Alec N. Salt *and*
Jeffery T. Lichtenhan

*Department of Otolaryngology*
*Washington University*
*School of Medicine*
*St. Louis, MO 63110*



# How Does Wind Turbine Noise Affect People?

***The many ways by which unheard infrasound and low-frequency sound from wind turbines could distress people living nearby are described.***

## Introduction

Recent articles in *Acoustics Today* have reviewed a number of difficult issues concerning wind turbine noise and how it can affect people living nearby (Leventhall 2013, Schomer 2013; Timmerman 2013). Here we present potential mechanisms by which effects could occur.

The essence of the current debate is that on one hand you have the well-funded wind industry **1.** advocating that infrasound be ignored because the measured levels are below the threshold of human hearing, allowing noise levels to be adequately documented through A-weighted sound measurements, **2.** dismissing the possibility that any variants of wind turbine syndrome exist (Pierpont 2009) even when physicians (e.g., Steven D. Rauch, M.D. at Harvard Medical School) cannot otherwise explain some patients' symptoms, and, **3.** arguing that it is unnecessary to separate wind turbines and homes based on prevailing sound levels.

On the other hand you have many people who claim to be so distressed by the effects of wind-turbine noise that they cannot tolerate living in their homes. Some move away, either at financial loss or bought-out by the turbine operators. Others live with the discomfort, often requiring medical therapies to deal with their symptoms. Some, even members of the same family, may be unaffected. Below is a description of the disturbance experienced by a woman in Europe we received a few weeks ago as part of an unsolicited e-mail.

*"From the moment that the turbines began working I experienced vertigo-like symptoms on an ongoing basis. In many respects, what I am experiencing now is actually worse than the 'dizziness' I have previously experienced, as the associated nausea is much more intense. For me the pulsating, humming, noise that the turbines emit is the predominant sound that I hear and that really seems to affect me.*

*While the Chief Scientist [the person who came to take sound measurements in her house] undertaking the measurement informed me that he was aware of the low frequency hum the turbines produced (he lives close to a wind farm himself and had recorded the humming noise levels indoors in his own home) he advised that I could tune this noise out and that any adverse symptoms I was experiencing were simply psychosomatic."*

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We asked how she felt when she was away from the wind turbines, to which she replied:

*"I did manage to take a vacation towards the end of August and for the two weeks we were away I was perfectly fine."*

The goal of our work in this field is to understand whether the physiology of the ear can, or cannot, explain the symptoms people attribute to wind turbine noise. As it is generally the case when debate influences a specific industry's financial interests and legal well-being, the scientific objectivity of those associated with the industry can be questioned. Liability, damage claims, and large amounts of money can hang in the balance of results from empirical studies. Whether it is a chemical industry blamed for contaminating groundwater with cancer-causing dioxin, the tobacco industry accused of contributing to lung cancer, or athletes of the National Football League (NFL) putatively being susceptible to brain damage, it can be extremely difficult to establish the truth when some have an agenda to protect the status quo. It is only when sufficient scientific evidence is compiled by those not working for the industry that the issue is considered seriously.

## Origins of Our Involvement in Infrasound from Wind Turbines

What is the evidence leading us to conclude that unheard infrasounds are part of the wind turbine problem, and how did we become involved in this debate? We are small group of basic and applied scientists, which means that our work addresses fundamental questions on how the ear works in normal and diseased states. While developing paradigms for our studies, we had been using a classic technique called "low-frequency biasing" – measurement of auditory responses to a test sound within the range of audibility, while simultaneously presenting a low-frequency tone (e.g., 4.8 to 50 Hz) to displace the sensory organ of the inner ear. Some auditory responses saturate when displaced by the bias tone, which can be used to establish whether the sensory organ is vibrating symmetrically or whether a fluid disturbance has displaced it to one side. A condition called "endolymphatic hydrops," which is found in humans with Ménière's disease, can displace the sensory organ as the space containing the fluid called endolymph swells. In our animal experiments we initially used 20 to 50 Hz bias tones, but for many reasons, and in large part based on a study in which we found that the ear responded down to 1 Hz (Salt and DeMott, 1999), we started using the lowest frequency our hardware could generate, 4.8 Hz, a frequency considered to be infrasound. Over the course of hundreds of experiments, we have found numerous biasing effects with 4.8 Hz tones at levels of 80 to 90 dB SPL (i.e., -13 to -3 dBA). We also found that the ear became about 20 dB more sensitive to infrasonic bias tones when the fluid spaces in the cochlear apex were partially occluded, as occurs with endolymphatic hydrops.

In late 2009, the first author received a report of a woman with Ménière's disease whose symptoms – primarily dizziness and nausea – were severely exacerbated when she was in the vicinity of wind turbines. From our animal data, we knew this woman was likely hypersensitive to very low-frequency sounds. Our subsequent review of the literature on wind-turbine noise revealed two aspects that were absolutely astounding:

**1.** Almost all measurements of wind turbine noise are A-weighted, making the unjustified assumption that hearing is the only way by which infrasound generates physiologic effects. The few studies that reported un-weighted measurements of wind-turbine noise, or recalculated spectra by removing the A-weighting from published A-weighted spectra, clearly demonstrated increasing energy towards low frequencies with highest energy levels in the infrasound region. We were surprised that objective full-frequency measurements showed that wind turbines generate infrasound at levels capable of stimulating the ear in various ways. Under such circumstances, A-weighting measurements of turbine noise would be highly misleading.

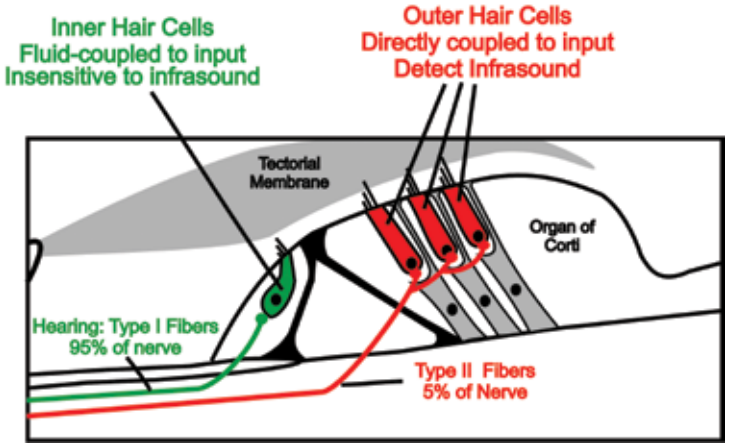


***Figure 1 :*** *The sensory organ of the cochlea, showing inner and outer hair cell and neural anatomy.*

**2.** Literature and websites from the wind industry often contained strong statements that wind turbine infrasound was of no significance. This view was largely based on publications by Leventhall (2006; 2007). Wind turbine noise was described as comparable to rustling leaves, flowing streams, air-conditioned offices or refrigerators heard from the next room. If wind turbine noise really was comparable to such sources then complaints would not be expected. But the turbines sounds are only comparable to these sources if the ultra-low frequencies emitted by the turbines are ignored through A-weighting. Stations that monitor infrasound or low frequency seismic (vibrational) noise for other purposes (for the detection of explosions, meteors, volcanic activity, atmospheric activity, etc.) are well-aware that low frequency sounds emanating from distant wind farms, or coupling to the ground as vibrations, can influence their measurements. The UK, Ministry of Defense has opposed wind turbines cited within 50 km of the Eskdalemuir Seismic Array. We have seen no reports of the Ministry opposing the presence of refrigerators in the region, suggesting they appreciate that sounds emitted from wind turbines and refrigerators are quite different. It was thus quite astounding to see the vast majority of wind turbine noise measurements excluding the low frequency noise content. Given the knowledge that the ear responds to low frequency sounds and infrasound, we knew that comparisons with benign sources were invalid and the logic to A-weight sound measurements was deeply flawed scientifically.

## The Ear's Response to Infrasound

Experimental measurements show robust electrical responses from the cochlea in response to infrasound (Salt and DeMott, 1999; Salt and Lichtenhan 2013). This finding was initially difficult to reconcile with measures showing that hearing was notably insensitive to such sounds but the explanation became clear from now-classic physiological studies of the ear showing that the two types of sensory cell in the cochlea had very different mechanical properties (Cheatham and Dallos 2001).

The auditory portion of the inner ear, the cochlea, has two types of sensory cell. The inner hair cells (IHC; shown green in *Figure 1*) are innervated by type I afferent nerve fibers that mediate hearing. The stereocilia (sensory hairs) of the IHCs are free-floating and do not contact the overlying gelatinous tectorial membrane (shown gray). They are mechanically displaced by fluid movements in the space below the membrane. As their input is fluid-coupled to the vibrations of the sensory organ they exhibit "velocity sensitive" responses. As the velocity of motions decreases for lower-frequency sounds, their fluid-coupled input renders the IHC insensitive to very low-frequency sounds. The other type of sensory cell, the outer hair cells (OHC; shown red in *Figure 1*) are innervated by type II afferent nerve fibers that are not as well understood as type I fibers and probably do not mediate conscious hearing per se. In contrast to the IHC, the stereocilia of the OHCs are inserted into the tectorial membrane. This direct mechanical coupling gives them "displacement sensitive" properties, meaning they respond well to low–frequency sounds and infrasound. The electrical responses of the ear we had been recording and studying originate from the sensitive OHCs. From this understanding we conclude that very low frequency sounds and infrasound, at levels well below those that are heard, readily stimulate the cochlea. Low frequency sounds and infrasound from wind turbines can therefore stimulate the ear at levels well below those that are heard.

The million-dollar question is whether the effects of wind turbine infrasound stimulation stay confined to the ear and have no other influence on the person or animal. At present, the stance of wind industry and its acoustician advisors is that there are no consequences to long-term low-frequency and infrasonic stimulation. This is not based on studies showing that long-term stimulation to low-level infrasound has no influ-

ence on humans or animals. No such studies have ever been performed. Their narrow perspective shows a remarkable lack of understanding of the sophistication of biological systems and is almost certainly incorrect. As we consider below, there are many physiologic mechanisms by which long-term infrasound stimulation of the cochlea could have effects.

One important aspect of wind turbine noise that is relevant to its physiological consequences is that the duration of exposure can be extremely long, 24 hours a day and lasting for days or longer, depending on prevailing wind conditions. This is considerably different from most industrial noise where 8 hour exposures are typically considered, interspersed by prolonged periods of quiet (i.e., quiet for 16 hours per day plus all weekends). There are numerous studies of exposures to higher level infrasound for periods of a few hours, but to date there have been no systematic studies of exposure to infrasound for a prolonged period. The degree of low-frequency cochlear stimulation generated by wind turbine noise is remarkably difficult to assess, due to the almost exclusive reporting of A-weighted sound level measurements. It certainly cannot be assumed that cochlear stimulation is negligible because A-weighted level measurements are low. For example, with 5 Hz stimulation cochlear responses are generated at -30 dBA and stimulation is sufficient to cause responses to saturate (indicating the transducer is being driven to its limit) at approximately 20 dBA (Salt and Lichtenhan, 2012; Salt et al., 2013). We have also shown that 125 Hz low-pass filtered noise at just 45 dBA produces larger responses than wide band noise with the same low-frequency content presented at 90 dBA (Salt and Lichtenhan 2012). We conclude that low frequency regions of the ear will be moderately to strongly stimulated for prolonged periods by wind turbine noise. There are a number of plausible mechanisms by which the stimulation could have effects:

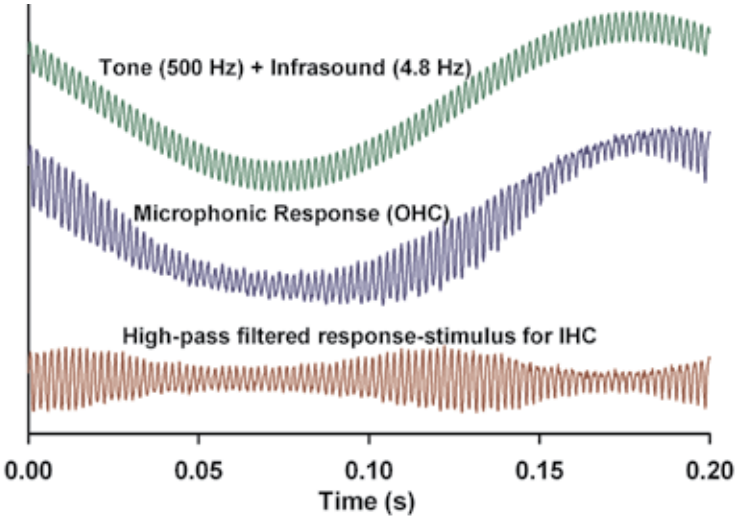


***Figure 2 :*** *Demonstration of biologically-generated amplitude modulation to a non-modulated stimulus consisting of an audible tone at 500 Hz tone summed with an infrasonic tone at 4.8 Hz. The cochlear microphonic response, which is generated by the OHC, includes low and high frequency components. The IHC detect only the high frequency component, which is amplitude modulated at twice the infrasound frequency for the stimuli in this example.*

**1. Amplitude Modulation: Low-Frequency Biasing of Audible Sounds**

Modulation of the biological mechano-electric transducer of the inner ear by infrasound is completely different from the amplitude modulation of audible sounds that can be measured with a sound level meter near wind turbines under some conditions. This can be demonstrated in low-frequency biasing paradigms in which a low-frequency tone and higher-frequency audible tone are presented simultaneously to a subject.

OHCs respond to both low- and high-frequency components and modulate the high-frequency components by either saturation of the mechano-electric transducer or by cyclically changing the mechanical amplification of high frequencies. IHCs, being insensitive to the low-frequency tone, see a high pass-filtered representation of the OHC response – an amplitude modulated version of the audible probe tone, as shown in *Figure 2*. As hearing is mediated through the IHCs that receive approximately 90-95% of afferent innervation of the auditory nerve, the subject hears the higher-frequency probe tone varying in amplitude, or loudness. A similar biasing influence on cochlear responses evoked by low-level tone pips was explained by the low-frequency bias tone changing OHC-based cochlear amplifier gain (Lichtenhan 2012). This same study also showed that the low frequency, apical regions of the ear were most sensitive to low-frequency biasing. Studies like this raise the possibility that the amplitude modulation of sounds, which people living near wind turbines report

as being so highly annoying, may not be easily explained by measurements with an A-weighted sound level meter. Rather, the low-frequency and infrasound levels need to be considered as contributing to the perceived phenomenon. Subjectively, the perceived fluctuation from an amplitude modulated sound and from a low-frequency biased sound are identical even though their mechanisms of generation are completely different. For the subject, the summed effects of both types of amplitude modulation will contribute to their perception of modulation. Acousticians therefore need to be aware that the degree of modulation perceived by humans and animals living near wind turbines may exceed that detected by a sound level meter.

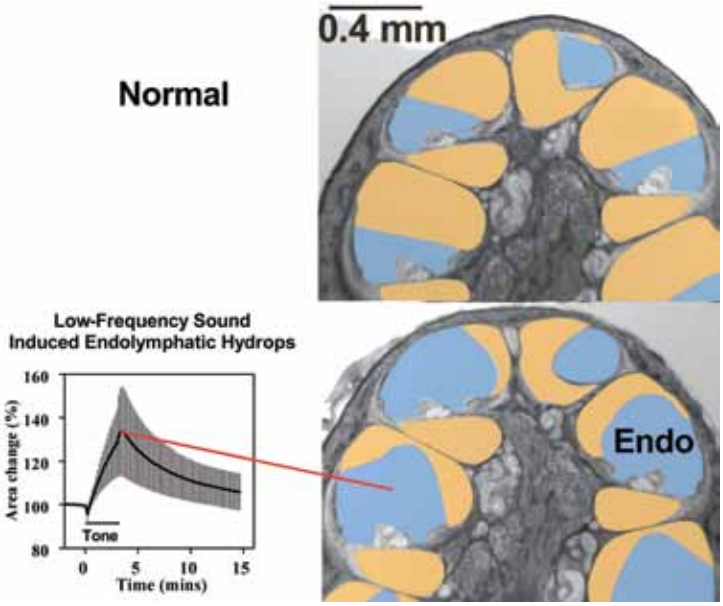


***Figure 3 :*** *Brief exposures to low-frequency tones cause endolymphatic hydrops in animals (Salt, 2004) and tinnitus and acoustic emission changes consistent with endolymphatic hydrops in humans (Drexel et al, 2013). The anatomic pictures at the right show the difference between the normal (upper) and hydropic (lower) cochleae The endolymphatic space (shown blue) is enlarged in the hydropic cochlea, generated surgically in this case.*

**2. Endolymphatic Hydrops Induced by Low Frequency Tones**

As mentioned above, endolymphatic hydrops is a swelling of the innermost, membrane bound fluid compartment of the inner ear. Low-frequency tones presented at moderate to moderately-intense levels for just 1.5 to 3 minutes can induce hydrops *(Figure 3)*, tinnitus (ringing in the ears) and changes in auditory potentials and acoustic emissions that are physiological hallmarks of endolymphatic hydrops (Salt, 2004, Drexl et al. 2013).

Unlike the hearing loss caused by loud sounds, the symptoms resulting from endolymphatic hydrops are not permanent and can disappear, or at least fluctuate, as the degree of hydrops changes. Return to quiet *(as in Figure 3)* or relocation away from the low-frequency noise environment allow the hydrops, and the symptoms of hydrops, to resolve. This which would be consistent with the woman's description of her symptoms given earlier. As hydrops is a mechanical swelling of the membrane-bound endolymphatic space, it affects the most distensible regions first – known to be the cochlear apex and vestibular sacculus. Patients with saccular disturbances typically experience a sensation of subjective vertigo, which would be accompanied by unsteadiness and nausea. As we mentioned above, an ear that has developed endolymphatic low frequencies – has to be considered. To date, all studies of low-frequency tone-induced hydrops have used very short duration (1-2 min) exposures. In humans, this is partly due to ethical concerns about the potential long-term consequences of more prolonged exposures (Drexel et al., 2013). Endolymphatic hydrops induced by prolonged exposures to moderate levels of low-frequency sound therefore remains a real possibility.

**3. Excitation of Outer Hair Cell Afferent Nerve Pathways**

Approximately 5-10% of the afferent nerve fibers (which send signals from the cochlea to the brain - the type II fibers mentioned above) synapse on OHCs. These fibers do not respond well to sounds in the normal acoustic range and they are not considered to be associated with conscious hearing. Excitation of the fibers may generate other percepts, such as feelings of aural fullness or tinnitus. Moreover, it appears that infrasound is the ideal stimulus to excite OHC afferent fibers given what has been learned about these neurons from *in vitro* recordings (Weisz et al, 2012; Lichtenhan and Salt, 2013). *In vivo* excitation of OHC afferents has yet to be attempted with infrasound, but comparable fibers in birds have been shown to be highly sensitive to infrasound (Schermuly and Klinke, 1990). OHC afferents innervate cells of the cochlear nucleus that have a role in selective attention and alerting, which may explain the sleep disturbances that some people living

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near wind turbines report (Nissenbaum et al. 2012). The likelihood that OHC afferents are involved in the effects of low-frequency noise is further supported by observations that type II innervation is greatest in the low-frequency cochlear regions that are excited most by infrasound (Liberman et al. 1990, Salt et al. 2009).

**4. Exacerbation of Noise Induced Hearing Loss**

Some years ago we performed experiments to test a hypothesis that infrasound was protective against noise damage (Harding et al. 2007). We reasoned that low-frequency biasing would periodically close the mechano-electric transducer channels of the sensory organ (reducing electrical responses as shown in the biasing studies above), and consequently reduce the amount of time that hair cells were exposed to the damaging overstimulation associated with noise exposure. The experimental study found that just the opposite was true. We found that simultaneous presentation of infrasound and loud noise actually exacerbated noise-induced lesions, as compared to when loud noise was presented without infrasound. Our interpretation was that low-frequency sound produced an intermixing of fluids (endolymph and perilymph) at the sites of hair cell loss resulting in lesions that were larger. A possibility to be considered is therefore that long-term exposure to infrasound from wind turbines could exacerbate presbycusis and noise-induced hearing loss. Because these forms of hearing loss develop and progress slowly over decades, this could be a lurking consequence to human exposures to infrasound that will take years to become apparent.

**5. Infrasound Stimulation of the Vestibular Sense Organs**

Recent exchanges in this journal between Drs. Leventhall and Schomer concerning the direct stimulation of vestibular receptors by sound at low and infrasonic frequencies deserve comment. Dr. Leventhall asserts that both Drs. Schomer and Pierpont are incorrect in suggesting that wind turbine infrasound could stimulate vestibular receptors, citing work by Todd in which the ear's sensitivity was measured in response to mechanical low-frequency stimulation applied by bone conduction. Leventhall fails to make clear that there are no *studies* reporting either vestibular responses, or the absence of vestibular responses, to acoustically-delivered infrasound. This means that for all his strong assertions, Leventhall cannot refer to any study conclusively demonstrating that vestibular receptors of the ear do *not* respond to infrasound. Numerous studies have reported measurements of saccular and utricular responses to audible sound. Indeed, such measurements are the basis of clinical tests of saccular and utricular function through the VEMP (vestibular-evoked myogenic potentials). Some of these studies have shown that sensitivity to acoustic stimulation initially declines as frequency is lowered. On the other hand, *in vitro* experiments demonstrate that vestibular hair cells are maximally sensitive to infrasonic frequencies (~1 – 10 Hz). Thus, sensitivity to acoustic stimulation may increase as stimulus frequency is lowered into the infrasonic range. Direct *in vivo* vestibular excitation therefore remains a possibility until it has been shown that the saccule and other vestibular receptors specifically do not respond to this stimulation.

Low-frequency tone-induced endolymph hydrops, as discussed above, could increase the amount of saccular stimulation by acoustic input. Hydrops causes the compliant saccular membrane to expand, in many cases to the point where it directly contacts the stapes footplate. This was the basis of the now superseded "tack" procedure for Ménière's disease, in which a sharp prosthesis was implanted in the stapes footplate to perforate the enlarging saccule (Schuknecht et al., 1970). When the saccule is enlarged, vibrations will be applied to endolymph, not perilymph, potentially making acoustic stimulation of the receptor more effective. There may also be certain clinical groups whose vestibular systems are hypersensitive to very low-frequency sound and infrasound stimulation. For example, it is known that patients with superior canal dehiscence syndrome are made dizzy by acoustic stimulation. Subclinical groups with mild or incomplete dehiscence could exist in which vestibular organs are more sensitive to low frequency sounds than the general population.

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**6. Potential Protective Therapy Against Infrasound**

A commonly-used clinical treatment could potentially solve the problem of clinical sensitivity to infrasound. Tympanostomy tubes are small rubber "grommets" placed in a myringotomy (small incision) in the tympanic membrane (eardrum) to keep the perforation open. They are routinely used in children to treat middle ear disease and have been used successfully to treat cases of Ménière's disease. Placement of tympanostomy tubes is a straightforward office procedure. Although tympanostomy tubes have negligible influence on hearing in speech frequencies, they drastically attenuate sensitivity to low frequency sounds (Voss et al., 2001) by allowing pressure to equilibrate between the ear canal and the middle ear. The effective level of infrasound reaching the inner ear could be reduced by 40 dB or more by this treatment. Tympanostomy tubes are not permanent but typically extrude themselves after a period of months, or can be removed by the physician. No one has ever evaluated whether tympanostomy tubes alleviate the symptoms of those living near wind turbines. From the patient's perspective, this may be preferable to moving out of their homes or using medical treatments for vertigo, nausea, and/or sleep disturbance. The results of such treatment, whether positive, negative, would likely have considerable scientific influence on the wind turbine noise debate.

## Conclusions and Concerns

We have described multiple ways in which infrasound and low-frequency sounds could affect the ear and give rise to the symptoms that some people living near wind turbines report. If, in time, the symptoms of those living near the turbines are demonstrated to have a physiological basis, it will become apparent that the years of assertions from the wind industry's acousticians that "what you can't hear can't affect you" or that symptoms are psychosomatic or a nocebo effect was a great injustice. The current highly-polarized situation has arisen because our understanding of the consequences of long-term infrasound stimulation remains at a very primitive level. Based on well-established principles of the physiology of the ear and how it responds to very low-frequency sounds, there is ample justification to take this problem more seriously than it has been to date. There are many important scientific issues that can only be resolved through careful and objective research. Although infrasound generation in the laboratory is technically difficult, some research groups are already in the process of designing the required equipment to perform controlled experiments in humans.

One area of concern is the role that some acousticians and societies of acousticians have played. The primary role of acousticians should be to protect and serve society from negative influences of noise exposure. In the case of wind turbine noise, it appears that many have been failing in that role. For years, they have sheltered behind the mantra, now shown to be false, that has been presented repeatedly in many forms such as "What you can't hear, can't affect you."; "If you cannot hear a sound you cannot perceive it in other ways and it does not affect you."; "Infrasound from wind turbines is below the audible threshold and of no consequence."; "Infrasound is negligible from this type of turbine."; "I can state categorically that there is no significant infrasound from current designs of wind turbines." All of these statements assume that hearing, derived from low-frequency-insensitive IHC responses, is the only mechanism by which low frequency sound can affect the body. We know this assumption is false and blame its origin on a lack of detailed understanding of the physiology of the ear.

Another concern that must be dealt with is the development of wind turbine noise measurements that have clinical relevance. The use of A-weighting must be reassessed as it is based on insensitive, IHC-mediated hearing and grossly misrepresents inner ear stimulation generated by the noise. In the scientific domain, A-weighting sound measurements would be

unacceptable when many elements of the ear exhibit a higher sensitivity than hearing. The wind industry should be held to the same high standards. Full-spectrum monitoring, which has been adopted in some reports, is essential.

In the coming years, as we experiment to better understand the effects of prolonged low-frequency sound on humans, it will be possible to reassess the roles played by acousticians and professional groups who partner with the wind industry. Given the present evidence, it seems risky at best to continue the current gamble that infrasound stimulation of the ear stays confined to the ear and has no other effects on the body. For this to be true, all the mechanisms we have outlined (low-frequency-induced amplitude modulation, low frequency sound-induced endolymph volume changes, infrasound stimulation of type II afferent nerves, infrasound exacerbation of noise-induced damage and direct infrasound stimulation of vestibular organs) would have to be insignificant. We know this is highly unlikely and we anticipate novel findings in the coming years that will influence the debate.

From our perspective, based on our knowledge of the physiology of the ear, we agree with the insight of Nancy Timmerman that the time has come to "acknowledge the problem and work to eliminate it".

## Biosketches

***Alec N. Salt*** is Professor of Otolaryngology at Washington University. He is a long-term member of the Acoustical Society of America, the Association for Research in Otolaryngology, and the American Otological Society. His research covers broad aspects of system-level cochlear physiology, with a major focus on the inner ear fluids, drug delivery to the inner ear, and low-frequency sound effects on the ear.

***Jeffery T. Lichtenhan*** is Assistant Professor of Otolaryngology at Washington University in St. Louis. He recently completed his postdoctoral fellowship in the Eaton-Peabody Laboratory of Auditory Physiology at Harvard Medical School. His research addresses questions on the mechanics of hearing to low-frequency acoustic sound, and the auditory efferent system. Ultimately, his work aims to improve the differential diagnostics of sensorineural hearing loss.

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