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Commentary

Brief Note on Presbycusis: An Age-Related Hearing Loss

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DESCRIPTION

Presbycusis, also known as Age-Related Hearing Loss (ARHL), is the gradual loss of hearing that most people experience as they get older. Debilitating hearing loss affects about one-third of people over the age of 65. In 2025, there will be 1.2 billion people over the age of 60 on the planet, with more than 500 million of them suffering from presbycusis. ARHL is a progressive, irreversible, and symmetrical bilateral neuro-sensory hearing loss caused by cochlear degeneration (where sound-induced vibrations are encoded by sensory hair cells into electrical signals in cochlear neurons that relay the information to the brain) or auditory nerve fiber loss during cochlear ageing. Hearing loss starts in the high-frequency range of the auditory spectrum and progresses to the low-frequency range as people get older. Threshold sensitivity declines with age, which is connected with problems in speech discrimination, as well as sound detection and localization, especially in noise. Males are more affected than females in general. Presbycusis, if left untreated, can lead to social isolation, depression, and dementia.

Hearing sensitivity declines faster in men over the age of 20 to 30, and in women over the age of 50, according to epidemiologic research in large groups of unscreened old persons. Men's average hearing thresholds show a dramatic increase in hearing loss in the high frequency range, but women's audiograms show a more progressive rise. Surprisingly, a large percentage of individuals reported noise exposure, otologic disease, and ototoxicity, implying that the cause of hearing loss in unscreened populations is not solely due to ageing.

Researchers proposed three major forms of ARHL based on temporal bone analyses correlating the patterns of hearing loss with defect location:

- Sensory presbycusis, characterized by an abrupt pure-tone threshold elevation in the high frequencies and hair-cell loss at the basal end of the cochlea.
- Strial presbycusis, characterized by a flat- or slightly descending pure-tone audiogram and stria vascularis atrophy.
- The exact mechanisms behind age-related deterioration of various cochlear components are unknown. This owes in part to the intricacy of each causal element, but more significantly, the combination of the various mechanistic pathways that can result in age-related hearing loss.

Sensory hair cells are vulnerable to a variety of ailments over time, including direct mechanical injury, mitochondrial oxidative stress from noise, ototoxic drugs such as aminoglycosides and cisplatin, and other unknown causes. The aggregation of multitudinous noise-induced loss of afferent dendrites may drive Spiral Ganglion Neuron (SGN) degeneration. Surprisingly, cochlear neurons with high thresholds and low spontaneous rates are the most sensitive to noise and ageing. Although these low-Spontaneous Rate (SR) fibers do not contribute to threshold detection in quiet settings, they do contribute to transient stimulus coding in the presence of continuous background noise, giving rise to the new idea of concealed hearing loss. For those patients who exhibit difficulty but have relatively normal thresholds in calm, tone in noise detection may be a useful metric in detecting age-related hearing losses.

This review summarizes our current knowledge of the effects of biological cochlear ageing on hearing, as well as the extrinsic and intrinsic risk factors that exacerbate age-related hearing function, as well as the molecular pathways that mediate cochlear cell senescence and degeneration as a result of ageing, injury (noise, ototoxic drugs), and genetic predisposition. We also go through some of the most recent breakthroughs in hearing restoration research.

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