## Journal of Bone and Mineral Metabolism Best Paper Award 2014

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The Journal of Bone and Mineral Metabolism Best Paper Award was established in 2008. Candidates for the award must be members of the Japanese Society of Bone and Mineral Research, and the winner is honored at the Society's Annual Meeting.

We are pleased to announce that the following article has received JBMM Best Paper Award.

"Characterization of the osteoblast-specific transmembrane protein IFITM5 and analysis of IFITM5-deficient mice"

by Nobutaka Hanagata, Xianglan Li, Hiromi Morita, Taro Takemura, Jie Li, Takashi Minowa J Bone Miner Metab (2011) 29:279–290



Dr. Nobutaka Hanagata

Abstract Interferon-inducible transmembrane protein 5 (IFITM5) is an osteoblast-specific membrane protein whose expression peaks around the early mineralization stage during the osteoblast maturation process. To investigate IFITM5 function, we first sought to identify which proteins interact with IFITM5. Liquid chromatography mass spectrometry revealed that FK506-binding protein 11 (FKBP11) co-immunoprecipitated with IFITM5. FKBP11 is the only protein it was found to interact with in osteoblasts, while IFITM5 interacts with several proteins in fibroblasts. FKBPs are involved in protein folding and immunosuppressant binding, but we could not be sure that IFITM5 participated in these activities when bound to FKBP11. Thus, we generated *Ifitm5*-deficient mice and analyzed their skeletal phenotypes. The skeletons, especially the long bones, of homozygous mutants (*Ifitm5* <sup>-/-</sup>) were smaller than those of heterozygous mutants ( $Ifitm5^{+/-}$ ), although we did not observe any significant differences in bone morphometric parameters. The effect of *Ifitm5* deficiency on bone formation was more significant in newborns than in young and adult mice, suggesting that *Ifitm5* deficiency might have a greater effect on prenatal bone development. Overall, the effect of *Ifitm5* deficiency on bone formation was less than we expected. We hypothesize that this may have resulted from a compensatory mechanism in *Ifitm5*-deficient mice.

We offer our sincere congratulations on behalf of the Journal of Bone and Mineral Metabolism, with best wishes for further development of the author's research.

Yoshiki Seino Editor-in-Chief

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