

LECTURE PRESENTATION

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Age-related hearing loss: biological aspects

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Presbycusis is one of the more prevalent neurodegenerative disease of aging. There are many studies about the influence of environmental and genetic factors. Age-related hearing loss is caused by changes in peripheral (cell loss in organ of Corti, spiral ganglion and stria vascularis) and central auditory systems (consequent to peripheral modifications or for changes in the neurobiologic activity underlying central processing of auditory informations) [1]. Consequences are reduced sensitivity, tuning sharpness, compression, and reduced signal-to-noise ratios, deficits in auditory discrimination, temporal processing, processing of degraded auditory signals or when embedded in competing acoustic signals. Approaching biology of age-related hearing loss is complex: it needs to clarify some peripheral aspects with different cochlear structure and cellular type affected, and some others central auditory processing aspects. There are some peripherally induced central effects and others direct neurodegenerative changes in the brain. Moreover biochemical and mechanical injury in life course can represent a risk factor for auditory function particularly for organ of Corti. This complicates the attempt of separate pure presbycusis from socioacusis. Research indicated some "longevity genes" and longevity-promoting life-styles (obesity and correlated conditions like hyperlipidemia, hypercholesterolemia, hypertension, hyperhomocysteinemia and cardiovascular disease, smoking, diet and diabetes [2,3]. Age-related hearing loss seems to occur more frequently in industrial population than in non-industrial [4]. The relation between alleles pro or against-aging and environment maybe play a determinant role in the evolution of hearing with aging. Until such genes are identified, the best strategy is to reduce

environmental risk factors (noise exposure, ototoxic drugs, industrial solvents or combinations of these).

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