The Role of Matrix Metalloproteinase-13 in Osteocytic Perilacunar Remodeling and Bone Quality in the Cochlea.

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Abstract

Objective: The quality of cochlear bone is crucial for hearing. Although the mechanisms controlling bone health are unclear, recent studies suggested osteocytic remodeling of the bone is vital for maintaining hearing. We hypothesized the role of MMP13 in the cochlear remolding. The study seeks to determine the role of MMP13 in the maintenance of cochlear bone health and hearing. We examined if MMP13-deficient mice show long bones with decreased resistance to fracture, disorganized canalicular network, collagen and aberrant mineralization.

Methods: Ongoing studies examine the ability of other PLR enzymes to compensate for MMP13-deficiency to PLR via MMP13. MMP13 deficiency did not lead to irregular bone matrix or impaired hearing.

Conclusions: MMP13-/- mice (n=5) showed increased variability relative to wild-type littermates. Sensorineural hearing deficit did not appear related to bone remodeling in MMP13-/- mice. Histological results, the presence of a normal canalicular network, and mineralized bone matrix in all groups shows that the role of MMP13 in bone is crucial to hearing.

Discussion

In long bones, MMP13-mediated PLR is necessary to maintain normal bone health. Without it, mice deficient in MMP13 show long bones with a decreased resistance to fracture, disorganized canalicular network, disorganized collagen, and aberrant mineralization. When we examine these same hallmarks of PLR in the cochlea of MMP13-deficient mice, we surprisingly see that some same signs of dysregulation of the ECM are not present. The cochlea is able to maintain its normal hearing function and structure despite the lack of MMP13.

These results suggest a novel mechanism at is work to protect the crucial hearing function of the cochlea provides, independent of MMP13 as well as osteoclast- and osteoblast-mediated bone remodeling.

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References