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Review Article

Cochlear Synaptopathy and Noise-Induced Hidden Hearing Loss

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Recent studies on animal models have shown that noise exposure that does not lead to permanent threshold shift (PTS) can cause considerable damage around the synapses between inner hair cells (IHCs) and type-I afferent auditory nerve fibers (ANFs). Disruption of these synapses not only disables the innervated ANFs but also results in the slow degeneration of spiral ganglion neurons if the synapses are not reestablished. Such a loss of ANFs should result in signal coding deficits, which are exacerbated by the bias of the damage toward synapses connecting low-spontaneous-rate (SR) ANFs, which are known to be vital for signal coding in noisy background. As there is no PTS, these functional deficits cannot be detected using routine audiological evaluations and may be unknown to subjects who have them. Such functional deficits in hearing without changes in sensitivity are generally called "noise-induced hidden hearing loss (NIHHL)." Here, we provide a brief review to address several critical issues related to NIHHL: (1) the mechanism of noise induced synaptic damage, (2) reversibility of the synaptic damage, (3) the functional deficits as the nature of NIHHL in animal studies, (4) evidence of NIHHL in human subjects, and (5) peripheral and central contribution of NIHHL.

1. Noise-Induced Hidden Hearing Loss (NIHHL)

Noise-induced hidden hearing loss (NIHHL) refers to any functional impairment seen in subjects with noise exposing history but no permanent threshold shift (PTS). This is different from the conventional definition of noise-induced hearing loss (NIHL), which is based on changes in auditory sensitivity or threshold shift [1]. Therefore, noise exposure recommendations are based on the likelihood that a particular dose of exposure will result in a PTS. Noise exposures that are not expected to cause PTS are thus considered safe.

Physiologically, variations in auditory sensitivity following exposure to noise are largely due to the functional status of outer hair cells (OHCs) in the cochlea, which provide mechanical amplification of soft sounds [2, 3]. Noise exposures that result in only a temporary threshold shift (TTS) have a reversible impact on OHC function, which is manifested by the recovery of otoacoustic emissions (OAE) [4–6] and cochlear microphonics (CM) [7–11]. The functional changes in these measures parallel the recovery of hearing thresholds, as well as the repair of structures such as stereocilia and the tectorial membrane [7, 12]. By contrast, noise exposure at higher levels and/or for longer durations can cause permanent damage to, or even the death of, OHCs and, hence, lead to PTS. Therefore, the OHCs and the structures surrounding them, including the tectorial membrane and the supporting cells, are considered to be the major loci of cochlear damage that result in noise-induced threshold shifts [13, 14].

Although some early reports claimed that reversible noise-induced IHC pathologies were responsible for TTS [15, 16], IHCs are relatively insensitive to noise-induced cell death. However, it has long been recognized that the synapse between IHCs and primary spiral ganglion neurons (SGNs) can be damaged by noise [17–19]. These early studies showed that this manifests mainly as damage to the postsynaptic terminals; however, there is clear evidence from more recent studies that noise induces damage to both pre- and postsynaptic structures. More importantly, disruption of the synapses can be permanent, resulting in degenerative death of SGNs [6]. The finding that damage to ribbon synapses

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impairment are aphasia, The Relation of Human Language to Human Emotion.Disorders impairing a patient's communication abilities may involve voice, speech, language, hearing, and/or cognition. Recognizing and.Items 1 - 24 of 24 Ed. Vol. 1. Thousand Oaks: SAGE Publications, Inc., The range of speech communication disorders that arise during a . There is a strong reason to believe that human children are . The lifetime incidence of the disorder is estimated to be 5%, with a 1% prevalence rate in the population.