RAT COCHLEA EXPOSED TO LOW FREQUENCY NOISE

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Introduction. Vibroacoustic disease (VAD) is an extra-aural, noise-induced pathology, caused by long-term exposure to low frequency noise (LFN) (≤500 Hz, including infrasound) [1]. Common auditory complaints of VAD patients include "hearing too much" and "not being able to stand any type of noise, not even television or music". However, their audiograms only present hearing losses within the lower frequency bands (250 Hz, 500 Hz), and their tympanograms are normal [1]. This noise intolerance has been understood by specialists as a pseudo-recruitment or recruitment-like situation. The goal of this study is to better understand the effects of LFN on the auditory system through the use of animal models and electron microscopy. Methods. Animals. Ten age-matched Wistar rats were exposed to LFN in an occupationally-simulated schedule: 8 hrs/day, 5 days/week, weekends in silence. Control rats were kept in equal living conditions, but in continuous silence. All animals were fed standard rat food, had unrestrained access to water, and were treated in accordance with applicable legislation (86/609/CE). Noise Exposure. Fig. 1 shows the overall linear and A-weighted noise and spectral analysis collected inside the rat chamber using a digital real time analyzer (B&K 2144). An analog noise generator produced an amplified and frequency filtered acoustic signal so that acoustic energy was highly concentrated in the lower frequency bands, 50 Hz to 500 Hz, exceeding 90dB_{Lin}. The overall linear sound pressure level was above 109dB_{Lin}, and the A-weighted level was around 98dBA. Microscopy. Animals were sacrificed by a lethal intravenous injection of sodium-pentobarbital after a cumulative 4399 hours of LFN exposure. Both cochlea of exposed and controls were removed and isolated. Specimens prepared for scanning electron microscopy (SEM) (JEOL JSM-35C) were dehydrated, critical point-dried, coated with gold-palladium and examined with the electron microscope at an accelerating voltage of 15 kV. Results. In control rats, cochlear cilia were lost with the normal aging
process (Fig. 2). In the exposed rats, cilia fused among themselves and with the upper tectorial membrane (Fig. 3). Cilia in the exposed rats also appeared shaggy, and no loss of ciliated cells was observed, despite being age-matched with controls. Ciliary fusion and/or shaggyness was observed along the entire cochlea. Control rats, independent of age, react to the sound of a blown kiss by becoming tense and discretely irritated. In the exposed rats, just before sacrifice, exposure to the sound of a blown kiss produced a dramatic startle response, some of them losing balance, and falling backwards. **Discussion.** Cochlea stereocilia are actin-based structures which fuse as a response to LFN exposure. This feature, if it also occurs in humans, may explain the unusual auditory complaints of VAD patients. If fused among themselves and to the tectorial membrane, cilia cannot freely vibrate as is intended when the sound pressure wave is transduced within the cochlea. In fact, by becoming a rigid structure, any attempt at vibrating them might, understandably, produce discomfort. How closely related this phenomenon is to the concept of “annoyance” is still unclear, however a relationship is clearly suggested, especially since annoyance has already been specifically associated with the presence of LFN [2]. Fusion of actin-based structures in LFN-exposed rodents can be observed in other organs. Respiratory tract brush cells possess a tuft of individual strands of actin-based microvilli, uniformly distributed over the apical surface of the cell that is open to the airway. These actin-based structures also fuse in the presence of LFN [3]. The behavior of a variety of other LFN-exposed actin-based structures is currently under study. Altered cilia are found along the entire cochlea and not merely at the apex, where LF sound waves (with a larger wavelength) are supposed have a more evident effect. This is an interesting feature that has led to the design of a new research program to evaluate if there is a topographic specificity in the progression of these LFN-induced morphological changes.

**Keywords:** vibroacoustic disease, actin, cilia, annoyance, electron microscopy, noise exposure