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Role of osteocyte apoptosis in peculiar ossicles of the hearing sense organ: preliminary observations on hearing loss and osteoporosis

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Starting point of the present study is the osteocyte role in bone remodelling that allows bone adaptation to mechanical load [1-3]. Bone remodelling has been investigated in relation to the occurrence of apoptosis [4] to understand if and how the process of programmed cell death interferes with bone turnover. In 1998, in a study on human middle ear, Marotti et al. [5] demonstrated that: 1) over 40% of osteocytes are dead within the 2nd year of age (but the authors were not able to demonstrate if osteocyte death occurred by degeneration or apoptosis); 2) bone remodelling occurs only occasionally. Recently [6], we showed that: 1) osteocytes of human auditory ossicles die by apoptosis; 2) also osteocytes located inside scleral ossicles of lower vertebrate eye (reptiles and birds) phylogenetically so far from human auditory ossicles are widely affected by apoptosis (about 60%); 3) in scleral ossicles bone turnover never occur. It is to be noted that both auditory ossicles of human ear and scleral ossicles of vertebrate eye are peculiar bony segments continuously submitted to stereotyped stresses and strains, with specialized functions: the first are involved in sound wave transmission and the latter protect the eyeball against deformation during the movement and have a role in visual accomodation, providing attachment for the ciliary muscles. In both cases, bone remodelling might severely impair, by resorption, the mechanical resistance of these extremely small specialized bony segments. Thus, we suggested that in auditory and scleral ossicles, submitted to stereotyped loading for all life, bone mechanical adaptation is not needed and osteocyte programmed death could represent the mechanism to avoid bone remodelling and to make stable, when necessary, bone structure and mechanical resistance. More recently, to confirm this hypothesis, clinical data were collected from a cohort of patients aged 55-85 years affected by hearing loss. The main target of the present study is to exclude any correlation between hearing loss and osteoporosis. During osteoporosis, unbalanced bone turnover causes the bone depletion in skeletal segments; such condition, in the peculiar ossicles of human middle ear, should imply hearing impairment. Our preliminary observations indicate, instead, that osteoporotic patients do not show higher percentage of hearing loss with respect to non osteoporotic ones. This evidence is ascribable to osteocyte apoptosis of auditory ossicles that avoid bone remodelling, thus assuring the integrity of such bony segments also in osteoporotic conditions.

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