Cellular mechanism alterations in the aging cochlea and implications for future therapies.

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Age-related hearing loss (presbyacusis), a progressive neurodegenerative disorder, is a global public health concern that negatively impacts communication, health, and quality of life of many older adults. Presbyacusis is a multifactorial complex disorder that can be associated with loss and/or dysfunction of multiple cell types in several cochlear regions. Dysfunction of neural-crest derived glial cells and resident immune cells such as microglia/macrophages, together with dysregulation of the complement cascade, play a vital role in the development of several age-related neurodegenerative disorders. The complement system, a fundamental element of the innate immune system, regulates the ability of antibodies and phagocytic cells (including tissue macrophages) to clear damaged cells from an organism. Recent studies from our and other laboratories have revealed that glial cells and macrophages undergo structural alterations with increasing age and in other pathological conditions, indicative of functional changes, and that these alterations are closely associated with pathological changes in several cochlear regions in mouse and in human temporal bone tissues, e.g., dysmyelination of the auditory nerve and disruption of the microvasculature in the cochlear lateral wall. In addition, gene expression changes in numerous molecules that were associated with innate immune response and complement cascade pathways were identified in the auditory nerve and cochlear lateral wall tissues of aged mouse ears. Discussion will focus on how macrophages and complement related signaling molecules can be used to identify targets for preventing the progression of age-dependent alterations in the cochlea and reduce subsequent functional declines. [Supported (in part) by grants from NIH].

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Present podium (Invited)