Conclusions This experiment confirms that the styrene exposure is responsible for cochlear dysfunctionality. A quantitative relation between the styrene exposure and the reduction of cochlear functionality, expressed in terms of DPOAE amplitude, can be found. Exposure-induced damage of the cochlear amplifier is shown in the mid-frequency range, confirming the results of animal experiments, in which hair cells in the middle turn of the cochlea were damaged. Hearing damage is consistent with the outer hair cell apoptosis pathway associated with oxidative stress.

1685d COCHLEOTOXICITY AND NEUROPHARMACOLOGY OF AROMATIC SOLVENTS CAN POTENTIATE NOISE-INDUCED HEARING LOSS

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The toxic mechanisms involved in solvent-induced hearing loss are not well-known, even though, today, the disturbances of K⁺ recycling and the generation of reactive oxygen species in the organ of Corti are considered as determinant. The cochleotoxic effects of these pollutants shows up after a long-lasting exposure and ends up by the phagocytosis of outer hair cells (OHCs) initiated by the supporting cells. Because of the lipid peroxidation which takes place within the OHC membranes, the latter are more vulnerable to noise. As a result, a solvent exposure can exacerbate the traumatic effects of noise at the level of the organ of Corti. Such a phenomenon is not the only one capable of explaining the synergistic effects of a co-exposure to noise and solvents. Indeed, solvents can also disturb the function of the middle-ear acoustic reflex which protects against continuous high-intensity noises by contracting the stapes muscle and thereby reduces the amount of acoustic energy penetrating into the cochlea.

Recently, it has been shown that solvents like benzene, toluene, ethylbenzene and xylenes disturb the function of the middle-ear reflex and therefore allow noise to be more cochlea-traumatic. These neuropharmacological effects are likely due to an action of the pollutants on the targeted neuroreceptors of the acoustic reflex circuit. The interaction 'pollutants vs receptors' would be determined by the stereo-specificity rather than by the lipophilicity of the molecules. Both cochleotoxic and neuropharmacological phenomena can induce the traumatic effect of the noise on the organ of Corti. Partial disruption of the organ of Corti, or temporary disturbances of the reflex by solvents, both can increase the risk of occupational deafness encountered by co-exposed workers. Despite these statements, the European hearing conservation programs have not changed their concerns about the co-exposures to noise and solvents.

1685e NANOTECHNOLOGY IN EAR DISEASES: PROMISING AND CHALLENGING ISSUES

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Introduction Nanotechnology and nanomedicine are innovative and rapidly developing areas, aimed to develop nanoscale structures and devices whose physico-chemical properties may be useful for pathological diagnosis and treatment. To date, almost no effective remedies are available for inner ear diseases, i.e. sensorineural hearing loss and vertigo, that are common and disabling conditions caused by the degeneration of sensory-neural transducer epithelial cells and nerve cells. In this scenario, nanotechnology attracted increasing scientific interests for its potential to improve existing treatments, but also raised concerns on possible, still not-fully explored, adverse effects exerted by nanomaterials on the auditory system. Therefore, aim of this work is to provide a comprehensive overview of existing evidence concerning biological interactions between nano-based applications and ear structures.

Methods A systematic search and revision of experimental *in vitro* and *in vivo* studies addressing possible effects exerted by nano-enhanced remedies on auditory system was performed in the Pubmed, Scopus, and ISI Web of Science databases.

Results Nanoparticle-based systems proved a high potential for inner ear delivery of various therapeutic agents in animal models and were reported to exert a significant protection against drugs- (i.e. cisplatin) as well as noise-induced hearing loss. However, nanomaterials were also able to induce ototoxic effects on cultured cochlear epithelium, as well as middle and inner ear mucosa permeability changes, associated with hearing loss in animals trans-tympanically treated with nanoparticles.

Discussion Conflicting results emerged concerning nanotechnology applications in otology. Nano-enhanced systems may provide benefits to ear disease treatments, overcoming inner ear anatomic inaccessibility, minimising systemic treatment side effects, allowing a specific and sustained drug release in inner ear fluids. However, other investigations are necessary to deeply assess nano-ototoxicity risks particularly in relation to nanomaterial physico-chemical characterisation, and verify issues of biocompatibility, drug release profile, and biosafety before achieving a successful clinical application.

1685f HOW DOES MECHANICAL VIBRATION REACH THE COCHLEA?

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Introduction Several epidemiological studies highlighted a synergistic interaction between noise and mechanical vibration exposure.¹ The etiologic mechanism is still missing. Moreover, the measured transmissibility from the hand to the head seems to be poor. At the shoulder, frequency over 30 Hz are practically suppressed. If there are not any vibration left, how can mechanical energy interact with the cochlea? The aim of this speculation is to approach the transmission of vibration from entering point to the cochlea from a different point of view.

Proposed methods In the seminar on hand-arm vibration exposure³ to isolated and repeated shock vibrations held in October 2015 in Beijing it was suggested that a wider spectral component of mechanical vibration may travel in the blood vessels and impair vasoregulation and nerve terminations in