Wind Turbine Syndrome

A Report on a Natural Experiment

March 7, 2009, pre-publication draft

Nina Pierpont, MD, PhD

March 7, 2009, the following is a pre-publication draft, with pages missing from WTS for Clinicians & Tables

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PREFACE

I wrote this report because I saw a medical problem which few clinicians were paying attention to or, for that matter, seemed to understand. Dr. Amanda Harry in the United Kingdom led the way in recognizing the cluster of symptoms people experience around wind turbines.[1] I, myself, began encountering the problem from numerous e-mails and telephone calls I received, 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 quickly became apparent. It didn't take long to realize the potential for a relationship between these complaints, on the one hand, and *migraine, motion sickness, vertigo, noise and visual and gastrointestinal sensitivity, and anxiety* which, all together, form a coherent and interconnected neurologic complex in medical practice.

The 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. Best of all, the interview introduced 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 worked to understand these complaints, I have benefited from new research allowing us to better understand neurologic phenomena like spatial memory loss and fear reactions in people with balance problems—symptoms that often "bored and baffled" clinicians, as one of my referees put it.[3] Some wind developers and acousticians are even less charitable.

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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.

> It's … worth noting that studies have shown that a person's attitude toward a sound—meaning whether it's a "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.[4]

> Their patients [the people living near wind turbines and reported on by Dr. Osborne and Dr. Harry] may well have been experiencing adverse symptoms, but we have to keep in mind that people who have failed, for whatever reason, in strong objections to a development, build up in themselves a level of unfulfilled expectations and consequent stress, which peaks after the failure and can overload their coping capabilities. This leads them to lay the blame on whatever straw they can clutch. This is especially so in group activities, where mutual support may turn to a mutual, interacting misery, which worsens the situation….The very low levels of low frequency noise and infrasound which occur from wind turbines will not normally cause problems. If problems have occurred, it is possibly for some other stress-related reason.[5]

> Brian Howe, a consulting engineer in acoustics for 20 years for HGC Engineering, said Ontario's guidelines for turbine noise are adequate and consistent with Health Canada studies. Most people near wind turbines aren't complaining about the noise, Howe said. In some cases, noise complaints could reflect higher anxiety levels from people who had unrealistic expectations of hearing virtually no sound, he said.[6]

Responses like these are a pity—because they're rubbish. There is nothing "psychosomatic" or malingering about it. Research clearly shows there are precise and definable neurologic connections that explain how sensory signals can derail normal psychological and cognitive function and, in fact, trigger physical symptoms. (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,

[4] Noble Environmental Power, LLC. Wind fact sheet #5: Are modern wind turbines noisy? http://www.noblepower.com/faqs/documents/06-08-23NEP-SoundFromWindTurbines-FS5-G.pdf, p. 2.
[5] Leventhall, Geoff. 2004. Notes on low frequency noise from wind turbines with special reference to the Genesis Power Ltd. Proposal near Waiuku, NZ. Prepared for Genesis Power/Hegley Acoustic Consultants, June 4, p. 7.
[6] Rennie, Gary. 2009. Wind farm noise limits urged. The Windsor (Ontario, Canada) Star. February 24.

of course, neurology.[7] Much of the research on vestibular function, whereon I draw heavily, is even more recent, conducted within just the last 10-15 years.)

Leaving that bit of pop psychology behind us, let's move on to evidence-based science. In the world of medicine my study is properly called a "case series," 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 raised, 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 gravity of symptoms that force a family to vacate its home or perform expensive renovations aimed solely at noise exclusion.

Which 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 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.

[7] 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.

The ecological dimension in the phrase *natural experiment* is worth emphasizing, for the simple reason that 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 also suggests pathophysiologic mechanisms.

It would be difficult to do a conventional epidemiologic study of the health effects of wind turbines, at least in the United States, 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) or case and control populations identified. 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 prohibit 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. In an epidemiologic study based on interviews or questionnaires these clauses could easily distort answers or skew participation, invalidating a 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, the turbines on your cousin's land happen to be 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.*

Non-clinicians should realize that, in medicine, many conditions (ailments) are diagnosed mostly by *medical history*. This includes migraine and other headaches, tinnitus, and sleep disturbance. For, of course, the doctor cannot tell objectively (by any sort of clinical test) 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 he (she) asks of the patient. This is the part I could credibly do by telephone.

My study subjects also told me about other kinds of problems that seemed to worsen 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 and case series. (I have included them in a separate section of the *Results* because I think they may need attention from the medical research community.)

This study also does not tell us how many people are affected within a certain distance of wind turbines. But it does offer a framework for what to pursue in such a study (meaning, the next phase: epidemiologic studies), such as what symptoms to study and what aspects of the exposure to measure.

Shifting, now, to the format of the book. I have written it as a (long) 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 sample 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, and a list of *Abbreviations*.

There are two sets of *Tables*. The regular *Tables* (numbered 1 A-C, 2, and 3) are compilations of data found in the *Results* section. What I call the *Family Tables* are the raw narrative data of each individual subject's symptoms and statements, organized one person per table with separate columns for *before*, *during*, and *after* exposure and separate lines for each organ or functional system (e.g., sleep, headache, cognition, balance/equilibrium, ears/hearing, etc.).

The *Family Tables* are presented together at the end of the clinical text. They are the backbone, the substance, of my report. I deeply appreciate my subjects' willingness to be included herein.

The book is intended for physicians and other professionals and individuals who wish to better understand the wind turbine-associated symptom complex. This posed a dilemma: writing in the specialized language of clinical medicine and science is very different from the language one uses for lay readers. Yet my goal is to reach both audiences. I solved the problem by adding (at my editor's insistence) a more conversational, parallel text, which I christened *WTS for Non-Clinicians.*

The result is a book with two, tandem texts. They both say the same thing. One says it in the language of the clinician (*WTS for Clinicians*), the other in the everyday language of—well—my editor (*WTS for Non-Clinicians*).

The goal of *WTS for Clinicians* is scientific precision, including frequent expressions of my degree of certainty or uncertainty. Since the physics and the physiology I invoke are complex and not widely known among clinicians, I explain them in this text. Here, likewise, I quote and summarize numerous scientific articles, and I use numbers and statistics (albeit the simplest type known).

WTS for Non-Clinicians says it all over again, this time in English my mother-in-law would understand. To accomplish this I had to sacrifice a degree of scientific precision, since *plain English* and *scientific precision* don't always mix. I freely acknowledge that *WTS for Non-Clinicians* might set some clinicians' teeth on edge. For this I beg their indulgence.

A second disclaimer. Readers should understand that Wind Turbine Syndrome is not the same as Vibroacoustic Disease.[8] 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, inner 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 (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, it appears that even low frequency noise or vibration too weak to hear can still stimulate the human vestibular system, opening the door for the symptoms I call Wind Turbine Syndrome. I am happy to report, there is now direct experimental evidence of such vestibular sensitivity in normal humans.[9]

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.[10] The suspected agent is high amplitude (high power or 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 that may be worthy of further clinical investigation, especially with regard to asthma and lower respiratory infections.

Moving on, I have been asked if Wind Turbine Syndrome could be caused by magnetic or electric fields. I have no reason to think so. There has been extensive epidemiologic research since 1979 on magnetic fields and health, comparing people who live close to high power lines or work in electrical utilities or work in other industries where magnetic field exposure is likely to be high, to those who do not.[11] This substantial body of research has produced no good evidence that magnetic field exposure causes cancer in

[8] Castelo Branco NAA , Alves-Pereira M. 2004. Vibroacoustic disease. Noise Health 6(23): 3-20.
[9] Todd NPMc, Rosengren SM, Colebatch JG. 2008. Tuning and sensitivity of the human vestibular system to low-frequency vibration. Neuroscience Letters 444: 36-41.
[10] Castelo Branco NAA , Alves-Pereira M. 2004. Vibroacoustic disease. Noise Health 6(23): 3-20.
[11] Ahlbom IC, Cardis E, Green A, Linet M, Savitz D, Swerdlow A; INCIRP (International Commission for Non-Ionizing Radiation Protection) Standing Committee on Epidemiology. 2001. Review of the epidemiologic literature on EMF and health. Environ Health Perspect 109 (Suppl 6): 911-33.

children or adults, cardiac or psychiatric disease, dementia, or multiple sclerosis.[12,13] After three decades of research, there is still no experimental evidence for a physiologic mechanism for any of the proposed effects of magnetic fields.[14]

This makes it very hard to do epidemiologic studies, since researchers don't know what exposure to measure, or what exposure period (e.g., last week or five years ago) might be relevant.[15] An association has been shown between higher magnetic field exposure in utility workers and amyotrophic lateral sclerosis (ALS), a neurodegenerative disease, but this is most likely due to more frequent electric shocks in these settings, not to the magnetic fields.[16] Claims that voltage and frequency irregularities in household alternating currents (what some refer to as "dirty electricity") create a wide, non-specific swath of medical problems – from ADHD to rashes to diabetes to cancer – are completely unsubstantiated, and also have no plausible biologic mechanisms.[17]

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."

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.

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[12] Ahlbom IC et al. 2001.
[13] Johansen C. 2004. Electromagnetic fields and health effects – epidemiologic studies of cancer, diseases of the central nervous system and arrhythmia-related heart disease. Scand J Work Environ Health 30 Suppl 1: 1-30.
[14] Ahlbom IC et al. 2001.
[15] Ahlbom IC et al. 2001.
[16] Johansen C. 2004.
[17] I have asked Prof. Magda Havas, Environmental and Resource Studies, Trent University, Ontario, Canada to remove references to Wind Turbine Syndrome from her PowerPoint presentation on hypothesized wind turbine health effects, because these references are inaccurate.

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, or if different lines of research point to the same conclusions.

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 Ralph Katz (epidemiology) and Henry Horn (ecology) 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 Katz and Horn, for which I am most grateful. Barbara Frey (biomedical librarian) edited the manuscript and provided many essential references.

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.[18] The expanded paper is posted on the Wind Turbine Syndrome website.[19]

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. Likewise, 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

[18] Kamperman GW, James RR, Simple guidelines for siting wind turbines to prevent health risks. Noise-Con, July 28-31, 2008, Institute of Noise Control Engineering/USA.
[19] 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.[20]

I 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,[21] 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. 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 other studies.[22] 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) greater 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) greater 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, greater than the 2-3.5 km. (1.24-2.2 mi.) at which there were symptomatic subjects in Professor Robyn Phipps's New Zealand study.[23]

[20] Sinclair, Upton. 1935. *I, Candidate for Governor: And How I Got Licked*.
[21] 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.
[22] 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.
[23] See *Introduction*, p. x, for discussion and references.

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) 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.

For those who read this report and recognize their own symptoms, the appropriate medical specialist to consult would be a neurotologist (or otoneurologist), who is an otolaryngologist (ears, nose, and throat doctor) 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 and recognizing your own symptoms within these pages: you are not crazy and not fabricating them. Your symptoms 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."[24]

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, 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 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.

[24] 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.

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. 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 pressure (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. These companies 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 rank-and-file 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

Winter 2009

As you read, note that there are missing pages in this typescript ms.

Wind Turbine Syndrome for Clinicians

Abstract

This report documents a consistent and often debilitating complex of symptoms experienced by adults and children while living near large industrial wind turbines (1.5-3 MW). It 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 that arise while awake or asleep.

The study is a case series of 10 affected families, with 38 members age <1 to 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 (a) before turbines were erected near their homes, (b) while living near operating turbines, and (c) 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 when low frequency noise or vibration stimulates 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 spatial awareness, spatial memory, spatial problem-solving, fear, anxiety, autonomic functions, and aversive learning, providing a robust neural framework for the symptom associations in Wind Turbine Syndrome. Further research is needed to prove causes and physiologic mechanisms, establish prevalence, and to explore effects in special populations, including children. This and other studies suggest that safe setbacks will be at least 2 km (1.24 mi), and will be longer for larger turbines and in more varied topography.

Santa Fe, NM: K-Selected Books, 2009

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] Developers assure prospective communities that turbines are no louder than a 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.

[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

This set of symptoms stands 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 that people 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 at such relatively low sound levels. I can 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]

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 been dominated to date 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,

[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.

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, sleeplessness, panic episodes, disrupted children, or memory deficits that they must move or abandon their homes to get away from wind turbine noise and vibration, then that noise and vibration is by definition significant, because the symptoms it causes are significant. The 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 living 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,

[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.

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

depression, tinnitus, hearing loss, and palpitations. Respondents described a similar set of symptoms and many of the same experiences that I document in this report, 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% farther 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 at distances even 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 visibility of turbines in Sweden[15,16,17] and the Netherlands.[18,19,20] The study in the Netherlands included questions on health and stress (see below, *Discussion*). Both sets of studies, the Swedish and

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[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.

[16] Pedersen E. 2007. Human response to wind turbine noise: perception, annoyance and moderating factors. Dissertation, Occupational and Environmental Medicine, Department of Public Health and Community Medicine, Goteborg University, Goteborg, Sweden. 86 pp.

[17] Pedersen E, Persson Waye K. 2007. Wind turbine noise, annoyance and self-reported health and wellbeing in different living environments. Occup Environ Med 64(7): 480-6.

[18] Pedersen E, Bouma J, Bakker R, van den Berg GP. 2008. Response to wind turbine noise in the Netherlands. J Acoust Soc Am 123(5): 3536 (abstract).

[19] van den Berg GP, Pedersen E, Bakker R, Bouma J. 2008. Wind farm aural and visual impact in the Netherlands. J Acoust Soc Am 123(5): 3682 (abstract).

[20] van den Berg GP, Pedersen E, Bouma J, Bakker R. 2008. Project WINDFARMperception: visual and acoustic impact of wind turbine farms on residents. Final report, June 3, 2008. 63 pp. Summary: http://umcg.wewi.eldoc.ub.rug.nl/FILES/root/Rapporten/2008/WINDFARMperception/WFp-final-summary.pdf
Entire report: https://dspace.hh.se/dspace/bitstream/2082/2176/1/WFp-final.pdf

Dutch, make important contributions to the rational setting of noise limits near wind turbines (see *Discussion*).

With regard to official opinion, the National Academy of Medicine in France recommended in 2005 that industrial wind turbines be sited at least 1.5 km (0.93 mi) from human habitation due to health effects of low frequency noise produced by the turbines.[21]

Current wind turbines have three airfoil-shaped rotor blades attached by a hub to gears and a generator, which are housed in a bus-sized box (nacelle) at the top of a nearly cylindrical, hollow steel tower. The nacelle is rotated mechanically to face the blades into the wind. The blades spin upwind of the tower. The tower is anchored in a steel-reinforced concrete foundation. Turbines heights in this study were 100 to 135 m (328 to 443 ft) with hub heights 59 to 90 m (194 to 295 ft) and blade lengths 33 to 45 m (108 to 148 ft). Individual turbine powers were 1.5 to 3 MW. Clusters contained from 8 to 45 individual turbines (see Table 1B).

In this study, participants from all families described good and bad symptomatic periods correlated with particular sounds from the turbine installations, rate of turbine spin, or whether the turbines were turned towards, away from, or sideways relative to their homes. All participants identified wind directions and intensities that exacerbated their problems and others that brought relief. Many subjects described a quality of invasiveness in wind turbine noise, more disturbing than other noises like trains. Some stated that the noise wouldn't sound loud to people who did not live with it, or that noises described with benign-sounding terms like "swish" or "hum" were in reality very disturbing. Several were disturbed specifically by shadow flicker, which is the flashing of light in a room as the slanting sun shines through moving turbine blades, or the repetitive movement of the shadows across yards and walls. (These observations are documented in the individual accounts in the *Family Tables*.)

Wind turbines generate sound across the spectrum from the infrasonic to the ultrasonic,[22] and also produce ground-born or seismic vibration.[23] "In the broadest sense, a sound wave is any disturbance that is propagated in an elastic medium, which may be a gas, a liquid, or a solid. Ultrasonic, sonic, and infrasonic waves are included in this definition… Sonic waves [are] those waves that can be perceived by

[21] Le retentissement du fonctionnement des éoliennes sur la santé de l'homme, le Rapport, ses Annexes et les Recommandations de l'Académie nationale de medicine, 3/14/2006. 17 pp. http://www.academie-medecine.fr/sites_thematiques/EOLIENNES/chouard_rapp_14mars_2006.htm

[22] van den Berg 2004

[23] Style, P, Stimpson I, Toon S, England R, and Wright M. 2005. Microseismic and infrasound monitoring of low frequency noise and vibrations from wind farms. Recommendations on the siting of wind farms in the vicinity of Eskdalemuir, Scotland. 125 pp. http://www.esci.keele.ac.uk/geophysics/News/windfarm_monitoring.html

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 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. Conversely, a vibrating solid object, such as the strings on a violin, can create sound waves in air. There is 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 in solids.

Energy in either form (sound or vibration) 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 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 *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 in the room with locations where the intersecting, reverberating sound waves reinforce each other. Resonance also occurs inside air-filled body cavities such as the lungs, trachea, pharynx, middle ear, mastoid, and gastrointestinal tract. The elasticity of the walls and density of the contents of these spaces affect the dynamics of sound waves inside them. The orbits (bones surrounding the eyes) and cranial vault (braincase) are also resonance chambers, because of the lower density of their contents compared to the bones that surround them. There are also vibratory resonance patterns along the spine (which is elastic), including a resonance

[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

involving the movement of the head relative to the shoulders. Von Gierke[26,27] and Rasmussen[28] have described the resonant frequencies of different 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. A-weighting augments the contributions of sounds in the 1000 to 6000 Hz range (from C two octaves above middle C, key 64 on the piano, to F# above the highest note on the piano), and progressively reduces the contributions of lower frequencies below about 800 Hz (G-G# 1½ octaves above middle C, keys 59-60). At 100 Hz, where the human inner ear vestibular organ has a peak response to vibration[29] (G-G# 1½ octaves below middle C, keys 23-24), A-weighting reduces measurement by a factor of 1000 (30 dB). At 31 Hz (B, the second-to-bottom white key, key 3), A-weighting reduces sound measurement by a factor of 10,000 (40 dB). Thus A-weighting preferentially captures the high sounds used in language recognition, to which the human cochlea is indeed very sensitive, but reduces the contribution of mid- and lower-range audible sounds, as well as infrasound (defined as 20 Hz and below).

Linear (lin) measurements use no weighting network, so the frequency responses are limited by other aspects of the system, such as microphone sensitivity. Linear measurements may capture low-frequency sounds but are not standardized – different sound level meters yield different results. As a result, the standardized and commonly available C weighting network is preferred for measuring environmental noise with low-frequency components, such as noise from wind turbines. The C weighting network has a flat response (meaning that it does not reduce or enhance the contributions of different frequencies) over the audible frequency range and a well-defined decreasing response below 31 Hz.

One third (1/3) octave band studies are used to describe sound pressure levels by frequency, and are presented as a graph rather than a single number. One third (1/3) octave bands can also be measured linearly or with weighting networks.

[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.
[29] Todd NPMc, Rosengren SM, Colebatch JG. 2008. Tuning and sensitivity of the human vestibular system to low-frequency vibration. Neuroscience Letters 444: 36-41.

telephone about further developments. All ten families have reviewed the information presented about them and signed permission for anonymous publication.

I use simple statistical tests (2x2 χ^2) to examine associations among symptoms and between pre-existing conditions and symptoms during exposure.[31] Degrees of freedom (df) is 2 for all the χ^2 results in this report. Children were excluded from the analysis of adult symptoms if no child younger than a certain age had the symptom in question. Study children were categorized into developmental-age blocks (see Table 1C). When I excluded children from an analysis, I excluded all the children in that age block and below. Excluding children from adult symptom analyses avoided inflating the no symptom/absent pre-existing condition box of the 2x2 χ^2 contingency tables, which could artificially increase the χ^2 value.

Results

I interviewed 23 adult and teenage members of 10 families, collecting information on all 38 adult, teen, and child family members. One family member was a baby born a few days before the family (A) moved out, so there is no data for this child on sleep or behavior during exposure (which was in utero). Thus the sample size of subjects for whom we have information about experiences or behavior during exposure is 37.

Residence status and family composition are detailed in Table 1A; turbine, terrain, and house characteristics in Table 1B; and the age and sex distribution of subjects in Table 1C. Twenty subjects were male and 18 female, ranging in age from <1 to 75. Seventeen subjects were age 21 and below, and 21 subjects were age 32 and above. There is a gap in the 20's and a preponderance of subjects in their 50's. Wind turbine brands to which study subjects were exposed included Gamesa, General Electric, Repower, Bonus (Siemens), and Vestas.

Individual accounts of baseline health status and pre-exposure, during exposure, and post-exposure symptoms or absence of symptoms are presented in Family Tables A through J, with a separate sub-table for each individual. I encourage the reader to read these, because they highlight the before-during-after comparisons for each person, show how the symptoms fit together for individuals, reveal family patterns, and provide subjects' own words for what they feel and detect. When individuals are referred to in the

[31] Sokal RR, Rohlf FJ. 1969. *Biometry*. WH Freeman and Co., San Francisco. 776 pp.

text, the letter and number in parentheses (e.g., A1, C2) refers to the family sub-table in which that subject's information is found.

Baseline conditions

Eight adult subjects had serious baseline medical conditions, including lupus (1), breast cancer (2), diabetes (1), coronary artery disease (2), hypertension (1), atrial fibrillation with anticoagulation (1), Parkinson's disease (1), ulcer (1), and fibromyalgia (2). Two were male (age 56-64) and six female (age 51-75). Other baseline medical conditions are listed in Table 2. Four subjects smoked at the beginning of exposure, and five others had smoked in the past (Table 2). There were no seriously ill children in the sample.

Seven subjects had histories of mental health disorders including depression, anxiety, post-traumatic stress disorder (PTSD), and bipolar disorder. Three were male (age 42-56) and four female (age 32-64). One of these men (age 56) also had Alzheimer's disease. There were no children with mental health disorders or developmental disabilities in this sample.

Eight subjects had pre-existing migraine disorder (including two with previous severe sporadic headaches that I interpreted as migraine). Four were male (age 19-42) and four female (age 12-42). An additional seven subjects, age <1 to 17, were children of migraineurs who had not experienced migraines themselves at baseline.

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 lawn mowers 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 that included all subjects age 5 and older (N=34), since the younger children in the study (age <1 to 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 (19) of the 34 study participants age 5 and older experienced unusually severe headaches during exposure. Migraine was a statistically significant risk factor but was present in less than half (8) of the 19 subjects with worsened headache. Children and teens up to age 21 with headaches either had known migraine or were the children of migraineurs. Nine of the19 headache subjects were adults without clear risk factors, showing that while people with migraine are more likely to have headaches of unusual intensity, duration, or frequency around turbines, so can other adults without identified risk factors.

Tinnitus and ear sensations. Fourteen subjects (nine males age 19-64 and five females age 33-57) experienced tinnitus that was new or worse in severity or duration than at baseline. For two men (age 55 and 64; B1, D1), the tinnitus at times interfered with their ability to understand conversation. Four of the 14 subjects experienced particularly disturbing kinds of tinnitus or noise which was perceived to be inside the head (two men age 42-55 and two women age 52-57; B1, F1, H2, I2). This sensation was painful for two subjects. Tinnitus tended to resolve over several hours after exposure ended.

Tinnitus risk factors were examined in subjects age 16 and older, since the youngest person with tinnitus was in this age group. The subject with dementia (E1) was excluded, since there was no information on his hearing status or tinnitus. Sample size was 24 subjects. The occurrence of new or worsened tinnitus in the presence of turbines was significantly correlated with previous noise exposure ($\chi^2 = 6.17$, $p = 0.013$), tinnitus prior to exposure ($\chi^2 = 5.71$, $p = 0.017$), and baseline hearing loss ($\chi^2 = 4.20$, $p = 0.040$). Tinnitus during exposure did not show a significant relationship with pre-existing migraine or motion sensitivity, or with headache or VVVD during exposure. Tinnitus during exposure showed a weak correlation with dizziness/disequilibrium during exposure ($\chi^2 = 3.60$, $p = 0.058$)

Eleven subjects during exposure experienced ear popping, ear or mastoid area pressure, ear pain without infection, or a sensation that the eardrum was moving but not producing a sensation of sound (six males age 2-55 and five females age 19-57). The 2½ year old (A3) pulled on his ears and got cranky repeatedly at the same time as his grandmother's (B2) exacerbations of headache, tinnitus, and ear pain. Five subjects experienced tickling, blowing, or undefined sensations in the external auditory canal, or increased wax production (two men age 42-55 and three women age 52-75).

Balance and equilibrium. Sixteen subjects (seven males age 19-64 and nine females age 12-64) experienced disturbance to their balance or sense of equilibrium during exposure, describing dizziness, light-headedness, unsteadiness, or spinning sensations. One of them, a 42-year-old woman (C2), described how a friend, sitting next to her in her turbine-exposed home, remarked how her eyes appeared to be bouncing back and forth (nystagmus). Ten of these 16 subjects also experienced nausea during exposure to turbines, during or separate from dizziness. No children under the age of 12 had symptoms of dizziness, disequilibrium, or nausea during exposure, except for the usual nausea of acute gastrointestinal and other infections.

Risk factors for dizziness/disequilbrium in the presence of turbines were analyzed using subjects age 12 and up, since this was the youngest age child with this type of symptom. The subject with Parkinson's disease and dementia (E1) was excluded because his baseline balance problems and inability to express himself made it hard for his wife (the informant) to tell if he had worsened symptoms during exposure or not. The remaining sample was 24 subjects. Disequilibrium during exposure is significantly correlated with headaches during exposure ($\chi^2 = 5.08$, $p = 0.024$) and baseline motion sensitivity ($\chi^2 = 4.20$, $p = 0.040$). Disequilibrium during exposure is weakly correlated with tinnitus during exposure ($\chi^2 = 3.60$, $p = 0.054$); inspection of the data shows that these are primarily ataxic (unsteady) subjects. Dizziness/ disequilbrium during exposure is not correlated with VVVD during exposure, pre-existing migraine disorder, previous noise exposure, or prior tinnitus or hearing loss.

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 from 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:

- Dr. J's (J1, age 49) "jittery" feeling includes being "real anxious," irritable, and "no fun to be around." He interrupts outdoor and family activities to sequester himself in his well-insulated house. When the turbine blades are spinning fast and he detects certain types of noise and vibration as he arrives home from work, he gets queasy and loses his appetite. He awakens from sleep with the "jittery" feeling and tachycardia, and may need to go downstairs to a cot in the 55 degree root cellar (the only place on his property where he cannot hear or feel the turbines) to be able to fall back to sleep. He often takes deep breaths or sighs when in the "jittery" state.

- Mrs. I (I2, age 52) describes episodic "queasiness and nausea" with loss of appetite, "trembling in arms, legs, fingers," "strong mental and physical agitation," and frequent unexpected crying. On noisy nights she awakens after four hours of sleep, weeping in the night. "When I wake up, [there is] more a feeling of pressure and tightness in my chest; it makes me panic and feel afraid." It is "a startling sort of waking up, a feeling there was something and I don't know what it was." Once she awoke thinking there had been an earth tremor (there had not), and twice she has awakened with tachycardia, the "feeling your heart is beating very fast and very loud, so I can feel the blood pumping." Feelings of panic keep her from going back to sleep.

- Mrs. H (H2, age 57) awakens 5-6 times per night with a feeling of fear and a compulsion to check the house. She describes it as a "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." She finds it hard to fall back to sleep and describes herself as irritable and angry, shouting more at her family members.

- Mr. G (G1, age 35) described the noise outside his home and the noise that awakened him at night "stressful."

- Mrs. G (G2, age 32) was, during exposure, irritable, angry, and worried about the future and her children. She awoke often at night because her children woke up, when she cared for their fears, mentioning none of her own.

- Mrs. F (F2, age 51) described a "feeling of unease all the time." At night she startles awake with heart pounding, a feeling of fear, and a compulsion to check the house. The feeling of alarm keeps her from being able to go back to sleep.

- Mrs. E (E2, age 56) did not express anxiety or fear, but she awakened repeatedly at night and was unable to get back to sleep on nights when the turbines were facing the house.

- Mr. D (D1, age 64) described how he has to "calm down" from the "tremor." 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." Mood has worsened with increased anger, frustration, and aggression. Tachycardia accompanies the "tremor" at times: "My heart feels like it's starting to race like crazy and I have these tremors going through my body." Mr. D pants or hyperventilates when the tremor and tachycardia occur, and consciously slows his breathing when calming down.

- Mr. C (C1, age 45) was unable to rest, relax, or recuperate in his home, where his body was "always in a state of defense." He had to drive away in his car to rest.

- Mrs. B (B2, age 53) became "upset and in a turmoil" when her symptoms were worse, leaving her house and tasks repeatedly to get relief.

- Mr. B (B1, age 55) described stress, "lots, pretty near more'n I could take, it just burnt me, the noise and run-around." He was prescribed an anxiolytic, and spent more time at the shore and his boat for symptom relief.

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 subjects) or past histories (5 subjects) 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, became routinely 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.

- 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 5-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, and she repeatedly had tantrums over schoolwork at home during the exposure period. Two months after moving, despite no change in her ears (on a waiting list for pressure equalization tubes), she was more patient and could work longer on homework. Her mother noted that her "schoolwork has improved massively."

- An 8-year old boy (H3) had an excellent memory and did well in reading, spelling, and math prior to exposure. During exposure he became resistant to doing homework, with tantrums, and his teacher told him he was not concentrating and needed to go to bed earlier.

In comparing the 20 subjects with salient concentration or memory changes to the 14 who had no change from baseline or vague/minimal difficulties, there are significant relationships with 1) baseline cognition, in that those without memory or concentration deficits at baseline are more likely to notice such deficits during exposure ($\chi^2 = 4.86$, $p = 0.027$); and 2) fatigue or loss of energy or enjoyment for usual activities during exposure ($\chi^2 = 5.61$, $p = 0.018$). There is no significant relationship between salient concentration or memory changes and pre-existing psychiatric diagnoses, migraine, motion sensitivity, or noise exposure, or between salient concentration or memory changes and headache, tinnitus, VVVD, or irritability during exposure.

In addition to the statistical association between fatigue and concentration disturbance, a number of subjects directly attributed their concentration problems to their sleep deprivation or disturbance. Several aspects of the data, however, suggest that other factors may also be involved.

First, one subject, Mrs. E (E2, age 56) could not do certain mental tasks requiring concentration when the turbines were turned towards her house, but could do them when the turbines were not turned towards the house. Mr. C (C1, age 45), Mr. I (I1, age 59), and Dr. J (J1, age 49) also had concentration problems closely linked in time and space to direct exposure to turbine noise.

Second, some of the problems described by subjects, such as Mrs. F (F2, age 51) and the members of families A and B, are more extreme than I expect from sleep deprivation. The degree of thinking dysfunction involved in not being able to follow a recipe or assemble a piece of furniture, in a woman both highly educated and involved in several practical professions (nursing and farming), does not match

my expectation of sleep deprivation from the experience, for example, of both younger and older physicians, who often function under sleep deprivation.

Third, some subjects had concentration problems without obvious sleep problems. All four members of family J had concentration problems, but only Dr. J (J1, age 49) was sleep deprived. Mrs. J (J2, age 47) fell asleep easily and usually went back to sleep if awakened, but still had problems with memory and focus in her home activities that she had noticed and attempted to treat. Their 13-year-old son (J3) needed white noise or music to drown out turbine noise to fall asleep, but went to sleep promptly, slept through the night, and did not complain in the morning of being tired or having slept poorly. His school performance and his level of distractibility at home, however, were both markedly different than at baseline. The younger son, age 8 (J4), continued to sleep well, but still had a surprising decline in school performance, though milder and of shorter duration than his brother's.

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 also 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. Four children (three boys age 8-9 and a girl age 5; C7, G3, H3, 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),[32] or other types of secondary effects (asthma).[33]

Respiratory infection/inflammation cluster: Seven subjects had unusual or prolonged lower respiratory infections during exposure (A2, B1, C2, E2, F1, F3, F4), and two of these also had prolonged asthma exacerbations (F1, F3). 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

[32] Castelo Branco NAA , Alves-Pereira M. 2004. Vibroacoustic disease. Noise Health 6(23): 3-20.
[33] Beasley R, Clayton T, Crane J, von Mutius E, Lai CK, Montefort S, Stewart A; ISAAC Phase Three Study Group. 2008. Association between paracetamol use in infancy and childhood, and the risk of asthma, rhinoconjunctivitis, and eczema in children aged 6-7 years: analysis from Phase Three of the ISAAC programme. Lancet 372(9643): 1039-48.

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/ 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 the move. Two women (age 56-57; E2, H2) had exacerbations of fibromyalgia, both of which resolved after moving or during times away from their exposed home.

Diabetes control. A 56-year-old man with Type II diabetes (E1), stable on oral medications and insulin before exposure, had marked glucose instability accompanied by visual blurring, retinal changes, and polyuria during exposure.

Anticoagulation: A 75-year-old woman with atrial fibrillation (F4) had stable INR values on 2-4 mcg warfarin daily for 10 years. By 16 months of exposure, her warfarin dose had been increased to 8-9 mcg daily in response to decreasing INR values.

Auditory processing cluster: A woman (age 33, A2) had progressively worsening tinnitus during her five months of exposure. After she moved away, the tinnitus resolved and she noticed she had a new difficulty understanding conversation in a noisy room, now needing to watch the speaker's face carefully. Her son, age 27-32 months during exposure, did not confuse sounds before exposure but began to do so during exposure, and continued to do so when I interviewed mother six weeks after the exposure ended (A3). The child's language development was otherwise good. One woman (age 42, C2) had tinnitus throughout her 21 month exposure period without hearing changes. After she moved and the tinnitus resolved, she noted hyperacusis. Another woman (age 32, G2) experienced hyperacusis during exposure, but no tinnitus. The hyperacusis resolved after the family moved.

Ocular cluster: Three subjects exposed to the same turbines (two men age 32-55 and one woman age 53; A1, B1, B2) had ocular pain, pressure, and/or burning synchronously with headache and tinnitus. Mr. D

(D1, age 64) had a painless retinal stroke, losing half the vision in his left eye. Mr. D had a normal CT scan of the brain and was examined by an ophthalmologist.

Complex migraine phenomena. A 19-year-old fisherman (C4) with migraine at baseline had complex visual symptoms with flashes in square patterns in one eye at a time (scintillating scotoma), evolving to blurring and visual loss for 30 seconds to 2 minutes, also in one eye at a time (amaurosis fugax), right more than left, repetitively during the last month of his 15-21 month exposure until 8-12 months after exposure ended, with a decrease in frequency by 7 months after moving out. These events happened at any time of day and rarely overlapped with headaches or tinnitus. He had normal ophthalmologic exams, normal MRI and MRA scans of the brain and associated arteries, and a normal evaluation for clotting abnormalities and vasculitis. The events resolved completely with normal vision. The same man experienced repetitive complex basilar migraines with aura after the first few months of his 15-21 month turbine exposure, involving daily bilateral paresis and paresthesias of his legs and occasional headache, tinnitus, and light-headedness. The leg symptoms resolved on the same schedule as the eye symptoms, though headaches and nausea continue to be triggered regularly by seasickness.

Discussion

The core symptoms of Wind Turbine Syndrome are sleep disturbance, headache, tinnitus, other ear and hearing sensations, disturbances to balance and equilibrium, nausea, anxiety, irritability, energy loss, 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. The subjects of this study had other types of health problems during exposure, discussed in "Other symptom clusters and isolated problems," but different types of study will be needed to find out if there is a link between these problems and wind turbine exposure.

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 directly 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 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 exposure or chemotherapy.

Visceral vibratory vestibular disturbance (VVVD)

The work of Mittelstaedt on visceral detectors of gravity,[34] and Balaban and others on balance-anxiety linkages,[35,36,37,38,39] opens a window on the VVVD symptom set. Balaban, a neuroscientist, has localized

[34] Mittelstaedt H. 1996. Somatic graviception. Biol Psychol 42(1-2): 53-74.
[35] 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.
[36] Balaban CD. 2002. Neural substrates linking balance control and anxiety. Physiology and Behavior 77: 469-75.
[37] Furman JM, Balaban CD, Jacob RG. 2001. Interface between vestibular dysfunction and anxiety: more than just psychogenicity. Otol Neurotol 22(3): 426-7.
[38] 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.
[39] Halberstadt A, Balaban CD. 2003. Organization of projections from the raphe nuclei to the vestibular nuclei in rats. Neuroscience 120(2): 573-94.

and described the neural connections among the vestibular organs of the middle ear, brain nuclei involved with balance processing, autonomic and somatic sensory inflow and outflow, the fear and anxiety associated with vertigo or a sudden feeling of postural instability, and aversive learning.[40] These form a coordinated, neurologically integrated system based in the parabrachial nucleus of the brainstem and an associated neural network.[41,42] Several aspects of this system need to be considered 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 (meaning 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.[43] Such receptors have been found in the kidneys and in structures supporting the great vessels in the mediastinum, among other locations.[44] Von Gierke (an older dean of vibration studies for the US space program) considers the inter-modality sensory conflict between the abdominal visceral graviceptors and the otolith organs to be a possible cause of motion sickness.[45]

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 brain then sends back out to the body. Balaban and colleagues describe how the parabrachial nucleus network receives motion and position information from visual, vestibular (inner ear), somatosensory, and visceral sensory input, and is linked to brain centers and circuits that mediate anxiety and fear, including the amygdala, a key mediator of fear reactions, and serotonin and norepinephrine-bearing neurons radiating from the midbrain.[46,47,48] Our sense of balance and stability in space is closely connected – neurologically – to fear and anxiety.

[40] Balaban and Yates 2004
[41] Balaban CD, Thayer JF. 2001. Neurological bases for balance-anxiety links. J Anx Disord 15: 53-79.
[42] Balaban 2002
[43] Balaban and Yates 2004
[44] 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.
[45] 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.
[46] Balaban and Thayer 2001
[47] Balaban 2002
[48] Halberstadt and Balaban 2003.

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 the sense of movement is ongoing and cannot be integrated with the evidence of the other senses, as happens in vertigo, there is a more prolonged fear reaction. The association of fear with vertigo has been known since ancient times.[49]

The third critical element is integrated neurologic outflow to the body from the parabrachial nucleus network to both the somatic (conscious, voluntary) and visceral (autonomic) effector systems. The somatic musculature is responsible for that fast foot on the brake, for righting movements of limbs, torso, and neck, and for breathing motions of the diaphragm and chest wall. The autonomic system is responsible for blood flow, heart rate, blood pressure, sweating, nausea, and other automatic, non-conscious modifications to visceral functioning. In a fear response, there is integrated outflow to these two systems. The parabrachial nucleus network is also involved in aversive learning, [50] an experience in which nausea, if present, plays a large role.

In VVVD, subjects detect unusual types of movement (pulsation, internal vibration, internal quivering) or other sensations (pressure, a sense of fighting something to breathe, pins and needles) in the chest or in the coordinated chest-abdominal internal space. The chest and abdomen are separated and unified by the diaphragm, which, as a striated somatic muscle, has fine-grained sensitivity to motion and stretch. The diaphragm sends signals to the brain which are specific and localizable in time and space, as opposed to visceral receptors, which send signals that are vague, like discomfort, malaise, fullness, or nausea. The diaphragm is tightly bound to one of the largest abdominal organs, the liver, and they move as a unit during breathing.

The chest, via the mouth, nose, trachea, smaller airways, and air sacs of the lungs, is open to the air. Pressure fluctuations in the air (sound waves) have free access to this airspace within the body when we breathe. Pressure fluctuations in the air also have access to the ear, which is designed to funnel them to the tympanic membrane, which concentrates their energy and transmits it to the inner ear. The ear and the

[49] Balaban and Thayer 2001
[50] Balaban and Thayer 2001

chest are different size spaces with walls of different mobility and elasticity. Hence they respond differently to air pressure fluctuations (sound waves) of different sizes.

Studies of whole body vibration focus on the easily mobile diaphragm and coupled abdominal organs. Being mobile, with the air of the lungs on one side and the soft abdominal wall on the other, this thoraco-abdominal system is easily set in motion by lower energy (amplitude) vibrations than are required to perturb other parts of the body.[51] Each part of the body has its own resonance frequency with regard to vibration. When an object is vibrated at its resonance frequency, the vibration is amplified. The resonant frequency of the thoraco-abdominal system, as it moves vertically towards and away from the lungs, lies between 4 and 8 Hz for adult humans.[52] Vibrations between 4 and 6 Hz set up resonances in the trunk with amplification up to 200%.[53] Related chest and abdominal effects are found in the same frequency range. Vibrations in the 4-8 Hz range influence breathing movements, 5-7 Hz can cause chest pains, 4-10 Hz abdominal pains, and 4-9 Hz a general feeling of discomfort.[54] In small children under 40 pounds, the vertical resonance or power absorption peaks at 7.5 Hz, as opposed to 4-5 Hz for adults.[55]

Low frequency noise can cause the human body to vibrate, as quantified by researchers in Japan.[56] The degree to which the body surface is induced to vibrate by low frequency noise is correlated with subjective unpleasantness (a sensation suggesting visceral as well as surface/somatic stimulation by the noise) .[57]

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

[51] Coermann RR, Ziegenruecker GH, Wittwer AL, von Gierke HE. 1960. The passive dynamic mechanical properties of the human thorax-abdominal system and of the whole body system. Aerospace Medicine 31(6): 443-55.
[52] von Gierke and Parker 1994
[53] Hedge, Alan, 2007
[54] Rasmussen 1982
[55] Giacomin J. 2005. Absorbed power of small children. Clin Biomech 20(4): 372-80.
[56] 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.
[57] 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.

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.

Perturbing the inner ear

I propose that disrupted stimulation of other channels of the balance system, especially the inner ear vestibular organs, is also likely to play a role in Wind Turbine Syndrome. Altogether, in subjects with or without VVVD, the Wind Turbine Syndrome core symptoms resemble the symptoms of a balance or vestibular disorder, meaning malfunctioning of the inner ear motion-detecting organs (peripheral vestibular dysfunction) or of brain processing of balance-related neural signals (central balance dysfunction). Near wind turbines, I suggest that these symptoms may arise through disturbed input to the classical pathways of motion and position perception (the visual, vestibular, and somatosensory channels), perhaps in an additive fashion. If several pathways are abnormally stimulated at the same time, it becomes even more likely that multisensory vestibular brain centers[58] will receive disordered or

[58] Dieterich M, Brandt T. 2008. Functional brain imaging of peripheral and central vestibular disorders. Brain, E-pub ahead of print, May 30, 2008, pp. 1-15.

including humans. These structures are used by fish (like us) to sense acceleration and tilt relative to gravity, but also to detect nearby perturbations in the water ("near-field sound") with peak sensitivities in the low frequency range between 40 and 120 Hz.[66] Certain fish are also known detect distant low-frequency sound, which they use to navigate relative to distant shores where waves are breaking.[67]

Most exciting, new research provides direct experimental evidence that normal human vestibular organs are sensitive to low-frequency vibration (which is the same as bone-conducted sound) and *much more sensitive than the cochlea*.[68] Among normal subjects, this sensitivity is "tuned" (has a sharp peak of sensitivity and response) at 100 Hz (G-G# 1½ octaves below middle C, keys 23-24 on a piano). Researchers applied carefully calibrated force and vibration frequency directly to the bony mastoid prominence behind the subjects' ears. They were able to elicit and measure responses of the vestibulo-ocular reflex at vibration intensities 15 dB below the subjects' hearing thresholds. In other words, the subjects could no longer hear the bone-conducted sound created by applying vibration to their mastoids, but the vestibular parts of the inner ear still picked up the vibration and transmitted signals into the balance and motion networks in the brain, resulting in specific types of eye muscle activation. Since dB is a base 10 logarithmic measure, *15 dB below* means a signal 0.0316 ($10^{-1.5}$), or about 3%, of the power or amplitude of the signal these normal subjects could hear.

The researchers note that "the very low thresholds we found are remarkable as they suggest that humans possess a frog- or fish-like sensory mechanism which appears to exceed the cochlea for detection of substrate-borne low-frequency vibration and which until now has not been properly recognized."[69] Thus the potential exists, in normal humans, for stimulation and disruption of balance signals from the inner ear by low-frequency noise and vibration, even when the noise or vibration does not seem especially loud.

Central balance processing

When there is conflict in healthy people among the signals coming from the different balance channels, the brain areas that integrate the information quickly compensate by suppressing or down-weighting the anomalous channel.[70] On functional brain scans, vestibular and visual cortical areas show a pattern of

[66] Fay RR, Simmons AM. 1999. The sense of hearing and fishes and amphibians. In *Comparative Hearing: Fish and Amphibians*, ed. Fay RR, Popper AN, pp. 269-317. Springer-Verlag, New York.
[67] Sand O, Karlsen HE. 1986. Detection of infrasound by the Atlantic cod. J Exp Biol 125: 197-204.
[68] Todd NPMc, Rosengren SM, Colebatch JG. 2008. Tuning and sensitivity of the human vestibular system to low-frequency vibration. Neuroscience Letters 444: 36-41.
[69] Todd et al. 2008, p. 41.
[70] Jacob RG, Redfern MS, Furman JM. 2008. Space and motion discomfort (SMD) and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry, E-pub ahead of print, July 24, 2008, pp. 1-20.

inverse activation and deactivation, such that vestibular activation deactivates visual cortex and vice versa.[71,72] In people with vestibular organ damage, long-term compensation promotes reliance on vision ("visual dependence") or on somatosensory input from muscles, tendons, joints, and skin ("surface dependence"). A visually dependent vestibular patient cannot adequately suppress visual input and up-weight vestibular signals because of pre-existing problems with the vestibular channel,[73] leaving the person dependent on visual perception of motion and position even in environments where the visual information is ambiguous. This can create fear of heights. It can also cause Space and Motion Discomfort, [74] a condition of discomfort in situations challenging to motion and position sense such as looking up at tall buildings, scanning shelves in a supermarket, closing eyes in the shower, leaning far back in a chair, driving through tunnels, riding in an elevator, riding in the back seat of a car, or reading in the car.[75]

Even without vestibular organ disease, some people have Space and Motion Discomfort due to central (brain) difficulty integrating balance signals into a coherent, moment-to-moment internal representation of position and motion. Balance testing with posturography shows that such people have difficulty down-weighting anomalous information from either the visual or somatosensory channel, or have a mild, central disorder of balance control with increased postural sway even under non-challenging conditions.[76,77,78]

Space and Motion Discomfort is common in patients with anxiety disorders,[79,80] migrainous vertigo,[81] and migraine-anxiety related dizziness.[82] Vertigo is especially characteristic of migraine and may at times occur as a migraine aura with or without headache.[83] In one study, dizziness or vertigo was found in 54%

[71] Brandt T, Bartenstein P, Janek A, Dieterich M. 1998. Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. Brain 121(Pt. 9): 1749-58.
[72] Brandt T, Dieterich M. 1999. The vestibular cortex: its locations, functions, and disorders. Ann NY Acad Sci 871: 293-312.
[73] Redfern MS, Yardley L, Bronstein AM. 2001. Visual influences on balance. J Anxiety Disord 15(1-2): 81-94.
[74] Jacob RG, Woody SR, Clark DB, Lilienfeld SO, Hirsch BE, Kucera GD, Furman JM, Durrant JD. 1993. Discomfort with space and motion: a possible marker of vestibular dysfunction assessed by the Situational Characteristics Questionnaire. J Psychopathol Behav Assess 15(4): 299-324.
[75] Jacob RG, Redfern MS, Furman JM. 2008. Space and motion discomfort (SMD) and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry, E-pub ahead of print, July 24, 2008, pp. 1-20. As a rural physician, I might also ask about driving past rows of parallel trees, especially with the low winter sun flashing between the trunks, as the rural equivalent of looking at lights on the wall of a tunnel.
[76] Redfern MS, Furman JM, Jacob RG. 2007. Visually induced postural sway in anxiety disorders. J Anxiety Disord 21(5): 704–16. NIH Public Access Author Manuscript, pp. 1-14.
[77] Jacob et al. 2008
[78] Furman JM, Balaban CD, Jacob RG, Marcus DA. 2005. Migraine-anxiety related dizziness (MARD): a new disorder? J Neurol Neurosurg Psychiatry 76: 1-8.
[79] Jacob et al. 2008
[80] Redfern et al. 2007
[81] Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. 2001. The interactions of migraine, vertigo, and migrainous vertigo. Neurology 56: 436-41.
[82] Furman et al. 2005
[83] Furman et al. 2005

of 200 migraine patients, half of whom also had a history of motion sickness, compared with 30% of those with tension-type headaches.[84] In a study of 72 patients with isolated recurrent vertigo, 61% were found to have migraine, compared to 10% in a control group of orthopedic patients.[85] Abnormal balance testing is seen in patients with migraine but not in those with tension-type headaches.[86] Balance testing shows that both peripheral and central balance abnormalities exist in migraine patients, both more prominent if dizziness or vertigo is an associated symptom.[87]

The dizziness associated with anxiety is not necessarily created by the anxiety, as is often assumed, but may have a component of disturbed balance control.[88,89] For example, the presence of panic or fear of heights is significantly associated with abnormalities on vestibular caloric testing.[90] A positive questionnaire for Space and Motion Discomfort is significantly associated with abnormalities on posturography showing either surface[91] or visual[92] dependence. In other types of balance testing, anxiety patients have been found to have greater vestibular sensitivity than normal controls.[93] Balance assessments of patients diagnosed with panic attacks or agoraphobia (fear of leaving the house) show a high proportion with vestibular abnormalities, in some studies greater than 80%, especially if the patients have episodes of dizziness between panic attacks.[94,95,96,97]

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, because their medical histories reveal a variety of risks for mild baseline balance dysfunction. These risks include motion sensitivity, migraine disorder, prior damage to inner ear organs from industrial noise

[84] Kayan A, Hood JD. 1984. Neuro-otological manifestations of migraine. Brain 107: 1123-42.
[85] Lee H, Sohn SI, Jung DK, Cho YW, Lim JG, Yi SD, Yi HA. 2002. Migraine and isolated recurrent vertigo of unknown cause. Neurol Res 24(7): 663-5.
[86] Ishizaki K, Mori N, Takeshima T,Fukuhara Y, Ijiri T, Kusumi M, Yasui K, Kowa H, Nakashima K.. 2002. Static stabilometry in patients with migraine and tension-type headache during a headache-free period. Psychiatry Clin Neurosci 56(1): 85-90.
[87] Furman et al. 2005
[88] Furman et al. 2005
[89] Eckhardt-Henn A, Breuer P, Thomalske C, Hoffmann SO, Hopf HC. 2003. Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. J Anxiety Disord 17(4): 369-88.
[90] Jacob et al. 2008
[91] Jacob et al. 2008
[92] Redfern et al. 2007
[93] Furman JM, Redfern MS, Jacob RG. 2006. Vestibulo-ocular function in anxiety disorders. J Vestib Res 16: 209-15.
[94] 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.
[95] Jacob RG, Furman JM, Durrant JD, Turner SM. 1996. Panic, agoraphobia, and vestibular dysfunction. Am J Psychiatry 153(4): 503-12.
[96] 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.
[97] 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.

exposure or chemotherapy, autoimmune disease,[98] fibromyalgia,[99] normal aging (over 50), and normal early childhood.[100,101] Other potential risks for chronic vestibular dysfunction are whiplash injury and head injury, including concussions and milder head impacts without loss of consciousness.[102,103,104]

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 familiar with their struggles with short-term memory, concentration, multitasking, arithmetic, and reading.[105,106] In the perilymphatic fistula syndrome, for example (a form of inner ear pathology that can follow whiplash, minor head injuries, or pressure trauma to the ear), symptoms of dizziness, headache, stiff neck, and disturbed sleep are accompanied by marked mental performance deficiencies compared to the patient's baseline.[107] Such cognitive symptoms are difficult to evaluate clinically and are often dismissed as psychological in origin.[108] However, recent research using imaging and other modalities shows that vestibular function exerts a powerful influence over human thinking and memory.

The vestibular system is ancient in the vertebrate lineage (as reviewed above). Its neural connections ramify widely in both older and more recently evolved parts of the brain, including the brainstem, midbrain, cerebellum, and occipital, parietal, and frontal cortex.[109] Vestibular injury causes specific deficits, but not general cognitive impairment.[110] Vestibular effects on cognition are often attributed to competing stimuli (meaning challenges to movement and position sense draw attention away from

[98] 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.
[99] Rosenhall U, Johansson G, Orndahl G. 1996. Otoneurologic and audiologic findings in fibromyalgia. Scand J Rehabil Med 28(4): 225-32.
[100] Foudriat BA, Di Fabio RP, Anderson JH. 1993. Sensory organization of balance responses in children 3-6 years of age: a normative study with diagnostic implications. Int J Pediatr Otorhinolaryngol 27(3): 255-71.
[101] Steindl R, Kunz K, Schrott-Fischer A, Scholtz AW. 2006. Effect of age and sex on maturation of sensory systems and balance control. Dev Med Child Neurol 48(6): 477-82.
[102] Grimm RJ, Hemenway WG, Lebray PR, Black FO. 1989. The perilymph fistula syndrome defined in mild head trauma. Acta Otolaryngol Suppl 464: 1-40.
[103] 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.
[104] Claussen CF, Claussen E. 1995. Neurootological contributions to the diagnostic follow-up after whiplash injuries. Acta Otolaryngol Suppl 520, Pt. 1: 53-6.
[105] Hanes and McCollum 2006
[106] Grimm et al. 1989
[107] Grimm et al. 1989
[108] Hanes and McCollum 2006
[109] Dieterich and Brandt 2008
[110] Hanes and McCollum 2006

cognitive tasks) but may actually reflect, instead, the direct dependence of certain cognitive operations on the vestibular system.[111]

Vestibular input is critical for spatial thinking, body and spatial awareness, spatial memory, and complex spatial or map calculations.[112] 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.[113] Patients with 5-10 year histories of bilateral vestibular loss showed 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).[114] In a test of general memory, however, these patients were no different from controls.[115] Vestibular signaling to the hippocampus is known to occur in both humans and other primates via a direct, two-neuron linkage through the posterior thalamus; there are also other proposed neural pathways.[116]

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.[117] This is true 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.[118] Vestibular stimulation in both humans and other primates activates a variety of areas in the parietal cortex, including 1) a core vestibular processing area (posterior insula), 2) the somatosensory strip, 3) areas involved in hemineglect in stroke patients (ventral parietal), 4) and a region "known to be involved in multimodal coordinate transformations and representation of space" (intraparietal sulcus), which is a principal site for arithmetic and counting tasks.[119] Hemineglect is a condition after right-sided parietal stroke in which a patient can have so much unawareness of the left side of space that he is oblivious to his 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

[111] Hanes and McCollum 2006
[112] Hanes and McCollum 2006
[113] 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.
[114] Brandt et al. 2005
[115] Brandt et al. 2005
[116] Brandt et al. 2005
[117] 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.
[118] Mast et al. 2006. 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.
[119] Hanes and McCollum 2006, p. 82.

representation of space.[120,121] Vestibular stimulation also improves hemineglect patients' performance on tasks of visual localization and visual-spatial memory retrieval. At baseline, and again 24 hrs after the experiement, their responses were biased away from the left side, but this bias was corrected or improved immediately after left vestibular stimulation.[122]

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,[123,124] clock representations of time,[125] and spelling at the beginnings (left) or ends (right) of words (errors occur opposite to the side of the parietal lesion).[126] 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.[127] At the other extreme of mental functioning, it has been found that great mathematicians think of numbers in spatial terms,[128] which "may be more efficient because it is grounded in the actual neural representation of numbers."[129] A recent study of outstanding human memorizers shows that spatially oriented strategies are also critical to good memory, by providing an efficient framework for memory organization and retrieval.[130]

In summary, 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) and 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, to figure out the most efficient path for a set of errands, remember the path and images of the items to be obtained, search for the items on the shelf, and judge 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 imagine what has gone wrong inside a device and initiate a

[120] Geminiani G, Bottini G. 1992. Mental representation and temporary recovery from unilateral neglect after vestibular stimulation. J Neurol Neurosurg Psychiatry 55(4): 332-3.
[121] Cappa S, Sterzi R, Vallar G, Bisiach E. 1987. Remission of hemineglect and anosognosia during vestibular stimulation. Neuropsychologia 25: 775-82.
[122] Geminiani and Bottini 1992
[123] Zorzi M, Priftis K, Umilta C. 2002. Brain damage: Neglect disrupts the mental number line. Nature 417: 138-9.
[124] Vuilleumier P, Ortigue S, Brugger P. 2004. The number space and neglect. Cortex 40(2): 399-410.
[125] Vuilleumier et al. 2004
[126] Hillis HE, Caramazza A. 1995. Spatially specific deficits in processing graphemic representations in reading and writing. Brain Lang 48 (3): 263-308.
[127] Zorzi et al. 2002
[128] Hadamard J. 1996. *The Mathematician's Mind: The Psychology of Invention in the Mathematical Field*. Princeton University Press, NJ. *In* Zorzi et al. 2002.
[129] Zorzi et al. 2002
[130] Maguire EA, Valentine ER, Wilding JM, Kapur N. 2003. Routes to remembering: the brains behind superior memory. Nat Neurosci 6(1): 90-5.

repair. It is used for understanding the images in a movie or TV show with the sensitivity not to miss subtle visual clues. In this context, it is easy to see how vestibular disturbance could impact concentration (which means the ability to perform thinking tasks successfully and efficiently) and memory. Vestibular disturbance also has the potential to affect reading directly via the reflex control exerted by semicircular canal and otolith organs over eye movements (vestibulo-ocular reflex).

Effects on concentration and memory were nearly ubiquitous in the present study, if one includes all subjects that told me about any problem in this area. For some subjects the deficits were dramatic compared to baseline (before exposure), including the 7 out of 10 school-age children and teens who showed 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 ($p = 0.018$). Though sleep deprivation and disturbance undoubtedly play a role, qualitative aspects of the mental performance deficiencies suggests a mechanism other than sleep disturbance 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, etc.) and they actively explore the physical world through this 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 play 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.[131] The authors use this as evidence that "predictive mechanisms of physical laws of motion are represented in the human brain"[132] 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

[131] 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.
[132] Indovina et al. 2005

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,[133] and is correlated with measures of language processing such as speech recognition.[134] Airplane noise, which has a large low frequency component, has a stronger effect than traffic noise in some studies,[135] but traffic noise is also shown to have modest effects on memory in quieter communities.[136] Most studies are cross-sectional, but a longitudinal or cohort study, done when an airport was closed in one location and opened in another, showed similar effects on reading acquisition.[137] 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.[138] These effects on reading occur at sound levels far less than those needed to produce hearing damage.[139] Children with pre-existing reading deficiencies and children at higher grade levels are more affected, and longer exposure produces larger deficits.[140]

Effects suggestive 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

[133] 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.
[134] Evans GW, Maxwell L. 1997. Chronic noise exposure and reading deficits: the mediating effects of language acquisition. Environment and Behavior 29(5): 638-56.
[135] 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.
[136] Lercher P, Evans GW, Meis M. Ambient noise and cognitive processes among primary schoolchildren. Environment and Behavior 35(6): 725-35.
[137] 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.
[138] Cohen S, Glass DC, Singer JE. 1973. Apartment noise, auditory discrimination, and reading ability in children. Journal of Experimental Social Psychology 9: 407-22.
[139] Evans GW. 2006. Child development and the physical environment. Annu Rev Psychol 57: 423-51.
[140] Evans 2006, p. 426.

superficial. The results also suggest that the health status sampling was inadequate. For example, the study found that only 2% of respondents indicated they had chronic migraine disorder,[189] whereas the population prevalence is probably 12-13%. Likewise, tinnitus prevalence in this study was 2%, whereas 4% is a more likely population figure for the average age of 54.[190] Tinnitus prevalence was not correlated with age in this sample,[191] while in reality tinnitus has a well-documented pattern of increasing prevalence with advancing age.[192] In sum, this survey did not adequately sample health status, and was not designed with comparison groups so as to detect differences.

Recommendations

For physicians practicing near wind turbine installations, I suggest incorporating proximity to turbines into the personal and social history in a neutral and non-suggestive way, especially for the types of symptoms described in this report.

With regard to turbine setback from dwellings: In Table 1B we see that the subjects in the current study lived between 305 m (1000 ft) and 1.5 km (4900 ft or 0.93 mi) from the closest turbine. There were three severely affected families at 930-1000 m (3000-3300 ft) from turbines. This study suggests that communities that allow 1000-1500 ft (305-457 m) setbacks from houses may have families who need to move after turbines go into operation. All turbine ordinances, I believe, should establish mechanisms to ensure that turbine developers will buy out any affected family at the full pre-turbine value of their home, so that people are not trapped between unlivable lives and destitution through home abandonment. By shifting the burden of this expense to turbine developers, I would hope that developers might have a stronger incentive to improve their techniques for noise prediction and to accept noise level criteria recommended by such agencies as the World Health Organization and the International Standards Organization,[193] and fortified by the findings of Pedersen and van den Berg (above). With regard to families already affected, developer and permitting agencies share the responsibility for turbines built too close to homes, and together need to provide the financial means for these families to reestablish their lives at their previous level of health, comfort, and prosperity.

[189] van den Berg et al. 2008b, p. 48.
[190] National Institute on Deafness and Other Communication Disorders, USA, website, "Prevalence of Chronic Tinnitus." 2009. http://www.nidcd.nih.gov/health/statistics/prevalence.htm
[191] van den Berg et al. 2008b, p. 47.
[192] National Institute on Deafness and Other Communication Disorders, USA, website, "Prevalence of Chronic Tinnitus." 2009. http://www.nidcd.nih.gov/health/statistics/prevalence.htm
[193] See George Kamperman and Richard James, *The "How To" guide to siting wind turbines to prevent health risks from sound,* 47 pp., at http://www.windturbinesyndrome.com/?p=925 (2008).

I support the recommendations for noise level criteria and procedures for noise monitoring by George Kamperman and Richard James.[194] A single setback distance may not be both protective and fair in all environments with all types of turbines, but it is clear, from the current study and others, that minimum protective distances need to be 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, more than the 1.6 km (5250 ft or 1 mi) at which there were affected subjects in Dr. Harry's UK study, [195] and, in mountainous terrain, more than the 2-3.5 km (1.24-2.2 mi) at which there were symptomatic subjects in Professor Robyn Phipps's New Zealand study. [196] Two kilometers, or 1.24 miles, remains the baseline shortest setback from residences (and hospitals, school, nursing homes, etc.) that communities should consider. In mountainous terrain, 2 miles (3.2 km) 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.

Suggestions for further research

- Epidemiologic studies comparing populations exposed and not exposed to wind turbines with regard to the prevalence of specific symptoms, such as tinnitus and balance disorders. Such studies might be best conducted in European countries that have both national health data systems and significant numbers of wind turbines.

- Case series by neurotologists internationally, who are able to do appropriate objective examination and testing in addition to clinical history.

- Collaboration between physicians and independent noise engineers to correlate specific frequencies and intensities of sound and vibration with subjects' symptoms, and to establish a standard protocol for noise sampling that captures the elements relevant to health effects.

- Further clinical/laboratory research on the effects of low frequency noise and vibration on the human vestibular system.

[194] Kamperman GW, James RR, *The "How To" guide to siting wind turbines to prevent health risks from sound*, 47 pp., at http://www.windturbinesyndrome.com/?p=925 (2008). Presented in shorter form, 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.
[195] Harry, Amanda, 2007.
[196] Phipps, Robyn, 2007.

Wind Turbine Syndrome for Non-Clinicians

Abstract

I interviewed 10 families living near large (1.5 to 3 MW) wind turbines, all of which were built since 2004. This gave me 38 people, from infants to age 75. Their symptoms formed a cluster (see Glossary for clinical terms):

1) sleep disturbance
2) headache
3) tinnitus (pronounced "tin´-ah-tus": ringing or buzzing in the ears)
4) ear pressure
5) dizziness (a general term that includes vertigo, lightheadedness, sensation of almost fainting, etc.)
6) vertigo (clinically, vertigo refers to the sensation of spinning, or the room moving)
7) nausea
8) visual blurring
9) tachycardia (rapid heart rate)
10) irritability
11) problems with concentration and memory
12) panic episodes associated with sensations of internal pulsation or quivering, which arise while awake or asleep

The families not only lived near turbines and developed new symptoms; they moved away from the turbines (because they were so troubled, often abandoning their homes) and the symptoms, significantly, went away.

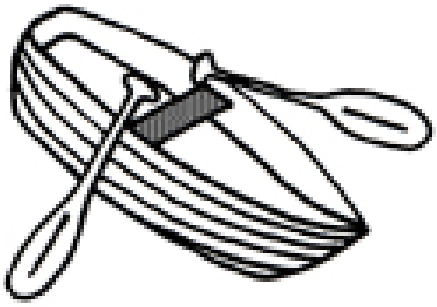
Hence, the definitive result of my report is that wind turbines cause the symptoms of Wind Turbine Syndrome (WTS).

Let's clarify something immediately. Not everyone living near turbines gets these symptoms. As a solo, unfunded researcher I could not get the samples needed to figure out what percentages of people at what distances get the symptoms. This needs to be done next. But I could (and did) look at the question of why some people are susceptible and others not, plus who is susceptible, and I used these patterns to explore the pathophysiology of Wind Turbine Syndrome: what's going on inside people to cause these specific symptoms.

I would like readers to be able to look at this study—including the detailed accounts I provide of people's experiences around turbines and their health backgrounds—and be able to make their own decisions about whether they should be exposed to these machines.

That said, I was able to prove mathematically that people with pre-existing migraines, motion sensitivity (such as car-sickness or seasickness), or inner ear damage are especially vulnerable to these symptoms. Equally as interesting, I was able to demonstrate that people with anxiety or other preexisting mental health problems are not especially susceptible to these symptoms.

This contradicts wind industry literature, which argues that people who worry about or otherwise dislike turbines ringing their homes are the ones getting ill. I show this to be complete nonsense.



Here is what's going on, as I put together the evidence. *Low frequency noise tricks the body's balance system into thinking it's moving*. Like seasickness. (It's vital to understand that the human balance system is a complex brain system receiving nerve signals from the inner ear, eyes, muscles and joints, and inside the chest and abdomen. Because the eyes are involved, visual disturbance from the blades' shadow flicker adds to the balance disturbance.)

Let me repeat this, because its significance is huge. *Low frequency noise from turbines appears to deceive the body into thinking it's moving*. So what, you say? Not so fast! Research within the last 10 years has demonstrated conclusively that *the way our bodies register balance and motion directly affects an astonishing array of brain functions*.

How? By direct neurologic linkages connecting the organs of balance to various, seemingly unrelated brain functions.

I'll rephrase this, since it's critical to the argument of this report. *The way our bodies perceive balance and motion in turn influences a host of brain functions which at first glance might appear to be entirely unrelated to balance and motion*. As I said, this is what the latest "balance" research tells us—more accurately, balance research combined with psychiatric, neurologic and cognitive research.

Incidentally, the people specializing in this kind of research are called *neuro-otologists*—from *neuro* for brain, and *oto* for ear.

And what are these seemingly unrelated brain functions affected by our perception of balance and motion?

a) *Alerting and awakening*

b) *Memory*

c) *Spatial processing*. Spatial processing is defined as the ability to 1) picture things, 2) remember where things are, 3) remember how to get somewhere, 4) understand how things work, 5) figure out how to put something together or fix it, 6) figure out the most efficient order and timing of something (such as work around the kitchen, farm, fishing boat, property, or a series of errands), 7) remember what you're looking for when you get someplace (such as errands in town), 8) understand math concepts, 9) along with a host of other critical thinking functions.

d) *The physiologic manifestations of fear*. This means increased heart rate and blood pressure, sweating, nausea, and hyper-alertness.

e) *Aversive learning*. This is when something bad happens and as a result of the experience you now know to avoid the situation in future. The classic illustration in both animals and people comes from vomiting after you eat something. You avoid that food afterwards for a long time. (Remember that experience as a child?) Aversive learning is so imprinted on mammals (we are mammals) that even the environment associated with this experience can trigger the feelings of nausea—to continue the nausea illustration. Merely smelling that particular food, or being in the same restaurant, or just thinking about that particular food, or seeing the food—makes you queasy. Aversive learning is potent and, clearly, an evolutionarily old reflex in mammals.

Okay. *Alerting and awakening*, *memory*, *spatial processing*, the *physiological manifestations of fear*, and *aversive learning*. All five brain functions are profoundly affected by our sense of balance and motion. All five get messed up when our sense of balance and motion is thrown off.

Back to wind turbines. Open any online newspaper article discussing Wind Turbine Syndrome and you almost invariably discover that someone has posted a comment ridiculing the whole idea for the obvious reason that there's no conceivable way such a disparate range of health problems—memory deficits, spatial processing deficits, anxiety and fear and panic, and aversive learning—could possibly be triggered by a wind turbine, of all things. Clearly, continues our brilliant curbside commentator, people who live near turbines and report these symptoms are making them up (probably because they don't like the darn things), and just as clearly the doctor who takes these seriously (that would be me) is a fake.

To which I respond: Clearly the authors of these brilliant gems of logic (forgive my cynicism) are neither neurobiologists nor clinicians—nor are they experiencing the symptoms which are clearly, unambiguously reported by many people living in the shadow (as it were) of industrial wind turbines.

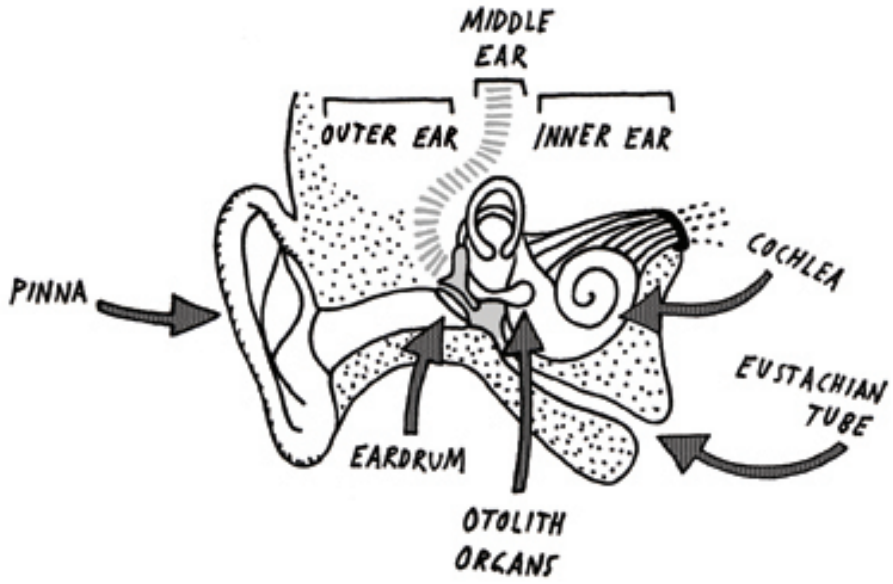
Back to real medicine. Wind Turbine Syndrome symptoms, outlined above, occur together *because humans are hardwired to exhibit these precise symptoms when their balance and motion sensors are dis-regulated*—as happens to many people living near wind turbines.

It's important to emphasize these symptoms are not psychological (as if people are fabricating them); they are neurological. People have no control whatsoever over their response to the turbines. It happens automatically. One can't turn on and turn off these symptoms.

We can be emphatic about this because *balance signals* (called *vestibular* signals) *are the one kind of sensory signal we simply cannot tune out*. You can tune out (ignore) what you see and hear—but not what comes in from your sense of balance. Call it a law of nature, if you like.

And what provides our sense of balance? Balance comes from a combination of signals. I'll rephrase this: balance comes from *clusters of signals from different body organs*. One source being, of course, the inner ear.

Stop. We need to review the anatomy of the inner ear, since it's essential to understanding Wind Turbine Syndrome.



Start with the weird flap of skin on the side of your head, necessary for holding up your glasses and, naturally, earrings. This is not the outer ear; it's the pinna. (Boxers get cabbage pinna.) The outer ear is where you put Q-Tips, and where your two-year old stores beads and other treasures, where earwax lives, and where water gets lodged when you shower—and you have to shake it out. The outer ear is a blind pouch ending at the eardrum, sealing off the pouch at the inner end.

Then comes the middle ear: the place between the eardrum and what's called the oval window. This is the part of the ear that gets infected in little kids. (Moms, remember all those times you took Johnny to the doctor and she said, "Yup, Johnny has an ear infection." This is after Johnny woke up screaming in the night, after having a cold for three days.) The middle ear is open to the air, through the Eustachian (pronounced "U-station") tube from the back of the throat (up behind the nose). And the middle ear houses those wonderful three little bones, incus ("ink-us"), malleus ("mal-ee-us"), and stapes ("stay-peas"), that are linked in a chain. Incus, malleus, and stapes transmit the energy of the vibrating eardrum to the inner ear.

This brings us to our destination in this mini-lesson: the inner ear. The inner ear consists of the semicircular canals (which you remember from high school biology) and the so-called otolith organs (which you probably don't remember from high school biology). The otolith organs are one of the keys to understanding Wind Turbine Syndrome. They consist of two little membranous sacs which are attached to the cochlea ("coke-leah") (the spiral-shaped, membranous organ that transduces the mechanical energy of sound into neural signals) and to the semicircular canals (membranous organs

which make a semi-circle in each of the three planes of movement—vertical forward, vertical sideways, and horizontal—and transduce angular acceleration: if you nod or turn your head, they detect it).

Bear with me a moment longer; we're almost there. Embedded in the two otolith organs are—believe it or not—rocks. (Remember when your teacher accused you having rocks in your head?) Well, not really rocks; they're tiny. In fact they're microscopic stones made of calcium carbonate (yes, chalk, or clam shells). The weight of these stones allows us to detect gravity and linear acceleration, by sending signals to the brain via minute neural (nerve) hair cells.

In people predisposed to Wind Turbine Syndrome the otolith organs (utricle and saccule) are (I propose) being rocked abnormally, thus sending aberrant motion signals to the brain. Either this, or their inner ear organs in general—which are all interconnected as a "membranous labyrinth"—may be hyper-sensitive to pressure changes, such as those produced by turbines.

We're in the presence, here, of a truly ancient anatomical structure. Many millions of years old. Biologists call it the macule ("mack-ewell"), which just means the "spot." The macule is critical in fish and us, and everything in between.

The macule: the membranous structure with hair cells embedded in the membrane and the otoliths fixed on top in a protein matrix. Mother Nature loved the macule so much that she conserved it through eons of evolution. Fish use these organs to figure out, instantaneously, which way is up, just like us. Fish use the macule for detecting pressure shifts in the water, such as the waves made by nearby predators or prey, or the low frequency noise traveling over long distances from waves breaking on shorelines, thus helping them navigate during migration.

Otoliths. The macule. Very ancient. Ancient and fundamental organs of balance and motion that appear to be thrown out of whack by wind turbines, with consequences I have called Wind Turbine Syndrome.

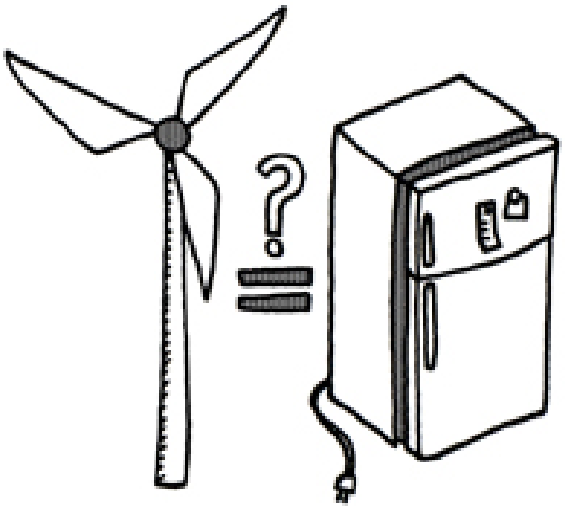
Back, now, to what provides us with our sense of balance. I said balance comes from a combination of signals, and I just explained how some of them originate in the inner ear. Besides the inner ear, the eyes also send motion and position signals to the brain. So, too, do muscles and joints all over the body, involving what are called "stretch" receptors, telling us where we are in space.

And lastly, we maintain our balance by newly discovered stretch and pressure receptors in the chest and abdomen. These tiny receptors rely on various organs, including blood vessels and blood itself, as weights to detect the body's orientation to gravity and other forms of acceleration.

All this is the proper context for studying people's health complaints from wind turbines. Health complaints that are routinely dismissed by the wind industry as nonsense. (Not unlike the tobacco industry dismissing health issues from smoking.) The wind industry, however, is not made up of clinicians, nor is it made up of people suffering from wind turbines.

In time, I hope, researchers will be able to measure and correlate wind turbine noise (audible and sub-audible) and vibration with the symptoms people experience in real time—that is, while they're actually experiencing the symptoms. Until that happens, I offer this report as a pilot study.

Introduction and Background



Developers say turbines are quiet. No louder than a household refrigerator. With this (false) claim, they easily convince local governments it's okay to erect turbines mere hundreds of feet from people's homes. Nearly in their backyards, in many instances.

Wind turbine setbacks, in other words, are wind-industry-driven. Virtually no government regulation.

This is where my phone (and email) starts ringing. People from around the world contacting me to say, often with great emotion in their voice, they haven't slept well (if at all) since the turbines were installed 1500 feet (and more) from their back door. Not just insomnia, but a host of health issues, again, since the turbines in the neighbor's field began operation.

For over four years I've been listening to their complaints. Describing symptoms that are remarkably consistent, person to person. Consistent and, often, debilitating. Symptoms, I began realizing, that suggest people's balance systems are getting messed up.

I realized what's needed is a clinical definition of the way people are getting sick when they live near wind turbines. If we can nail down the pathophysiology of their illness, we're in a better position to figure out,

a) precisely what's causing it
b) how many people are getting it
c) who is susceptible
d) how to control or prevent it.

This became my goal: figure out the pathophysiology of the illness cluster they all describe.

So let's begin. Except immediately there's a problem. Which is, developers focus on noise. They hire an acoustician to measure noise levels (incidentally, there are many ways to slice and dice noise measurements), who then writes a report saying, in effect,

a) the turbines are emitting this (whatever) dB of noise
b) the conventional acoustical wisdom about this range of dB says it doesn't create health problems
c) hence, we conclude these people are faking their symptoms
d) end of story

I turn the above sequence inside out. We need to begin with c) *symptoms*, not a) *noise levels*. The symptoms are consistent person to person, no matter if it's England or Canada or what have you. Furthermore, the symptom cluster fits with known clinical mechanisms. There is no mystery here.

Hence, the symptom cluster becomes—must become—the chief reference point.

When one measures noise, one must refine noise measurements so as to answer what it is about the noise spectrum *at this moment, when people are actually getting symptoms*, versus that other moment when they're not getting symptoms. *This* is the value of noise measurements.

Other published reports on health and wind turbines, by the way, find the identical set of symptoms to what I found. In my report I review papers by Dr. Amanda Harry, Barbara Frey and Peter Hadden, and Prof. Robyn Phipps.

a) Harry found all the same problems, and, interestingly, her group of patients was shifted toward older folks. Moreover, she sampled the same way I did: she talked to people who were bothered.
b) Frey and Hadden document the same symptoms in people's own narratives.
c) Phipps mailed questionnaires to everyone living within 9.3 miles of turbines. She got affirmative responses about unpleasant physical symptoms from people living at least 1.24 miles from turbines, on up to 2.2 miles. Some even further. Phipps got even more detail about what they were experiencing because (almost) 7% were so distressed, they telephoned her to describe more specifically their problems from turbine noise and vibration—almost all of them exhibiting disturbed sleep.

My own subjects make it clear their problems are caused by noise and vibration and, in some instances, moving blade shadows. What's more, my subjects notice that symptoms came and went with the wind's direction and strength, turbine spinning speed, which way the turbines were facing, and particular sounds coming from the turbines. In other words, they see their symptoms going up and down depending on what the turbines are doing. They also know that the quality of noise is strange and bothersome even compared to other loud noises, like nearby trains or cars. A few people were specifically bothered by the shadow flicker in rooms or blade shadows sweeping the landscape.

Above all, the symptoms went away when they left home and the turbines, and symptoms returned when they came back home.

Again, the only rational way to study the problem is *symptoms first*, *noise measurements second*, not the reverse.

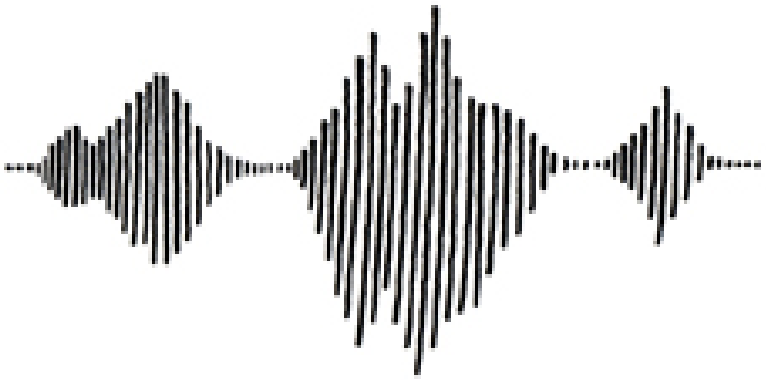
Noise. You, dear reader, need to understand what noise is before we go further. If you're confident your grasp of noise is sophisticated, then skip the next few paragraphs. Otherwise, here we go.

Wind turbines indisputably make noise from infrasonic (below what you hear), through the range you can hear (audible, in other words), to ultrasonic (above what you hear). This is well established. By "above" and "below" we mean "pitch." "Frequency" means "pitch." Hence, low frequency noise (LFN) means "low-pitched," like the low notes on a piano. High frequency means high-pitched, like the "s" sounds in human speech.

Noise also has a quality of intensity or power which, if the sound is within the hearing range, we call "loudness." Loudness or intensity is measured as "decibels" or "sound pressure level." These are both measures of how much energy, or power, is in the sound wave, and is also called “amplitude.”

Next definition: wavelength. A high frequency wave means a short wavelength (as with ocean waves: when the waves arrive in rapid succession the distance between the wave peaks is short). Low frequency means a long wavelength: peaks further apart, although the waves travel at the same speed.

Now things get interesting. *A sound wave in the air is a sequence of pressure changes*. A sound wave in a solid is more like a vibration. (In fact the word "vibration" is technically used to refer only to what happens in solids.)



(As an aside, I will often talk about noise and vibration together because I'm talking about a continuum of energy as it passes through different substances. For instance, a sound wave coming through the air, hitting a building, can make the walls vibrate, which in turn sets up sound waves inside the room.)

When symptoms of the sort we’re dealing with, here, have been medically studied, they are typically associated with lower sound frequency ranges—below hearing range or in the lower part of the hearing range. With further research into Wind Turbine Syndrome it may turn out that some of the turbine noise

in the higher frequencies is also causing symptoms; however, the chief noise culprit, from past clinical studies, appears to be low frequency noise.

Loudness, or intensity, also matters. How loud does a low frequency noise have to be to trigger health effects? Acousticians are taught, "*If you can't hear it, it can't hurt you!*" This, however, is an oversimplification of how the body works. Noise health standards focus on protecting people's ears from loud noise that could damage their hearing. *What these standards ignore is pressure detection by the balance mechanism or pressure or vibration effects on other parts of the body*.

There's the rub.

When we decide to look at symptoms first, the noise issue becomes very simple. People's symptoms come and go. Acousticians need to measure noise levels when symptoms are present and compare these to noise levels when symptoms are absent. In this manner they can find out exactly *what frequencies* at *what intensities* are causing symptoms.

In the *Discussion* section of my clinical report, I give two examples of published accounts by German noise control engineers correlating symptoms with their noise measurements. In each case the symptoms (very similar to Wind Turbine Syndrome, incidentally) were due to very low frequency noise. In one case the noise was identified but not the noise source; in the other case the source was a large building ventilator fan.

Back to my crash course on noise. (Yes, the pun is intended.) Resonance. Resonance is what happens inside the body of a guitar or violin after a string is plucked or disturbed by a bow. It's like an echo inside a space. Thus certain wavelengths bounce back and forth very efficiently, given the size of that space. The walls of the space tend to vibrate at particular frequencies, and if the natural vibration frequency of the wall is the same as the incoming sound frequency, the wall itself (guitar wall, violin wall) can give an added "punch" to the sound, making it louder.

This is a lot like pumping a swing. (We all did this as children.) Swinging is a kind of wave function, like sound, with frequency and amplitude. The frequency of the swing is how many times per minute it's going back and forth. Frequency depends on the length of the ropes—a short swing swings faster. Amplitude is how high the child is swinging. Resonance is a child who knows how to pump (add some

energy to the swinging) at exactly the right time to increase the amplitude (swing higher). The frequency stays the same, but, as the child pumps, she swings higher and higher.

The child pumping is like the wall of a resonant chamber; it provides a little push to the "wave" at exactly the right time.

Okay, course on noise is over. Now let's apply it to Wind Turbine Syndrome.

Resonances occur inside body spaces and in solid but flexible or elastic parts of the body, such as along the spine. Different parts of the body have different resonant frequencies. Many of these are in the low frequency range. When a sound wave or vibration hits the body, it's more likely to set up vibrations in a body part with a matching resonant frequency.

In Wind Turbine Syndrome, an important body resonance is the resonance of the chest and abdominal space. The chest wall is made of elastic muscles, bones, cartilage, tendons, and ligaments, giving the chest a natural recoil we use in breathing. We use energy to expand the chest to breathe in, but much of the force to push the air back out comes from the elastic recoil of the chest.

One of the important parts of the breathing mechanism is the diaphragm muscle at the bottom of the chest. It's dome-shaped, like the top of an egg. When you take a breath, the diaphragm flattens. As it flattens, it pulls down, thus expanding the chest space and pushing on the abdominal space. The abdominal space is very soft and flexible, the front being thin sheets of muscle, skin, and other soft tissues, without bone or cartilage. So when you breathe in, your stomach sticks out. When you relax the diaphragm muscle, it springs back to its dome shape and it pushes air out. Natural elasticity at work.

Hence, when air pressure waves enter the lung, it takes very little energy in the air pressure waves to set this very mobile system vibrating. At frequencies between 4 and 8 times per second, the diaphragm will vibrate. Frequencies 4-8 times/second are low frequency noise or infrasound, below hearing range.

Not only the diaphragm vibrates, but the entire mass of internal organs in the abdomen swing up and down with diaphragm movements. One of the largest abdominal organs, the liver, is attached to the underside of the diaphragm.

There are other places in the body with resonance, including the eyes (globes with bone around them and less dense material inside) and the brain case. Even the spine (backbone) has a resonance frequency. The spine is elastic. If it's vibrated at a particular frequency it can set up a vertical vibration along the spine.

Even very small body parts, like blood vessels inside the brain, have resonances that influence the damage caused by air pressure waves from a nearby blast, for example.

In sum, what we casually call *noise* can have a powerful impact on numerous internal structures and cavities. We will see the significance of all this in the Discussion, below.

Before moving on to the Methods section, a few words about measuring sound power and what's called "A-weighting" and "C-weighting." It's difficult to measure the loudness, or energy, of sound in consistent, reproducible ways, especially at low frequencies. A-weighted and C-weighted "networks" in sound-measuring equipment screen sound energy (i.e., loudness) according to frequency. A-weighting screens out most of the low frequency and high frequency sounds; it's biased toward what the human ear hears best. C-weighting includes more of the low frequency sounds, but not the very lowest frequencies.

It's easy to obtain standardized measuring equipment with these two weighting networks (A and C), but measuring the power of the lowest frequency sound requires expensive and specialized equipment that's not standardized among models. Nevertheless, if we are to fully understand Wind Turbine Syndrome, it's at this lowest of low frequencies that measurements must be made.

Methods

I used what's called a *case series* as my research protocol. (Remember my definition of a case series from the Preface: "*A descriptive account of a series of individuals with the same new medical problem.*")

In medical research, *case series* don't usually have control (comparison) groups. However, I added a new wrinkle to my study, based on my training in field ecology: despite not having a formal control (comparison) group, I chose subjects and arranged the way I collected information so I could create comparisons.

First, to call this a wind-turbine-associated problem at all, I compared how people were *during exposure* to how they were when *not exposed*, and I specified that "not exposed" meant both *before* and *after* living near turbines. *All my subjects saw their problems start soon after turbines went on-line near their homes, and all saw their problems go away when they were away from the turbines.*

Second, to discover medical risk factors, I compared subjects who exhibited particular symptoms more intensely, to those who did not. Then I looked at whether these differences were influenced by age, underlying health conditions, etc.

There was a third type of implicit comparison going on—to the population at large. For example, both I and Dr. Harry sampled in the same way—we interviewed affected adults—and we both wound up with samples shifted toward people in their 50's or older. This suggests that older people are more often affected, since older people are over-represented in our samples. (This makes medical sense, and also corresponds to patterns of noise complaints in other, non-wind-turbine settings).

Additionally, in my study there are more people with underlying migraine than in the general population, suggesting that people with migraine are, like older people, more susceptible.

Hence, the more elderly and migraineurs (people who get migraines) stand out in my investigations.

Now, let's consider what a standard epidemiologic study of Wind Turbine Syndrome might look like, as distinct from my *case series* approach. When a scientist sets out to perform an epidemiologic study, he (she) begins by defining two identical groups to be studied. Notice, this is *before* either group is exposed to the (supposedly) disease-causing agent. One group being the *study group*, the other the *control group*. The study group, of course, is the so-called guinea pig: the individuals about to be exposed to the potential problem, in this case wind turbines. The control group is identical in every conceivable way to the study group: by age, by sex, and (in the case of wind turbines) similar rural areas with similar occupations, etc.

Then exposure starts. In this case, the turbines are built and turned on—though only, as I said, for the study group. The researchers monitor what happens to everybody in both groups—guinea pigs, non-guinea pigs—and make comparisons and draw conclusions.

But there's a problem. A problem in what's called medical ethics. *It's unethical to design a study to expose people to something already suspected to be harmful*.

So another type of epidemiologic study, a case-control study, comes to the rescue. In a case-control study, people meeting the case definition for Wind Turbine Syndrome would be enrolled. For each case, two or three other similar people not living near turbines would also be enrolled. These are the controls. The researcher would collect the same types of data on all cases and controls, and make comparisons.

But to do this we need to know what a case is: *we need a case definition*. A case definition describes the symptoms and findings, proves there's consistency from person to person, and presents ideas or hypotheses about what causes the newly defined illness.

That is what my study has done—*created a case definition.* The next step is an epidemiologic study, *but the case definition comes first*.

Back to an epidemiologic study. A standard epidemiologic study would require several years of study, a huge research grant, and a small army of researchers. Besides this, as I explained in the Preface, the gag clauses found in wind leases and so-called Good Neighbor Agreements, plus the desire to sell one's home yet not reveal the noise toxicity and thus scuttle a potential sale, and the trickiness of community relationships—all these considerations would make an epidemiologic study highly difficult and perhaps doomed.

Nevertheless, epidemiologic studies should be undertaken. At least the attempt should be made.

My report should be seen as a necessary preliminary step to this much larger, more ambitious, more long term study. However, no government or private agency will undertake this Big Study without a preliminary project like mine—let's call it a pilot study—showing there is in fact something worth studying.

Back to my report. The problem in any clinical study is figuring out which new symptoms are due to a new exposure and which are not. In an epidemiologic study this is worked out by having parallel groups, with one group not getting exposed. Since I didn't have the resources to do such a study, I insisted that among my study subjects there be a post-exposure period—a time after exposure ended, during which the symptoms disappeared. *Wind Turbine Syndrome is defined only as those symptoms which came on*

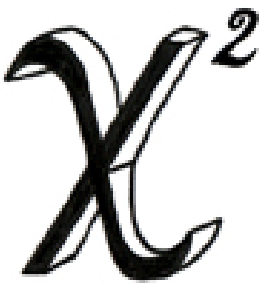
during exposure and abated only after exposure ended. It may not capture all the health effects of wind turbine exposure, because of the limitations built into my study design. But it certainly captured a significant set of symptoms.

There's an additional way I generated a comparison group. I either interviewed every family member or, in the case of a 5-year-old and the elderly senile, I got information on these people. In this way I discovered that not everyone in the family was equally affected, despite living in the same house at the same distance from the turbines. I used comparisons between affected and non-affected people to figure out which parts of their pre-exposure medical history predicted which symptoms during exposure.

With this in mind, notice how I chose my study subjects:

a) at least one family member was severely affected by living near turbines
b) the family either had to have left the home or spent sufficient time away to experience relief from symptoms
c) the people I interviewed had to be able to say clearly, consistently, and in detail what had happened to them, under what conditions and at what time
d) they all live near turbines put into operation between 2004 and 2007
e) if they had already moved out when interviewed, it was less than 6 weeks since they'd moved out
f) they had to have taken serious action to protect themselves from the turbine exposure (generally identified as noise):

    a. some moved out
    b. some purchased a second home in anticipation of moving out
    c. some left home for months
    d. one family renovated the house in an effort to mitigate the noise
    e. and one man took to sleeping in his root cellar



A final point. This squiggly symbol, χ^2 , is called "chi squared" (pronounced "keye," as in "eye"). Don't panic! It's a simple statistical test. I'll illustrate with an example.

1) You have a group of people
2) You classify them as tall or short, with blue eyes or brown eyes

3) A χ^2 statistic lets you say if blue eyes are associated with being tall or short in anything other than a random way
4) Since everyone knows that having blue or brown eyes has nothing to do with whether you're tall or short, if you do a χ^2 statistic on, say, 20 people, categorized for both of these qualities (eye color and height), it would come out to be non-significant
5) End of illustration. Now, that wasn't so hard, was it?

Notice, when you read my full report you encounter what are called p (probability) values in parentheses, together with χ^2 values. Again, don't panic. The p is the probability that the relationship between the two variables (eye color and height) is random. In other words, that being tall does not increase your probability of having blue eyes, or that height and eye color are totally unrelated.

P goes between 0 (zero) and 1 (one). Very low p values mean *there's a significant correlation between the two variables*. "Very low" would be less than 0.05. Less than 0.01 means there's an even stronger likelihood the two variables (eye color and height) occur together more than by chance.

Okay, you can breathe again; we're done with the math. This is precisely how I identify "risk factors" in my study. (Risk factor is something in your medical history or makeup that makes you susceptible, in this case, to Wind Turbine Syndrome when exposed to turbines.) I applied a χ^2 analysis. For instance, I look at whether a person has or does not have tinnitus when exposed to turbines. I compare that to whether they do or don't have a history of industrial noise exposure. I discovered, in this particular example, that a significant relationship does exist.

We'll come back to this in the Results section, below.

Results

My study demonstrated the following to be the core symptoms of Wind Turbine Syndrome.

1) First, *almost everyone had disturbed sleep*. Two particularly interesting patterns emerged in the disturbed sleep.

a. The first being what we might call "fear" patterns. This would be childhood night terrors and adults waking up alarmed and hyper-alert (i.e., felt they had to check and see if someone had broken into the home, even though they knew they had been awakened by turbine noise). At times adults woke up with a racing heart at night.
b. The second was a tendency to urinate a lot at night. For adults this meant getting up frequently, and for one child it involved bed wetting (which resolved whenever she was away from the turbines).

I didn't look for risk factors for sleep disturbance since virtually everyone interviewed had disturbed sleep.

2) *Headaches*. Slightly more than half the study group had headaches that were worse than what they normally experienced before and after turbine exposure. Headaches that lasted longer and were more severe, in other words.

Half of the people getting headaches were people with pre-existing migraine disorder (i.e., a hereditary tendency to get severe headaches, along with dizziness, nausea, visual changes, or strong avoidance of light and sound during these headache episodes). All the children who got headaches during turbine exposure either had migraine disorder themselves or their parents had migraine disorder.

About half the adults who got headaches during exposure had no risk factors for headache which I could identify. This means that anyone can get severe headaches when exposed to turbines.

3) *Ear symptoms*. Tinnitus was a dominant symptom during exposure. Tinnitus: ringing, buzzing, a waterfall noise, or even a buzzing that seems to be inside the head. Risk factors for tinnitus during exposure were:

a. having some tinnitus before exposure
b. having some hearing loss before exposure
c. a previous industrial noise exposure

All these suggest previous damage to the inner ear, which could come from noise exposure, chemotherapy, certain antibiotics, head injury, or even whiplash injury.

People also experienced pain and popping and a feeling of pressure in their ears.

4) The fourth core symptom I am calling VVVD. *Visceral Vibratory Vestibular Disturbance*. This is a new symptom to medicine, I believe. Before reading further, you should study the VVVD symptom accounts in the Results section (page xxx), so you have a mental picture of what people say they're experiencing.

   Once you've looked over those accounts we can move on to consider how the symptoms of VVVD can occur together. The symptoms being:

   a. A feeling of internal pulsation, quivering or vibration. For some, breathing feels controlled or restricted.
   b. Nervousness or jitteriness. Fear. The urge to flee. The urge to check the house for safety.
   c. Shaking
   d. Rapid heartbeat
   e. Nausea

   VVVD is essentially the *symptoms of a panic attack associated with feelings of movement inside the chest in people who have never had panic attacks before* (none of my subjects had).

   Because VVVD is so similar to panic attacks, I looked for a correlation between VVVD and a history of any other kind of anxiety or depression or mental health disorder. I found there is no relationship. However *there was a highly significant correlation between VVVD and pre-existing motion sensitivity* (i.e., people who get car-sick, seasick, or had a history of repeated episodes of vertigo).

   Out of the 21 adults (age 22 and up) in the study, 14 had VVVD. The two toddlers in the study looked like they had something similar. Though we don't know exactly what they felt, they woke up screaming several times per night, and were inconsolable and hard to get back to bed or to sleep. The two 5-year-olds in the study also awoke fearful in the night.

5) *Concentration and memory*. Almost everyone in the study had some kind of problem with concentration and memory. The more severe concentration problems were linked with a general loss of energy and motivation. What's noteworthy, among many of my subjects, was the degree to which they lost basic skills they had prior to turbine exposure, and the way teachers noticed new problems with kids' schoolwork and sent notes home. (Here, you should read the Concentration and Memory symptom accounts in the Results section, on page xxx, and the accounts of recovery from these symptoms on page xxx.)

   For some people, these problems with thinking resolved as soon as they got away from the turbines, or even if the turbines turned in another direction. For others, they did not resolve immediately, but improved gradually over time. To me this suggests that the memory and concentration difficulties were not solely due to sleep disturbance.

   I see the cognitive problems as the most worrisome of the whole constellation of Wind Turbine Syndrome symptoms. Somehow the brain seems to be conditioned into new patterns, as though thinking patterns have changed with the distorted vestibular input or prolonged disturbance of sleep. More about this later.

6) The remaining core symptoms were *irritability and anger*, which occurred in most of my subjects, including the children. Often it was the children's behavior and school problems, their irritability and loss of social coping skills, that drove families to move out of their homes and away from the turbines.

7) Most subjects had *fatigue* – sometimes a distinctly leaden feeling – *and loss of enjoyment and motivation for usual activities*. For most this cleared up soon after they got away from the turbines.

8) Finally, I listed clusters of symptoms that subjects told me about, but would require other modes of study (including physical exams or lab tests or x-rays, and a case-control format) to find out if they are connected to turbines. These symptoms occurred in low numbers in my study. They included *lower respiratory infections* (bronchitis, pneumonia, pleurisy) that were unusual for the people who got them, *worsened asthma*, *unusual middle ear fluid or infections*, and *ocular stroke*.

Though my study cannot prove a connection, I think they are worth attention in a large-scale study of wind turbine health effects.

Discussion

This section is about how I think Wind Turbine Syndrome works, and the ideas I got from the medical literature and my referees. This is the most interesting section of the report—where we join the dots.

I recognized the symptoms of Wind Turbine Syndrome as being something coherent, something that hangs together, because I already knew about what's called *migrainous vertigo* or *migraine-anxiety associated dizziness*.

Migraine is not just a bad headache. It has many other peculiar symptoms associated with it. My husband has had migraines since he was a teen, but he never gets headaches. He gets dizziness, tiredness, and patches where he can't see (scotoma). He has to lie down till it goes away. Some years ago he had a terrible episode of nauseating vertigo (a spinning kind of dizziness), tinnitus, and anxiety that developed into depression. The person who figured out what was wrong with him was the otolaryngologist to whom this book is dedicated, Dr. Dudley Weider.

Dr. Weider taught me how migraine, vertigo, tinnitus, and anxiety are neurologically related—and he treated my husband successfully. I might add that my husband has always been motion sensitive. And I learned that this commonly goes along with migraine.

When I started hearing about the symptoms in Wind Turbine Syndrome, I recognized it as a related complex of symptoms. I had hoped to share this report with Dr. Weider, but, alas, he had passed away. Instead I had the pleasure of sharing it with a group of his former colleagues in otolaryngology. Read through the list of referees and readers of this report, and you will discover it's a Dudley Weider reunion. They taught me many other important matters regarding balance and the inner ear, which I've incorporated into this report.

First, Drs. Lehrer and Black recognized the symptom complex of Wind Turbine Syndrome as similar to the symptom complex of an inner ear problem called endolymphatic hydrops (EH). In the case of EH the

symptoms are continuous or vary for unknown reasons. In Wind Turbine Syndrome these symptoms come and go depending on whether people are near or far from the turbines. Or whether the turbines are making a particular kind of noise, or facing in certain directions.

EH, which includes Meniere's Disease (pronounced "Man-ears") and perilymphatic fistula (where the fluid is leaking from the inner ear into the middle ear), involves distorted pressure relationships between the two fluid compartments in the inner ear: the endolymph and perilymph. This causes erratic and distorted balance and (often) hearing signals to be sent to the brain.

This brings us to the balance system and how it works. It's a complex system that penetrates many areas of the brain and draws sensory signals from all over the body. Other senses have only one kind of sensory input, whereas the balance system has four.

By balance system I mean a) *how the body maintains its upright posture* and also b) *everything to do with motion and position awareness*. For example, the balance system is highly active during the turns and twists of diving or gymnastics, even though a person is not staying upright.

Why all this focus on the balance system? Because I think that *people susceptible to imbalance are especially susceptible to Wind Turbine Syndrome*. So I need to explain the different ways people become imbalanced, and how the air pressure variations, or sound, from wind turbines may be triggering an abnormal sense of motion in susceptible people.

As I mentioned before, *motion* and *position* signals come from four discrete body systems and are integrated by balance centers in the brain:

1) eyes (the visual center)
2) dedicated motion and position sensing organs in the inner ear (the vestibular center)
3) stretch receptors from muscles and joints all over the body, and touch receptors in the skin (the somato-sensory center)
4) stretch and pressure receptors associated with organs in the chest and abdomen

The balance system requires that at least two of the first three channels, that we will call visual, vestibular, and somato-sensory, be working every moment if we are to maintain balance. Hang onto this point; it's extremely important. We might call it the *Law of Balance*.

For example, the vestibular organs in the inner ear tend not to work so well in older folks. If the inner ear is not sending correct signals, people are more dependent on what they can see and on what their feet and legs are feeling, to keep their balance.

Since two channels have to be sending signals for balance to work, these people are in trouble in the dark.

If you have good balance, try this experiment: stand on one foot and feel all the little corrective movements your foot and ankle are making to keep you upright. People with normal balance can stand on one foot indefinitely.

Now, close your eyes. See how long before you have to put your other foot down to keep from falling over.

You can't keep your balance in this situation because you've deprived yourself of both vision and adequate somato-sensory input from the legs—and one system, the vestibular input from the inner ear, is not enough. (If you don't have good balance, keep both feet on the floor when you close your eyes, and you still may notice a difference.)

Variations in balance function seem to fall into four broad categories.

1) *The first is very young age*. Little children fall down a lot. As kids get older and improve their balance, they can do more complex things without falling. At very young ages, children are mapping their entire sensory system onto the world. For example, an infant figures out how far he has to reach his arm to touch something, and what that looks and feels like. This gives him a sense of distance, mapping that concept of distance onto his visual sensors and the coordinated stretch receptors of his arm and shoulder.

   This process of learning where the parts of the body are in space, through increasingly complex activities, continues through childhood. In its early stages, children are more susceptible to balance disturbance.

2) *A second origin of balance variation is differences in the central (brain) processing of balance and motion-related signals*. People who are motion sensitive, which includes many people with

migraine disorder as well as other people, have difficulty successfully integrating the signals from the different sensory channels of balance. Their brains tend to over-emphasize or under-emphasize certain channels.

For example, in a person with migrainous vertigo and tinnitus—like my husband—the signals from the inner ear may be turned up too loud. So, centrally, the brain has to turn these down. It has to deal with the over-intensity of one signal. Or it may not be that they're too loud, but distorted, in which case there's an even greater need for the brain to down-weight the signals from that channel.

When we turn down the signals from the inner ear, we become more dependent on the visual channel or the somato-sensory channel. People who are visually dependent for balance often have trouble with heights, like being high up in a building or on a cliff. (Witness my husband.) This is because, when everything is far away, there's less visual position information that can be drawn from what one sees (less retinal slip and parallax changes as one moves, for example).

Someone who is surface dependent, on the other hand, may be in more trouble when the surface is slippery, because he is relies more on the position information coming from his muscles and joints. These signals are distorted by the slippery surface.

3) *The third source of balance variation or dysfunction is inner ear damage, or congenital or developmental malformations of the inner ear*. Damage may come from loud noise or blast exposures, head or neck injury (including minor ones like whiplash or a concussion), complications of repeated or chronic middle ear infections in childhood, or exposure to certain chemicals (aminoglycoside antibiotics or certain kinds of chemotherapy). There is also endolymphatic hydrops, an inner ear pathology (described above) that includes Meniere's disease and perilymphatic fistula. Autoimmune disorders like lupus can also cause endolymphatic hydrops.

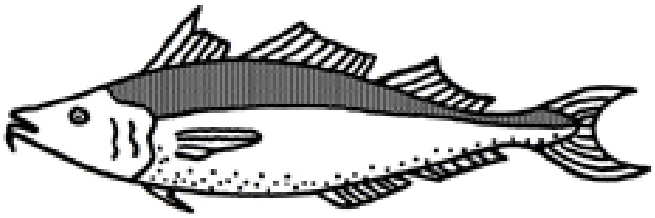
4) *The fourth source of balance variation or dysfunction is older age*. There seems to be deterioration of inner ear function after about age 50, varying among people, of course.

This brings us to *compensated vs. uncompensated balance dysfunction*. If you happen to have a balance dysfunction and yet are able to compensate for it, you feel fine: you keep your balance. On the other

hand, if there's an additional challenge, or distortion from another channel, then you're off balance—you feel unsteady or dizzy, or have vertigo or motion sickness. This is *uncompensated balance dysfunction*. Because of what's called "the system's redundancy" (the "system" in this case being the brain) brain balance centers can ignore or down-weight anomalous (false) signals from one channel, *but they can't for two channels*.

People who suffer from Wind Turbine Syndrome have, I believe, a compensated balance problem at baseline, in one of the four ways described above. By *baseline* I mean: they had a compensated balance problem before they were exposed to wind turbines. *Exposure to wind turbines pushes them over the edge, since, as I said above, the brain can't ignore disorienting signals from two channels*. (Remember, one set of false signals is now coming from the turbines. The other set of disorienting signals is coming from any of the four categories described immediately above.)

How, you ask, might sound pulsations (coming from wind turbines) be disrupting human balance signals?



Good question. Let's start with fish. The otolith organs of the vestibular system are highly conserved through evolution. Meaning, they're similar in fish and all other vertebrates—amphibians, reptiles, mammals, and birds—that evolved from fish. In fish these organs detect upright position (a fish's version of balance), and also low frequency noise and pressure variations in the water.

This detection of pressure variations and noise, including low frequency noise, is important for detecting the movements of other animals to escape being eaten, catch prey, and navigate through the oceans using the low frequency sound from distant waves breaking on shores. ("No, Virginia, they don't use Google maps or GPS locators.")

Thus the otoliths have a long evolutionary history of being sensitive both to gravity and noise/pressure variations. It makes sense that a system with a critical role in escaping predation would be hardwired into the brain's networks for fear and alerting, for fast escapes. Think too about all those stories about animals

detecting and fleeing earthquakes, tsunamis, incipient volcanoes, and ice breakup—things that rumble or make low frequency noise and vibration—long before human beings become aware of them.

Detection of this kind of signal is tied to fear responses: the animals flee. Animals apparently pick up this kind of signal through the ground, but there is also evidence that animals, including us, are sensitive to air pressure changes which are not sound. Weather changes influence mood and energy in many people, with higher barometric pressure associated with better mood and more energy. Birds sense big snowstorms coming, and feed like crazy before the storm rolls in.

What about that fourth balance channel, the stretch and pressure receptors in the internal organs? This fourth one, called *visceral graviceptors* and discovered relatively recently (in the 1990s, by a German researcher), is especially fascinating. (This is the balance channel many physicians are unaware of, since we were all taught in medical school that only three senses feed into balance.)

Visceral graviceptors are based on stretch and pressure receptors in and around internal organs. These receptors can let you know you're upside-down because there's more blood in the chest, making the blood vessels weigh more. Or they let you know by increasing the pressure of blood inside organs or blood vessels. This is thought to be a reason why astronauts hurling through outer space can have the sensation they're upside down, because gravity is no longer pulling so much blood into the legs and more of it is in the organs and vessels of the chest.

There are suggestions in the balance literature that visceral graviceptors play an important role in car-sickness and seasickness, by being the detectors for unusual up-and-down motions at odds with what the rest of the balance system is saying. It helps in seasickness, for instance, to stand up and look out at the horizon. This brings information from the eyes and stretch receptors in legs in line with the vestibular and visceral motion signals.

The point being that anomalous (unusual) signals, including from the newly discovered visceral graviceptors, disturb the balance system.

The VVVD story also involves understanding how the chest is a receptor for air pressure fluctuations. (Every form of sound in air, from low frequency to high frequency, consists of strings of air pressure pulses.) Briefly, when we breathe, our airways and lungs, which fill most of the chest, are open to the air.

Sound pressure waves can easily enter, and can set this elastic and mobile system moving with very little energy. (For a description of chest wall and diaphragm dynamics, see the explanation on page xxx.)

The internal graviceptors provide a potential link between the chest pressure fluctuations and the set of sensations and symptoms I call VVVD—the same set of neurologic symptoms seen in chronic inner ear disorders. The internal graviceptors may well provide the neural connection between chest pressure and weird discordances picked up by the balance system, producing similar sets of symptoms in these two situations. Or, as suggested by Dr. Owen Black (a balance and inner ear researcher), there may also be a relationship between pressure in the chest, the fluid around the brain, and the fluid in the inner ears in some people with particular inner ear problems.

Now consider how the balance system in the brain is neurologically tied in with fear and anxiety. Here we look to the work of Dr. Carey Balaban, a brain researcher. Balaban studies the neural networks linking balance with the brain centers controlling anxiety and fear, and with the autonomic responses and aversive learning that are also part of VVVD and Wind Turbine Syndrome. (The *autonomic nervous system* controls all the bodily functions that you don't have to think about and, in fact, you can't control. Such as blood pressure, heart rate, sweating, and digestion.) Disordered balance signals feed directly into fear and anxiety. *It's not because you start out scared or negative; it's because of a physiologic reaction to being off balance*. This is Balaban's point. He shows the actual nerve networks mediating these communications in the brain.

Balaban illustrates with a story. Imagine you're stopped in your car on a hill. Say, San Francisco. Out of the corner of your eye you see the truck next to you starting to inch forward. This immediately gives you impression you're starting to slip backwards! You panic! You jam your foot on the brake! The fear subsides as you realize you are in fact … not moving.

Balaban's story underscores that when you sense you're not stable in space—you're going to fall, you're moving when you don't expect it—it grabs all your attention, immediately, with alerting and fear. He also points out that when the sense of moving unexpectedly goes on over a long time, as in vertigo, so does the sense of fear.

Studies by psychiatrists and balance specialists show how the linkages between anxiety and balance problems play out clinically and in real life. A mild form of balance disorder is called *space and motion discomfort*, where people feel uncomfortable or dizzy in situations like looking up at tall buildings, closing their eyes in the shower, leaning far back in a chair, driving through tunnels, riding in an elevator, or reading in the car. These people also have abnormalities on balance testing. It's usually a central balance problem, meaning the brain has difficulty integrating all the different signals coming into the balance system, and deciding which ones to ignore if they don't match.

Space and motion discomfort is common in people with migraine disorders. So are dizziness, vertigo (spinning dizziness), and motion sickness. Balance testing is abnormal in people with migraine disorders compared to people who get other kinds of headaches, especially if the migraine patients tend to get dizziness or vertigo. The balance problems in migraine disorder, incidentally, can be inner ear or central (brain-based).

Anxiety problems are also associated with migraine, sharing a common thread in the serotonin systems of the brain. Space and motion discomfort is common in people with anxiety disorders. Balance testing shows that anxiety patients have higher vestibular (inner ear) sensitivity than people without anxiety problems. When balance testing is done in people diagnosed with panic attacks or agoraphobia (fear of leaving the house), a high number are found to have abnormalities of vestibular (inner ear) function—more than 80% in some studies. This is especially true if the people have episodes of dizziness between panic attacks.

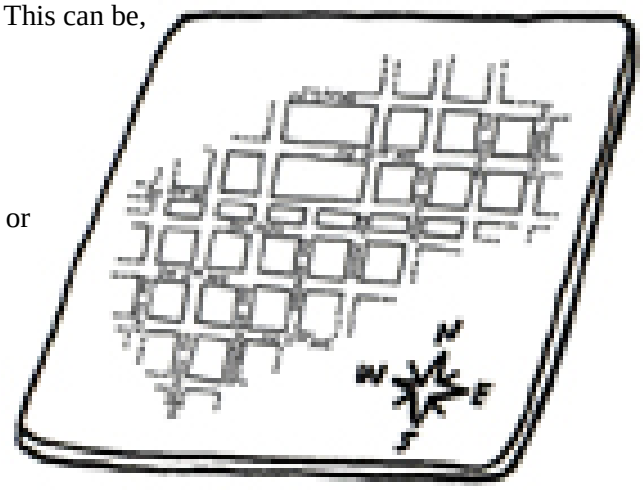
In sum, *there is a robust clinical literature supporting the biological connection between balance problems and anxiety, and between balance problems and panic attacks, in particular*. It makes eminent clinical sense that *disturbing a person's balance system can lead to fear, alerting, and panic, including physical symptoms like fast heartbeat and shakiness*.

I've talked about how the visceral graviceptors in the chest might be stimulated and lead to balance disturbance, but not so much about the other channels of balance. In my study, two subjects, both adult women already prone to vertigo, were very sensitive to the visual channel. Both developed severe headaches when exposed to the moving shadows of turbine blades.

Some subjects were able to feel vibrations from the ground in their lower legs, opening the possibility that the somatosensory channel (stretch scepters from the muscles and joints, and touch receptors from the skin) could be disturbed.

Many of my subjects had ear symptoms, including tinnitus, ear pressure, ear pain, or a physical feeling of noise inside the head. If the air pressure fluctuations from the turbines can produce these symptoms, which are all different from hearing the noise, might they also perturb the balance organs of the inner ear? The labyrinthine (inner ear) organs are delicate, interconnected membranous sacs with fluid inside and outside, sensitive to minute shifts in pressure and position. Audible sounds stimulate the cochlea, but certain sounds can also stimulate the saccule in humans (the otolith organ that tells us if we are upright). The otolith organs of certain fish, which are similar to ours, are known to detect low-frequency sound, aiding in navigation. This gives us reason to think, for both animals and humans, that certain types of noise may be able to stimulate the balance parts of the inner ear, especially the saccule, causing disturbance to motion and position sense.

The plot thickens. Thinking and memory: current research demonstrates that these, too, depend on coherent vestibular signaling. If you don't know which way is up, literally, at all times, your brain can't figure out a multitude of things related to position in space. This can be,



a) *position in real space*, like
    a. remembering how to get somewhere or
    b. figuring out how to put something together, or
b) *position in conceptual space*, like
    a. the distance between two numbers or
    b. the position of events in time or
    c. the categorization of objects in memory

Research is supporting what doctors who treat balance problems have seen for years: *struggles with short-term memory, concentration, multi-tasking, arithmetic, and reading are common in patients with balance disorders*.

Neuroscientists have recently shown that nerves from the vestibular system follow a direct path to the hippocampus, a brain structure critical for memory in general and spatial learning in particular. People with no inner ear input to the brain at all (the nerves having been cut years before to remove tumors)

cannot do experimental tasks involving navigation, and their hippocampi (plural of hippocampus) are smaller than normal. (Conversely, taxicab drivers in London have extra-large hippocampi, the size depending on how many years they have been driving and storing in their brains their personal map data of locations, shortcuts, and one-way streets.)

Functional MRI and PET scans (PET scans don't scan your pet, just as CAT scans don't scan your kitty; see Glossary) now allow researchers to see which parts of the brain are used for different tasks by awake humans while they are doing things. Stimulating the vestibular (inner ear balance) system lights up many areas in the brain, including those used for mental representations of space and mathematical thinking.

If the vestibular input is distorted (for example, by putting ice water in one ear), people make more mistakes in purely mental spatial tasks, like imagining a certain object in detail or imagining rotating it. These people were sitting still at the time, eyes closed, just thinking, not trying to keep their balance or having to judge where they were in space at all. Nonetheless, when signals came from one inner ear indicating movement—signals out of whack with all the other signals their balance centers were receiving—they remembered the objects less accurately and made mistakes when imagining them in different positions.

Disordered signaling from the inner ear at the time of spatial thinking, in other words, degrades both memory and concentration.

A cluster of brain centers that receive signals from the inner ear (meaning, they become active on functional MRI or PET studies when the vestibular organs are stimulated) is in the right parietal ("pair-eye-ital") lobe of the brain, towards the upper middle on the right side. There can be some very weird outcomes if these centers are lost to a right parietal stroke. Called "hemineglect" (hemi-neglect: meaning neglect of half the body and half of space), these poor people can have so much unawareness of the left side of space that they can be unaware that their left arm is paralyzed or the left side of their body undressed. Vestibular stimulation, however, temporarily reverses the neglect, so that these people become aware of the left side again in a more normal way.

People with hemineglect have typical types of errors on visual search and visual memory tasks, with answers biased away from the left and towards the right sides of images. Left vestibular stimulation corrects or improves performance on these tasks.

Other studies of people with hemineglect let us see what other kinds of mental tasks are "spatialized," meaning, they require the spatial types of thinking done in these right parietal lobe centers linked to the vestibular system. Spatialized thinking includes mathematical operations like forming a mental image of a ruler (lower numbers on the left, higher on the right), and imagining the midpoint between two numbers. It also includes clock representations of time, and spelling at the beginning (left) and ending (right) of words. Studies of powerful thinkers also show how important spatial thinking is: great mathematicians think of math in spatial terms (which is efficient, because the actual neural representation of numbers is spatial), and outstanding memorizers use spatially oriented strategies.

The punchline being, when there's a lack of input from the vestibular system, spatial thinking gets shut down. Whereas when the input from the vestibular system is confused (disordered), spatial thinking gets mixed up. Spatial thinking absolutely requires vestibular stimulation. Disordered vestibular information knocks it off balance, so to speak, rendering it less efficient and inaccurate.

Spatial thinking, it must be emphasized, is much of what we do with our brains.

Now, think about the specific tasks my study subjects had trouble with—what they spontaneously told me about themselves and their children, along the lines of,

a) "I can't believe I can't manage something this simple anymore!"
b) "He (my child) knew how to do this, and now he can't do it at all and gets really mad and frustrated when I make him keep trying!"

The letter and number refers the person's Family Tables. I put a description of the *spatial quality* of each task in italics:

A1 Remembering what he had come to get when he arrived at a store. *Spatial memory for the image of what he was searching for*.

B2 Remembering a series of errands and things to get in town. *Spatial memory for the objects and places to get them, spatial calculation of the most efficient path and order*.

C1, D1, G3 Reading. *Conversion of spatial input (words on page) to language and then to concepts and imagery (which are also spatial). There is also direct vestibular control of eye movements*.

C2, G2 Multitasking in kitchen and household. *Having an internal map of the locations and timing of multiple things at once, inserting tasks and events into the map and not losing awareness of them when out of sight*.

C7 Math—lost skills and forgot math facts. *Spatial representation of numbers and number relationships*.

E2 Spelling, writing. *Putting letters in the right order so the word looks right; changing language into a visual representation*.

F2 Assembling furniture. *Being able to convert written instructions or diagrams to 3-dimensional mental representations of what she was supposed to do with the pieces*.

F2 Following the steps in a simple recipe. *Picturing and ordering the steps in mind from the written instructions*.

F2 Following the plot of a TV mystery. *Noticing, remembering, and putting together visual clues*.

F3 Did worse than in past on national exams. *Outstanding memorizers use spatial strategies, as described above*.

H3 Reading, spelling, math. *All these have significant spatial components*.

I1 Professional landscaping and gardening—loss of concentration. *Planning and arranging things in space, remembering where you put down a tool, judging if something you're building is*

turning out right and how to fix it, planning steps of tasks efficiently in time and space, not forgetting steps.

J1 Paying bills. *Mathematics, memory for objects and services purchased, mental calculation of future needs*.

These are not all the tasks people said they were having trouble with, but they are the majority. Making sense? *Balance signals messed up, spatial thinking full of errors and inefficiencies, and people are enormously frustrated over normal, common sense things they suddenly can't do efficiently*. ("Common sense" has a big spatial thinking component, too.) Early school learning is thrown off, as well as reading and certain higher memory and problem-solving tasks in adults.

Interference of noise with reading and children's learning is not a new discovery; there is an extensive literature on it. In brief, environmental noise, like airport or traffic noise, makes children learn to read more slowly. In these studies, large numbers of children were studied in carefully controlled exposed and non-exposed groups, by choosing school districts at different locations relative to airports. Children were exposed to the extra noise both in school and at home.

In one study, a city closed an old airport and built a new one, and researchers had the opportunity to follow the reading skills of both sets of children over time. The ones living near the airport that closed showed improvements in their reading. The ones near the new airport showed slower learning after planes started flying in and out.

One study looked at children living in an apartment building next to a busy highway. Those on the higher floors, where it was quieter, had better reading scores and better ability to tell word sounds apart.

The effects of noise on reading ability go beyond the distracting effects of noise, and are linked to problems with language processing—like differentiating between sounds in noisy environments.

Noise can affect thinking in adults, too, at loudness levels nowhere near the levels that harm hearing. In one study, industrial workers worked on psychological tests while exposed to 50 dBA broadband noise (like white noise or machine noise) with or without low frequency components. The noise with low frequency components interfered with test performance more than the noise without the low frequencies, especially in individuals who rated themselves as sensitive to low frequency noise. Neither type of noise

was considered more annoying than the other, nor did subjects become accustomed or sensitized to the noise.

Many environmental noise studies look at effects on sleep, stress hormone (adrenalin, cortisol) output, blood pressure, and cardiovascular risk factors in general. Noise at night can significantly disturb sleep even when the person does not remember waking up. Since the sorting and storage of daily memories occurs during sleep, especially dreaming or what's called REM sleep (rapid eye movement), sleep disturbance by noise, even without known awakening, interferes with memory and learning.

In children, nighttime low frequency (rumbling/vibrating) noise from trucks has been shown to produce more stress hormone at a time of night when it's not usually produced (i.e., the wrong time of night), than gets produced in the same children exposed to regular nighttime car traffic noise. *In other words, low frequency nose stimulates elevated stress hormone production in children during nighttime sleep.* (Elevated cortisol levels are also known to harm memory and learning, reducing the survival of new hippocampal memory cells, as in chronically abused children.) High adrenalin or cortisol levels elevate blood sugar and increase blood pressure, increasing cardiovascular risk.

Interestingly, the levels of noise that disturb sleep are quite low. Noise events of 32 dBA cause people to move in sleep, showing a low level of arousal. Noise events of 35 dBA cause arousals that can be seen on a brain wave study (EEG). Conscious awakenings occur at 42 dBA. This is why the World Health Organization (WHO) recommends 30 dBA as an acceptable indoor nighttime noise level.

I don't present noise analyses in this paper—something that clearly needs to be done, but requires resources I didn't have—but I find that published descriptions of people's experiences in documented low frequency noise are very similar to what my study subjects noticed and described to me. If you haven't already done so, I recommend you read the section of clinical text called "Low-frequency noise" (pages xxx-xxx).

First, I quote Dr. Birgitta Berglund (the dean of community noise studies and lead editor of the 1999 World Health Organization *Guidelines for Community Noise*) on the reasons she thinks many of the adverse effects of noise in general are due to its low frequency components. She calls attention to how low frequency noise travels farther than other noise without losing its power, travels through walls and hearing protectors, rattles objects, sets up vibrations and resonances in the human body, and is linked to motion sickness even when vibration is not present. The lower frequencies can make it hard to

distinguish sounds at higher frequencies, like speech. Noise with low frequency components is experienced as louder and more annoying than noise at the same dBA level without low frequency components. All this is discussed in the "Low frequency noise" section in the clinical text. Again, it's well worth reading.

It's important to remember that the term "annoyance" in community noise surveys is used as a shorthand for a variety of negative reactions. Some of them severe. "Apart from 'annoyance,'" states the WHO, "people… exposed to community noise… report anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation, or exhaustion."

In the clinical report, I quote as well several other small studies of situations wherein people were exposed to documented low frequency noise. For instance, the symptoms that healthy young men felt when exposed to high amplitude low frequency noise, for only 2-3 minutes, in a NASA test facility in the 1960s, included fatigue, reduced efficiency at performing tasks, tickling in the ear, chest vibrations, and a feeling of fullness in the throat—all symptoms I heard about from my study's participants.

Indeed a case report from Germany in 1996 may well be Wind Turbine Syndrome, since the source of the low frequency noise (actually infrasound, below 10 Hz) was never identified. It's an especially interesting story. The couple's symptoms and the intensity of noise below 10 Hz both varied with the wind and weather, and were worse in winter. Their symptoms were,

a) sleep disturbance
b) headache
c) ear pressure
d) not feeling well in a general way
e) decreased ability/efficiency in doing things
f) chest symptoms described as shortness of breath and a tingling/crawling sensation

Symptoms occurred when the sound pressure level at 1 Hz was 65 dB, well below the couple's own hearing thresholds measured in a sound lab. All the frequencies responsible for the symptoms, which were all below 10 Hz, had sound pressure levels below 80 dB.

We now know that sound levels near turbines easily fall within these ranges, as measured by a Dutch physicist several years ago.

The 1996 German case, above, and another series of cases, also by German noise control officials, both *emphasize how the symptoms and the degree to which the people were bothered increased over time after they moved into the home or apartment with low-frequency noise. They did not get used to the noise. In fact, the opposite: they became sensitized to it over time*. At first it wasn't so bad, but it grew worse and worse.

My study subjects said the same thing, as they compared turbine noise to other types of noise, like traffic, that they easily got used to. Many said that wind turbine noise would not sound loud to people who did not live with it, but several also mentioned visitors being bothered if they spent the night. When they moved away from their turbine-exposed homes, all the families moved into towns and villages with more traffic noise, but no risk of turbines being build next door.

Hence, glib claims that "you will get used to wind turbine noise," are contradicted both by people who struggle to live with it, and by clinical evidence.

Both German case studies focused on the ability of low frequency noise, with its long wavelengths, to pass through walls and then reverberate or set up resonances inside rooms. The authors of the case series measured the difference in low frequency noise intensity near walls and away from walls, picking up nodes of higher intensity away from walls, like a standing wave in a stream.

In my study, Mr. and Mrs. G (G1 and G2) both identified a spot in one room where they got symptoms, a feeling of internal vibration for Mrs. G and the beginnings of nausea for her husband. They could not feel any vibrations with their hands if they touched walls or furniture. I think this was one of those places where the low frequency sound (air pressure) waves overlapped in such a way, as they bounced around the room, that they made a stable spot or standing wave of increased intensity.

Swedish researchers verified in a survey study of hundreds of households that the amount of noise needed for a wind turbine to cause severe annoyance was much lower than the amount of noise road traffic, airplanes, or trains would have to make to be severely annoying to as many people. "Amount of noise" was measured in dBA, which filters out the effect of any low-frequency components, and averaged over time. Fifteen percent of people were highly annoyed at 38 dBA of wind turbine noise, compared to 57 dBA for aircraft, 63 dBA for road traffic, and 70 dBA for trains. By the time the wind turbine noise level

reached 41 dBA, 35% of people were highly annoyed. Sixteen percent reported sleep disturbance over 35 dBA of outdoor turbine noise.

When these researchers interviewed some of the people surveyed, they found the same sorts of problems I encountered in my study, including people who had moved out of their homes because of the noise or rebuilt their homes to try to exclude the noise. Some reported feeling invaded or violated by turbine noise, being sensitive to blade motion as well as noise, and the loss of their ability to rest and feel restored at home.

From this one can reasonably conclude that, for wind turbines, perhaps unlike other sources of noise, *community standards allowing 45-55 dBA outside neighboring homes are asking for trouble*.

George Kamperman and Rick James, two independent American noise control engineers with decades of experience working with industrial noise and communities, recommend a noise standard based on quietest background ambient noise and using C-weighted as well as A-weighted measurements, so that the low frequency components are also controlled. Their specific recommendations—for how the noise measurements should be done and how the procedures should be spelled out in a local ordinance—were presented at the annual conference of the Institute of Noise Control Engineering/USA in 2008 and are posted on the Wind Turbine Syndrome website at http://www.windturbinesyndrome.com/?p=925. An important part of Kamperman and James's method is that as turbines get larger, setbacks will have to be longer.

But the simple answer is: keep wind turbines at least 2 km (1¼ miles) away on the flat, and 3.2 km (2 miles) in mountains. These are minimum distances; Kamperman and James's methods will in all likelihood recommend longer setbacks, especially in rural areas that are very quiet at baseline. Secondly, all wind turbine ordinances should hold developers responsible for a full price (pre-turbine) buy-out of any family whose lives are ruined by turbines—to prod developers to follow realistic health-based rules and prevent the extreme economic loss of home abandonment.

Notice, while you're reading, that there are missing pages from this typescript ms.

Table 1B: Cases: physical attributes

Case	Distance to closest turbine	# turbines	MW per turbine	Year placed in operation	Hub height	Total height	Terrain	Configuration of turbines	House construction
A	1000 m (3281 ft)	10	3	2007	90 m	135 m	Hilly with rocky ridges	10 in line point at house at hub level	Wood frame
B*	1000 m (3281 ft)	10	3	2007	90 m	135 m	Hilly with rocky ridges	10 in line point at house at hub level	Wood frame
C	305 m (1000 ft)	17	1.8	2004-05	80 m	125 m	Rocky peninsula	On three sides	Wood frame
D	548 m (1798 ft)	22	1.8	2006	78 m	117 m	Flat farmland	Group on one side	Wood frame
E	423 m (1388 ft)	45	1.5	2006	87 m	120 m	Flat farmland, swamp	On three sides	Wood frame with stone front
F	930 m (3051 ft)	8	2	2006	59 m	100 m	Flat farmland	5 in line point at house	Brick on cement slab
G	596 m (1955 ft)	32	3	2006	80 m	125 m	Rocky hills	Above house on three sides	Stone cottage, walls 2 ft. thick
H	1500 m (4921 ft)	11	2.3	2005	80 m	121 m	Rocky hills	Above house on three sides	Stone cottage, cement slab
I	350 m (1148 ft)	10	2	2006	78 m	121 m	Rocky hills	Across valley at higher elevation	Wood frame
J	732 m (2400 ft)	40	2	2007	80 m	123 m	Ridges and valleys	6 in L above house on two sides	Wood frame

*Families A and B are related and own separate homes on the same property

Table 1C: Cases: demographics

Age	Male	Female	Total
<1	1	1	2
1-3	1	1	2
4-6	2	1	3
7-11	3	0	3
12-15	1	2	3
16-21	2	2	4
22-29	0	0	0
30-39	2	2	4
40-49	3	2	5
50-59	4	5	9
60-69	1	1	2
70-79	1	0	1
Totals	21	17	38

Family Table A1 (page 1 of 2)

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 A2 (page 1 of 2)

Person
Mrs. A
Age
33
Pre-exposure health status
Good. Pregnant during exposure and delivered at term 4 days before moving.
Health history
Polycystic ovarian syndrome and metabolic syndrome. Caesarian section for first delivery.
Previous noise exposure
Worked at biomedical chemical plant for 5 yrs with 1-2 hrs/wk exposure to noisy areas.
Time to onset of symptoms
Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Normal. Sleeps through noises other than children.	Frequent awakening	Normal, resolved
Headache	Rare, mild	Occasional headache	At baseline
Cognition	Concentration "great," works as accountant	Noticed concentration problem at work when training someone; working to focus; trainee had to help	Resolved
Mood	Good, including during and after first pregnancy	Irritable	Resolved
Balance / equilibrium	Gets seasick but not carsick	Slight unsteadiness	Resolved
Ear / hearing	Normal hearing. Persistent middle ear fluid in late 20's, resolved. Tinnitus in past when emerging from noisy plant.	Repetitive popping in ears and decreased hearing for first 3 weeks, then tinnitus began. Tinnitus varied with exposure and worsened over time.	Tinnitus resolved, but has new difficulty understanding conversation in a noisy room. Has to watch speaker's face.
Eye / vision	Wears glasses. Eyes water if strained.	No change	No change
Other neurological	Normal, no concussion	No change	No change
Cardiovascular	Normal except h/o temporary stress-related hypertension at age 22.	Normal	Normal
Gastrointestinal	Nausea and GER during pregnancy	No change	Resolved after delivery

Family Table A2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Respiratory	Normal, no asthma or smoking.	Lower respiratory infection for 6 weeks not treated until after delivery and move.	Resolved
Other		"Not noisy like a chainsaw, more like pulsating annoyance. To another person it wouldn't sound loud."	

Animals	Dog barks at windmills and up more at night	Improved dog behavior

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Family Table A3

Person
Son A
Age
2½
Pre-exposure health status
Good
Health history
Term birth, normal growth and development.
Previous noise exposure
No significant
Time to onset of symptoms
Immediate

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Slept through night 12 hrs without awakening. Always a good sleeper.	Night terrors 2-5 times each night, 30 min to calm down and return to quiet sleep.	At baseline. Night terrors resolved. Awakes once briefly for drink and goes back to sleep.
Headache	None	No apparent headaches.	None
Cognition	Good speech development with lots of words and no sound confusion.	Began to confuse *t* with *k* sounds and *w* with *l* sounds.	Vocabulary, sentences, and conversational skills are good but still confusing sounds.
Mood	Good-natured, sensitive, bright, listened well for age.	Oppositional, cranky, "a completely different kid for a few months."	"Instantaneous" resolution when moved, resumed former behavior.
Balance / equilibrium	Normal for age.	No change	No change
Ear / hearing	Normal hearing test at birth. One episode of otitis media.	Pulled ears and got cranky synchronously with adult episodes of headache and tinnitus.	Resolved
Eye / vision	Normal	No change	No change
Other neurological	Normal	No change	No change
Cardiovascular	Normal	No change	No change
Gastrointestinal	Normal without h/o GER.	No change	No change
Respiratory	Normal without h/o asthma.	No change	No change

*Exposure period 5 months, age 27-32 months.

**Information provided by parents 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

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.

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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 C2 (page 1 of 2)

Person
Mrs. C
Age
42
Pre-exposure health status
Good
Health history
Migraine disorder, 6 healthy term pregnancies without hypertension
Previous noise exposure
No significant
Time to onset of symptoms
Immediate when first turbines operational, with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good	Delayed onset, frequent awakening, hyperalert when awakened, nocturia; "no good rest in 10 months."	Resolved including nocturia.
Headache	Migraine frequency varied, never awoke her at night; headache onset in childhood.	Headache onset day or night, 5-6 nights/wk at maximum.	Resolved, no migraines.
Cognition	Normal, very organized mother of 6 children, "ready a month in advance for birthday parties."	Disorganized; could not handle as many things at once; difficult to plan and track cooking; "I thought I was half losing my mind"	Resolved including ability to multitask.
Mood	Good, lots of energy.	Tired, anxious, irritable.	Improved, but still sadness and stress related to loss of home and living with parents.
Balance / equilibrium	Lifelong motion sensitivity in cars, boats, swings, standing on wharf seeing boats go up and down. No vertigo.	Frequent dizziness, vertigo, and nausea preceding headaches.	Resolved.

Family Table C2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Ear / hearing	Normal hearing, no tinnitus.	Tinnitus began when first 2 turbines operational; no change in hearing.	Hyperacusis
Eye / vision	Normal, no glasses.	Nystagmus, subjective blurring.	Persistent subjective blurring
Other neurological	Normal, no concussion.	No change	No change
Cardiovascular	Normal including BP during pregnancies and at other times.	Hypertension and episodes of tachycardia.	Persistent BP elevation 180/102, started medications. Rare palpitations.
Gastrointestinal	Normal	Frequent nausea with dizziness and headache.	Resolved
Respiratory	Normal, never smoked.	Pneumonia with pleurisy twice in first 3 months of exposure to all turbines.	Resolved
Other	Hand and foot eczema	Exacerbation	Persistent increased itching.
		• At sunset, strobe effect inside or moving shadows outside triggered dizziness, nausea, and headache. • Occasional sensation of vibration in feet and legs outside house.	Resolved

*Exposure period 15 months to all turbines, 21 months to at least 2 operating turbines. Interviewed 2 weeks before move and 18 and 21 months after move.

** Limited ongoing exposure of several hours per week when goes to house to get things, but stopped going to house by 25 months after moving.

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 2 of 3)

	Pre-exposure	During exposure*	Post-exposure**
Balance / equilibrium	Never carsick but badly seasick once as a child. Avoided water ever after and disliked crossing bridges. No vertigo.	After retinal stroke, episodes of "numbness coming over my head. It seems to be my brain. Light-headed, not dizzy, I don't stagger. I can hear, I can talk, everything works for me properly, it's just that I get light-headed." No vertigo.	Does not occur away from home.
Ear / hearing	Some hearing loss but no difficulty understanding conversation. Skillfully differentiates machine noises in all settings. Has background tinnitus.	Background tinnitus is louder and higher, a "squeal," when turbines in operation. Drops in pitch when turbines are off and changes intensity when turbines change direction. When louder, the tinnitus interferes with hearing. No other sensations in ears.	Tinnitus at baseline when away from home.
Eye / vision	Wears glasses and has early cataracts.	Painless retinal stroke at night during sleep. Lost over half of vision in left eye. Confirmed by ophthalmologist, who talked to Mr. D about muscles squeezing off blood vessels in his eye. Normal CT.	No change
Other neurological	Normal without history of seizure or tremor.	After 16 mos: "Right arm jumps all over on its own…it just sits and bounces...hand shaking fierce just hanging onto the phone…started with feeling of satin or silk between the fingers…feels like it's wore out, like you're grabbing something real tight all the time...muscle spasms"; had nerve conduction studies [results unknown] and normal MRI of brain.	Arm calmed down during 5 days away and worsened on return.
Cardiovascular	Normal including BP, no palpitations.	Episodic tachycardia: "My heart feels like it's starting to race like crazy and I have these tremors going through my body and I was getting into a light pain on the left side of my chest." Symptoms exacerbated by nitro spray. Stress test terminated in 30 seconds. Scheduled for cardiac imaging test.	Does not occur away from home.
Gastrointestinal	Uses laxative to counteract opiate effect. Ulcer 2 years before while taking aspirin.	Stool again positive for blood; omeprazole started, endoscopy scheduled; bowels too loose or too firm.	No information.
Respiratory	Normal except smoking age 15-44, no asthma.	Pants or hyperventilates when tremor and tachycardia occur, and consciously slows his breathing when calming down.	Does not occur away from home.
Endocrinologic	No diabetes or other problem.	No change.	No change.

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 E2 (page 1 of 2)

Person
Mrs. E
Age
56
Pre-exposure health status
Fibromyalgia vs. reflex sympathetic dystrophy
Health history
4 term births, appendectomy, hysterectomy with "nerve damage" at age 38
Previous noise exposure
No significant
Time to onset of symptoms
Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Normal except after hysterectomy	Onset delayed up to 3 hours, multiple awakenings, nocturia (no glucosuria). At times awake all night, worse when blades facing NW.	Sleeps well, no nocturia
Headache	Rare, mild. Only one previous similar headache, when landing in a jet with nose and ears plugged from allergy.	Headache whenever turbines were generating. "In the wintertime, the strobing in the house and on property built up such pressure in my head you'd think it was going to blow off the top."	No headaches.
Cognition	Normal: retired teacher, organizes community activities	When blades facing house, could not spell, write letters, or keep her train of thought on the telephone, but was able work when blades not facing house.	Resolved; no concentration or memory difficulties.
Mood	Mild anxiety with chronic low-dose anxiolytic at bedtime	Episode of depression.	At baseline.
Balance / equilibrium	Never carsick or seasick. Vertigo twice in past, each episode 1-2 weeks.	"Lightheadedness, head kind of swimming." Less steady on feet depending on direction blades facing, especially outside	Resolved.
Ear / hearing	Normal, tested	Occasional sensation like insect crawling in ear; no tinnitus or change in hearing	Resolved.
Eye / vision	Normal, glasses for reading only	No change	No change
Other neurological	Painful right leg and abdomen ascribed to nerve damage, uses TENS unit; no concussion	Pain worse, increased use of TENS unit	Resolved when away even for short periods.
Cardiovascular	Normal including BP	"Heart synchronized to rhythm of blades." When lying on back, felt "ticking" or "pulsing" in chest in rhythm with swish of the blades. Could make it stop by getting up and moving around, but started again when she lay down. Occurred more at night. No change in BP.	Resolved.

Family Table E2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Gastrointestinal	GER resolved with diet intervention.	Nauseated when she had a pounding headache.	Resolved.
Respiratory	Normal, never smoked. Soprano in church choir.	More coughing illnesses as opposed to URIs, one lasting 6 weeks. Lost ability to sing.	Both resolved.
Rheumatologic	Fibromyalgia; osteoarthritis in hands	Diffuse muscle aches, "thought my fibromyalgia had really flared up."	Resolved when away even for short periods
Animals	Anxious dog	Dog did not sleep, wet floor 9/10 nights	Dog dry and no longer anxious

*Exposure period 17 months.

**While away on trips of 12 days to 3 weeks and after final move 1 month before 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.

Family Table J1 (page 2of 2)

	Pre-exposure	During exposure*	Post-exposure**
Balance / equilibrium	Good, seasick once in life	3 episodes of transient vertigo/dizziness while in tree stands late in day	No dizziness or vertigo
Ear / hearing	Slight left hearing loss on test 10 years prior; tinnitus during sinus infections	No subjective change in hearing; occasional tinnitus outdoors when turbines spinning rapidly.	At baseline; no tinnitus
Eye / vision	Normal with glasses	Developing presbyopia (expected for age)	No change
Other neurological	Normal with mild concussion age 7	No change	No change
Cardiovascular	Normal including BP; no palpitations.	BP normal but not measured during "jittery" episodes; awakens with rapid or pounding heart and "jittery" sensations when turbines noisy.	No "jittery" episodes
Gastrointestinal	Normal without GER or nausea	Queasiness and reduced appetite in evening with onset as he arrives home from work	No nausea, appetite good
Respiratory	Normal without asthma; smoked age 18-23	No change	No change
Other	Farming, building, and hunting activities for relaxation at home	Home more stressful than work; driven inside from farming activities, picnics, playing with sons, and hunting by turbine noise provoking symptomatic episodes.	Able to relax outdoors

Animals	Horse, 5 beef cattle, ducks unaffected	

*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.

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.

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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.

ABBREVIATIONS

χ^2	chi-squared statistic or test
CT	computerized tomography
dB	decibels
dBA	decibels measured with an A-weighted filter
dBC	decibels measured with a C-weighted filter
CSF	cerebrospinal fluid
EEG	electroencephalogram
EH	endolymphatic hydrops
ft	feet
Hz	Hertz (frequency in per second or sec^{-1})
INCE	Institute of Noise Control Engineering
INR	international normalized ratio of prothrombin times (see *Glossary*: anticoagulation)
km	kilometers (1000 m)
m	meters
mcg	micrograms
mg	milligrams
mi	miles
MRA	magnetic resonance angiography
MRI	magnetic resonance imaging
MW	megawatts
p	when used in the context of a statistical test, p means probability that the compared distributions are no different from each other
P.E.	professional engineer
PET	positron emission tomography
PTSD	post-traumatic stress disorder

VVVD visceral vibratory vestibular disturbance (defined in this paper, p. 11)

WHO World Health Organization

WTS Wind Turbine Syndrome

Nina Pierpont, MD, PhD

Fellow of the American Academy of Pediatrics (FAAP)

19 Clay St.
Malone, NY 12953

(518) 483-6481 phone and fax
pierpont@twcny.rr.com
www.ninapierpont.com

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HOME ADDRESS

Same as office.

PERSONAL

Place of birth: Stamford, CT
Date of birth: May 18, 1955
Married with two adult stepchildren

EDUCATION AND TRAINING

Education

1991	M.D.	The Johns Hopkins University School of Medicine
1985	Ph.D.	Princeton University (Ecology, Evolution, and Behavior)
1981	M.A.	Princeton University (Ecology, Evolution, and Behavior)
1977	B.A.	Yale University (cum laude)
1973		Milton Academy, Milton, Mass.
1970		New Canaan Country School, Conn.

Post-Doctoral Training

1992 to 94	Pediatrics	Dartmouth-Hitchcock Medical Center, Lebanon, NH
1991 to 92	Pediatrics	Children's National Medical Center, Washington, DC
1985 to 86	Ornithology	American Museum of Natural History, New York, NY

Licensure and Certification

1997	Licensed Physician, New York
1997	Licensed Physician, New Hampshire (expired)
1995	Pediatric Advanced Life Support (recertified 2002)
1994	Diplomate, American Board of Pediatrics (recertified 2008, expires 2015)
1994	Licensed Physician, Alaska (expired)
1994	DEA Registration
1994	Advanced Trauma Life Support Provider (expired)

1994 Advanced Cardiac Life Support Provider (expired)
1992 Neonatal Advanced Life Support Provider (recertified 2003)

Continuing Education

2008 Intermediate Training in the Psychological Treatment of Children with Trauma-Attachment Problems - Daniel A. Hughes, Ph.D. (32 hours)
2007 Training in the Psychological Treatment of Children with Trauma-Attachment Problems - Daniel A. Hughes, Ph.D. (32 hours)
2006 Workshop in Basic Pediatric Hypnosis (20 hours)
2006 Introductory Theraplay Training (27 hours)
2005 Psychiatry: Comprehensive Update and Board Preparation (Harvard, 51 hours)
2005 ADHD Across the Life Span (Harvard, 22 hours)
2004 Gesell Developmental Evaluation, Anthony Malone, M.D., Latham, NY (6 days)
2002 Promoting Student Success (Melvin Levine, M.D., U. of N. Carolina, 20.5 hours)
2002 Psychiatric Neuroscience Home Study Course (Harvard, 16.5 hours)
2000 Child and Adolescent Psychopharmacology (Harvard, 20 hours)
1998 Clinical Diagnosis and Treatment of Fetal Alcohol Syndrome (7.5 hours)
1997 Pediatric and Adolescent Gynecology (Harvard, 14 hours)

PROFESSIONAL APPOINTMENTS

Hospital or Affiliated Institution Appointments

2004 to Consulting Pediatrician Alice Hyde Medical Center, Malone, NY
2000 to 03 Senior Attending in Pediatrics Bassett Healthcare, Cooperstown, NY
1997 to 00 Attending Pediatrician Alice Hyde Medical Center, Malone, NY
1995 to 96 Chief of Pediatrics Yukon-Kuskokwim Delta Regional Hospital, Bethel, AK (Yup'ik Eskimo)
1994 to 95 Staff Pediatrician Yukon-Kuskokwim Delta Regional Hospital, Bethel, AK

Other Professional Positions

1998 to 00 Private Practice (Solo) Pediatrics Malone, NY
1997 to 00 Staff Pediatrician St. Regis Mohawk Health Services, Hogansburg, NY
1997 to 98 Staff Pediatrician North Country Children's Clinic, Malone, NY

Academic Appointments

2000 to 03 Assistant Clinical Professor of Pediatrics Columbia University College of Physicians and Surgeons
1980 to 85 Teaching Assistant Princeton University
1978 Teacher Children's School of Science, Woods Hole, MA
1977 to 78 Research Assistant Yale University

LANGUAGES SPOKEN

Spanish, French

AWARDS AND HONORS

1984 National Science Foundation Dissertation Grant (Princeton)
1979 to 82 National Science Foundation Predoctoral Fellowship (Princeton)
1979, 80 Dunlop Prize, Biology Department, Princeton University

1981 to 83	Research grants from the National Academy of Sciences, American Museum of Natural History, American Ornithologists' Union, and others
1973	National Merit Scholar to Yale University

MAJOR ADMINISTRATIVE RESPONSIBILITIES

1995 to 96	Chief of Pediatrics Yukon-Kuskokwim Delta Regional Hospital, Bethel, AK

PROFESSIONAL SOCIETY INVOLVEMENT

1997 to	American Academy of Pediatrics Fellow
2000 to	Medical Society of the State of New York
2006 to	Franklin County Medical Society
2000 to 03	Otsego County Medical Society

COMMUNITY SERVICE

1998 to 00	Physician member, Child Abuse Response Team, Franklin County, NY
1994 to 96	Physician member, Child Abuse Response Team, Yukon-Kuskokwim Delta, AK

GRAND ROUNDS

May 1994	"Infectious Diseases in Yup'ik Eskimos" at Dartmouth-Hitchcock Medical Center (Lebanon, NH)
May 2001	"Vaccinations: The Debate" at Bassett Healthcare (Cooperstown, NY)
March 2002	"Evaluation of Children and Adolescents with Behavior and Learning Problems" at Bassett Healthcare (Cooperstown, NY)
April 2002	"Vaccinations: An Overview for Family Practitioners" at Bassett Hospital of Schoharie County (Cobleskill, NY)
Feb 2003	"A Neurodevelopmental Approach to ADHD" at Bassett Healthcare (Cooperstown, NY)

BIBLIOGRAPHY

Original reports

Pierpont N, Fitzpatrick JW. Specific status and behavior of *Cymbilaimus sanctaemariae*, the Bamboo Antshrike, from southwestern Amazonia. Auk (Journal of the American Ornithologists' Union) 1983; 100: 645-52.

Terborgh JT, Robinson SK, Parker TA, Munn CA, Pierpont N. Structure and organization of an Amazonian forest bird community. Ecological Monographs 1990; 60: 213-38.

Proceedings of Meetings

Pierpont, N. Competencia y coexistencia de trepadores [Competition and coexistence of woodcreepers]. In: Rios MA, ed. Reporte Manu. Lima, Peru: Centro de Datos para Conservacion, 1985, 13 pp.

Abstracts

Pierpont N. Interspecific dominance and the structure of woodcreeper guilds [Abstract]. American Zoologist 1983; 23: 1001.

Pierpont N. Interspecific aggression and the ecology of woodcreepers (Aves: Dendrocolaptidae). Ph.D. Dissertation, Princeton University, Princeton, NJ. Ann Arbor, Michigan: University Microfilms, 1986; Abstract No. 8602443.

Pierpont N. Habitat selection induced by interspecific aggression in Amazonian birds [Abstract]. Proceedings of the IV International Congress of Ecology, Syracuse, N.Y., 1986, p. 271.

Karron RA, Singleton RJ, Petersen KM, Bonilla D, Hughes B, Davidson N, Bulkow L, Pierpont N, Wainwright R, Santosham M, Harrison LH. RSV is a frequent cause of hospitalization in Alaska Native infants. Poster, ICAAC meeting, 1995.

Newspaper column, The Malone (NY) Telegram

9/9/06	ADHD: Young Children, Self-Regulation, and Look-Alike Problems
9/23/06	ADHD: Older Children: Mental Energy and Consistency
10/7/06	ADHD: In One Ear and Out the Other (Processing Controls)
10/23/06	What Elephants Teach Us about Children
11/4/06	ADHD: Look Before You Leap (Production Controls)
11/18/06	Mapping the World onto the Brain: Neurological Templates for Learning
12/2/06	Childhood Adverse Experiences and Long-Term Health (ACE Study)
12/16/06	Autism from the Inside (Temple Grandin)
1/7/07	Mirror Neurons and Autism
1/20/06	Autism, Asperger's, and Non-Verbal Learning Disabilities
2/3/07	Concussions: Short- and Long-Term Effects
2/17/03	Play + Therapy = Theraplay
3/3/07	Sick Of Poverty: Poverty, Stress, and Health
3/17/07	TV, Video Games, and Kids
4/3/07	Punished by Rewards: Research on Behaviorism
4/21/07	The Genius of Inner Motivation
5/12/07	Warbler Wave: Healing and Nature
5/26/07	Plan B: Collaborative Problem Solving

6/9/07 Try Collaborative Problem Solving

Wind energy

Pierpont N. Health, hazard, and quality of life near wind power installations: How close is too close? Malone (New York, USA) Telegram, March 2, 2005, p. 5.

Pierpont N. Health effects of wind turbine noise. Report presented to NY State legislators, February 3, 2006.

Pierpont N. Wind turbine syndrome. Testimony before the New York State Legislature Energy Committee, March 7, 2006.