A second disclaimer. Readers should understand that Wind Turbine Syndrome is not the same as Vibroacoustic Disease.[10] 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 be heard 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.[11]

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.[12] 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

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

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

[12] Castelo Branco and Alves-Pereira 2004.

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 people who do not.[13] This substantial body of research has produced no good evidence that magnetic field exposure causes cancer in children or adults, cardiac or psychiatric disease, dementia, or multiple sclerosis.[14,15] After three decades of research, there is still no experimental evidence for a physiologic mechanism for any of the proposed effects of magnetic fields.[16]

This makes it difficult 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.[17] 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

[13] 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.

[14] Ahlbom et al. 2001.

[15] 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.

[16] Ahlbom et al. 2001.

[17] Ahlbom et al. 2001.

that synchronizes with the feeling of pulsation some subjects felt in their chests. Coming from several towers at once, these low frequency air pressure fluctuations may synchronize and reinforce, depending on the orientation of the towers and house and the timing of the individual turbines. Three families in this study (A, B, and F) lived in houses nearly in line with a row of turbines. For families A and B, the area's worst storms, "nor'easters," swept right down the line towards their houses, which were built on a hill at the level of the turbine hubs. These two families, though they were a kilometer (about 3300 feet) from the closest of the 10 turbines, moved out faster—in five months—than any of the other families, and had particularly severe symptoms.

Studies of turbine noise also show that noise carries farther than predicted by conventional industry modeling. This has to do not only with the low frequency components of the noise, which attenuate less with distance, but also with layering of the atmosphere at night, which creates cool still air at ground level and brisk, laminar airflow at turbine hub heights.[191] Industry models do not take these factors into account. Nor do they allow for a noise source more than 30 m above the ground. (Turbine hub heights in this study were 59-90 m.) Nor do they allow for increased transmission of sound in front of and behind the blades (with less sound transmission in the plane of the blades, including under the turbines), sky reflections, or weather conditions that focus the noise transmissions.[192]

Vibroacoustic Disease (VAD) model

High intensities of low frequency noise over prolonged time periods may cause marked neurologic damage, as described

[191] van den Berg 2004b.

[192] Richard James, INCE Full Member, personal communication, 5/11/08.

by the Vibroacoustic Disease (VAD) group in Portugal.[193] This is a provocative body of research, full of interesting case descriptions and pathology studies, but compromised by absence of specified study group criteria, absence of control groups, and lack of quantification. The study group consists of 140 aircraft maintenance and repair technicians in the Portuguese Air Force, of whom 22 (15.7%) had adult-onset epilepsy, compared to a national prevalence of 0.2%.[194] Some of the case descriptions of the subjects with epilepsy also include cognitive decline, depression, paranoia, and rage attacks.[195] The descriptions are similar to those of retired professional football players with histories of multiple concussions.[196,197] The vibroacoustic disease researchers ascribe VAD pathology to whole-body vibration induced by the noise, with the pathology of each body part induced by vibration of that part. Neurologic effects may be due to neuronal or axonal shearing, as in the multiple concussions scenario, or due to microangiopathy in the brain, meaning, effects on and occlusion of small blood vessels.[198]

With regard to the chest, the VAD researchers have used human autopsy and biopsy and animal rearing studies to describe loss of

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

[194] Castelo Branco and Alves-Pereira 2004.

[195] Martinho Pimenta AJ, Castelo Branco NAA. 1999. Neurological aspects of vibroacoustic disease. Aviat Space Environ Med 70(3): A91-95.

[196] Omalu BI, DeKosky ST, Minster RL, Kamboh MI, Hamilton RL, Wecht CH. 2005. Chronic traumatic encephalopathy in a National Football League player. Neurosurgery 57: 128-34.

[197] Omalu BI, DeKosky ST, Hamilton RL, Minster RL, Kamboh MI, Shakir AM, Wecht CH. 2006. Chronic traumatic encephalopathy in a National Football League player: part II. Neurosurgery 59: 1086-93.

[198] Martinho Pimenta and Castelo Branco 1999.

cilia and microvilli from epithelial surfaces of the bronchi,[199–201] pleura,[202] and pericardium.[203] They also describe thickening of bronchial epithelial basement membrane,[204] pericardium,[205] and blood vessel walls[206] by extra, organized collagen and elastin. Several of the animal-rearing studies on bronchial epithelial changes are well controlled and convincing.[207,208]

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

[199] Oliveira MJR, Pereira AS, Ferreira PG, Guinaraes L, Freitas D, Carvalho APO, Grande NR, Aguas AP. 2004. Arrest in ciliated cell expansion on the bronchial lining of adult rats caused by chronic exposure to industrial noise. Environ Res 97: 282–86.

[200] Oliveira MJR, Pereira AS, Castelo Branco NAA, Grande NR, Aguas AP. 2002. In utero and postnatal exposure of Wistar rats to low frequency/high intensity noise depletes the tracheal epithelium of ciliated cells. Lung 179: 225–32.

[201] Monteiro M, Ferreira JR, Alves-Pereira M, Castelo Branco NAA. 2007. Bronchoscopy in vibroacoustic disease I: "pink lesions." Inter-Noise 2007, August 28–31, Istanbul, Turkey.

[202] Pereira AS, Grande NR, Monteiro E, Castelo Branco MSN, Castelo Branco NAA. 1999. Morphofunctional study of rat pleural mesothelial cells exposed to low frequency noise. Aviat Space Environ Med 70(3): A78–85.

[203] Castelo Branco NAA, Aguas AP, Pereira AS, Monteiro E, Fragata JIG, Tavares F, Grande NR. 1999. The human pericardium in vibroacoustic disease. Aviat Space Environ Med 70(3): A54–62.

[204] Castelo Branco NAA, Monteiro M, Ferreira JR, Monteiro E, Alves-Pereira M. 2007. Bronchoscopy in vibroacoustic disease III: electron microscopy. Inter-Noise 2007, August 28–31, Istanbul, Turkey.

[205] Castelo Branco et al. 1999.

[206] Castelo Branco NAA. 1999. A unique case of vibroacoustic disease: a tribute to an extraordinary patient. Aviat Space Environ Med 70(3): A27–31.

[207] Oliveira et al. 2004.

[208] Oliveira et al. 2002.

to the same process, leading to prolonged middle ear effusions and unusual acute infections.

The increased asthma seen in subjects F1 and F3 may also have a connection to their frequent use of paracetemol (acetaminophen) for headaches during turbine exposure.[209]

Community noise studies and *annoyance*

Studies of community noise frequently assess a quality called *annoyance*. "Apart from 'annoyance,'" the World Health Organization writes, "people may feel a variety of negative emotions when exposed to community noise, and may report anger, disappointment, dissatisfaction, withdrawal, helplessness, depression, anxiety, distraction, agitation, or exhaustion."[210]

Beyond even these negative emotions, moving out of an owned home indicates that people feel sick and under threat, judging that their survival and well-being, and that of their children, will be enhanced by moving out—even as they exhaust limited resources to do so and face unrecompensed loss of their major asset, their home.

Sick and *annoyed* are not the same thing. In English, *annoyance* carries an air of triviality, like a mosquito buzzing around one's head. *Sickness* threatens survival itself.

Pedersen and Persson Waye assessed annoyance (which may be a shorthand for the above list of negative emotions, but remains different from sickness) among 351 households near wind turbines in Sweden in 2000. They used a mailed survey and compared annoyance to modeled A-weighted sound pressure levels they

[209] Beasley et al. 2008.

[210] World Health Organization 1999, *Guidelines for Community Noise*, p. 50.