b-series gangliosides deficiency in mice resulted in the prevention of age-related bone loss

Kazunori Hamamura¹, Shoyoku Yo^{1,2}, Koichi Furukawa³, Ken Miyazawa², Shigemi Goto², Akifumi Togari¹

¹*Dept. Pharmacol., Sch. Dent., Aichi Gakuin Univ.*, ²*Dept. Orthodontics, Sch. Dent., Aichi Gakuin Univ.*, ³*Dept. Biomed. Sci., Chubu Univ.*

Purpose: b-series gangliosides are involved in the regulation of cell growth, neurite extension, and apoptosis. However, little is known about their roles in bone metabolism. In this study, we investigated effects of deletion of b-series gangliosides in bone metabolism.

Material & Methods: We examined expression levels of b-series gangliosides (GD3, GD2, GD1b, and GT1b) in MC3T3E1 osteoblast-like cells, RAW264.7 pre-osteoclast, and primary bone marrow cells using flow cytometry. To determine whether b-series gangliosides are involved in bone metabolism, we analyzed bone phenotype of GD3 synthase-knockout (GD3S KO) mice lacking all b-series gangliosides using mCT.

Results & Conclusion: b-series gangliosides were not detected in MC3T3E1 cells. On the other hand, they were detectable in both RAW264.7 cells and primary bone marrow cells. However their expression was reduced after induction of osteoclastogenesis. No differences in bone phenotype between GD3S KO and wild type mice at the age of 15 weeks were detected. However, bone volume (BV/TV) in GD3S KO mice at the age of 40 weeks was higher than that in wild type mice. Correctively, these results suggest that b-series gangliosides may prevent age-related bone loss.