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Title: Enhanced BMP signaling via ALK2 in osteoclasts decreases bone density in mice

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Abstract

Bone remodeling is a complex biological process that has been extensively studied. Bone Morphogenetic Proteins (BMPs) are recognized as one of the critical growth factors that coordinate bone remodeling. Previous studies have demonstrated that BMP signaling in osteoclasts has a positive effect on osteoclast function. However, little is known about how each BMP type I receptors control osteoclastogenesis. To investigate this question, we utilized the Cre-LoxP system to specifically activate BMP signaling through ALK2 in mice. We utilized Cathepsin K (Ctsk)-Cre driver to activate BMP signaling in osteoclasts in mice. Compared with aged- and gender-matched controls, gain-of-function of BMP mutant mice (hereafter ca-ALK2:Ctsk-Cre) displayed bone loss. Consistent with this gross morphology, the number of tartrate-resistant acid phosphatase (TRAP)-positive osteoclasts was significantly increased in ca-ALK2:Ctsk-Cre mice. These results indicate that enhanced BMP signaling through ALK2 promotes osteoclast function, leading to impaired bone remodeling and thus reducing bone density. Since multiple BMP pharmacological inhibitors have been approved by the FDA, our study has the potential to provide mechanistic insights for exploring treatment options for osteoporosis.