reactions in people with balance problems – symptoms that heretofore have often "bored and baffled" clinicians, as one of my referees put it.[3] These are symptoms that tend to get dismissed under the disparaging rubric of "psychosomatic" – medically, popularly, and by wind industry consultants[4] – whereas research now shows there are precise and definable neurologic connections to explain how certain sensory signals can derail normal psychological and cognitive function and trigger physical symptoms.

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[1] Harry, Amanda. February 2007. Wind turbines, noise, and health. 32 pp. http://www.windturbinenoisehealthhumanrights.com/wtnoise_health_2007_a_barry.pdf

[2] National Research Council. 2007. *Environmental Impacts of Wind-Energy Projects.* The National Academies Press, Washington, DC. 185 pp, p. 109.

[3] I review and discuss this research in the *Discussion* section, pp. xx.

[4] See, for example, *Wind Fact Sheet #5: Are modern wind turbines noisy*? Noble Environmental Power, LLC, http://www.noblepower.com/faqs/documents/06-08-23NEP-SoundFromWindTurbines-FS5-G.pdf, p. 2.

To dismiss people's symptoms with "It's all in the head" of course turns back upon itself in an absurd tautology. For the brain, our most complex organ, is indeed situated "in the head." Like any other organ, everything the brain does is physiological and dependent upon cellular and membrane-based interactions, resulting in neural signals to other organs and in cognitive and emotional performance. It's worth pointing out that our understanding of brain function has progressed by leaps and bounds in the last 25 years, radically changing the landscape of psychology and psychiatry and, of course, neurology.[5] Much of the research on vestibular function, whereon I draw heavily, is even more recent, conducted within just the last 10-15 years.

In the world of medicine my study is properly called a "case series," being defined as *a descriptive account of a series of individuals with the same new medical problem*. Let me be clear: a case series is a standard and valid form of medical research. New illnesses are often introduced with case series, whose role is to define an illness, suggest causation, and alert the medical and research profession to its existence. (This, simply put, is the purpose of this report.) After an illness is defined and awareness cultivated, it becomes more feasible to do larger, more expensive studies to explore etiology (causation), pathophysiology, and epidemiologic characteristics.

Case series do not typically have control groups, nevertheless I saw I needed a comparison group of similar, though unexposed, people to distinguish which symptoms were due to turbine exposure. The most similar unexposed people, of course, were my study subjects themselves prior to turbine exposure and after the end of exposure. I therefore set up a *before-during-after* study format, interviewing families who had already moved out of their homes due to symptoms, or who were planning to move and had already spent periods away from home during which turbine-associated symptoms abated.

This format served a three-fold purpose: 1) it ensured there was an "after" phase for each family, 2) it guaranteed that at least one member of each family was severely affected, enough to need to move, and 3) it provided validation for participant statements, since one can hardly discount the seriousness of symptoms that force a family to vacate its home or perform expensive renovations aimed solely at noise exclusion.

This brings us to what is known in science as a "natural experiment." A natural experiment being *a circumstance wherein subjects are exposed to experimental conditions both inadvertently and ecologically (within their own homes and environment*s). Obviously it would be unethical to expose

[5] See, for example, Schore, Allan N., 1994. *Affect Regulation and the Origin of the Self: The Neurobiology of Emotional Development.* Lawrence Earlbaum Associates, Hillsdale, NJ. 700 pp.

people deliberately to potentially harmful interventions. Hence natural experiments, while less controlled, have an important role in clarifying the impacts of potentially toxic, man-made exposures. The ecological dimension is also significant, since many elements of an exposure may not be reproducible in a laboratory, such as round-the-clock exposure, exposure over months, or impacts on customary activities. For symptoms related to wind turbine sound, there are also technical difficulties in reproducing in a laboratory the types of sound, air pressure variation, and vibration that my subjects' observations suggest are involved. Failure to provoke the same symptoms in a laboratory setting may tell us more about the limitations of the laboratory situation than about the real-world effects.

To further create comparison groups, I collected information on all members in the ten families, not just the most affected. This widened the age range of subjects and gave me information on variably affected people who were all exposed to turbine noise capable of causing severe symptoms. I then used the natural variation within the study group to examine which elements of the *pre-exposure* medical history predicted which parts of the *during-exposure* symptom complex. By this method, the study begins to answer the intriguing question of why some individuals are affected more than others by living near wind turbines, and which individuals in the general population are at notable risk for symptoms.

It would be extremely difficult to do a conventional epidemiologic study of the health effects of wind turbines, even if one were blessed with substantial funding and institutional backing, as I was not. By "epidemiologic" I mean studies in which random or regular sampling is used, as for example assessing everyone within three miles of a set of turbines, or every fourth name in an alphabetical listing of everyone within three miles. The difficulty comes from the legal and financial stone wall of the *gag clause*.

In the course of this study I repeatedly encountered these clauses in leases between wind developers and landowners, in "good neighbor" contracts between wind developers and neighbors to leaseholders, and in court decisions following citizen challenges to wind turbine development. Gag clauses prohibiting people who receive payments from wind companies, or who have lost legal challenges, from saying anything negative about the turbines or developer. The prohibition includes matters of health, of course. In an epidemiologic study these clauses could easily distort answers or skew participation, invalidating the random sample. Some people informed me, as well, that they didn't want to talk about their problems because they hoped to sell their homes in order to flee the turbines next door. (No better way to kill a real estate deal than to leak the news that one's home is toxic.) There is also the matter of relationships and

family ties within small communities, where folks are often reluctant to reveal a problem because, let's say, one's cousin's turbines are the source of it.

In this manner has the wind industry both shattered many rural communities and thwarted research like mine.

Despite what I see as the virtues of my approach, this study has clear limitations. One being that it was conducted entirely by clinical interview, over the telephone. This had the benefit of allowing me to have an international group of subjects. On the other hand it limited the type of data I could collect. As a result my ability to say that a certain symptom during exposure is due to turbines is confined to medical conditions that are diagnosable by medical history – the medical history being *all the information a patient tells the doctor about his illness, his past health and experiences, and habits*. Some conditions are diagnosed mostly by history, such as migraine and other headaches, tinnitus, and sleep disturbance. For, of course, the doctor cannot tell objectively if a patient has headache, tinnitus, or sleep disturbance, and much of what the physician figures out about the causes of these symptoms will come from the other questions she asks. This is the part I could do by telephone.

My study subjects also told me about other kinds of problems that seemed to them to be accentuated during exposure, including asthma, pneumonia, pleurisy, stroke, and changes in coagulation or blood sugar. I did not include these problems in Wind Turbine Syndrome, since my method of study did not allow me to determine whether in fact the wind turbines played a role in these conditions during exposure. These conditions would require other kinds of study over and above the clinical interview. (I have included them in a separate section of the *Results* because I think they may need attention from the medical research community.)

The format of the book is that of a scientific article, beginning with an *Abstract* or brief summary, followed by an *Introduction* to the problem and background information, a description of the *Methods* used (including study population selection), a presentation of the *Results* (which is the data secured during the study and its analysis), and a *Discussion* of the results with interpretation of their meaning in the context of current medical knowledge. *References* are footnoted in the text and listed together at the end. I added a *Glossary* of medical and technical terms to make the book more intelligible to non-medical readers.

The book, in fact, is intended for physicians and other professionals and individuals who wish to better understand the wind turbine-associated symptom complex.

Wind Turbine Syndrome is not the same as Vibroacoustic Disease.[6] I say this because the two are often equated in the popular media. The proposed mechanisms are different, and the noise amplitudes are probably different as well. Wind Turbine Syndrome, I propose, is mediated by the vestibular system – by disturbed sensory input to eyes, ears, and stretch and pressure receptors in a variety of body locations. These feed-back neurologically onto a person's sense of position and motion in space, which is in turn connected in multiple ways to brain functions as disparate as spatial memory and anxiety. Several lines of evidence suggest that the amplitude (i.e., power or intensity) of low frequency noise and vibration needed to create these effects may be even lower than the auditory threshold at the same low frequencies. Re-stating this, one does not have to be able to hear the low frequency noise and vibration to experience the vestibular effects described as Wind Turbine Syndrome.

Vibroacoustic Disease, on the other hand, is hypothesized to be caused by direct tissue damage to a variety of organs, creating thickening of supporting structures and other pathological changes. It is caused by high amplitude (high power or high intensity) low frequency noise. Given my research protocol, described above, my study is of course unable to demonstrate whether wind turbine exposure causes the types of pathologies found in Vibroacoustic Disease, although there are similarities worthy of further clinical investigation, especially with regard to asthma and lower respiratory infections.

A few words about peer review. Peer review is quite simple, contrary to the mystique it has acquired among wind developers (most of whom probably have a fanciful idea of what it is). Peer review *consists of sending a scholarly manuscript to experts in that particular field of knowledge, who are asked to judge whether it merits publication*. Simple as that. The identity of reviewers (also called "referees") can be either known to the author (this is often the case with book manuscripts, where authors are routinely asked by the editor to submit a list of possible referees) or kept confidential.

If the referees (usually consisting of two or three) manage to convince the editor that the manuscript is not worthy of publication, the editor contacts the author and rejects the manuscript. If, on the other hand, the referees feel the manuscript merits publication subject to certain revisions and perhaps additions, the editor will forward their reports to the author and ask for a response. "Are you willing to make these changes? Do you agree with these criticisms? If not, give me compelling reasons why not."

[6] Castelo Branco NAA , Alves-Pereira M. 2004. Vibroacoustic disease. Noise Health 6(23): 3-20.

The author then revises the manuscript accordingly, except where she feels her referees are wrong – and manages so to convince the editor. Once the editor feels the author has addressed criticisms and suggestions adequately, he (she) proceeds with publication.

Lastly, referees do not have to agree with the author's arguments or conclusions. This is worth emphasizing. Their purpose is merely to certify that a) the manuscript conforms to conventional standards of scholarly or clinical research appropriate for the discipline, and, perhaps most important, b) the manuscript is a significant contribution to knowledge.

In the case of this book, a variety of scientists and physicians, all professors at medical schools or university departments of biology, read and commented on the manuscript and recommended it as an important contribution to knowledge and as conforming to the canons of clinical and scientific research. Moreover, they did in fact suggest revisions, even substantial revisions and additions – all of which I made. Some gave me written reports to include in the book itself. Others offered to review the book after it was published.

With that said, the litmus test of scientific validity is not peer review (which, after all, is not infallible, as the history of science amply demonstrates). Peer review is an important first step in judging scientific or scholarly merit, however the ultimate test is whether other scientists can follow the author's research protocol and get the same results.

That, of course, remains to be seen with this report.

I thank Dr. Joel Lehrer in particular for providing me with new information regarding vestibular function, contributions echoed by Drs. Owen Black and Abraham Shulman (all in otolaryngology/neurotology). I thank Professors Henry Horn (ecology) and Ralph Katz (epidemiology) for discussion of scientific method and presentation. Dr. Jerome Haller (neurology) and Professor Robert May (theoretical ecology and epidemiology, past president of the Royal Society of London) read the manuscript and provided commentary to be included in the book, as did Dr. Lehrer and Professors Horn and Katz, for which I am most grateful. Barbara Frey (biomedical librarian) edited the manuscript. Other readers read and discussed the manuscript with me and advised on routes of publication. These included Professor Carey Balaban (neuroscience), Dr. Rolf Jacob (psychiatry/neurotology interface), Dr. John Modlin (pediatrics/ infectious diseases), and Dr. Anne Gadomski (pediatrics/public health). I thank them all, as well as

Christina Ransom and William McCall, librarians of the Champlain Valley Physician's Hospital in Plattsburgh, NY, and the FYI Hospital Library Circuit Rider Program.

George Kamperman and Rick James, INCE (Institute of Noise Control Engineering) certified noise control engineers, edited the sections describing noise measurement and modeling. They also analyzed noise studies done at the homes of several affected families, while developing standards and protocols for the assessment and control of noise from industrial wind turbines. Kamperman and James presented their standards and rationale at the Noise-Con 2008 meeting of the Institute of Noise Control Engineering (USA) in July 2008, then expanded their paper with a detailed discussion of noise measurement protocols and a model wind turbine ordinance.[7] The expanded paper is posted on the Wind Turbine Syndrome website.[8]

Some are surprised that I chose to publish this study as a book rather than an article. My reason is straightforward: it's too long for a medical or scientific journal. The problem is the incompressible yet indispensable narrative data – people's accounts of their sensations, experiences, symptoms, and history. It would be impossible to present these accounts in a 3000 or 7000-word article, yet they are essential as evidence for qualitative changes around turbines. For example, to support a summary statement like, "The noise from wind turbines has a different and disturbing quality, even when it does not seem loud," I must present the descriptions given by multiple study participants. To describe a symptom new to medicine, such as the feeling of internal vibration or pulsation, I again need the words of multiple participants. Because I could not do testing to examine thinking and memory abilities, for example, I need to recount the subjects' own evidence, consisting of their descriptions of things they used to do easily but now cannot do, or of loss and recovery in their children's school functioning.

Many of my reviewers suggested ways to split the study into shorter papers – a segment on migraine, a segment on tinnitus, a segment on methodology, for example. However, I feel that keeping the entire study in one piece makes for a more powerful and intelligible document, allowing readers to appreciate the intertwined nature of individual symptoms and the way they fit with new neural models of vestibular function.

As for the reception I anticipate for this report, I don't flatter myself that it will be greeted with loud hosannas from the wind industry. Keep in mind that wind developers have what is called in science a

[7] Kamperman GW, James RR, "Simple guidelines for siting wind turbines to prevent health risks," at the annual conference of the Institute of Noise Control Engineering/USA, Noise-Con, July 28-31, 2008.
[8] See "How loud is too loud?" www.windturbinesyndrome.com.

"conflict of interest." Meaning, their judgment is unduly influenced by money. "It's difficult to get a man to understand something when his salary depends upon his not understanding it," wryly observed Upton Sinclair.[9]

I, on the other hand, have no conflicts of interest. This research was unfunded, and neither my small village property, my town, nor the Adirondack Park bordering my town is a likely candidate for a wind farm. Is a fondness for bats and other interesting, highly evolved animals a conflict of interest? I wouldn't think so. Admittedly, I am distressed to hear about bats dying of internal hemorrhage as they fly near wind turbines, just as I am distressed to hear that people are forced from their homes or endure cognitive impairment of uncertain reversibility in order to remain in the only home they can afford.[10] I have spoken and written earnestly and vigorously about wind developers because of their stubborn refusal to acknowledge health problems amply documented in this and numerous other studies.[11] Such stonewalling would test the patience of a saint – and I am no saint.

My hope is that this report will balance the risk-benefit picture of wind turbines more realistically, and help those individuals, such as George Kamperman and Rick James, who are actively promoting noise control criteria that will prevent the health and home abandonment problems documented here.

Kamperman and James have convinced me that a single, one-size-fits-all setback distance may not be both protective and fair in all environments with all types of turbines. Even so, it is clear from this study and others that minimum protective distances need to be:

a) more than the 1-1.5 km (3280-4900 ft or 0.62-0.93 mi) at which there were severely affected subjects in this study,
b) more than the 1.6 km (5250 ft or 1 mi) at which there were affected subjects in Dr. Harry's UK study,
c) and, in mountainous terrain, more than the 2-3.5 km (1.24-2.2 mi) in which there were symptomatic subjects in Professor Robyn Phipps's New Zealand study.[12]

Two kilometers, or 1.24 miles, remains the baseline, shortest setback from residences (and hospitals, schools, nursing homes, etc.) that communities should consider. In mountainous terrain, 2 miles (3.2 km)

[9] Sinclair, Upton, 1935. *I, Candidate for Governor: And How I Got Licked.*
[10] Baerwald EF, D'Amours GH, Klug BJ, Barclay RM. 2008. Barotrauma is a significant cause of bat fatalities at wind turbines. Curr Biol 18(16): R695-6. Due to air pressure shifts near moving turbine blades, blood vessels in bats' lungs and abdomen are disrupted, which produces fatal internal hemorrhage.
[11] In anticipation of wind industry blowback, I imagine it may once again publicize that it thinks *I think* wind turbines cause mad cow disease. I do not and never did. My reply to this canard – now a family joke – was published several years ago (www.windturbinesyndrome.com/?p=84). My previous reports and papers on Wind Turbine Syndrome and the wind industry can be found on www.windturbinesyndrome.com.
[12] See *Introduction*, p. 4, for discussion and references.

is probably a better guideline. Setbacks may well need to be longer than these minima, as guided by the noise criteria developed by Kamperman and James.

The shorter setbacks currently in use in the USA and elsewhere – 1000 to 1500 ft (305-457 m) – are a convenience and financial advantage for wind developers and participating landowners. They have no basis in research on safety and health, and they do not make clinical sense.

My next step in this project will include 1) amassing a larger series of cases to further clarify setback needs relative to turbine power and other characteristics, 2) initiating a standard symptom survey tool for physicians to use in their practices or citizens in their communities after turbines are built, and 3) working with neurotologists to explore objective testing protocols for Wind Turbine Syndrome.

For those who, reading this, recognize their own symptoms, the appropriate medical specialist to consult would be a neurotologist, who is an otolaryngologist (ears, nose, and throat doctor) or neurologist who specializes in balance, the inner ear, and its neurological connections. When I sent this report out for critical review, these were the physicians who recognized a remarkably similar symptom complex from cases familiar to them – such as certain inner ear pathologies.

To those of you living near turbines who recognize your own symptoms within these pages, you are not crazy and not fabricating them. They are clinically valid – and unnecessary. While wind developers rush headlong into yet more projects, you unfortunates will have to exercise patience as the medical profession catches up with what is ailing you. Meanwhile, my advice is: Speak out. In *The Tyranny of Noise*, Robert Alex Baron calls for an end to "our passive acceptance of industry's acoustic waste products."[13] This will happen only when the suffering refuse to be silenced.

By the time I finished interviewing (February 2008) and moved on to data analysis and writing, six of the ten families in this study had moved out of their homes because of turbine-associated symptoms. Three months later (May 2008), when the first draft was complete and I contacted the families for their approval and permission to publish the information about them, two more had moved out because of their turbine-associated symptoms – bringing the total to eight of the ten. The ninth family could not afford to move, but had done extensive renovations in an effort to keep the noise out. (Renovations, ironically, that made

[13] Baron, Robert Alex, 1970. *The Tyranny of Noise: The World's Most Prevalent Pollution, Who Causes It, How It's Hurting You, and How to Fight It*. St. Martin's Press, New York. P. 12.

the house worse to live in, since they could no longer heat it properly.) As of this writing, family number ten is struggling to remain in their home.

Behold ten families whose lives have been turned upside down because of the wind industry's acoustic waste products.

Finally, ask yourself why a country doctor practicing in the poorest county in New York State did this study, and not the Centers for Disease Control or some other relevant government agency. It's a fair question and a troubling one. I ask it myself.

It is well known that wind developers target impoverished communities for their wind farms. This explains the "poorest county" part of my question, and likewise why wind turbines quickly became a looming issue in my life four years ago. But it leaves unanswered the part about, Why did I write this report, and not the government?

To answer that would of necessity catapult this report (and me) into the treacherous territory of public policy. One would like to think science is not beholden (craven?) to public policy, but that would be naïve, would it not? Moreover, while the scientist in me would like to imagine that I can write this report and remain above the hurly burly of public policy, I know this, too, is naïve. Wind Turbine Syndrome is an industrial plague. It is man-made and easily fixed. Proper setbacks are the best cure I know of; they do the job just fine. If I could scrawl this on a prescription pad and hand it to my subjects in this report, I would do so. No brilliant scientist needs to discover a new antibiotic or vaccine or sleeping pill to treat it.

Setbacks, however, are not considered matters of public health, but matters of public policy – what is called "politics." And right there is the rub. Right now, in the global rush to wind energy, there is almost no voice heard for public health repercussions. Where it is heard – at town meetings, on the Internet, in Letters to the Editor, in courtrooms – it is routinely ridiculed. I speak from experience.

Wind energy is being promoted by every state and national government I know of – under intense lobbying by wind development companies generally owned or otherwise capitalized by powerful investment banks which in turn take large tax write-offs and reap large government subsidies for their wind farm projects, who then turn around and sell carbon credits (green credits). Perhaps this helps explain why no provision is made for clinical caution?

And perhaps this goes some way toward explaining why a pediatrician in rural NYS and a general practitioner in Cornwall, England – along with a handful of physicians elsewhere in the UK and Australia and who knows where else – are the ones funding this research and writing these reports.

Then so be it.

Nina Pierpont, MD, PhD
Malone, NY
Autumn 2008

Note: This page is in manuscript format, pending publication of the book.

ABSTRACT

This report documents a consistent, often debilitating, complex of symptoms experienced by adults and children while living near large (1.5-3 MW) industrial wind turbines, examines patterns of individual susceptibility, and proposes pathophysiologic mechanisms. Symptoms include sleep disturbance, headache, tinnitus, ear pressure, dizziness, vertigo, nausea, visual blurring, tachycardia, irritability, problems with concentration and memory, and panic episodes associated with sensations of internal pulsation or quivering which arise while awake or asleep.

The study is a case series of 10 affected families, with 38 members age 0-75, living 305 m to 1.5 km (1000 to 4900 ft) from wind turbines erected since 2004. All competent and available adults and older teens completed a detailed clinical interview about their own and their children's symptoms, sensations, and medical conditions before turbines were erected near their homes, while living near operating turbines, and after leaving their homes or spending a prolonged period away.

Statistically significant risk factors for symptoms during exposure include pre-existing migraine disorder, motion sensitivity, or inner ear damage (pre-existing tinnitus, hearing loss, or industrial noise exposure). Symptoms are not statistically associated with pre-existing anxiety or other mental health disorders. The symptom complex resembles syndromes caused by vestibular dysfunction. People without known risk factors are also affected.

The proposed pathophysiology posits disturbance to balance and position sense due to low frequency noise or vibration stimulating receptors for the balance system (vestibular, somatosensory, or visceral sensory, as well as visual stimulation from moving shadows) in a discordant fashion. Vestibular neural signals are known to affect a variety of brain areas and functions, including memory, spatial processing, complex problem-solving, fear, autonomic effects, and aversive learning, providing a robust neural framework for the symptom associations in Wind Turbine Syndrome. Further research is needed to establish prevalence and to explore effects in special populations, including children. This and other studies suggest that safe setbacks will at least 2 km (1.24 mi), and will be longer for larger turbines and in more varied topography.

DEDICATION

This study is dedicated to the memory of Dudley Weider, MD, Otolaryngologist at the Dartmouth-Hitchcock Medical Center, who sent me to Alaska, diagnosed and cured my husband, and taught me about migraine and dizziness. We miss him.

Note: These are sample, manuscript pages from the body of the report. Be aware that there are gaps between pages, pending publication of the book.

INTRODUCTION AND BACKGROUND

Policy initiatives in the United States and abroad currently encourage the construction of extremely large wind-powered electric generation plants (wind turbines) in rural areas. In its current format, wind electric generation is a variably regulated, multi-billion dollar a year industry. Wind turbines are now commonly placed close to homes. Usual setbacks in New York State, for example, are 305-457 m (1000-1500 ft) from houses.[1] Developer statements and preconstruction modeling lead communities to believe that disturbances from noise and vibration will be negligible or nonexistent.[2,3,4] Ahead of time, developers compare the noise the turbines will make to one's refrigerator, a library reading room, or the rustling of tree leaves, which they say easily obscures turbine noise.[5]

Despite these assurances, some people experience significant symptoms after wind turbines are placed in operation near their homes. The purpose of this study is to establish a case definition for the consistent, frequently debilitating, set of symptoms experienced by people while living near wind turbine installations, and to place this symptom complex within the context of known pathophysiology. A case definition is needed to allow studies of causation, epidemiology, and outcomes to go forward, and to establish adequate community controls.

This set of symptoms stand out in the context of noise control practice. George Kamperman, P.E., INCE Bd. Cert., past member of the acoustics firm Bolt, Beranek and Newman (USA), wrote, "After the first day of digging into the wind turbine noise impact problems in different countries, it became clear the health impact on persons living within about two miles from 'wind farms' all had similar complaints and health problems. I have never seen this type of phenomenon [in] over fifty plus years of consulting on industrial noise problems. The magnitude of the impact is far above anything I have seen before… I can

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[1] Town of Ellenburg, NY wind law—1000 ft (305 m); Town of Clinton, NY wind law—1200 ft (366 m); Town of Martinsburg, NY wind law—1500 ft (457 m). For other examples in and outside NY State, see *Wind Energy Development: A Guide for Local Authorities in New York*, New York State Energy Research and Development Authority, October 2002, p. 27. http://text.nyserda.org/programs/pdfs/windguide.pdf.

[2] "The GE 1.5 MW wind turbine, which is in use in Fenner, New York, is generally no louder than 50 decibels (dBA) at a distance of 1,000 feet (the closest we would propose siting a turbine to a residence). Governmental and scientific agencies have described 50 dBA as being equivalent to a 'quiet room.' Please keep in mind that these turbines only turn when the wind blows, and the sound of the wind itself is often louder than 50 dBA. Our own experience, and that of many others who live near or have visited the Fenner windfarm, is that the turbines can only be heard when it is otherwise dead quiet, and even then it is very faint, especially at a distance." Letter from Noble Environmental Power, LLC, to residents of Churubusco (Town of Clinton), New York, 7/31/2005.

[3] "Virtually everything with moving parts will make some sound, and wind turbines are no exception. However, well-designed wind turbines are generally quiet in operation, and compared to the noise of road traffic, trains, aircraft, and construction activities, to name but a few, the noise from wind turbines is very low... Today, an operating wind farm at a distance of 750 to 1,000 feet is no noisier than a kitchen refrigerator or a moderately quiet room." *Facts about wind energy and noise*. American Wind Energy Association, August 2008, p. 2. http://www.awea.org/pubs/factsheets/WE_Noise.pdf.

[4] "In general, wind plants are not noisy, and wind is a good neighbor. Complaints about noise from wind projects are rare, and can usually be satisfactorily resolved." *Facts about wind energy and noise*. American Wind Energy Association, August 2008, p. 4. http://www.awea.org/pubs/factsheets/WE_Noise.pdf.

[5] "Outside the nearest houses, which are at least 300 metres away, and more often further, the sound of a wind turbine generating electricity is likely to be about the same level as noise from a flowing stream about 50-100 metres away or the noise of leaves rustling in a gentle breeze. This is similar to the sound level inside a typical living room with a gas fire switched on, or the reading room of a library or in an unoccupied, quiet, air-conditioned office... Even when the wind speed increases, it is difficult to detect any increase in turbine sound above the increase in normal background sound, such as the noise the wind itself makes and the rustling of trees." *Noise from wind turbines: the facts.* British Wind Energy Association, August 2008. http://www.bwea.com/ref/noise.html

see the devastating health impact from wind turbine noise but I can only comment on the physical noise exposure. From my viewpoint we desperately need noise exposure level criteria."[6]

What is remarkable, and different from previous industrial noise control problems, is that a broad cross-section of people with regard to age, neurologic vulnerabilities, and state of health are being exposed over prolonged periods, often around the clock, to types of noise and vibration previously confined to industrial, military, or aeronautical settings. For this reason, this study also contributes significantly to the science of noise control in general.

I named this complex of symptoms "Wind Turbine Syndrome" in a preliminary fashion in testimony before the Energy Committee of the New York State Legislature on March 7, 2006. My observation that people can feel vibration or pulsations from wind turbines, and find it disturbing, was quoted in the brief section "Impacts on Human Health and Well-Being" in the report *Environmental Impacts of Wind-Energy Projects* of the National Academy of Science, published in May 2007. No other medical information was cited in this report. The authors asked for more information to better understand these effects.[7]

Debates about wind turbine-associated health problems have to date been dominated by noise control engineers, or acousticians, which is problematic in part because the acoustics field at present is dominated by the wind turbine industry,[8] and in part because acousticians are not trained in medicine. A typical approach to wind turbine disturbance complaints, world-wide, is noise first, symptoms second: if an acoustician can demonstrate with noise measurements that there is no noise considered significant in a setting, then the symptoms experienced by people in that setting can be, and frequently are, dismissed. This has been the experience of seven of the ten families in this study in the United States, Canada, Ireland, and Italy.[9] At least one developer has put forward the hypothesis that a negative attitude or worry towards turbines is what leads people to be disturbed by turbine noise.[10]

A reorientation is in order. If people are so disturbed by their headaches, tinnitus, panic, sleeplessness, or disrupted children that they must move or abandon their homes to get away from wind turbine noise and vibration, then that noise and vibration is significant, because the symptoms it causes are significant. The

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[6] George Kamperman, personal communication, 2/21/2008. See http://www.kamperman.com/index.htm.

[7] National Research Council. 2007. *Environmental Impacts of Wind-Energy Projects*. The National Academies Press, Washington, DC. 185 pp, p. 109.

[8] George Kamperman, personal communication, 2/23/2008.

[9] A notable exception to this pattern is the work of GP van den Berg, PhD, who, as a graduate student and a member of the Science Shop for Physics of the University of Groningen in the Netherlands, investigated noise complaints near a wind turbine park and devised new models of atmospheric noise propagation to fit the phenomena he observed. References: 1) van den Berg, GP. 2004. Effects of the wind profile at night on wind turbine sound. Journal of Sound and Vibration 277: 955-970; 2) van den Berg, GP. 2004. Do wind turbines produce significant low frequency sound levels? 11th International Meeting on Low Frequency Noise and Vibration and Its Control, Maastricht, The Netherlands, 30 August to 1 September 2004, 8 pp.; 3) van den Berg, GP. 2005. The beat is getting stronger: the effect of atmospheric stability on low frequency modulated sound of wind turbines. Journal of Low Frequency Noise, Vibration, and Active Control, 24(1): 1-24; 4) van den Berg, GP. 2006. The sound of high winds: the effect of atmospheric stability on wind turbine sound and microphone noise. PhD dissertation, University of Groningen, The Netherlands. 177 pp. http://irs.ub.rug.nl/ppn/294294104

[10] "We often use the word 'noise' to refer to 'any unwanted sound.' It's true that wind turbines make sounds... but whether or not those sounds are 'noisy' has a lot to do with who's listening. It's also worth noting that studies have shown [no references provided in source document] that a person's attitude toward a sound – meaning whether it's not 'wanted' or 'unwanted' sound – depends a great deal on what they think and how they feel about the source of the sound. In other words, if someone has a negative attitude to wind turbines, or is worried about them, this will affect how they feel about the sound. However, if someone has a positive attitude toward wind energy, it's very unlikely that the sounds will bother them at all." *Wind fact sheet #5: Are modern wind turbines noisy*? Noble Environmental Power, LLC, http://www.noblepower.com/faqs/documents/06-08-23NEP-SoundFromWindTurbines-FS5-G.pdf, p. 2.

role of an ethical acoustician is to figure out what type and intensity of noise or vibration creates particular symptoms, and to propose effective control measures.

My study subjects make it clear that their problems are caused by noise and vibration. Some symptoms in some subjects are also triggered by moving blade shadows. However, I do not present or analyze noise data in this study, because noise is not my training. (Conversely, symptoms and disease are not the training of acousticians.) I focus on detailed symptomatic descriptions and statistical evaluation of medical susceptibility factors within the study group. Correlating the noise and vibration characteristics of the turbine-exposed homes with the symptoms of the people in the homes is an area ripe for collaboration between medical researchers and independent noise control engineers.

Other than articles on the Internet, there is currently no published research on wind turbine associated symptoms. A UK physician, Dr. Amanda Harry, whose practice includes patients who live near wind turbines, has published online the results of a checklist survey, documenting specific symptoms among 42 adults who identified themselves to her as having problems while living 300 m to 1.6 km (984 ft to 1 mi) from turbines.[11] She found a high prevalence of sleep disturbance, fatigue, headache, migraine, anxiety, depression, tinnitus, hearing loss, and palpitations. Respondents describe a similar set of symptoms and many of the same experiences that I document in this paper, including having to move out of their homes because of symptoms. Respondents were mostly older adults: 42% were age 60 or older, 40% age 45-60, 12% age 30-45, and 5% age 18-30. A biomedical librarian, Barbara Frey, working with this physician and others, has published online a compilation of other personal accounts of symptoms and sensations near wind turbines.[12] These also mirror what I document.

Robyn Phipps, PhD, a New Zealand scientist specializing in health in indoor environments, systematically surveyed residents up to 15 km (9.3 mi) from operating wind turbine installations, asking both positive and negative questions about visual, noise, and vibration experiences.[13] All respondents (614 or 56% of the 1100 households to whom surveys were mailed) lived at least 2 km (1.24 mi) from turbines, with 85% of respondents living 2-3.5 km (1.24-2.2 mi) from turbines and 15% further away. Among other questions, the survey asked about unpleasant physical sensations from turbine noise, which were experienced by 2.1% of respondents, even at these distances. Forty-one respondents (6.7%) spontaneously telephoned Dr. Phipps to tell her more than was asked on the survey about their distress due to turbine noise and vibration, nearly all (39) with disturbed sleep.[14] Symptoms were not further differentiated in this study, but clearly may occur even at distances greater than 2 km (1.24 mi) from turbines.

Published survey studies have examined residents' reactions to wind turbines relative to modeled noise levels and views of turbines in Sweden[15,16,17] and the Netherlands.[18,19,20] The studies in the Netherlands

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[11] Harry, Amanda. February 2007. Wind turbines, noise, and health. 32 pp. http://www.windturbinenoisehealthhumanrights.com/wtnoise_health_2007_a_barry.pdf

[12] Frey, Barbara J and Hadden, Peter J. February 2007. Noise radiation from wind turbines installed near homes: effects on health. 137 pp. http://www.windturbinenoisehealthhumanrights.com/wtnhhr_june2007.pdf

[13] Phipps, Robyn. 2007. Evidence of Dr. Robyn Phipps, in the matter of Moturimu wind farm application, heard before the Joint Commissioners 8th-26th March 2007, Palmerston North [New Zealand]. 43 pp. http://www.wind-watch.org/documents/wp-content/uploads/phipps-moturimutestimony.pdf

[14] Phipps 2007

[15] Pedersen E, Persson Waye K. 2004. Perceptions and annoyance due to wind turbine noise – a dose-response relationship. J Acoust Soc Am 116(6): 3460-70.

the hearing sense of the human being. Noise is defined as any perceived sound that is objectionable to a human being."[24] Following the usual usage in noise literature, I use the word *vibration* to refer only to disturbances in solid media, such as the ground, house structures, or the human body. When air-borne sound waves of particular energy (power) and frequency meet a solid object, they may set the object vibrating. A vibrating solid object, such as the strings on a violin, can conversely create sound waves in air. There can be energy transfer in both directions between air-borne or fluid-borne sound waves and the vibration of solids. When I talk about noise and vibration together, I am referring to this continuum of mechanical energy in the air and mechanical energy in solids.

Energy in either pathway can impinge on the human body, and there may be multiple exchanges between air and solids in the path between a source and a human. The tissues of humans and other animals are also semi-liquid to varying degrees, and have fluid-filled and air-filled spaces within them, as well as solid structures like bones. As an example of such energy transfer, a sound wave in the air, encountering a house, may set up vibrations in the structure of the house. These vibrations, in walls or windows, may set up air pressure (sound) waves in rooms, which in turn can transmit mechanical energy to the tympanic membrane and middle ear, to the airways and lungs, and to body surfaces. Alternatively, vibrations in house structures or the ground may transmit energy directly to the body by solid-to-solid contact and be conducted through the body by bone conduction.

All parts of the body, and indeed all objects, have specific resonance frequencies, meaning that particular frequencies or wavelengths of sound will be amplified in that body part.[25] If the wavelength of a sound or its harmonic matches the dimensions of a room, it may set up standing waves inside the room in which the power or amplitude of the wave reinforces and is increased at certain locations in the room. Likewise inside air-filled body cavities such as the lungs, trachea, pharynx, middle ear, mastoid, and gastrointestinal tract, though in these cases the elasticity of walls and variations in the density of the contents affects the dynamics of the sound waves. The orbits and cranial vault are resonance chambers of sorts, because of the lesser density of their contents compared to the bones that surround them. There are also resonance patterns along the spine, including a resonance involving the movement of the head relative to the shoulders. Von Gierke[26,27] and Rasmussen[28] have described the resonant frequencies of parts of the human body.

Noise intensity is measured in decibels (dB), a logarithmic scale of sound pressure amplitude. Single noise measurements or integrated measurements over time combine the energies of a range of frequencies into a single number, as defined by the filter or weighting network used during the measurement. The A weighting network is the most common in studies of community noise. It is designed to duplicate the frequency response of human hearing, so it does not capture the low frequencies to which human hearing is relatively insensitive. Linear (lin) measurements use no weighting network, so the frequency responses

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[24] Beranek, LL. 2006. Basic acoustical quantities: levels and decibels. Chapter 1 in Ver IL, Beranek LL, *Noise and Vibration Control and Engineering: Principles and Applications.* John Wiley and Sons, Hoboken, NJ. 976 pp., p. 1.

[25] Hedge, Alan, Professor, Cornell University, Department of Design and Environmental Analysis, syllabus/lecture notes for DEA 350: Whole-Body Vibration, January 2007, found at http://ergo.human.cornell.edu/studentdownloads/DEA325pdfs/Human%20Vibration.pdf

[26] von Gierke HE, Parker DE. 1994. Differences in otolith and abdominal viscera graviceptor dynamics: implications for motion sickness and perceived body position. Aviat Space Environ Med 65(8): 747-51.

[27] von Gierke HE. 1971. Biodynamic models and their applications. J Acoust Soc Am 50(6): 1397-413.

[28] Rasmussen G. 1982. Human body vibration exposure and its measurement. Bruel and Kjaer Technical Paper No. 1, Naerum, Denmark. Abstract: Rasmussen G. 1983. Human body vibration exposure and its measurement. J Acoust Soc Am 73(6): 2229.

Eight subjects had permanent hearing impairments, defined subjectively or objectively, including mild losses, losses limited to one ear, or impairments of binaural processing. Six were male (age 32-64) and two female (age 51-57).

Six subjects had continuous tinnitus or a history of multiple, discrete episodes of tinnitus prior to exposure. Four were male (age 19-64) and two female (age 33-57).

Twelve subjects had significant previous noise exposure, defined as working in noisy industrial or construction settings; working on or in a diesel boat, truck, bus, farm equipment, or aircraft; a military tour of duty; or operating lawn mowers and chain saws for work. Not included were home or sporadic use of lawnmowers and chain saws, commuting by train or airplane, urban living in general, or playing or listening to music. Nine of the noise-exposed subjects were male (age 19-64) and three female (age 33-53).

Eighteen subjects were known to be motion sensitive prior to exposure, as defined by carsickness as a child or adult, any episode of seasickness, or a history of two or more episodes of vertigo. Ten were male (age 6-64) and eight female (age 12-57).

Seven subjects had a remembered history of a single concussion, and none had a history of a more severe head injury. Six were male (age 19-59) and one female (age 12). I did not collect information on whiplash injury.

Core symptoms

Core symptoms are defined as: 1) common and widely described by study participants; 2) closely linked in time and space to turbine exposure; and 3) amenable to diagnosis by medical history. Core symptoms include sleep disturbance, headache, tinnitus, other ear and hearing sensations, disturbances to balance and equilibrium, nausea, anxiety, irritability, energy loss, motivation loss, and disturbances to memory and concentration.

An additional core symptom is a new type of internal or visceral sensation which has no name in the medical lexicon. Subjects struggled to explain these sensations, often apologizing for how strange their words sounded. A physician subject called it “feeling jittery inside” or “internal quivering.” Other subjects chose similar words, while others talked about feeling pulsation or beating inside. The physical sensations of quivering, jitteriness, or pulsation are accompanied by acute anxiety, fearfulness, or agitation, irritability, sleep disturbance (since the symptom arises during sleep or wakefulness), and episodes of tachycardia. I call this sensation and accompanying symptoms *visceral vibratory vestibular disturbance* (VVVD). It is described further below.

Core symptoms are closely correlated with exposure, including being at home, the direction and strength of the wind, whether turbines are facing the home, and the presence of moving blade shadows. Core symptoms all resolve immediately or within hours away from the turbines, with the exception of disturbances of concentration and memory, which resolved immediately in some cases or improved over weeks to months in others.

Sleep disturbance. Thirty-two subjects (17 males age 2-64 and 15 females age 2-75) had disturbed sleep. Types of sleep disturbance included: difficulty getting to sleep, frequent or prolonged awakening by turbine noise, frequent or prolonged awakening by awakened children, night terrors (both 2½-year-olds, B3 and G5), nocturnal enuresis (one 5-year-old girl, G4), nocturia (six women age 42-75 and one man age 64; B2, C2, E2, F2, F4, H2, D1), excessive movement during sleep (one 8-year-old boy, H3), excessive nighttime fears (two 5-year-olds, a girl and a boy, C8 and G4), and abrupt arousals from sleep in states of fear and alarm (four women age 42-57; C2, F2, H2, I2). Other adults, though not fearful when they woke up, awoke with physical symptoms similar to their daytime symptoms of anxiety/agitation/internal quivering (three men age 42-64 and two women age 32-53; D1, F1, J1, B2, G2). Four people slept well, including the one infant (G6), a 19-year-old woman (B3), a 47-year-old woman (J2) and her 8-year-old son (J4). It was unclear whether a 56 year-old man with dementia, bipolar disorder, Parkinson's disease, and disturbed sleep at baseline (E1) slept worse than usual or not.

With three exceptions, all types of sleep disturbance resolved immediately whenever subjects slept away from their turbine-exposed homes, including the adult nocturia and the 5-year-old's nocturnal enuresis. A 49-year-old man with a preexisting sleep disturbance (J1) took two nights to get back to his baseline, and a 45-year-old man (C1) and a 42-year-old man (F1) did not improve all the way to baseline, thought to be due to coexisting depression after abandoning their homes.

Headache. Nineteen subjects experienced headaches that were increased in frequency, intensity, and/or duration compared to baseline for that person. Eight were male (age 6-55) and eleven female (age 12-57). Eight had pre-existing migraine (C2, C3, C4, C5, C6, F1, G1, G2). Two women (one a migraineur, one not; C2, E2) had severe headaches provoked by shadow flicker. All other exposure-related headaches were triggered by noise alone. Recovery from headaches generally took several hours after the exposure ended.

Headache risk factors were examined in a subset of the study group which included all subjects age 5 and older (N=34), since the younger children in the study (age 0-2) were not reliable sources of information on headache. The occurrence of unusually severe or frequent headaches during exposure was significantly associated with pre-existing migraine disorder ($\chi^2 = 8.26$, $p = 0.004$). All 8 subjects with pre-existing migraine experienced headaches that were unusually intense, frequent, or prolonged compared to their baseline headaches. Of the 26 subjects without pre-existing migraine, 11 also experienced unusual or severe headaches during exposure. Two of these were children of migraineurs not known to have migraine themselves (a girl age 17 and a boy age 6; F3, G3). All children or teens (through age 21) who had headaches during exposure were migraineurs or children of migraineurs.

Once migraine was factored out as a risk factor, 9 of 17 subjects over age 22 without a history of migraine still had headaches of increased intensity, duration, or frequency during exposure to turbines. I found no significant correlation within this group between headache and the presence of serious underlying medical illness ($\chi^2 = 0.486$, $p = 0.486$), present or past mental health disorder ($\chi^2 = 0.476$, $p = 0.490$), tinnitus or hearing loss at baseline, motion sensitivity at baseline, or tinnitus, disequilibrium, or VVVD during exposure.

In summary, a little more than half the study group (19/34) experienced unusually severe headaches during exposure. Migraine was a statistically significant risk factor but was present in less than half (8/19) of subjects with worsened headache. Children and teens up to age 21 with headaches either had

Internal quivering, vibration, or pulsation. Eleven adult subjects described these uncomfortable, unfamiliar, and hard-to-explain sensations:

- Dr. J (J1, age 49) described "internal quivering" as part of the "jittery feeling" he has when the turbines are turning fast.
- Mrs. I (I2, age 52) said the noise inside her house is "low, pulsating, almost a vibration," not shut out by earplugs. She gets a sensation inside her chest like "pins and needles" and chest tightness on awakening at night to noise. "It affects my body – this is the feeling I get when I say I'm agitated or jittery. It's this that gives me pressure or ringing in my ears." "A feeling someone has invaded not only my health and my territory, but my body."
- Mrs. H (H2, age 57) described a pulsation that prevented sleep in the "unnatural" noise from the turbines.
- Mr. G (G1, age 35) described feeling disoriented and "very strange" in certain parts of the house where he could "feel rumbling." If he did not move quickly away from these locations, the feeling would progress to nausea. He described the noise as "at times very invasive. Train noise has a different quality, and is not invasive."
- Mrs. G (G2, age 32) felt disoriented, "light-headed," dizzy, and nauseated in her garden and in specific parts of the house where she detected vibration. She felt her body vibrating "inside," but when she put her hand on walls, windows, or objects, they did not seem to be vibrating.
- Mrs. F (F2, age 51) described a physical sensation of noise "like a heavy rock concert," saying the "hum makes you feel sick."
- Mrs. E (E2, age 56), when supine, felt a "ticking" or "pulsing" in her chest in rhythm with the audible swish of the turbine blades. She interpreted this as her "heart synchronized to the rhythm of the blades," but there is no information (such as a pulse rate from the wrist at the same time) to determine whether this was true or not, or whether she detected a separate type of pulsation. Mrs. E could make these sensations go away by getting up and moving around, but they started again when she lay back down.
- Mr. D (D1, age 64) felt pulsations when he lay down in bed. In addition, "When the turbines get into a particular position (facing me), I get real nervous, almost like tremors going through your body…it's more like a vibration from outside…your whole body feels it, as if something was vibrating me, like sitting in a vibrating chair but my body's not moving." This occurs day or night, but not if the turbines are facing "off to the side."
- Mr. C (C1, age 45) felt pulsations in his chest that would induce him to hold his breath, fight the sensation in his chest, and not breathe "naturally." Chest pulsations interrupted his sleep and ability to read. He also described a sensation of "energy coming within me…like being cooked alive in a microwave."
- Mrs. B (B2, age 53) described her breath being "short every once in a while, like [while] falling asleep, my breathing wanted to catch up with something."
- Mr. B (B1, age 55) had two episodes of feeling weight on his chest while lying down, which resolved when he stood up. Other than this, he experienced the invasive quality of the noise in his head and ears: "That stuff [turbine noise] doesn't get out of your head, it gets in there and just sits there – it's horrible."

Agitation, anxiety, alarm, irritability, nausea, tachycardia, and sleep disturbance are associated with internal vibration or pulsation:

The internal quivering, vibration, or pulsation and the associated complex of agitation, anxiety, alarm, irritability, tachycardia, nausea, and sleep disturbance together make up what I refer to as *visceral vibratory vestibular disturbance* (VVVD). Fourteen adult subjects (six men age 35-64 and eight women age 32-75) had VVVD during exposure, including the eleven quoted above and Mr. F (F1, age 42), Mrs. F Senior (F4, age 75), and Mrs. C (C2, age 42). Mr. I (I1, age 59) had partial symptoms, with urge to escape, noise-induced nausea, and sleep disturbance, but no feeling of internal movement. VVVD resolves immediately upon leaving the vicinity of the turbines, when the turbines are still and silent, and under favorable weather conditions at each locality.

Because VVVD is in part a panic attack, accompanied by other physical and mental symptoms, I examined the relationships among VVVD and panic disorder, other mental health diagnoses, and other risk factors. The sample for this analysis was 21 adults ages 22 and above (since the study had no participants age 22-29, this is the same for this study as starting with the age group of the youngest symptomatic subjects, who were 32).

No study subjects had pre-existing panic disorder or previous isolated episodes of panic, so there was no correlation between pre-existing panic and VVVD. Seven subjects had immediate pre-exposure (2) or past histories (5) of mental health disorders including depression, anxiety, post-traumatic stress disorder (PTSD), and bipolar disorder. There was no correlation between immediate pre-exposure or past mental health disorder and VVVD ($\chi^2 = 0.429$, $p = 0.513$). There was, however, a highly significant correlation between VVVD and motion sensitivity ($\chi^2 = 7.88$, $p = 0.005$).

There was also a moderately significant correlation between VVVD and headaches during exposure ($\chi^2 = 4.95$, $p = 0.026$). There was no correlation between VVVD and dizziness or tinnitus during exposure, or between VVVD and pre-existing migraine, tinnitus, or hearing loss.

Concentration and memory. Twenty of the 34 study subjects age 4 and up (eleven males age 6-64 and nine females aged 5-56) had salient problems with concentration or memory during exposure to wind turbines compared to pre- and/or post-exposure. This is a conservative count, including only subjects whose accounts included specific information on decline in school and homework performance (for children and teens) or details on loss of function for adults. Eight other subjects had some disturbance to concentration and memory, but symptoms were milder or the descriptions more vague (in their own or parents' accounts). Five others subjects, all older adults, noted no change compared to pre-existing memory problems. This leaves only one subject, a 19-year-old woman home from college and minimally exposed (B3), who did not have baseline deficits and was unaffected.

Pre-exposure cognitive, educational, and work accomplishments, specific difficulties related to concentration and memory during exposure, and degree and timing of post-exposure recovery are documented in the Family Tables for each individual, under "Cognition." Difficulties are often striking compared to the subject's usual state of functioning:

- Mr. A (A1, age 32), a professional fishermen with his own boat, who had an isolated difficulty with memory for names and faces prior to exposure, was unable to remember what he meant to get when he arrived at a store unless he had written it down.

- Mrs. B (B2, age 53), a homemaker, got confused when she went to town for errands unless she had written down what she was going to do, and had to return home to get her list. When interviewed six weeks after moving, she reported that she had improved to being able to manage three things to do without a list.
- Mr. C (C1, age 45) had to put reading aside because he could not concentrate whenever he felt pulsations.
- Mrs. C (C2, age 42), a very organized mother of six who was "ready a month in advance for birthday parties" prior to exposure, became disorganized and had difficulty tracking multiple tasks at once, including while cooking, repeatedly boiling the water away from pots on the stove. She remarked, "I thought I was half losing my mind."
- Mr. D (D1, age 64), a disabled, retired industrial engineer, noticed progressive slowing of memory recall speed and more difficulty remembering what he had read.
- Mrs. E (E2, age 56), a retired teacher active in community affairs, could not spell, write e-mails, or keep her train of thought on the telephone when the turbine blades were turned towards the house, but was able to do these things when the blades were not facing the house.
- Mrs. F (F2, age 51), a nurse, child development specialist, midwife, and Masters level health administrator, could not follow recipes, the plots of TV shows, or furniture assembly instructions during exposure.
- Mrs. G (G2, age 32), a well-organized mother of four, was forgetful, had to write everything down, could not concentrate, and could not get organized. She forgot a child's hearing test appointment. She did not have memory or concentration problems during a previous depression at age 18, and described her experience as "different this time."
- Mr. I (I1, age 59), a professional gardener, could not concentrate on his outdoor gardening and building tasks if the turbines were noisy, saying "after half an hour you have to leave, escape, close the door."
- Dr. J. (J1, page 49), a physician, noticed marked concentration problems when he sat down to pay bills in a small home office with a window towards the turbines.

Decline in school performance compared to pre-exposure, or marked improvement in school performance after moving away from turbines, was noted for 7 of the 10 study children and teens attending school (age 5-17; C7, F3, G3, G4, H3, J3, J4). For example:

- A 17-year-old girl (F3), a diligent student, was not concerned about the turbines and thought her parents were overdoing their concern until she unexpectedly did worse on national exams than the previous year, surprising her school, family, and self. At this point she began accompanying her parents to their sleeping house, and her performance improved.
- A 9-year-old boy (C7) whose school work was satisfactory without need for extra help prior to exposure, failed tests, lost his math skills, and forgot his math facts. He could not maintain his train of thought during homework, losing track of where he was if he looked up from a problem.
- A 6-year-old boy (G3), described as an extremely focused child and advanced in reading prior to exposure, did not like to read during exposure. Two months post-exposure, now age 7, he would sit down to read on his own for an hour at a time, reading “quite a thick book” for his age.
- His five-year-old sister (G4) had a short attention span prior to exposure. Her hearing loss due to bilateral chronic serous otitis media was thought to be interfering with school work during exposure,

Fourth, the problems with concentration and memory resolve on a different schedule from the turbine-related sleep problems. Sleep problems resolve immediately except when accompanied by persistent depression (C1, F1). Problems with concentration and memory frequently took longer to improve, even in the absence of depression. To study resolution, we need to look at subjects who have moved away from their exposed homes or spent a prolonged period away that included work (families A, B, C, E, F, and G, and Mrs. I), since vacations do not provide the same challenges to concentration and memory. Of these 23 subjects over age 4, 13 had salient difficulties with concentration or memory:

- Mr. A (A1, age 32) rated his memory as 85% at baseline, 2% during exposure, and 10% six weeks after moving away.
- Mr. and Mrs. B (B1, B2, age 55 and 53) said their memories had partially recovered six weeks after moving.
- Mr. C (C1, now age 47), with continuing depression and ongoing exposure for house maintenance, noted 25 months after moving how bad his memory seemed.
- Mrs. C (C2, now age 44) felt she had recovered her memory and concentration 18 months after moving, despite ongoing stress from crowded living arrangements. Her affected son (now age 11, C7) had not completely recovered his school performance.
- Mrs. E (age 52) recovered immediately. She only experienced problems during exposure when the turbines were turned in a particular direction.
- Mr. and Mrs. F (F1, F2, ages 42 and 51) had moved away but still worked at their turbine-exposed home and farm during the day. Three months after they moved, both thought their concentration had improved, but not to baseline. Mr. F, with ongoing depression, did not perceive any memory recovery. I do not have information about their daughter's (F3, age 17) exam performance after moving.
- Mrs. G (G2, age 32) rated her memory as 10/10 at baseline, 2/10 during exposure, and 5/10 two months after moving away, at which point her depression was mostly resolved. Mrs. G's 5-year-old and 6-year-old children (G3, G4) showed marked improvements in concentration by two months after moving.

Only three subjects were clearly depressed during or after exposure. Mrs. G (G2, age 32) was becoming depressed at the time of the first (during exposure) interview. She remarked on the difference in her cognitive functioning between her current experience and a previous episode of depression at age 18, when she had no problem with her memory or concentration. Two other subjects, Mr. C (C1, age 45) and Mr. F (F1, age 42) developed depression after they had to abandon their homes, which was associated with prolonged memory difficulties. Both had ongoing exposure.

Irritability and anger. Twenty-eight subjects (fifteen male age 2-64 and thirteen female age 2-64) perceived themselves or were noted by parents to be more angry, irritable, easily frustrated, impatient, rude, defiant, or prone to outbursts or tantrums than at baseline. The adults were uniformly apologetic about their own irritability, and several described how careful they were to avoid acting irritable in their households. Three children (two boys age 8-9 and a girl age 5; C7, G3, G4) were markedly frustrated over homework. The young children of family G quarreled and had tantrums incessantly, and the six children/young adults in family C became angry, prickly, moody, defiant, or prone to fights at school. In families with children, the breakdown in children's behavior, social coping skills, and school performance was one of the strongest elements propelling them to move.

Fatigue and motivation. Twenty-one subjects felt or acted tired, and 24 had problems with motivation for usual, necessary, or formerly enjoyable activities (27 combined, fourteen male age 2-64 and thirteen female age 2-75). Like concentration and memory, these symptoms undoubtedly have a relationship with sleep deprivation, but certain subjects described leaden feelings around turbines that resolved as soon as they left the vicinity, such as Mr. A (A1, age 32), who said, "You feel different up there, draggy, worn out before you even start anything... it was a chore to walk across the yard." After driving an hour away to visit a family member, "I felt better all over, like you could do a cart wheel," and he felt well after moving.

When away from their turbine-exposed homes, most subjects recovered their baseline positive mood states, energy, and motivation immediately. Six adult subjects did not. These were Mr. B (B1, age 55), Mr. and Mrs. C (C1, C2, age 45 and 42), Mr. and Mrs. F (F1, F2, age 42 and 51), and Mrs. G (G2, age 32). By their own accounts, three (Mr. C, Mr. F, and Mrs. G) had unresolved or resolving depression. All but Mrs. G had ongoing anxiety and anger over abandoning their homes and their unresolved life situations.

Other symptom clusters and isolated problems

These symptoms and problems occurred in fewer subjects and typically require more than a medical history to diagnose. Several are exacerbations of pre-existing conditions with obvious connections to situations of high stress (cardiac arrhythmias, hypertension, irritable bowel, gastroesophageal reflux, glucose instability). Others are sequelae of core symptoms (auditory processing problems, unusual migraine aura). Others may indicate different kinds of direct effects of noise on body tissues, as in the vibroacoustic disease model of noise effects (respiratory infections, asthma, clotting abnormalities).[31]

Respiratory infection/inflammation cluster: Seven subjects had unusual or prolonged lower respiratory infections during exposure, and two of these also had prolonged asthma exacerbations (A2, B1, C2, E2, F1, F3, F4). Four subjects had unusually severe or prolonged middle ear problems (C7, F2, G3, G4).

Cardiovascular cluster: Two subjects had exacerbations of preexisting dysrhythmias (F1, J2). Two women had hypertension that increased during and after the exposure period, requiring medication after the end of the exposure period. Both still had considerable stress related to moving out and not being able to establish another regular home, and depressed husbands (C2, F2).

Gastrointestinal cluster. Four subjects had exacerbations of pre-existing gastroesophageal reflux (GER), ulcer, or irritable bowel, two with irritable bowel and upper gastrointestinal symptoms at the same time (D1, F1, F2, J2).

Arthralgia/arthritis/myalgia cluster. One healthy 32-year-old woman (G2) noted pain in one elbow while in her exposed house. It resolved when she went away for vacations with her family, and recurred when she returned. It resolved quickly when the family moved away, even though she did lots of lifting during

[31] Castelo Branco NAA , Alves-Pereira M. 2004. Vibroacoustic disease. Noise Health 6(23): 3-20.

motivation loss, disturbances to memory and concentration, and *visceral vibratory vestibular disturbance* (VVVD). Core symptoms are defined as common and widely described by study participants, closely linked in time and space to turbine exposure, and amenable to diagnosis by medical history. The latter was a particular requirement of this study. Wind turbine exposure is associated with other types of health problems, discussed in "Other symptom clusters and isolated problems," but different types of studies will be needed to determine if causal links exist for these other problems.

The most distinctive feature of Wind Turbine Syndrome is the group of symptoms I call *visceral vibratory vestibular disturbance*, or VVVD. The adults who experience this describe a feeling of internal pulsation, quivering, or jitteriness, accompanied by nervousness, anxiety, fear, a compulsion to flee or check the environment for safety, nausea, chest tightness, and tachycardia. The symptoms arise day or night, interrupting daytime activities and concentration, and interrupting sleep. Wakefulness is prolonged after this type of awakening. Subjects observe that their symptoms occur in association with specific types of turbine function: the turbines turned towards or away from them, running particularly fast, or making certain types of noise. The symptoms create aversive reactions to bedroom and house. Subjects tend to be irritable and frustrated, especially over the loss of their ability to rest and be revitalized at home. Subjects with VVVD are also prone to queasiness and loss of appetite even when the full set of symptoms is not present.

There is no statistical association in this study between VVVD and pre-existing panic episodes (which occurred in none of the subjects) or other mental health disorders, such as depression, anxiety, bipolar disorder, or posttraumatic stress disorder. There is a highly significant association between VVVD and pre-existing motion sensitivity ($p = 0.005$).

Headaches more frequent or severe than at baseline occurred in all migraineurs in the study, and all children with headaches in the study were migraineurs or the children of migraineurs. Non-migrainous adults also got severe headaches around turbines, and indeed about half the people with headache worse than at baseline (9 out of 19) were adults without history of migraine. Pre-exposure migraine is a significant risk factor for more severe or frequent headaches during turbine exposure ($p = 0.004$), but does not account for all the cases of headache.

Tinnitus occurred as a migraine aura in three subjects, but statistically in the study group tinnitus was not significantly associated with migraine, but rather with previous industrial noise exposure ($p = 0.013$), past history of tinnitus ($p = 0.017$), and baseline permanent hearing impairment ($p = 0.040$). I interpret tinnitus in these non-migrainous subjects as the direct impact of turbine noise on the cochlea, sensitized by previous inner ear damage from earlier noise exposures or chemotherapy.

Visceral vibratory vestibular disturbance (VVVD)

The work of Balaban and others on balance-anxiety linkages, both functional and neuroanatomic, provides a key to understanding the VVVD symptom set.[32,33,34,35,36] Citing recent work on the nature and

[32] Balaban CD, Yates BJ. 2004. The vestibuloautonomic interactions: a teleologic perspective. Chapter 7 in *The Vestibular System*, ed. SM Highstein, Fay RR, Popper AN, pp. 286-342. Springer-Verlag, New York.
[33] Balaban CD. 2002. Neural substrates linking balance control and anxiety. Physiology and Behavior 77: 469-75.

location of visceral detectors of gravity,[37] Balaban describes linkages among the vestibular system, autonomic and somatic sensory inflow and outflow, the fear and anxiety associated with vertigo or a sudden feeling of postural instability, and aversive learning.[38] These form a coordinated, neurologically integrated system based in the parabrachial nucleus and an associated neural network.[39,40] There are several critical elements here. First, there appear to be not three but four body systems for regulating balance, upright posture, and the sense of position and motion in space. The first three systems are the eyes, the semicircular canals and otolith organs of the inner ear, and somatic input from skin, skeletal muscles, tendons, and joints. The fourth system is visceral detection of gravity and acceleration (i.e., change in speed or direction of movement) by visceral graviceptors. These include stretch receptors in mesenteries or other connective tissue supporting organs or great vessels, and integrated systems of pressure detection in vessels and organs.[41] Such receptors have been found in the kidneys and in structures supporting the great vessels in the mediastinum, among other locations.[42] Von Gierke considers the inter-modality sensory conflict between the abdominal visceral graviceptors and the otolith organs to be a possible cause of motion sickness.[43]

The second critical element is central processing: how sensory information about motion and position is integrated by the brain, what other brain centers are activated, and what kinds of signals the system then sends back out to the body. Balaban and colleagues describe how the parabrachial nucleus network receives motion and position information from visual, vestibular, somatosensory, and visceral sensory input, and is linked to brain centers and circuits that mediate anxiety and fear, including serotonin and norepinephrine-bearing neurons from the midbrain and the amygdala, a key mediator of fear reactions.[44,45,46] Neurologically, fear or anxiety and a sense of balance or stability in space are closely connected.

Balaban illustrates with a story: he asks the reader to visualize waiting in traffic on a hill for a light to turn. Out of the corner of your eye you see the truck next to you starting to inch forward, and you jam your foot on the brake, since your sensory system has told you that you are starting to slip backwards. There's a bit of panic in that moment, quickly settled as you realize you are indeed stable in space and not moving. The story illustrates how a sensation of unexpected movement elicits alerting and fear. When a sense of movement is ongoing and cannot be integrated with the evidence of the other senses, as in

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[34] Furman JM, Balaban CD, Jacob RG. 2001. Interface between vestibular dysfunction and anxiety: more than just psychogenicity. Otol Neurotol 22(3): 426-7.
[35] Balaban CD. 2004. Projections from the parabrachial nucleus to the vestibular nuclei: potential substrates for autonomic and limbic influences on vestibular responses. Brain Res 996: 126-37.
[36] Halberstadt A, Balaban CD. 2003. Organization of projections from the raphe nuclei to the vestibular nuclei in rats. Neuroscience 120(2): 573-94.
[37] Mittelstaedt H. 1996. Somatic graviception. Biol Psychol 42(1-2): 53-74.
[38] Balaban and Yates 2004
[39] Balaban CD, Thayer JF. 2001. Neurological bases for balance-anxiety links. J Anx Disord 15: 53-79.
[40] Balaban 2002
[41] Balaban and Yates 2004
[42] Vaitl D, Mittelstaedt H, Baisch F. 2002. Shifts in blood volume alter the perception of posture: further evidence for somatic graviception. Int J Psychophysiol 44(1): 1-11.
[43] von Gierke HE, Parker DE. 1994. Differences in otolith and abdominal viscera graviceptor dynamics: implications for motion sickness and perceived body position. Aviat Space Environ Med 65(8): 747-51.
[44] Balaban and Thayer 2001
[45] Balaban 2002
[46] Halberstadt and Balaban 2003.

Low frequency noise can cause the human body to vibrate, as quantified by researchers in Japan.[54] The degree to which the body surface is induced to vibrate by low frequency noise is correlated with subjective unpleasantness.[55]

With this background, I hypothesize the following mechanism for VVVD. Air pressure fluctuations in the range of 4-8 Hz, which may be harmonics of the turbine blade passing frequency, may resonate (amplify) in the chest and be felt as vibrations or quivering of the diaphragm with its attached abdominal organ mass (liver). Slower air pressure fluctuations, which could be the blade passing frequencies themselves or a low harmonic (1-2 Hz), would be felt as pulsations, as opposed to the faster vibrations or quivering. (The vibrations or pressure fluctuations may also be occurring at different frequencies, without resonance amplification.) The pressure fluctuations in the chest could disturb visceral receptors, such as large vessel or pulmonary baroreceptors or mediastinal stretch receptors, which function as visceral graviceptors. These aberrant signals from the visceral graviceptors, not concordant with signals from the other parts of the motion-detecting system, have the potential to activate the integrated neural networks which link motion detection with somatic and autonomic outflow, emotional fear responses, and aversive learning. The people who are susceptible to responding in this way are those who in the past have become nauseated in response to other vertically oriented, anomalous environmental movements (seasickness or carsickness). Thus panic episodes with autonomic symptoms such as tachycardia and nausea arise during wakefulness or sleep in people with pre-existing motion sensitivity but without prior history of panic, anxiety, or other mental health disorders. Repeated triggering of these symptoms creates aversive learning, wherein the person begins to feel horror and dread of things associated with the physical sensations, such as his bedroom or house where he previously found comfort and regeneration.

VVVD was identified in the study in 14 out of 21 adult subjects. The behavior and experiences of other subjects, especially children, could be interpreted as partial manifestations of the same problem. For example, the two toddlers in the study, both aged 2½ (A3, G5), had night terrors. They awoke screaming multiple times per night, and were inconsolable and difficult to get back to sleep. The little girl (G5) would fight her mother, grabbing onto the posts of the bunk bed, to avoid going back into her own bed after awakening in this state. This shows clear parallels with the fear responses, prolonged awake periods, and aversive responses of the adults with VVVD. Both toddlers were agitated and irritable in the daytime, also similar to the adults in the study. Both 5-year-olds in the study, a boy and a girl (C7, G4), also frequently woke up fearful at night.

Other aspects of vestibular function

With regard to the other manifestations of wind turbine syndrome, I hypothesize that disrupted input from the other channels of the balance system may also play a role. Altogether, in subjects with or without VVVD, the turbine-associated symptom set resembles the symptoms of a balance or vestibular disorder, meaning malfunctioning of the inner ear balance organs (peripheral vestibular dysfunction) or of brain processing of balance-related neural signals (central balance dysfunction). For VVVD I proposed a

[54] Takahashi Y, Yonekawa Y, Kanada K, Maeda S. 1999. A pilot study on the human body vibration induced by low-frequency noise. Industrial Health 37: 28-35.
[55] Takahashi Y, Kanada K, Yonekawa Y, Harada N. 2005. A study on the relationship between subjective unpleasantness and body surface vibrations induced by high-level low-frequency pure tones. Industrial Health 43: 580-87, p. 580.

the house) show a high proportion with vestibular abnormalities, in some studies >80%, especially if patients have episodes of dizziness between panic attacks.[88,89,90,91]

If a person is already in a state of adaptation to ongoing vestibular or central balance deficits – even mild, fully compensated deficits – he or she is at particular risk for decompensation with exposure to new balance challenges. Many of the affected people in the present study, I suspect, were in this condition, their risks for mild baseline balance dysfunction including motion sensitivity, migraine disorder, prior damage to inner ear organs from industrial noise exposure or chemotherapy, autoimmune disease,[92] fibromyalgia,[93] normal aging (over 50), and normal early childhood (period of acquisition of walking and other upright and complex motor skills, as well as intense brain mapping of one's own body in space and the laws of the natural world; see below). Other potential risks for chronic vestibular dysfunction are whiplash injury and head injury, including concussions and milder head impacts without loss of consciousness.[94,95,96]

Cognition and vestibular function

It is now becoming apparent that a variety of cognitive functions depend on coherent vestibular signaling. Clinicians who work with balance-disordered patients are well acquainted with their struggles with short-term memory, concentration, multitasking, arithmetic, and reading.[97,98] In the perilymphatic fistula syndrome, for example, a form of inner ear pathology that can follow whiplash or minor head injuries, there are marked mental performance deficiencies compared to baseline functioning, associated with dizziness, headache, stiff neck, and sleep problems.[99] Cognitive symptoms are difficult to quantify in clinical practice and are often dismissed as psychological in origin.[100] Functional brain imaging by MRI or PET, however, has opened a window on the influence of vestibular function on human memory and thinking, by allowing examination of brain activation patterns during structured mental tasks or particular kinds of sensory stimulation.

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[88] Perna G, Dario A, Caldirola D, Stefania B, Cesarani A, Bellodi L. 2001. Panic disorder: the role of the balance system. J Psychiatr Res 35(5): 279-86.
[89] Jacob RG, Furman JM, Durrant JD, Turner SM. 1996. Panic, agoraphobia, and vestibular dysfunction. Am J Psychiatry 153(4): 503-12.
[90] Yardley L, Britton J, Lear S, Bird J, Luxon LM. 1995. Relationship between balance system function and agoraphobic avoidance. Behav Res Ther 33(4): 435-9.
[91] Yardley L, Luxon LM, Lear S, Britton J, Bird J. 1994. Vestibular and posturographic test results in people with symptoms of panic and agoraphobia. J Audiol Med 3: 58-65.
[92] Rinne T, Bronstein AM, Rudge P, Gresty MA, Luxon LM. 1998. Bilateral loss of vestibular function: clinical findings in 53 patients. J Neurol 245(6-7): 314-21.
[93] Rosenhall U, Johansson G, Orndahl G. 1996. Otoneurologic and audiologic findings in fibromyalgia. Scand J Rehabil Med 28(4): 225-32.
[94] Grimm RJ, Hemenway WG, Lebray PR, Black FO. 1989. The perilymph fistula syndrome defined in mild head trauma. Acta Otolaryngol Suppl 464: 1-40.
[95] Ernst A, Basta D, Seidl RO, Todt I, Scherer H, Clarke A. 2005. Management of posttraumatic vertigo. Otolaryngol Head Neck Surg 132(4): 554-8.
[96] Claussen CF, Claussen E. 1995. Neurootological contributions to the diagnostic follow-up after whiplash injuries. Acta Otolaryngol Suppl 520, Pt. 1: 53-6.
[97] Hanes and McCollum 2006
[98] Grimm et al. 1989
[99] Grimm et al. 1989
[100] Hanes and McCollum 2006

The vestibular system is ancient in the vertebrate lineage, and its connections ramify widely in both older and more recently evolved parts of the brain, including brainstem, midbrain, cerebellum, and occipital, parietal, and frontal cortex.[101] Vestibular injury causes specific deficits, but not general cognitive impairment.[102] Vestibular effects on cognition are often attributed to competing stimuli (i.e., challenges to movement and position sense draw attention away from cognitive tasks) but may also reflect direct dependence of certain cognitive operations on the vestibular system.[103]

Vestibular input is critical for spatial thinking, body and spatial awareness, spatial memory, and complex spatial or map calculations.[104] Dynamic, active vestibular signaling is needed for the acquisition, storage, and use of information with spatial components, such as building mental maps or deducing a novel path between two points.[105] Patients with 5-10 year histories of bilateral vestibular loss show marked deficits in a classic experimental task of spatial memory and navigation, accompanied, on average, by a 16.9% volume loss in the hippocampus (a temporal lobe structure essential for learning and memory).[106] In a test of general memory, however, these patients are no different from controls.[107] Vestibular signaling to the hippocampus is known to occur in both humans and non-human primates via a two-neuron linkage through the posterior thalamus or other proposed pathways.[108]

Disordered vestibular input increases error rates in purely mental tasks based on visualization of remembered objects, showing that coherent vestibular input is critical for thinking successfully and efficiently in spatial terms, even without using sight and beyond the period of memory storage. The tasks included detailed visualization, considered an occipital (visual) cortical task, and mental rotation, a parietal cortical task.[109] Vestibular stimulation in both humans and other primates activates a variety of areas in the parietal cortex, including a core vestibular processing area (posterior insula); the somatosensory strip; areas involved in hemineglect in stroke patients (ventral parietal); and a region "known to be involved in multimodal coordinate transformations and representation of space" (intraparietal sulcus), a principal site for arithmetic and counting tasks.[110] In hemineglect, patients after right-sided parietal strokes can have so much unawareness of the left side of space that they are oblivious to their own left-sided body parts being paralyzed, for example, or undressed. Vestibular stimulation temporarily corrects or improves this unawareness, in ways that suggest stimulation not only to general attention, but also to cerebral structures involved in the mental representation of space.[111,112] Vestibular stimulation also improves hemineglect patients' performance on tasks of visual localization and visual-spatial memory retrieval. At baseline, and again 24 hrs after stimulation, responses were biased away

[101] Dieterich and Brandt 2008
[102] Hanes and McCollum 2006
[103] Hanes and McCollum 2006
[104] Hanes and McCollum 2006
[105] Brandt T, Schautzer F, Hamilton DA, Bruning R, Markowitsch HJ, Kalla R, Darlington C, Smith P, Strupp M. 2005. Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. Brain 128: 2732-41.
[106] Brandt et al. 2005
[107] Brandt et al. 2005
[108] Brandt et al. 2005
[109] Mast FW, Merfeld DM, Kosslyn SM. 2006. Visual mental imagery during caloric vestibular stimulation. Neuropsychologia 44(1): 101–9. I wonder whether the detailed visualization task also included a parietal component, given the quantitative comparison the subjects had to make with the remembered image.
[110] Hanes and McCollum 2006, p. 82.
[111] Geminiani G, Bottini G. 1992. Mental representation and temporary recovery from unilateral neglect after vestibular stimulation. J Neurol Neurosurg Psychiatry 55(4): 332-3.
[112] Cappa S, Sterzi R, Vallar G, Bisiach E. 1987. Remission of hemineglect and anosognosia during vestibular stimulation. Neuropsychologia 25: 775-82.

from the left side, but this bias was corrected or ameliorated immediately after left vestibular stimulation.[113]

Studies of hemineglect patients have further shown that many mental operations are "spatialized" and dependent on parietal brain areas that have been lost, including mathematical operations involving a "mental number line" with lower numbers on the left,[114,115] clock representations of time,[116] and spelling at the beginnings (left) or ends (right) of words (errors occur opposite to the side of the parietal lesion).[117] In right-handed patients with right parietal strokes, there is no impairment to simple numeric calculation, a left-sided parietal function, but there is impairment to spatialized mathematical thinking, such as finding the midpoint between two numbers.[118] At the other extreme of mental functioning, great mathematicians think of numbers in spatial terms,[119] which "may be more efficient because it is grounded in the actual neural representation of numbers."[120] A recent study of outstanding human memorizers also shows that spatially oriented strategies are critical to good memory, providing an efficient framework for memory organization and retrieval.[121]

Coherent vestibular neural input is critical for spatialized forms of thinking and memory. Spatialized thinking and memory is intrinsic to many of the things we do with our minds, including mathematical thinking and memory organization as discussed above, but also the many forms of map-based or visually based problem-solving or short-term memory we do in everyday life. Spatial thinking is used, for example, in figuring out the most efficient path for a set of errands, remembering the path and images of the items to be obtained, searching for the items on a shelf, and judging if one was given the correct change. It is used for mental "maps" or calendars of one's day, week, or month and its appointments, to picture in three dimensions how to put something together, or to imagine what has gone wrong inside a device and initiate a repair. It is used for understanding the visual images in a movie or TV show, and for the sensitivity needed not to miss subtle visual clues. Disturbed vestibular signaling is also found experimentally to be a potent distractor from mental processing in general. In this context, it is easy to see how a situation of vestibular disturbance could impact concentration (which means the ability to perform thinking tasks successfully and efficiently) and memory. Vestibular disturbance may also affect reading directly via the direct reflex control exerted by semicircular canal and otolith organs over eye movements.

Effects on concentration and memory were nearly ubiquitous in this study, if one includes all subjects that told me about any problem in this area. For some subjects the deficits were dramatic relative to baseline, including the 7 out of 10 school-age children and teens who had a decline in their academic performance. Detrimental effects on concentration and memory were significantly associated with normal memory at baseline ($p = 0.027$) and with fatigue and loss of energy and motivation during exposure ($p = 0.018$).

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[113] Geminiani and Bottini 1992
[114] Zorzi M, Priftis K, Umilta C. 2002. nBrain damage: Neglect disrupts the mental number line. Nature 417: 138-9.
[115] Vuilleumier P, Ortigue S, Brugger P. 2004. The number space and neglect. Cortex 40(2): 399-410.
[116] Vuilleumier et al. 2004
[117] Hillis HE, Caramazza A. 1995. Spatially specific deficits in processing graphemic representations in reading and writing. Brain Lang 48 (3): 263-308.
[118] Zorzi et al. 2002
[119] Hadamard J. 1996. *The Mathematician's Mind: The Psychology of Invention in the Mathematical Field.* Princeton University Press, NJ. *In* Zorzi et al. 2002.
[120] Zorzi et al. 2002
[121] Maguire EA, Valentine ER, Wilding JM, Kapur N. 2003. Routes to remembering: the brains behind superior memory. Nat Neurosci 6(1): 90-5.

Though sleep deprivation undoubtedly plays a role, qualitative aspects of the mental performance deficiencies suggests a mechanism other than sleep deprivation alone. I propose that this mechanism is the effect of vestibular disturbance on cognition.

It is interesting here to examine a possible role of vestibular disturbance in the learning of very young children, in the toddler and preschool years. Mrs. G. (G2) volunteered that her 2½-year-old's (G5) irritability during turbine exposure was especially triggered by her older siblings' "unsteadying her" or coming so close that she thought she might be unsteadied. Children at this age are learning to keep their balance through a variety of different kinds of activities and postures. They are both fascinated and relaxed by vestibular stimulation (swinging, spinning, rolling, somersaults) and they actively explore the physical world through their play. The behavior of objects in gravity is another source of fascination, starting with babies' casting behavior and moving on to pouring water, sliding down slides, rolling things down inclines, building dams, floating toy boats, blowing bubbles, releasing helium balloons, etc. Vestibular input and processing plays a critical role in balance during movement; in the generation, storage, and use of internal maps; and in recognition of the behavior of objects under the influence of gravity. Indovina et al. measured brain activity by functional MRI while adult subjects watched the movement of simulated objects, finding that the vestibular network was selectively engaged when the acceleration of the object was consistent with natural gravity, even though the stimulus was only visual.[122] The authors use this as evidence that "predictive mechanisms of physical laws of motion are represented in the human brain"[123] under the influence of vestibular signaling of the vector of gravity. I suggest that these representations of the physical laws of motion are embedded in the human brain during early childhood as toddlers and children learn through experimentation (play) about the behavior of their bodies and other objects in gravity, and that coherent vestibular signaling is critical to this learning.

Environmental noise, learning, sleep, and health effects

Many studies have quantified the effects of environmental noise on children's learning. Reading acquisition, a language-intensive process, is especially sensitive to the effects of noise in school and at home. The effect is distinct from the effects of noise on attention or working memory,[124] and is correlated with measures of language processing such as speech recognition.[125] Airplane noise, which has a large low frequency component, has a stronger effect than traffic noise in some studies,[126] but traffic noise is also shown to have modest effects on memory in quieter communities.[127] Most studies are cross-sectional, but a prospective study, when an airport was closed in one location and opened in another,

[122] Indovina I, Maffei V, Bosco G, Zago M, Macaluso E, Lacquaniti F. 2005. Representation of visual gravitational motion in the human vestibular cortex. Science 308: 416-9.
[123] Indovina et al. 2005
[124] Haines MM, Stansfeld SA, Job RFS, Berglund B, Head J. 2001. A follow-up study of effects of chronic aircraft noise exposure on child stress responses and cognition. International Journal of Epidemiology 30: 839-45.
[125] Evans GW, Maxwell L. 1997. Chronic noise exposure and reading deficits: the mediating effects of language acquisition. Environment and Behavior 29(5): 638-56.
[126] Clark C, Martin R, van Kempen E, Alfred T, Head J, Davies HW, Haines MM, Barrio IL, Matheson M, Stansfeld SA. 2005. Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: the RANCH project. Am J Epidemiol 163: 27-37.
[127] Lercher P, Evans GW, Meis M. Ambient noise and cognitive processes among primary schoolchildren. Environment and Behavior 35(6): 725-35.

showed similar effects on reading acquisition longitudinally.[128] One study showed effects of noise on reading and auditory processing in children who lived in an apartment building next to a busy highway. The higher they lived in the building, the quieter were their apartments and the better their reading and auditory discrimination scores (e.g., distinguishing *goat* from *boat*). After controlling for parental education and income, the auditory discrimination scores largely explained the noise-reading linkage.[129] The effects on reading occur at sound levels far below those needed to produce hearing damage.[130] Children with pre-existing reading deficiencies and children at higher grade levels are more affected, and longer exposure produces larger deficits.[131]

suggestive
Effects ~~Ofggestive~~ of wind turbine noise impact on auditory discrimination or central auditory processing were found in the current study. During the period immediately after moving away from turbines and the cessation of her tinnitus, Mrs. A (A2, age 33) found she had a new difficulty understanding conversation in crowded, noisy places. Her son (A3, age 2½) began to confuse several consonant sounds during exposure, and continued to do so in the immediate post-exposure period.

Studies of adults in industrial settings show effects of noise on cognitive function when the noise is not considered loud and is nowhere near threshold for causing damage to hearing. Polish researchers exposed workers to 50 dB(A) broadband noise or 50 dB(A) broadband noise with low-frequency components (10-250 Hz) as they worked on standard psychological tests. Low-frequency noise impaired performance more than broadband noise without low-frequency components, especially in subjects who rated themselves as highly sensitive to low-frequency noise. There was no difference in the annoyance ratings for the two types of noise, nor evidence of either habituation or sensitization.[132]

Sleep deprivation is a primary focus of studies of community noise in general and was a major factor for the subjects in this study. The occurrence of VVVD contributes a distinctive quality to sleep disturbance and to the extent of sleep deprivation near wind turbines, since waking up in a physiologic state of panic leads to prolonged wakefulness or not returning to sleep at all. A second distinctive quality of wind turbine-associated sleep disturbance was nocturia (getting up repeatedly in the night to urinate), mostly in adult women, and nocturnal enuresis (bed-wetting) in a 5-year-old girl. Nocturia resolved immediately when subjects slept away from turbines. For the 5-year-old, the enuresis stopped during a family vacation, resumed on return home, and resolved fully when the family moved away. Studies of whole body vibration identify 10-18 Hz as frequencies likely to create the urge to urinate, [133] a possible mechanism for nocturia during exposure. Nocturnal enuresis may be a manifestation of the same direct vibratory stimulation in a child not yet developmentally ready to awaken to bladder signals, or it may instead be a parasomnia (like sleepwalking, sleep talking, and night terrors) that occurs during disordered

[128] Hygge S, Evans GW, Bullinger M. 2002. A prospective study of some effects of aircraft noise on cognitive performance in schoolchildren. Psychol Sci 13: 469-74.
[129] Cohen S, Glass DC, Singer JE. 1973. Apartment noise, auditory discrimination, and reading ability in children. Journal of Experimental Social Psychology 9: 407-22.
[130] Evans GW. 2006. Child development and the physical environment. Annu Rev Psychol 57: 423-51.
[131] Evans 2006, p. 426.
[132] Pawlaczyk-Luszczynska M, Dudarewicz A, Waszkowska M, Szymczak W, Sliwinska-Kowalska M. 2005. The impact of low-frequency noise on human mental performance. International Journal of Occupational Medicine and Environmental Health 18(2): 185-98.
[133] Rasmussen 1982

partial arousal from the deeper stages of sleep. Perilymphatic fistula syndrome, a vestibular disorder, includes nocturnal enuresis in adult women in its list of parasomnic manifestations.[134]

Noise at night is known to cause a variety of sleep disturbances, including delay of sleep onset, overt awakening, brief arousals seen on EEG, changes in length and timing of sleep stages, and premature final awakening. Short-term effects of noise during sleep include noise-induced body movements and modifications of autonomic functions such as heart rate, blood pressure, vasoconstriction, and respiratory rate. Noise-induced body movements indicate a low level of arousal from sleep, and occur with noise events as low as 32 dBA. Arousals detected by brain wave pattern on EEG occur with noise events as low as 35 dBA, and conscious awakenings with events of 42 dBA.[135]

Much of the extensive literature on community noise and sleep disturbance focuses on neuroendocrine changes in catecholamine and cortisol levels due to noise disturbance,[136] short-term changes in circulation, including blood pressure, heart rate, cardiac output, and vasoconstriction,[137,138] and the effects of long-term exposure on the risk of myocardial infarction.[139] There is a significant exposure-response relationship between exposure to nighttime aircraft noise, daily average road traffic noise, and hypertension.[140,141,142]

Most studies of sleep do not differentiate between low frequency and other types of noise, but there is a growing awareness of the particularly disturbing nature of the low-frequency components of community noise.[143] One study compared children sleeping with heavy trucks passing 2 m from the house walls every two minutes all night to children sleeping with traffic noise without the low frequency component. The low frequency noise-exposed children showed increased cortisol production during the first half of the night (an alteration in the normal circadian rhythm of secretion) compared to the other children.[144] Increased cortisol during the first half of the night was significantly related to restless sleep and difficulties in returning to sleep after awakening during the night.

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[134] Grimm et al. 1989

[135] Muzet A, Miedema H. 2005. Short-term effects of transportation noise on sleep with specific attention to mechanisms and possible health impact. Draft paper presented at the Third Meeting on Night Noise Guidelines, WHO European Center for Environment and Health, Lisbon, Portugal 26-28 April 2005. Pp. 5-7 in *Report on the Third Meeting on Night Noise Guidelines*, available at: http://www.euro.who.int/Document/NOH/3rd_NNG_final_rep_rev.pdf.

[136] 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.

[137] Babisch W. 2003. Stress hormones in the research on cardiovascular effects of noise. Noise Health 5(18): 1-11.

[138] Babisch W. 2005. Noise and health. Environmental Perspectives 113(1): A14-15.

[139] Babisch W, Beule B, Schust M, Kersten N, Ising H. 2005. Traffic noise and risk of myocardial infarction. Epidemiology 16(1): 33-40.

[140] Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, Dudley M-L, Savigny P, Seiffert I, Swart W, Breugelmans O, Bluhm G, Selander J, Haralabidis A, Dimakopoulou K, Sourtzi P, Velonakis M, Vigna-Taglianti F. 2008. Hypertension and exposure to noise near airports: the HYENA study. Environmental Health Perspectives 116(3): 329-33.

[141] Eriksson C, Rosenlund M, Pershagen G, Hilding A, Ostenson C-G, Bluhm G. 2007. Aircraft noise and incidence of hypertension. Epidemiology 18(6): 716-21.

[142] Haralabidis AS, Dimakopoulou K, Vigna-Taglianti F, Giampaolo M, Borgini A, Dudley M-L, Pershagen G, Bluhm G, Houthuijs D, Babisch W, Velonakis M, Katsouyanni K, Jarup L. 2008. Acute effects of night-time noise exposure on blood pressure in populations living near airports. European Heart Journal 29(5): 658-64.

[143] Persson Waye K. 2004. Effects of low frequency noise on sleep. Noise Health 6(23): 87-91.

[144] Ising H, Ising M. 2002. Chronic cortisol increases in the first half of the night caused by road traffic noise. Noise Health 4: 13-21.

Low-frequency noise

Birgitta Berglund, lead editor of the WHO *Guidelines for Community Noise,*[145] stated in a review of low-frequency noise effects: "Evidence suggests that a number of adverse effects of noise in general arise from exposure to low frequency noise: loudness judgments and annoyance reactions are sometimes reported to be greater for low-frequency noise than other noises for equal [A-weighted] sound-pressure level; annoyance is exacerbated by rattle or vibration induced by low-frequency noise; speech intelligibility may be reduced more by low-frequency noise than other noises except those in the frequency range of speech itself, because of the upward spread of masking... Low-frequency noise (infrasound included) is the superpower of the frequency range: it is attenuated less by walls and other structures; it can rattle walls and objects; it masks higher frequencies more than it is masked by them; it crosses great distances with little energy loss [from] atmospheric and ground attenuation; ear protection devices are much less effective against it; it is able to produce resonance in the human body; and it causes greater subjective reactions (in the laboratory and in the community studies) and to some extent physiological reactions in humans than mid- and high frequencies."[146] "Low-frequency noise also differs from other noise in producing vibrations of the human body and other objects… Rattle and vibration magnify reaction to the noise… Motion sickness has been linked to low-frequency noise even without accompanying vibration."[147]

Many subjects in the present study stated that turbine noise was different from other types of noise, using words like "invasive" and "unnatural," and saying that it was impossible to get used to this noise. Several said it wouldn't sound loud to people who did not live at their homes, or described a "swish" or "hum" as extremely bothersome noises. A number spoke favorably of living near heavily traveled roads or urban train lines, compared to living near wind turbines. All who moved, moved into villages, towns, or suburbs, where there was more traffic but no danger of turbines being built next to them. The descriptions make it clear that there is a disturbing quality about turbine noise which is more than its audible loudness.

In the present study, Mr. and Mrs. G described a resonance or standing wave phenomenon in one room of their turbine-exposed home. At one end of this room Mrs. G felt internal vibration, even though she could not feel any surfaces or objects vibrating when she put her hand on them. Mr. G felt peculiar in the same place, and always had to walk quickly away from that spot before his feeling progressed to nausea. In the home of family C, an audiologist detected vibration in the floor of a small room the family identified as having the worst problem in the home.[148]

At a NASA test facility in the 1960's, healthy young men were exposed to low frequency noise in the 1-50 Hz frequency range at 110 to 150 dB for 2-3 minutes (high amplitude and short duration). Over the full 1-50 Hz frequency range they experienced fatigue and took longer to perform assigned tasks. At frequencies less than 25 Hz there was an "annoying tickling" in the ear. In the same frequency range there were modulations of speech, moderate vibrations of the chest, and fullness in the hypopharynx with

[145] World Health Organization. 1999. *Guidelines for Community Noise*, ed. Berglund B, Lindvall T, Schwela DH. 159 pp. www.who.int/docstore/peh/noise/guidelines2.html
[146] Berglund B, Hassmen P, Job RFS. 1996. Sources and effects of low frequency noise. J Acoust Soc Am 99(5): 2985-3002.
[147] Berglund et al. 1996, p. 2993.
[148] Personal communication from acoustician; name withheld for confidentiality reasons.

an annoying gag sensation. "In regard to the opinions of those tested, it was indicated that the sensations involved were impressive."[149]

A case, similar to the cases presented in this paper, involved a couple in Germany in 1996. After moving into a new house outside a provincial city, the couple experienced symptoms with increasing intensity, including "indisposition, decrease in performance, sleep disturbance, headache, ear pressure, crawl parasthesy or shortness of breath." [150] Their case was intensely investigated with both A-weighted and linear measurements of noise indoors and outdoors, correlated in real time with the couple's symptoms. In time, the symptoms were correlated with intensity of noise below 10 Hz. The couple's symptoms and the intensity of noise below 10 Hz both varied with the wind and weather, and were worse in the winter. No plausible mechanism for production of such noises or correspondences to local sources of noise, such as the housing complex heating plant, was found. Symptoms occurred when the sound pressure level at 1 Hz was 65 dB, well below hearing threshold. None of the frequencies responsible for the symptoms, all below 10 Hz, had sound pressure levels above 80 dB. The decibel levels that affected the man and wife in their home were far less than their own threshold hearing levels measured in a sound lab. The authors hypothesized that infrasound with its very long wavelengths (10 Hz, for example, has a 34 m wavelength) causes strong pressure fluctuations in relatively small closed rooms, which are detected more by the whole body and its inner organs than by the ears.

Similar intensive investigations, using linear as well as A-weighted sound levels, 1/3 octave sound levels down to 1 Hz, indoor measurements, and assessments of wall vibration, have proved fruitful in other low-frequency noise complaint investigations.[151] These investigators, from a state environmental agency in Germany, paid attention to spontaneous statements by the affected people, to see whether perceptions of noise followed a systematic pattern. They found that "noises which in many cases induced vehement complaints were to a large extent of rather low sound levels,"[152] and that indoor ventilator noise and noises generated by structure-borne sound transmission were distinctly more disturbing than road traffic noise. These authors documented standing waves in rooms by measuring and comparing loudness in dB(A) and dB(lin) at the center of the room and near walls. They detected vibration in walls, and correlated the dominant frequency and its corresponding wavelength to the size of the room in discussing how a standing wave was established in the room.

For this kind of complaint, the authors noted,

> More than half …were made on the grounds of sleep disturbance. Quite often symptoms like "a roaring in the head, especially when lying down" were brought forward. Time and again, "a feeling of riding a lift [elevator]" was reported, and over and again the measuring team had the impression that the reported immissions [noise] meant a nerve-wracking experience for the exposed persons. Several complainants even got into a state of being aggressive. There were reports by a number of trustworthy persons on how they at first – for instance when moving into the flat – did not even notice any immissions. But in the course of a few weeks

[149] Edge PM, Mayes WH. 1966. Description of Langley low-frequency noise facility and study of human response to noise frequencies below 50 cps. NASA Technical Note, NASA TN D-3204. 11 pp.
[150] Feldmann J, Pitten FA. 2004. Effects of low-frequency noise on man – a case study. Noise Health 7(25): 23-8.
[151] Findeis H, Peters E. 2004. Disturbing effects of low-frequency sound immissions and vibrations in residential buildings. Noise Health 6(23): 29-35.
[152] Findeis and Peters 2004, p. 29.

> they began to perceive them distinctly and became intolerable after continued exposure. It was obvious that in these cases the sensibility of specific noise components had developed. Thus, it is understandable that non-exposed persons were at a difficulty to even acknowledge such noise immissions.[153]

Wind turbines produce noise in the low and infrasonic frequency ranges. The issue has not been whether they produce low frequency or infrasonic noise, but whether the amplitudes are sufficient to cause human effects. According to data published by van den Berg,[154] unweighted amplitudes at 1 Hz, at one wind park under one set of weather conditions, were in the 70-100 dB range, declining to the 55-75 dB range at 10 Hz and the 50-60 dB range at 100 Hz. Wind turbine noise has a pulsating quality, produced as the airfoil blades swing past the tower, compressing the air between blade and tower. These low-frequency pressure fluctuations, among other effects, modify the loudness of the higher-frequency sounds coming from the turbines, producing the audible "swish" that synchronizes, for example, with the feeling of pulsation some subjects felt in their chests. Coming from several towers at once, these low frequency air pressure fluctuations may synchronize and reinforce, depending on the orientation of the towers, the position of the house relative to the towers, and the timing of the individual turbines. Three families in this study (A, B, and F) lived in houses nearly in line with a row of turbines. For families A and B, the area's worst storms, "nor'easters," swept right down the line towards their houses, which were on a hill at the level of the turbine hubs. These two families, though they were a kilometer (about 3300 feet) from the closest of the 10 turbines, moved out faster – in five months – than any of the other families, and had particularly severe symptoms.

Studies of turbine noise also show that noise carries further than predicted by conventional industry modeling. This has to do not only with the low-frequency components of the noise, which attenuate less with distance, but also with the layering of the atmosphere at night, creating cool still air at ground level and brisk, laminar airflow at turbine hub heights.[155] Industry models also lack sophistication in other ways, such as not taking into account that the noise source is higher than 30 m, increased transmission of sound in front of and behind the blades with less sound transmission in the plane of the blades (including under the turbines), sky reflections, and weather conditions that focus the noise transmissions.[156]

Vibroacoustic Disease (VAD) model

High intensities of low-frequency noise over prolonged time periods may cause marked neurologic damage, as described by the Vibroacoustic Disease (VAD) group in Portugal.[157] This is a provocative body of research, full of interesting case descriptions and pathology studies, but plagued by absence of specified study group criteria, absence of control groups, and lack of quantification. The study group consists of 140 aircraft maintenance and repair technicians in the Portuguese Air Force, of whom 22 (15.7%) had adult-onset epilepsy, compared to a national prevalence of 0.2%.[158] Some of the case descriptions of the subjects with epilepsy also include cognitive decline, depression, paranoia, and rage

[153] Findeis and Peters 2004, p. 32.
[154] van den Berg 2004a
[155] van den Berg 2004b
[156] Richard James, INCE, personal communication, 5/11/08.
[157] Castelo Branco NAA , Alves-Pereira M. 2004. Vibroacoustic disease. Noise Health 6(23): 3-20.
[158] Castelo Branco and Alves-Pereira 2004

attacks.[159] The descriptions are similar to those of retired professional football players with histories of multiple concussions.[160,161] The vibroacoustic disease researchers ascribe VAD pathology to whole body vibration induced by the noise, the pathology of each body part induced by vibration of that part. Neurologic effects may be due to neuronal or axonal shearing, as in the multiple concussions scenario, or due to microangiopathy in the brain, meaning effects on/occlusion of small blood vessels.[162]

With regard to the chest, the VAD researchers have used human autopsy and biopsy and animal rearing studies to describe loss of cilia and microvilli from epithelial surfaces of the bronchi,[163,164,165] pleura,[166] and pericardium.[167] They also describe thickening of bronchial epithelial basement membrane,[168] pericardium,[169] and blood vessel walls[170] by extra, organized collagen and elastin. Several of the animal rearing studies on bronchial epithelial changes are well controlled and convincing.[171,172]

Based on the vibroacoustic disease research, I hypothesize that vibratory or pulsating air pressure fluctuations in subjects' airways in the present study may induce shearing of surface cilia, thus impairing the clearance of mucus and particulates from airways. This in turn could make subjects more susceptible to increased airway irritation and reactivity (asthma), decreased clearance of the mucus produced in asthma, and infection. The Eustachian tube and middle ear could be susceptible to the same process, leading to prolonged middle ear effusions and unusual acute infections.

Community noise studies and *annoyance*

Studies of community noise frequently assess a quality called *annoyance*. "Apart from 'annoyance,'" the World Health Organization writes, "people may feel a variety of negative emotions when exposed to community noise, and may report anger, disappointment, dissatisfaction, withdrawal, helplessness,

[159] Martinho Pimenta AJ, Castelo Branco NAA. 1999. Neurological aspects of vibroacoustic disease. Aviat Space Environ Med 70(3): A91-5.
[160] Omalu BI, DeKosky ST, Minster RL, Kamboh MI, Hamilton RL, Wecht CH. 2005. Chronic traumatic encephalopathy in a National Football League player. Neurosurgery 57: 128-34.
[161] Omalu BI, DeKosky ST, Hamilton RL, Minster RL, Kamboh MI, Shakir AM, Wecht CH. 2006. Chronic traumatic encephalopathy in a National Football League player: part II. Neurosurgery 59: 1086-93.
[162] Martinho Pimenta AJ, Castelo Branco NAA. 1999. Neurological aspects of vibroacoustic disease. Aviat Space Environ Med 70(3): A91-5.
[163] Oliveira MJR, Pereira AS, Ferreira PG, Guinaraes L, Freitas D, Carvalho APO, Grande NR, Aguas AP. 2004. Arrest in ciliated cell expansion on the bronchial lining of adult rats caused by chronic exposure to industrial noise. Environ Res 97: 282-6.
[164] Oliveira MJR, Pereira AS, Castelo Branco NAA, Grande NR, Aguas AP. 2002. In utero and postnatal exposure of Wistar rafts to low-frequency/high intensity noise depletes the tracheal epithelium of ciliated cells. Lung 179: 225-32.
[165] Monteiro M, Ferreira JR, Alves-Pereira M, Castelo Branco NAA. 2007. Bronchoscopy in vibroacoustic disease I – "pink lesions." Inter-Noise 2007, 28-31 August 2007, Istanbul, Turkey.
[166] Pereira AS, Grande NR, Monteiro E, Castelo Branco MSN, Castelo Branco NAA. 1999. Morphofunctional study of rat pleural mesothelial cells exposed to low frequency noise. Aviat Space Environ Med 70(3): A78-85.
[167] Castelo Branco NAA, Aguas AP, Pereira AS, Monteiro E, Fragata JIG, Tavares F, Grande NR. 1999. The human pericardium in vibroacoustic disease. Aviat Space Environ Med 70(3): A54-62.
[168] Castelo Branco NAA, Monteiro M, Ferreira JR, Monteiro E, Alves-Pereira M,. 2007. Bronchoscopy in vibroacoustic disease III – electron microscopy. Inter-Noise 2007, 28-31 August 2007, Istanbul, Turkey.
[169] Castelo Branco et al. 1999
[170] Castelo Branco NAA. 1999. A unique case of vibroacoustic disease: a tribute to an extraordinary patient. Aviat Space Environ Med 70(3): A27-31.
[171] Oliveira et al. 2004
[172] Oliveira et al. 2002

depression, anxiety, distraction, agitation, or exhaustion."[173] Beyond even these negative emotions, moving out of an owned home indicates that people feel sick and unable to cope, and that they judge their survival and well-being will be enhanced by moving out even though they exhaust limited resources to do so, in the face of being unable to recoup the value of their major asset, their home.

Pedersen and Persson Waye assessed *annoyance* (a shorthand for the above negative emotions) among 351 households near wind turbines in Sweden in 2000, using a survey and comparing *annoyance* to modeled A-weighted sound pressure levels they calculated to exist outside homes near clusters of one to five turbines of power 0.15 to 0.65 MW, based on the homes' distances from turbines.[174] They found people to be *highly annoyed* by wind turbine noise at sound pressure levels much lower than for other types of community noise. The A-weighted decibel level (in a measure averaged and weighted over time, L_{eq}) that corresponded to 15% of the people being *highly annoyed* by the noise was 38 dB(A) for wind turbines, 57 dB(A) for aircraft, 63 dB(A) for road traffic, and 70 dB(A) for railways. The curve for annoyance due to wind turbine noise had a steep slope, so that by 41 dB(A), 35% of people were *highly annoyed*. Sixteen percent of respondents over 35 dB(A) reported that their sleep was disturbed by wind turbine noise.

In a continuation study that involved interviewing participants, Pedersen found that some people had moved out of their homes, rebuilt their homes, or instituted legal proceedings because of problems associated with turbine exposure.[175] Pedersen and Persson Waye also detected informants who were sensitive to both noise and blade motion, felt violated or invaded by turbine noise, and found their houses to be places where they could no longer find restoration.[176]

Van den Berg, Pedersen, and others in 2007 surveyed 725 households in the Netherlands located up to 2.1 km (1.3 mi) of at least two turbines of power 0.5 to 3 MW.[177] Their questionnaire asked about visual and noise perception, annoyance, background health, psychological stress, sleep disturbance, and demographics. Owners of turbines, who lived the closest and by modeling were exposed to the loudest noise, were able to turn turbines off if they or their neighbors were bothered by the noise, a key difference between the Netherlands and the situation in the various countries in the present study. As a group, the people who lived closer to turbines were younger, healthier, and better educated. They tended to be farmers and to benefit economically from the turbines. Levels of modeled noise, degree of sleep disturbance, annoyance, and questionnaire measures of stress were positively correlated among people who did not receive economic benefit. Annoyance occurred at lower dBA levels than for road, rail, or air traffic noise, as in the Swedish study discussed above. Being awakened from sleep, difficulty falling asleep, and higher stress scores were associated with annoyance at wind turbine sound.

[173] World Health Organization 1999, *Guidelines for Community Noise*, p. 50.
[174] Pedersen E, Persson Waye K. 2004. Perceptions and annoyance due to wind turbine noise – a dose-response relationship. J Acoust Soc Am 116(6): 3460-70.
[175] Pedersen 2007
[176] Pedersen and Persson Waye 2007
[177] van den Berg et al. 2008b

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Family Table A1 (page 1 of 2)

Note: These are sample, manuscript pages from the tables presented in the report. Be aware that there are gaps between pages, pending publication of the book.

Person
Mr. A
Age
32
Pre-exposure health status
Good
Health history
No significant
Previous noise exposure
Diesel fishing boat from childhood
Time to onset of symptoms
Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good but always easily awakened by noise.	"I didn't really." Hard to fall asleep. Frequent awakening due to child's frequent awakening.	Good, at baseline. Child sleeping through night.
Headache	Rare, mild	Continuous headache at home which resolved after several hours away and resumed several hours after return, with onset 3 wks into turbine start-up process. OTC and prescription analgesics, addition of glasses not helpful.	Resolved
Cognition	Normal. Runs own fishing business. Mild difficulty with memory, especially for names and faces.	Memory problems: "You'd think I was 99." When arriving at a store or storage building, could not remember what he had come to get without a list.	Partial recovery: self-rated memory 80-85% at baseline, 2% during exposure, and 10% at 6 weeks after moving
Mood	Good. Usually does not show annoyance.	Loss of usual energy and enjoyment for spring fishing season. Mildly irritable.	Anger about home abandonment, otherwise resolved.
Balance / equilibrium	Normal, never carsick or seasick	"A little shaky on feet every now and then" at home.	Resolved

Family Table A1 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Ear / hearing	Mild subjective hearing loss attributed to diesel engine exposure, no tinnitus.	Repetitive popping in ears for first 3 weeks. Tinnitus started several weeks after headache onset and worsened over time.	Resolved
Eye / vision	Normal without glasses	Burning sensation in eyes. When headache and tinnitus were severe, eyes "felt like they were going to fall out on the table if you looked down." Had normal eye exam.	Resolved
Other neurological	Normal except mild concussion age 14	No change	No change
Cardiovascular	Normal including BP (110-120/80 in 2006)	Mild diastolic hypertension on one reading (128/94 on 4/4/07)	No further BP measurements obtained.
Gastrointestinal	Normal	Nausea when headache was severe. No vomiting or other gastrointestinal changes.	Resolved
Respiratory	Normal except smokes	No change	No change
Other		Symptoms were present in all wind directions, better during rain, and worse with wind from direction of turbines or from the 180 degree opposite direction.	
		"You feel different up there, draggy, worn out before you even start anything." "It was a chore to walk across the yard."	When visiting family 100 km away, "I felt better all over, like you could do a cartwheel." Feels well at new house.

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Family Table B1 (page 1 of 2)

Person
Mr. B
Age
55
Pre-exposure health status
Good
Health history
Surgery 4 times for benign prostatic hypertrophy, once for hand injury
Previous noise exposure
Diesel fishing boat since childhood
Time to onset of symptoms
Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good	Delayed onset and repeated awakenings; prescribed sleep aid.	Resolved
Headache	Rare, mild	Continuous, head and ears "sizzling." "It got in your head and would dang well stay there." Started "at back of head, then down sides, then affected right eye." Prescription and non-prescription analgesics minimally helpful.	At baseline
Cognition	Normal	"Trouble remembering;" "a little problem concentrating" blamed on sleep deprivation	"Pretty good, a little problem still."
Mood	Good	Stress, "lots, pretty near more'n I could take, it just burnt me, the noise and run-around"; prescribed anxiolytic.	Improved, still takes some anxiolytic.
Balance / equilibrium	Normal, never seasick or carsick, no vertigo.	Wobbly, staggering, off-balance "like had drunk." No falls. Occasionally felt dizzy.	Resolved, on roof shingling without problems.
Ear / hearing	Normal hearing on left and mild sensorineural loss at 4kHz on right in 2006. Intermittent left tinnitus since 2005.	Tinnitus continuous and bothersome, "ringing and sizzling," and interfering with conversation comprehension. Ears popped "like an airplane." Ear wax increased.	Resolved
Eye / vision	Normal with reading glasses.	Intermittent right eye pain "like a force on it, like pressure on the eye, the inside part, in the head." No change in vision. Eye pain/pressure synchronous with headache.	Resolved

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Family Table B1 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Other neurological	Normal, no concussion	No change	No change
Cardiovascular	Normal with BP 126/82, 126/88, 112/70 in 2006	Mild BP elevation 140/80, 132/90, 152/92. After started anxiolytic, BP 128/84.	Resolved, BP 110/68
Gastrointestinal	Normal, no GER, not prone to nausea.	Frequent nausea.	Resolved
Respiratory	Slight asthma as child. Never smoked.	Two episodes of feeling of weight on chest while lying on couch, which resolved when he stood up. Lower respiratory infection in 5th month of exposure.	Normal
Rheumatologic	Osteoarthritis	No change	No change
Other	Little road traffic or other noise	"That stuff [turbine noise] doesn't get out of your head, it gets in there and just sits there - it's horrible."	Not bothered by "all kinds of traffic" at new location; "after a while you don't hear it."
		He felt pulsation in ears and chest when there was fog in the valley between the turbines and the house and he was outside.	
		Hum heard and felt in double glazed picture window when turbines running.	
		Spent more time at shore at boat, away from house and property, for symptom relief.	

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Family Table B2 (page 1 of 2)

Person
Mrs. B
Age
53
Pre-exposure health status
Good
Health history
Hysterectomy and cholecystectomy, 4 births
Previous noise exposure
Diesel fishing boat intermittently for decades
Time to onset of symptoms
Several weeks, with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good	Delayed onset, repeated awakening, difficulty going back to sleep, nocturia. Ear plugs somewhat helpful.	Resolved
Headache	Rare, mild	Continuous except when left property or wind in favorable direction.	Resolved
Cognition	Normal	Concentration disturbed; confused if went on errands without list, had to return home.	Partly resolved at 6 wks, up to remembering three things without a list.
Mood	Good, hard worker, not moody.	Anxiety, guarding against irritability, upset and "in a turmoil" when symptoms worse.	Resolved
Balance / equilibrium	Normal, never carsick or seasick.	Some unsteadiness and gait change.	Resolved
Ear / hearing	Normal hearing test in 2005, no tinnitus.	Tinnitus and ear pain continuous except when left property or wind in favorable direction. Ear irrigation at clinic worsened tinnitus.	Resolved
Eye / vision	Normal with glasses	Eyes irritated, burning, runny. Ebb and flow of eye symptoms synchronous with headache and tinnitus.	Burning resolved but visual blurring noted when chemotherapy started.
Other neurological	Normal, no concussion	No change	No change
Cardiovascular	Normal including BP	Mild BP elevations 132-140/80-90	Unknown
Gastrointestinal	GER and post-tussive vomiting.	No change	Worsened with chemotherapy

Family Table B2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Respiratory	Chronic cough secondary to GER and smoking.	Breath "short every once in a while, like [while] falling asleep, breathing wanted to catch up with something, hard to explain."	Resolved, normal breathing pattern.
Oncologic	Felt well though had undiagnosed breast cancer.	Breast cancer diagnosed. Mastectomy 4 wks before end of exposure.	Chemotherapy started.
Other		Left house repeatedly to get relief of symptoms, interrupting work and tasks.	Resolved

Machines	Refrigerator quiet	Refrigerator became loud and was replaced, but new one was also loud.	New refrigerator was moved to new house and is quiet.
	Furnace quiet	Furnace became loud. Circulator was replaced and the furnace was still loud.	

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Family Table C4 (page 1 of 2)

Person
Second son C
Age
19
Pre-exposure health status
Good, strong and athletic
Health history
Migraines with vomiting as older child and teen; pneumonia once; mononucleosis
Previous noise exposure
Diesel fishing boat for several years
Time to onset of symptoms
Several months

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Sound sleeper with some sleep walking and talking, hard to arouse	Harder to get to sleep when he could hear turbines	Resolved
Headache	Headache with nausea and dizziness for first 2 days of each fishing trip	Occasional headache with tinnitus and dizziness on awakening	At baseline
Cognition	Normal, left school for fishing age 17; difficulty with memorization	Distracted by shadow flicker when present	Resolved
Mood	Easy-going, "jokey"	"Prickly," irritable	Resolved
Balance / equilibrium	Seasick and carsick as child with persistent symptoms on fishing trips	Occasional dizziness on awakening, as above	At baseline
Ear / hearing	Occasional tinnitus and headache from motor noise on fishing trips	Occasional tinnitus on awakening, as above	At baseline
Eye / vision	Normal, with acute peripheral and distance vision	In final month, intermittent flashes of light, then blurring, in one eye at a time at any time of day, with recovery; evolved to transient blindness (amaurosis fugax) lasting 30 sec-2 min, repetitively in each eye, right more than left; not associated with headache or tinnitus.	Persistent at 8 months and resolved at 12 months with normal vision.
Other neurological	Normal, h/o one concussion in hockey	After first few months, hard to move legs for first 2-5 minutes after awakening, then normal; not numb; occasional bilateral tingling around knees; knees buckled unexpectedly in daytime.	Resolved on same schedule as eye problems.
Cardiovascular	Normal including BP	No change	No change

Family Table C4 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Gastrointestinal	Normal except nausea with migraines	No change	No change
Respiratory	Normal, no asthma, smoked briefly in past	Occasionally felt pulsation in chest	Occasional difficulty taking deep breath at rest
Rheumatologic	Back injury in hockey years before with some residual pain	Exacerbation of back pain	At baseline
Other	Hand and foot eczema	Exacerbation of eczema	At baseline
		Slept in basement with fan on because of turbine noise	

* Exposure period 15 months to all turbines, 21 months to at least 2 operating turbines. Exposure mostly limited to nighttime in windowless basement bedroom. Interviewed 21 months after move. Information also provided by parents 2 weeks before move and 8, 12, 18, and 21 months after move.

** Moved away from immediate family when family left home.

Family Table D1 (page 1 of 3)

Person
Mr. D
Age
64
Pre-exposure health status
Disabled due to injury to back and neck in industrial accident, without paralysis
Health history
Ulcer age 61; current medications Tylenol #3, omeprazole, docuset, senecot, lovastatin
Previous noise exposure
Heavy industry age 16-37, including weaving mills, turbine and jet engine production
Time to onset of symptoms
Sleep disturbance immediate. Palpitations/tremors by 4-6 wks. Retinal stroke at 11 weeks. Diarrhea and GI bleeding by 4 months.

	Pre-exposure	During exposure*	Post-exposure**
Sleep	No sleep problems. One Tylenol with codeine at bedtime for back pain. Did not awaken or get up to urinate until morning.	Feels pulsation as soon as he lies down in bed. Frequent awakening, 6-12 per night. Nocturia 2-3 per night. "The worst sleep you ever heard of, up half the night." Gets to sleep using self-hypnosis he was taught for pain (counting backwards), but has to start at a higher number and count longer.	Sleeps well away from home, without nocturia.
Headache	Rare/mild. No migraine or sinus problems.	Not headache, not painful, but a "kind of numbness which sets over the head" [see below, Balance/equilibrium]	Does not occur away from home.
Cognition	Concentration and memory good. 2-year college degree in industrial engineering.	More difficulty remembering what he reads. In last 2-3 months "I notice a little more each time." "Once I had real fast recall, but now I have to think about things."	No information
Mood	No depression, anxiety, panic, or anger problems.	Frequent need to “calm down.” Angry, including in night when awakened. "I can get real aggressive now and I never used to. If something doesn't go my way, I get real flustered, and then start with that nervousness and I have to go calm myself down." Irritable. Anxious about his own and wife's health and well-being.	When away for weekend, "you get all relaxed and all of a sudden you're back in the same thing again." "Getting away calms you down."

Family Table D1 (page 3 of 3)

	Pre-exposure	During exposure*	Post-exposure**
Rheumatologic	Persistent neck and back pain due to injury at age 37. Two Tylenol with codeine daily, rarely more. No other joint problems.	No change.	No change
Other	Spent his time outside with ponies and traveled to Florida with wife for 6 weeks in winter.	"Now I don't go outside at all." At f/u interview, the couple had not taken their next winter trip to Florida because of Mr. D's health problems.	No information.
		"When turbines get into a particular position (facing me), I get real nervous, almost like tremors going through your body…it's more like a vibration from outside…your whole body feels it, as if something was vibrating me, like sitting in a vibrating chair but my body's not moving." Occurs day or night, but not if the turbines are facing "off to the side." If outside, "I come in, sit down in my chair and try to calm myself down. After an episode like that, I'm real tired."	Does not occur away from home.
		Two months of static electric charge in yard: hair on arms would stand up when he stood in a certain area.	Static charge resolved.
		F/u interview: had bought his own sound meter, registers 50-70 dB all the time.	
Animals	Ponies well trained for riding, jumping, and pulling cart.	Riding pony refused to leave barn, go up road, or go in field over jumps. Cart pony broke into sweats, trembled, ran uncontrolled through gates and fences with cart and harness attached. Both ponies were sold 8 wks into exposure period.	No information.
	Dog had 4 litters previously and did well.	Puppies 3 days old: mother had killed one large healthy puppy; she was staying with puppies and tolerating nursing but not licking or caring for pups.	No information.

*Exposure period 6 months by first interview and 16 months at f/u interview. Information is from first interview unless otherwise noted.

**Had purchased second house but not yet moved at f/u interview; away only for weekends or short trips.

Family Table F1 (page 1 of 2)

Person
Mr. F
Age
42
Pre-exposure health status
Good
Health history
No significant
Previous noise exposure
Farm equipment exposure since youth; uses hearing protection consistently
Time to onset of symptoms
3 days for sleep disturbance; 3 months for memory deficits

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Rare insomnia when worried.	Frequent abrupt awakening, focusing on noise, no sleep past 4 a.m. Tired and "feeling beat up" in morning. Prescribed anxiolytic.	Improved, still requires occasional anxiolytic.
Headache	As teen, occipital headaches triggered by studying. No severe headaches in 20 years.	2-3 per week, not all day, increased OTC analgesic use.	1-2 per week.
Cognition	Good; BSc and registered instructor in agronomy.	Memory deficits noted from 3 months into exposure, “frustrating at times;” noise draws attention at night; concentration problems attributed to poor sleep and the lack of resolution of problem	Concentration improved with improved sleep but memory still decreased; has ongoing depression.
Mood	Intermittent anxiety and depression since age 14, never medicated.	Depression, frustration, annoyance, anger. Unable to accomplish daily tasks.	Improved, not resolved; has more enthusiasm for doing things.
Balance / equilibrium	Slight carsickness in back seat or if reading in car. No h/o vertigo.	Occasionally off balance but not interfering with functioning.	Persists only during ongoing exposure.
Ears / hearing	"Reasonably keen hearing for age." No tinnitus.	Irritation and rumbling in ears with sensations of blowing in ears, of eardrum "moving without hearing it," and hearing noise "in center of head."	Improved with less exposure.
Eyes / vision	Normal, no glasses.	No change	No change
Other neurological	Normal, no concussion.	No change	No change
Cardiovascular	Infrequent tachycardia of short duration (5 sec), diagnosed at age 15.	Weekly episodes of tachycardia of increased duration, longest 15 minutes.	Frequency still increased.

Family Table F1 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Gastrointestinal	Intermittent GER and irritable bowel symptoms.	Increased frequency and intensity of GER and irritable bowel symptoms.	Unresolved.
Respiratory	Mild wheezing with URIs began about 6 yrs before, no medication prescribed.	Pneumonia and asthma diagnosed 6 weeks into exposure; thereafter, persistent wheezing requiring use of bronchodilator about twice a week.	Persistent opacity on chest x-ray and semiweekly wheezing.
Rheumatologic	Intermittent knee arthralgia since age 11 related to overuse.	No change	No change
Other		Detected indoor vibration/hum more after double-glazed windows installed in attempt to exclude noise.	

*Exposure period 7 months until rented “sleeping house” and 12 months until rented “sleeping and living house.”

**Ongoing exposure up to 8 hours per day while farming land and using farm office in home. Interviewed 3 months after second exposure reduction.

Family Table G1 (page 1 of 2)

Person
Mr. G
Age
32
Pre-exposure health status
Good
Health history
Chronic bilateral serous otitis media and conductive hearing loss treated with tubes at ages 7 and 9
Previous noise exposure
Ongoing exposure to airplane and train noise while commuting
Time to onset of symptoms
Noticed gradually

	Pre-exposure	During exposure*	Post-exposure
Sleep	Good	Delayed onset and increased awakening due to noise. Uses ear plugs.	Not disturbed by urban rail line outside window during work week. Slept well when away with family and after move.
Headache	A few bad headaches in life, not identified as migraines	If awakened by turbine noise, has headache at time of awakening and in morning	No headaches
Cognition	Good; computer programmer; long work days and commute	Tired at work, “concentration lacking in afternoons”	At baseline with same commute; tired but concentration fine
Mood	Good	Finds noise outside or noise which awakens him at night stressful. Worried about wife and family.	Feels more relaxed; situation resolved; wife and children all happier; home with family every day.
Balance / equilibrium	Always seasick; carsick if in back seat. Vertigo for a few wks at age 29.	Episodes of dizziness "like being spun fast in a circle"; disorientation/feeling "very strange" in certain parts of house at certain times where he can "feel rumbling."	Did not occur away from exposed home, when turbines off, or after moving

Family Table H2 (page 1 of 2)

Person
Mrs. H
Age
57
Pre-exposure health status
Lupus with arthritis and normal renal function; takes quinine; also diagnosed with fibromyalgia
Health history
Hysterectomy and lipoma removal
Previous noise exposure
Urban life to age 54
Time to onset of symptoms
Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Slept well through night	Delayed onset and frequent awakening, 5-6 per night; awakens with sense of fear, compulsion to check house, "very disturbed sort of waking up, you jolt awake, like someone has broken a pane of glass to get into the house; you know what it is but you've got to check it -- go open the front door -- it's horrific"; nocturia; unable to go back to sleep	Slept well through night
Headache	Rare, mild	Headaches continuous unless turbines off; takes analgesics only when headache very bad in night.	No headaches while away
Cognition	Concentration/memory problems when first diagnosed with lupus, then improved	Concentration/memory slightly worse, writing herself more reminders	At pre-exposure baseline
Mood	No h/o mood problem or anxiety	Irritable and angry, shouting more	Improved while away
Balance / equilibrium	Never carsick or seasick; 4 episodes of vertigo, all 6 or more yrs before exposure	10-20 min episodes of dizziness, sometimes with nausea	Did not occur while away

Family Table H2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Ear / hearing	Tinnitus and hearing loss	Ongoing tinnitus and 3 incidents, each 1 hr at 3-4 am, of "real high-pitched noise, holding my head, not in ears, just in head, not something I could hear." Also intermittent ear pain, "not earache."	Did not occur while away
Eye / vision	Diplopia requiring prisms in glasses began at least 6 yrs before exposure	Intermittent blurring (can't read letters on TV) and dry, sore feeling in eyes	Unknown
Other neurological	Diplopia; concussion around beginning of exposure period	No change	No change
Cardiovascular	Normal including BP	No change	No change
Gastrointestinal	Normal	Repetitive belching with feeling of air trapping and soreness of chest wall; rubs chest, lies on side to release air and obtain relief.	Unknown
Respiratory	Short of breath around polish or perfumes; feather allergy; smokes	No change	No change
Rheumatologic	Joint and muscle pains, not exacerbated by weather	Pain worse and continuous. Began with exacerbation of muscle and joint pains, then neck pain and headaches; when returned from trip, pain built back up over a week.	Improved to baseline level
Other	No problem with noise from truck traffic or living in flight path of small airport	Turbine noise different, "unnatural"; sounds like airplane stuck over house; pulsation prevents sleep; sound intensifies in cold weather.	"When I'm away it's so different, it's like I'm in a normal life"
	Cottage heated by two open coal fires	Both chimneys taken down and roof replaced, with extra insulation, in attempt to keep noise out; coal fires replaced by electric oil-filled radiators; cottage became damper.	

* Exposure period two years; family lived here one year before turbines went into operation.

** Family has not moved but was away from home for 2 weeks in the 2 months preceding the interview.

Family Table J1 (page 1of 2)

Person
Dr. J
Age
49
Pre-exposure health status
Good
Health history
Broken nose repair as teen; thyroglossal duct cyst excision as child
Previous noise exposure
Uses tractors and chain saws on property with hearing protection
Time to onset of symptoms
Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Long-term difficulty with returning to sleep started during medical training, had been improving	Delayed sleep onset and frequent awakening when turbines running fast; awakens with racing heart; can't get back to sleep; taking prescription sleep aid.	Improved sleep, no need for sleep aid
Headache	Infrequent sinus headache, no migraines	Bilateral temporal-parietal headaches 3-4 times a week; may follow a "jittery" episode.	No headaches
Cognition	Good; specialist physician	Difficulty with focus and mental energy after nights of poor sleep; marked concentration problem when doing accounts/bills at home.	Concentration seemed fine but demand low
Mood	Good, no history of anxiety or depression	"Jittery" episodes begin with sensation of "internal quivering" or awakening with rapid or pounding heart; gets "real anxious"; has to stop outdoor or family activities and go indoors; at night has to move to basement where the turbines cannot be heard or felt; on arriving home from work, he can judge whether symptoms will be triggered by the rotational speed of the turbines or the noise/feeling of vibration in the garage; increased irritability; taking two anti-anxiety medications.	No "jittery" episodes or anxiety when away or at work; no need for prn anxiety medication.

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Family Table J2 (page 1 of 2)

Person
Mrs. J
Age
47
Pre-exposure health status
Good
Health history
Two term births
Previous noise exposure
Aircraft during medical evacuations
Time to onset of symptoms
1-3 mos to headaches; 1-3 mos to concentration and memory problems; 4-5 mos to continuous palpitations; 6 mos to exacerbation of irritable bowel.

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Slept well under any circumstances	Falls asleep easily; if awakened, can usually go back to sleep	Slept well
Headache	No headaches	Evening headache at least every 2 wks requiring ibuprofen	No headaches
Cognition	Good; acute/critical care nurse; teaches nursing at university; organized mother; no problem with focus or memory.	Noticeable trouble focusing and remembering at home; has to write down what children tell her or any item to be picked up at store; easily distracted; started vitamins and supplements	Improved memory when away but not at baseline (also less demand)
Mood	Happy, energetic, busy, "up" person	Marked decrease in energy and motivation at home; frustrated; "on edge"; feels rejuvenated at work	Felt great, lots of energy
Balance / equilibrium	Never carsick or seasick, no h/o vertigo	No change	No change
Ear / hearing	Normal, tested yearly; no tinnitus	No change, no ear symptoms	No change
Eye / vision	Normal, wears contact lenses	No change	No change
Other neurological	Normal, no concussion	No change	No change

Family Table J2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Cardiovascular	Normal BP except during first pregnancy; dysrhythmia (trigeminy) 10/06 resolved with caffeine restriction.	Continuous palpitations began 10/07 and did not respond to caffeine restriction or trials of two medications; evaluated including electrophysiology; right ventricular focus.	Decreased frequency of palpitations
Gastrointestinal	Irritable bowel (cramping and diarrhea) since young adulthood with exacerbations before exams; normal colonoscopy x 2.	Continuous symptoms for 3 months before interview, except during week after return from vacation	Symptoms unchanged while away in tropical country
Respiratory	Normal, no asthma, never smoked	No change	No change
Other		• Feels vibration in feet/lower legs when stands still in house or barn, which feels like it is coming from vibrations in the structure; worse in barn, which is not insulated; does not feel this outside/on the ground. • Sounds like helicopter starting up or jet circling house every 3-4 seconds	

*Interviewed after 9 months of exposure. Family has not moved.

**Away for vacation for 2 weeks during the first 3 months of exposure and 10 days during the month before the interview.

Note: These pages are in manuscript format, pending publication of the book.

GLOSSARY

A-weighting network: see definition on p. 8.

Acute gastrointestinal infection: nausea, vomiting, abdominal pain, and diarrhea, generally self-limited and caused by a viral infection of the gastrointestinal tract.

Agoraphobia: an abnormal fear of leaving the house.

Airways: trachea, bronchi, and bronchioles, the tubular structures through which air passes to reach the air sacs or alveoli of the lungs.

Amaurosis fugax: temporary loss of vision in one eye.

Anticoagulation: use of medications such as heparin or warfarin to decrease the tendency of the blood to clot. Higher INR (international normalized ratio of prothrombin time) values, used in the monitoring of warfarin administration, indicate slower or less effective clotting.

Arthralgia: joint pain without objective signs of inflammation (see *arthritis*).

Arthritis: pain and/or stiffness in joints with accompanying objective signs of inflammation, such as redness or swelling.

Ataxia, **ataxic**: in reference to gait, unsteady on feet, difficulty with balance or coordination in walking, or difficulty maintaining posture, for neurologic reasons.

Asthma: intermittent and reversible respiratory difficulty caused by partial obstruction of small airways by inflammation/swelling and constriction of smooth muscle around the airways. Asthma attacks may be provoked by any kind of respiratory infection, allergic exposures, or irritant exposures.

Atrial fibrillation: an abnormal heart rhythm in which the small chambers do not pump rhythmically, but instead vibrate erratically, placing patients at risk for stroke from blood clots that can form inside the heart.

Autonomic nervous system: the involuntary part of the nervous system that regulates automatic body functions such as heart rate, blood pressure, gastrointestinal function, sweating, glandular output, pupillary reflexes, airway smooth muscle tone, and others. The autonomic system includes sensory receptors (for afferent signals or input to the central nervous system) and effector neurons (for efferent signals or output to organs). It consists of opposing sympathetic and parasympathetic networks. Sympathetic stimulation speeds the heart and readies the body for optimal "fight or flight" activity. Parasympathetic stimulation slows the heart, lowers blood pressure, and facilitates digestion.

Baroreceptors: pressure detectors, as in blood vessels or lungs.

Basilar migraine: migraine with auras representing brainstem effects, including vertigo, tinnitus, fluctuations in level of consciousness, and temporary motor deficits.

Binaural processing: brain integration of neural signals from both ears.

Bilateral: on both sides of the body.

Bone conduction: sound or vibratory stimuli reaching the inner ear via direct solid-to-solid transmission, without passing through or utilizing the tympanic membrane or middle ear ossicles.

C-weighting network: see definition on p. 8.

Caloric test: a test of semicircular canal function and the vestibulo-ocular response. In the caloric response to ice water in the external auditory canal, thermal convection induces fluid movement within the horizontal semicircular canal, creating an illusion of head movement that is reflected in eye movement via the vestibulo-ocular reflex.

Cardiac arrhythmia or **dysrhythmia**: specific types of irregular heartbeat, often occurring episodically.

Catecholamine: a class of biochemicals that function as neurotransmitters in the brain and as hormones produced by the sympathetic part of the autonomic nervous system, such as epinephrine (adrenalin), norepinephrine, and dopamine.

Central: occurring in the brain (central nervous system), as opposed to a peripheral neural receptor, effector, or organ. For example, central processing, central origin, central dysfunction.

Cerebellum, cerebellar: a posterior/inferior portion of the brain with important functions in coordination and integration of movement.

Chemotherapy: in this report, refers specifically to medications given for cancer treatment.

Cilium, cilia: actively motile, hair-like projections from epithelial cell surfaces in the airways and Eustachian tubes that beat in synchrony to move mucus out of these moist, air-filled spaces, towards the pharynx. Cilia occur on surfaces of other types of cell, including single-celled protozoa.

Circadian rhythm: a daily physiologic cycle, such as sleep and wakefulness or peaks and troughs of cortisol secretion.

Cochlea: spiral-shaped sensory organ of hearing, part of the inner ear membranous labyrinth. See p. 26.

Collagen: a protein which is the chief substance of connective tissue, cartilage, tendons, etc.

Concussion: mild brain injury produced by impact to the head resulting in brief unconsciousness, disorientation, or memory problem.

Coronary artery disease: partial obstruction or narrowing of the small arteries that supply the heart muscle.

Cortex, cortical: the outer cellular layers of the two cerebral hemispheres of the brain.

Cortisol: the major natural glucocorticoid hormone produced by the adrenal cortex in a regular daily rhythm and in response to stress, which exerts diverse effects on tissues and metabolic processes throughout the body.

Cranial vault: the space in the skull that contains the brain.

Diaphragm: the dome-shaped sheet of skeletal muscle that separates the thoracic (chest) and abdominal cavities and enables breathing.

Dysfunction: malfunction or poor functioning.

Elastin: an elastic connective tissue protein, which gives elasticity to certain structures, such as arterial walls.

Electroencephalogram (EEG): a recording of brain waves monitored in a specific fashion, used in studies of seizure disorder and sleep.

Epithelial basement membrane: a thin layer of extracellular proteins and mucopolysaccharides that lies at the base of and supports the layers of cells comprising an epithelium, such as the linings of airways, mouth, esophagus, intestine, pleura, etc.

Eustachian tube: a tube that connects the middle ear with the nasopharynx, or upper part of the throat behind the nose. It allows equalization of air pressure on either side of the tympanic membrane.

Fibromyalgia: a condition of chronic pain of muscles, ligaments, tendons of unclear origin, without inflammation.

Gastritis: inflammation of the lining of the stomach causing pain and nausea.

Gastroesophageal reflux: reflux or intrusion of acidic stomach contents into the esophagus; heartburn.

Gastrointestinal tract: stomach, small intestine, and colon or large intestine.

Glucose instability: in diabetes, fluctuating blood sugar levels that go too high or too low.

Graviceptors: neural detectors of gravity and acceleration; see definition on p. 23.

Great vessels: the large arteries and veins immediately around the heart, including the aorta, pulmonary artery, pulmonary veins, and superior and inferior vena cavae.

Hippocampus: a brain region in the medial temporal lobe critical to spatial navigation and formation of new episodic memories.

Hyperacusis: oversensitivity to sound, with normal sounds seeming painfully loud.

Hypopharynx: the lower part of the throat, just above the larynx (vocal cords).

Hypertension: high blood pressure.

Immissions: in acoustics, sound from the point of view of the person or location receiving the sound. *Emissions* in this context refers to the sound as it leaves the source.

Infrasonic: sound frequency below hearing range, generally considered to be 20 Hz or less.

In utero: in the uterus during pregnancy.

Irritable bowel syndrome: recurrent episodes of abdominal pain and diarrhea, often with alternating periods of constipation, without any pathologic or inflammatory changes in the gastrointestinal tract.

Labyrinthine organs, membranous labyrinth: the inner ear organs, including the cochlea, utricle, saccule, and semicircular canals. See *otolith organs* and *semicircular canals,* and p. 26.

Lower respiratory infection: bronchitis, pneumonia, or pneumonia with pleural effusion (pleurisy).

Lupus: systemic lupus erythematosus, a systemic inflammatory or autoimmune disease affecting the skin, joints, gastrointestinal tract, kidney, blood, and brain.

Magnetic resonance angiography (MRA): a noninvasive imaging method for examining the patency of blood vessels.

Magnetic resonance imaging (MRI): soft tissue imaging using magnetic fields, providing the most detailed images of living brain structure available. Functional magnetic resonance imaging (fMRI) quantifies blood flow to different brain structures during specific activities.

Malaise: a vague sense of not feeling well.

Mastoid: a bony structure immediately behind the ear that contains air-filled cells connected to the middle ear.

Mediastinum: the central portion of the chest or thorax between the lungs, containing the heart, great vessels, trachea, esophagus, lymph nodes, and other structures.

Mesentery: a fold of membranous tissue encasing and attaching the small intestine and other abdominal organs to the inside of the peritoneal (abdominal) cavity, also supporting blood vessels and nerves to the organs.

Microvilli: hair-like projections from epithelial cell surfaces that increase absorptive surface area, for example, in the small intestine.

Migraine: a hereditary, episodic, neurologic condition generally involving severe headaches that may be preceded by visual or other sensory phenomena such as tingling or numbness (aura), with symptoms of nausea and sensitivity to light and sound commonly accompanying headaches. A headache may be one-sided or pounding. Aura and accompanying symptoms may include vertigo, tinnitus, temporary focal weakness or paralysis, temporary loss of vision, vomiting, or loss of consciousness. Sensory sensitivities and triggers include motion, odors, a wide variety of foods (especially products of fermentation or aging, caffeine, chocolate, and varieties of plants), hormonal state, and sleep deprivation.

Migraineur: a person who gets migraines.

Myocardial infarction: heart attack, or obstructed coronary blood flow leading to death of cardiac muscle.

Neuroanatomic: referring to the anatomy of neural linkages in the brain.

Neuroendocrine: relating to cells or tissues that release hormones into the blood in response to a neural stimulus.

Night terror: a parasomnia, or sleep disturbance occurring during disordered arousal from the deeper stages of sleep, in which a person (usually a child) may scream, act afraid, say nonsensical things, or get up to do irrational or fearful things, all without memory in the morning.

Nocturia: awakening and getting up repeatedly in the night to urinate.

Nocturnal enuresis: bedwetting while asleep.

Norepinephrine: a central catecholamine neurotransmitter, sympathetic nervous system neurotransmitter, and vasoactive adrenal medullary hormone.

Nystagmus: a pattern of eye movement indicating a disordered vestibulo-ocular reflex that is often due to disordered vestibular signaling or processing, as in the caloric test.

Ocular: pertaining to the eyes.

Orbit: the eye socket or hollow space in the skull that contains the eyeball and its associated structures.

Otolith organs: the utricle and saccule, labyrinthine organs of the inner ear that detect linear acceleration, including gravity, by virtue of microscopic calcium carbonate stones or *otoconia* positioned in a protein matrix over the mechanically sensing hair cells. See p. 26.

Palpitations: irregular or pounding heart at times not expected from activity or exertion.

Panic attack: an episode of sudden intense fear out of proportion to circumstances, which may be accompanied by symptoms of dizziness, sweating, trembling, chest pain, palpitations, and the feeling of not being able to get enough breath.

Parabrachial nucleus: Pontine brain center involved in extended vestibular system influence

Parasomnia: a sleep disturbance occurring during disordered arousal from the deeper stages of sleep, such as sleep walking, sleep talking, and night terrors.

Paresthesia: tingling or "pins and needles" sensation, as when a numb extremity is waking up.

Parkinson's disease: a neurologic degenerative disease involving dopamine-producing neural tracts in the brain and affecting movement and psychiatric status.

Pericardium: the two-layered membranous sac that encloses the heart and the roots of the great vessels, in which the heart beats.

Perilymphatic fistula syndrome: see p. 30.

Pharynx: the throat.

Pleura: the outer epithelial surface of the lung and the lining of the thoracic cavity, providing low friction surfaces for lung movement.

Polyuria: excessive daily volume of urine, a typical sign of high glucose levels in diabetics.

Positron emission tomography (PET): a method of functional imaging that quantifies glucose uptake by different brain regions as a measure of activity.

Posturography: a form of balance testing that is sensitive to the vestibulo-spinal reflexes, including the influence of inner ear, visual, somatosensory, and central processing on the movements by which a subject remains balanced and upright.

Pressure equalization tube: a tube inserted through a small, surgically placed hole in the tympanic membrane after removal of middle ear fluid, to provide aeration.

Resonance: a property of sound; see pp. 7 and 25.

Retina, retinal: the light-sensing neural structure at the back of the eye.

Scotoma: temporary loss of vision in one part of the visual field.

Semicircular canals: bilateral labyrinthine organs of the inner ear that detect angular acceleration of the head by virtue of fluid shifts deflecting mechanically sensing hair cells. See p. 26 and *caloric test.*

Serotonin: a brain and gastrointestinal neurotransmitter.

Serous otitis media: viscous fluid in the middle ear (middle ear effusion) that may obstruct sound transmission, usually occurring after a series of acute ear infections.

Sequela, sequelae: a pathologic condition that develops from another pathologic condition, such as chronic middle ear fluid and hearing loss being sequelae of repeated acute ear infections.

Somatic nervous system: the sensory and motor nervous system from and to the skin, skeletal muscles, and associated tendons and ligaments, whose signals may be consciously perceived and voluntarily modified.

Somatosensory: sensory input from the skin, skeletal muscles, tendons, and ligaments.

Sonic: sound frequency in the range of human hearing.

Tachycardia: rapid heartbeat.

Taxon, taxa: a group or groups in the scientific categorization (Linnaean taxonomy) of living things.

Temporal bone: solid bone at the base of the skull, in which the labyrinthine organs lie.

Thalamus: a part of the brain involved in part in relaying sensory information to the cerebral cortex.

Tinnitus: "ringing in the ears," which may be a tonal sound, buzzing, white noise, or other types of sound heard in one or both ears. The sound itself is not present in the outside environment.

Trachea: the large central airway between the larynx (voice box) and the split or bifurcation of the right and left bronchi.

Tympanic membrane: eardrum; the layer of taut, thin tissue that separates the external auditory canal from the middle ear.

Ulcer: duodenal or gastric ulcer.

Ultrasonic: sound frequency above hearing range, generally considered to be 20,000 Hz or more.

Upper gastrointestinal symptoms: gastroesophageal reflux, gastritis, and/or ulcer.

Vasculitis: inflammation of blood vessels, which can cause restriction of blood flow.

Vasoconstriction: constriction of a blood vessel.

Vertigo: the spinning form of dizziness, in which the visual surround seems to move.

Vestibular: pertaining to the balance organs in the inner ear (utricle, saccule, and semicircular canals) or to the integrated balance system in general, as in "vestibular areas of the brain."

Visceral: pertaining to the internal organs.

Whiplash injury: an injury to the neck (cervical vertebrae) caused by abrupt acceleration or deceleration, as in an automobile accident.