Case Report: Cross-Sensitisation to infrasound and low frequency noise

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ABSTRACT

This Case Report describes an episode experienced by two noise-sensitised individuals during a field trip. Exposed to residential infrasound and low frequency noise due to coal mining activities, the subjects reacted suddenly, strongly and unexpectedly to pressure pulses generated by a wind farm located at a different town, approximately 160 km by road from their residence. Simultaneous physiological data obtained in one subject and subjective sensations occurring during the episode are reported. Acoustical evaluations of the location of the episode are also reported. The possibility of a nocebo effect as an etiological factor for their bodily reactions is cogently eliminated. The degree of cross-sensitisation to acoustical phenomenon depends on prior exposure histories, and on the temporal characteristics of the acoustical phenomenon.

BACKGROUND

Many families residing in the vicinity of coal mining operations in New South Wales, Australia, report adverse health effects in response to infrasound and low frequency noise (ILFN). Mr & Mrs T and their children moved to this area in December 2009. Subsequent to significant health deterioration in all members of the family, noise measurement equipment was installed in the T home starting in 2015, and infrasound and low frequency components were monitored on a 24-hr basis for several months. Medical histories of the adult family members were recorded. Within the scope of CSI-ACHE (Citizen Science Initiative – Acoustical Characterization of Human Environments [1]), the family volunteered to wear physiological monitoring devices while at home, so that specific acoustical environments could be correlated with specific physiological responses. CSI-ACHE also includes families whose residential exposure to ILFN is caused by industrial wind turbines (IWT), and who have been reporting similar symptoms as the T family. The goal of this report is to document the reactions endured by Mr and Mrs T, as observed by 3 of the 4 authors and as registered on electronic devices,
when in the presence of infrasound generated by a nearby wind farm. Informal acoustical evaluations are also presented.

**Female Subject – Mrs. T.**

Caucasian, age 44, homemaker, non-smoker, no alcoholic habits. Mother has history of heart disease, and worked at processed food factories and as a sewing machinist while pregnant with Mrs T. Father died of asbestos-related mesothelioma. As a child, Mrs T had her tonsils and appendix removed and, as a teenager, developed mononucleosis and herpes simplex 1. Subject began living in urban environments at age 16, in the suburbs of Sydney, Australia. Mrs T had a hairdresser apprenticeship at age 17, but developed severe dermatitis with associated chemicals. Began administrative office work. At this time, she lived next to railway lines and power lines “that hummed,” but moved out one year after the birth of her first child in 1993. Two years later, the family moved to another home, where mould was a significant issue and finally led to their departure. In 2002, Mrs T had her second child, and began shift-work in the food industry. Later, she became a homemaker and domestically raised poultry for eggs. In December 2009, the T family moved from suburban Sydney to the Central Tablelands, in New South Wales. No significant exposure to radiation or asbestos was identified.

**Male Subject – Mr. T.**

Caucasian, age 40, public employee, non-smoker, no alcoholic habits. Mother has heart disease and worked as a sewing machine operator until she discovered she was pregnant. Father has diabetes. Three of the subject's four grandparents died of smoking-related lung cancer. Until the age of 4, subject lived in busy western suburb of Sydney; then moved to a more rural suburb where he lived until age 18. No childhood or adolescent medical conditions of note. Started working on motorcycles at age 10. At 18 he worked in a refrigerated warehouse for approximately 3 years. Subsequently, he worked in the automobile restoration business for 10 yrs, during which protective equipment was always used. Periodic testing subsequent to chemical exposures during automotive restoration work always returned as “cleared.” Changed to an administrative job in the insurance sector and then obtained a teacher’s certificate for automotive restoration, painting and rectification work. After moving to the Central Tablelands in December 2009, he obtained a position as a public servant that he maintains to this day. Mr T has never worked the ‘graveyard’ shift. No significant exposure to radiation or asbestos was identified.

**HEALTH COMPLAINTS SINCE 2009**

After moving to the new home, Mr and Mrs T noticed that their house often vibrated, audibly rattling windows and the shower screen. Within a year, Mr and Mrs T began awakening in the middle of the night, sensing vibration through their bed and audibly hearing home structures rattling. By the end of 2010, Mrs T reported feeling vibration through her feet. Mr T’s systematic awakening in the night was attributed to his snoring, which was also blamed as the cause of Mrs T’s interrupted sleep. By 2011, Mrs T developed numbness in her face and fingers and had difficulty swallowing. Multiple sclerosis was suspected but, after medical testing, was eliminated as a cause of her symptoms. Sleep continued to be disrupted, but was mainly attributed to Mr T’s loud snoring. In 2012, Mrs T took a cleaning job at a local resort hotel. Although she wore gloves and no dermatitis developed, she felt sick and thought it was the chemicals, so she quit. Also in 2012, prior symptoms persisted, and new medical conditions developed, particularly in Mrs T: digestive problems, unexplained joint pain, shortness of breath and nausea. A colonoscopy revealed secol polyps; an inflammatory process in the stomach was identified (no *Helicobacter pylori* detected); it was suggested that
the shortness of breath was related to anxiety. Mrs T developed a “pulsating head pressure” which impeded her from lying down to sleep, and was forced to sleep sitting up. She also began developing balance disorders. In 2013, all these symptoms collectively persisted. In this same year, the T family took a family trip to another part of the country, and realized that their symptoms greatly subsided. This led them to first suspect their home environment as the cause of their health complaints. Since Mr T’s snoring was still the main contender for the couple’s sleep disruption, in late 2013, Mr T had his tonsils removed. While the snoring did indeed cease, awakenings in the middle of the night did not. Starting in 2014, Mrs T began developing the feeling of intense coldness. Today, Mrs T reports nausea, shortness of breath and difficulty swallowing that occur in wave-like episodes, and simultaneously with other members of the family; increased emotional instability and no stamina or will to complete tasks. Mr T reports pulsating head pressure, nausea, and shortness of breath; about his symptoms he says: “I am about one year behind my wife.” The T family is currently undergoing diagnostic testing for vibroacoustic disease [2-5].

RESIDENTIAL ILFN EXPOSURE

Acoustical monitoring conducted on the T family property (Nov 2015-Feb 2016, and then again Sept-Oct 2016) was performed in accordance with the requirements of Australian Standard AS1055-1997. Two sets of instrumentation were used: one set external to the dwelling, in free-field, and the other inside the master bedroom.

Instrumentation used for the measurements included a SVAN 979 sound level meter with a GRAS 40AZ microphone, set to record and sample measurements 10 times per second for statistical purposes and, simultaneously, to record 24-bit 10-minute wave files for subsequent analyses if required. The sound level meter was field calibrated using a sound level calibrator (B&K 4230), before and after measurements, and did not exhibit any deviation. A secondary system using an INFILTEC micro-barometer was used to record pressure variations in the infrasound region. The output of the INFILTEC unit was connected to a RAND ACOUSTICS serial data logger type SDL2. The logger was set to record data 50 times per second, and store data files in one-hour blocks. The micro-barometer and logger were calibrated using a GRAS 42AE calibrator. All instrumentation used for these sound level measurements hold current calibration to manufacturers’ specifications, as required by Australian Standard AS1259.

Results obtained in the T home have not yet been submitted for publication. Figure 1 is an informal representative sonogram from inside the master bedroom, captured shortly after midnight. This sonogram is the result of passing the signal from a SVAN 979 through a bank of 1/24-octave, narrow-band filters compliant with ANSI® S1.11-2004 standard, class 0. These are uncalibrated, relative levels of acoustical energy within the 10-100 Hz range.

The large number of horizontal lines indicates the presence of strong tonal components within the soundscape. The strongest is at 50 Hz, which would be assumed to come from the house’s electrical mains, as would any harmonics. Several of the other tones, however, appear to be harmonics of a fundamental frequency slightly over 10 Hz, which did not arise from any source within the house.
Figure 1: Sonogram showing the relative levels of acoustical energy within the 10-100 Hz range, as frequently encountered in the Master Bedroom of the T family home (around midnight). The predominance of horizontal lines indicates acoustic sources with distinctive tonal characteristics.

THE EXPERIMENT

Background

In 2015, the T family hosted a team of researchers who were taking measurements in parallel with those already in place and described above. One of these used a prototype microbarometer to measure infrasound in addition to ‘Biopatch’ physiological monitors (Medtronic Zephyr™ [6]) to record heart rate, heart rate variability, respiration rate, skin temperature, energy usage, movement and orientation.

Mr & Mrs T reported becoming acutely and severely distressed whilst driving through the township of Taralga (Southern Highlands, New South Wales) earlier that year. This location has an IWT development in its proximity (Fig 2). Mr T reported developing pressure bolts, headaches and nausea while Mrs T had developed a sudden, severe headache and unexplained anxiety. This report presented itself to CSI-ACHE as an opportunity to further explore the correlation between physiological response and specific acoustic components in the soundscape. On December 4th a field trip was made to several locations near IWT developments while Mr & Mrs T were each wearing a Biopatch.
The Episode

On December 4th, 2015, the recorded incident happened at the very end of a long day that had been disappointing scientifically in that very few of the IWTs in the visited areas were operating. No acute responses had been recorded up to that point. On the way home, a stop was made in Taralga to make use of the public toilets. These were located behind the Taralga Memorial Hall on Orchard Street. An aerial plan view from Google Earth has been annotated to show the relative placement of the facilities and the vehicle (Fig 3).

The hall is of solid brick construction with the facilities at the left side of the building towards the rear where there is a short corridor (Fig 4).
Mrs T and a member of the research team left the parked vehicle and walked to the toilets behind the hall. On turning right down the side of the hall and passing the reflecting wall, Mrs T experienced a sudden reaction of nausea and a feeling that she was swaying, “like being on a ship at sea,” requiring her to grasp the handrail to maintain her balance. She described “waves of pressure” sweeping over her, nausea, and a feeling of extreme anxiety. No obvious sources of ILFN were present on the day in question. Trees obscured the IWTs, and neither person was aware of their proximity.

Physiological Response During the Episode

Figure 5 shows the physiological responses of Mrs T between 5:00 pm to 6:00 pm, as obtained with the Biopatch (Medtronics, Zephyr™ [6]), during the episode described above.
At approximately 5:18 pm, Mrs T exited the vehicle and walked toward the public toilet entrance. At the precise moment she passed the reflecting wall (Fig 4C) she immediately experienced sudden tachycardia accompanied by feelings of vertigo and anxiety. Her heart rate variability spiked from 40 to 90 beats per minute (Fig 5A). After steadying herself using the handrail she managed to walk to the toilets and return to the vehicle a few minutes later. At this point a researcher retrieved the SAM infrasound unit [7] and returned to the start of the guardrail (Fig 4A), recording for about 12 minutes (until approx 5:32) in Mrs T’s presence. Her heart rate again spiked, as did her heart rate variability (Fig 5B). The team returned to the vehicle and drove off at around 5:33 pm, at which time Mrs T’s heart rate and heart rate variability returning to baseline values (Fig 5).

Mr T was driving. Having driven maybe 1 km, he looked to the right where a break in the houses allowed the public toilet entrance to become visible. He reacted violently and instantly. He stopped the vehicle, jumped out and, in the middle of the road, doubled up and underwent dry retching for some time. In the same time (5:36, Fig 5-C), Mrs T’s heart rate again spiked to 120 beats per minute (bpm) and her heart rate variability hit 90 bpm. The entire team was shocked at this physical reaction, as the onset was so rapid and so physiologically violent. Unfortunately, Mr T’s reaction was so severe that it dislodged the Biopatch sensor, so no usable data could be obtained for him from the incident. After a few minutes, Mr T returned to the vehicle, extremely embarrassed for his sudden, uncontrollable reaction. Team members offered to drive but he stated that he was better in control of the vehicle as he was feeling some vertigo. A second attempt to drive off only managed to move the car forward a few metres before Mr T again brought the vehicle to an emergency stop, this time running into the grassy side, where he continued to dry retch uncontrollably for about 5 minutes. At around 5:39 pm, Mr T had recovered sufficiently to attempt to drive off again, reporting that the symptoms did not finally subside until about one hour later. Mrs T’s heart rate returned to baseline values after the team had driven off, although she reported inexplicable anxiety and pressure for the next half hour. (NB: The Biopatch real-time clock was later found to be approximately 7 minutes slow.)

**Acoustical Analysis**

The results of the informal recordings with a prototype infrasound recorder unit [7] are shown here. Data was captured continuously during the journey with approximately 12 minutes being recorded at the toilet location. The resultant sonogram is shown in Fig 6 from the time the digital filters settled until the instrument was returned to the car. It represents non-calibrated, relative levels of acoustical energy.

The sonogram shows a large proportion of energy in near 1 Hz and below. Between about 1 Hz and 3 Hz vertical striping can be seen within the frequency bands. The striping occurs at frequencies below the band in which they appear, e.g., in the 1–1.5 Hz bands the striping occurs at about 3–4 second intervals. This indicates the presence of amplitude modulation.

Figure 7 shows a 5-minute sonogram obtained on the outskirts of Taralga during the time when Mr T was suffering his episode. The car motor was running during this time. The same features indicating amplitude modulation can be seen in the 1–3 Hz region.

In view of the discovery of this amplitude modulation, the time signals were isolated from Figure 7 for the 1 and 1.25 Hz 1/24-octave bands (Figure 8).

It can be seen that the infrasound levels at these two frequencies were being modulated at a frequency of about 0.25 Hz, or a pulse every four seconds. The modulation depth is of the order of 5 dB. This depth of modulation would be readily noticeable as a change in perceived loudness had the carrier frequencies been in the audible range instead of the infrasound.
range. It can also be seen that the sound level of the 1.25 Hz band dropped by 20 dB and returned despite the 1 Hz band remaining relatively unchanged. The infrasound unit did not record the audible frequency range and so it is not possible to state whether amplitude modulation also occurred in that region.

Figure 6: Sonogram at Taralga public toilets, showing the relative levels of acoustical energy within the 0.5–41 Hz frequency range.

Figure 7: 5-minute sonogram from the outskirts of Taralga showing the relative levels of acoustical energy within the 0.5-41 Hz frequency range.
DISCUSSION AND CONCLUSION

At present, Mr & Mrs T have been exposed to excessive levels of ILFN in their residence for seven years. This experiment documents how sensitised they had become to this acoustical agent of disease at the end of five years. The immediacy of the onset of extreme bodily symptoms speaks to the nature of the agent of disease, and to the importance of prior exposure histories when current reactions are examined.

Sensitisation to airborne pressure waves rich in low frequency components is a phenomenon well-known to acousticians working in the field of low-frequency noise and vibration. It was described in 1985 in the seminal work of Dr. Neil Kelley. [8] A recent animal study [9] suggests that the degree of impulsiveness of the sound, or sound character, is as important as the frequency and peak amplitude. In 2011 Gotz & Janik [9] showed that wild seals exposed to impulsive sound became progressively sensitised to the sound and distressed by it. In contrast, a control group of wild seals exposed to the same sound level, but without the impulsivity, habituated to the sound. Another example of animal sensitisation can be found in laboratory experiments with Wistar rats exposed to ILFN. Here, non-exposed animals react to the sound of a blown kiss by becoming tense and nervously looking around for its source. After exposure, the same sound of a blown kiss induces tremors, rising on the hind legs and falling backwards [10].

The concept of ‘noise annoyance’ is a form of noise sensitisation. Classical hearing loss does not necessarily imply the existence of ‘noise annoyance.’ Indeed, annoyance to noise (any noise) is often the first symptom associated with excessive ILFN exposure [3,4], as opposed to having hearing difficulties or decreased speech intelligibility, i.e., the typical initial symptoms of classical hearing impairment. Many authors [for example, 11] still defend that ‘annoyance’ occurs as the consequence of an indirect pathway associated with noise exposure. Normally, those authors place annoyance on par with a “cognitive and emotional response” [11]. As suggested in 2003 [10], the organic lesions seen in the actin-based, cochlear cilia of ILFN-exposed laboratory rats could provide a physical, anatomical aetiology for the phenomenon of noise annoyance [12].

The notion that a nocebo effect may have been the aetiological factor for Mr and Mrs T’s episode in Taralga is only here entertained due to suggestions made by other authors [13]. (This issue is discussed in more detail by these authors in [14].) In the episode described herein, no hint of a nocebo effect is possible, since it was already “on-the-way-home,” after the
The experiment had been officially terminated; there was no reason to believe that there would be any adverse elements in the acoustic soundscape at the toilet stop. The Gotz & Janik experiment with seals [9] provides further scientific evidence that a nocebo effect cannot be used to dismiss such reactions as psychosomatic in origin, as does the laboratory experiment with Wistar rats [10]. Studies that have provided objective, clinical diagnostic tests to ILFN-exposed individuals have also negated the possibility of a nocebo effect [2-5]. Emesis, or involuntary vomiting, is controlled by the autonomic nervous system, as is the neurological control of breathing. It is therefore interesting to note that ILFN-exposed individuals who have undergone lung functional tests including the pCO₂ respiratory drive, reveal an impaired neurological response to the presence of excessive CO₂ (i.e., no hyperventilation is triggered) [15]. As an ending note on the nocebo issue, Figure 9A shows the domestic poultry eggs collected by Mrs T at her home in 2015. The teratogenic effect in Mrs T’s chick (Fig 9B) is similar in nature to that observed in ILFN-exposed rats (Fig 9C) [16]. Increased frequency of sister chromatid exchanges, as seen in ILFN exposed workers and animals [17-20], corroborate the genotoxicity of ILFN.

**Figure 9:** Photographs taken by Mrs T (Dec 2015) of the domestic poultry gestated and raised on their property. A. Abnormal hatching of turkey eggs. B. Teratogenesis in a chick gestated and born on the T family property. C. Teratogenesis in a Wistar rat gestated and born in ILFN [16].

The evolution of symptoms in Mr T occurred more slowly than in Mrs T. This is unsurprising since Mrs T spends more time in the home than Mr T. Prior noise exposure histories of Mrs T suggest that she had already lived in a home where ILFN was an issue, and may have had non-trivial *in utero* exposure. The evolution of signs and symptoms (clinical stages) associated with occupational exposures to ILFN has already been identified [2]. Residential exposures to ILFN however, have been observed to have an earlier onset than occupational exposures and an accelerated evolution [21-24]. It is postulated that exposure to ILFN during sleep-time may be playing an important role in this matter. Mrs T seems to have a predisposition for immune system disorders, as gleaned from her anamnesis summarised above (i.e., medical and familial histories self-reported to clinically-trained professionals [25]), and that can be a concomitant situation when living in ILFN-contaminated homes. ILFN exposure has already been shown to modulate the immune system in both human [26, for example] and animal models [for example, 27,28]. Balance disorders and other neurological impairments have also been previously documented in ILFN-exposed workers [29,30].

As is so often the case with wind turbine emissions, the blade-pass frequency manifests as a modulation of the overall sound emitted. This can be likened to listening to a radio where the volume control is being slowly turned up and down at low infrasound frequencies [31]. Wind turbines are not thought of as emitting these low frequencies as separate tones, rather they are simply artefacts of the modulation of the total sound envelope. It is likely that when residents complain about infrasound or pulsing noise, it is the total sound envelope changing in volume in a periodic fashion that is being perceived. How the human body responds to modulated infrasound is still a matter of debate and further research is urgently needed to determine the exact biological mechanism of action.
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Disclaimer

The authors 1) Do not harbour anti-technology sentiments; 2) Consider industrial activities to be important to modern technological societies; 3) Have presented this report under one and only on agenda—pure scientific inquiry; 4) Are not producing a report arguing against industrial developments.

REFERENCES


[25] Anamnesis, or case histories, are sine qua non components of clinical medicine. These are not to be confused with anecdotal stories that are recounted amongst non-clinically-trained professionals. For more on this topic, please see reference [14].


