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Noise-induced hearing loss as a complex disorder influenced by environmental and genetic factors

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Noise-induced hearing loss (NIHL) is the second most common form of sensorineural hearing loss in adults, after presbyacusis. Approximately 25-30 million workers in Europe and 30 million workers in the US work in an environment with excessive noise levels. This number should be adjusted for a large population of individuals exposed to environmental noise, military noise and during the leisure activities. Besides well know parameters of noise contributing to occupational NIHL, such as the equivalent level of exposure to noise (LeqA) and years of exposure, some other factors may play a role. They include i.e. impulsiveness of noise (impulse noise is more harmful than steady-state at the same equivalent level), exposure paradigm (breaks in noise exposure allow for the recovery), occupational exposures to certain chemicals, i.e. organic solvents, asphyxiants and heavy metals, co-exposure to noise and vibration, ototoxic drugs (aminoglycosides, cis-platin and others), and smoking. Special care should be taken for individuals exposed to chemical substances, as it was lately shown for organic solvents.

Organic solvents are frequent contaminants of atmoshperes in industry, including paint and lacquer factories, dockyards, printing industry, plants manufacturing yachts, furniture, plastic, fibres, rubber tires and several other products. They have detrimental effects on both – peripheral and central part of the auditory pathway. A synergistic effect occurs in case of combined exposure to noise and solvents and this significantly increases the odds ratio of developing hearing loss. Thus, awareness should be raised among occupational physicians and decision makers that in solvent alone and combined with noise exposures, the current limits and hearing conservation programmes might be inadequate.

Remarkably, the individual susceptibility to NIHL varies greatly, and this inter-individual variation is due to an interaction of environmental factors and susceptibility genes. It has been demonstrated that mouse strains exhibiting age-related hearing loss are more susceptible to noise than other strains, while some knockout mice (SOD1-/, GPX1-/-, PMCA2-/- and CDH23+/-) were more sensitive to noise than their wild-type littermates. Very little is known about hereditary of NIHL in humans. An increased susceptibility to NIHL may rely on the Single Nucleotide Polymorphisms (SNPs) of several genes, including the groups of oxidative stress genes, potassium ions recycling genes, monogenic deafness genes and mitochondrial genes. Recent association studies indicate that SNPs in some potassium recycling genes - GJB2 (Cx26), GJB6 (Cx30), KCNQ1, KCNQ4 and KCNJ10 - may play an important role in determining the individual susceptibility to NIHL.
Identifying new environmental hazards and looking for intrinsic risk factors of developing NIHL, as well as more effective methods of prevention and medical treatment could have a beneficial impact both on health and economics.