BMC Geriatrics

LECTURE PRESENTATION Open Access

Age-related hearing loss: biological aspects

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From de Senectute: Age and Health Forum
Catanzaro, Italy. 5-7 December 2009

Presbycusis is one of the most prevalent neurodegenerative diseases of aging 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 due to 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. Moreover there are many studies about the influence of environmental and genetic factors. 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 other about central auditory processing. There are some peripherally induced central effects and others direct neurodegenerative changes in the brain. On the other side biochemical and mechanical injury in the course of life can represent risk factors relating to auditory function, particularly for organ of Corti. This complicates the attempt to 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 populations than in non-industrial [4]. The relation between genes pro or against-aging and environment may play a determinant role in the evolution of hearing with aging. Until such genes are identified, the best strategy is to reduce environmental and subjective risk factors (noise exposure, ototoxic drugs, industrial solvents or combinations of these).

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Published: 19 May 2010

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doi:10.1186/1471-2318-10-S1-L57
Cite this article as: Cassandro and Chiarella: Age-related hearing loss: biological aspects. *BMC Geriatrics* 2010 **10**(Suppl 1):L57.

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