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Sommario/riassunto	Worldwide, 278 million people are estimated to have moderate to profound hearing loss. Age-related hearing loss, also known as presbyacusis, affects approximately half of the population over 60 years old, making it the second most common cause of disability in older people. Hearing loss occurs when the sensory cells and neurons of the cochlea degenerate and die. The vestibular system, which holds the sense of balance, shares a common embryonic origin with the cochlea and together conform the inner ear. Balance problems are a trait of ageing to the point that balance ability is considered a sensor of physical decline and vestibular degeneration is the most common cause of falls in the elderly. Still the molecular bases of ageing in the vestibular system have not been studied in detail. Genetic and environmental factors contribute to the progression of age-related hearing loss (ARHL). Being noise the main environmental noxious agent for human hearing in the industrialized societies.There is no restorative treatment for deafness but functional replacement by means of

prosthesis. Therefore, prevention and treatment of hearing loss is an unmet medical need. To develop innovative medical strategies against hearing loss, it is critical to understand the causes of ARHL and the essential pathways responsible for the manifestation of this complex disease.In this research topic, experts will discuss the stages and molecular elements of the damage and repair processes involved in ARHL, from cellular processes involved in ageing as senescence and autophagy, to molecules essential for hearing as IGF-1 and neurotrophins. Neuroinflammation takes a central stage as an essential element in the progression of injury and cell loss, and a target for cell protection strategies. Neurogenesis is also essential to understand the adult cochlea self-repair potential. Finally, the mechanisms of action and the potential of novel therapies for hair cell repair and protection will be discussed along with drug delivery strategies.