KYUSHU UNIVERSITY

Public Relations Office, Kyushu University
744 Motooka Nishi-ku Fukuoka, 819-0395
TEL:092-802-2130 FAX:092-802-2139
MAIL:koho@jimu.kyushu-u.ac.jp
URL:http://www.kyushu-u.ac.jp

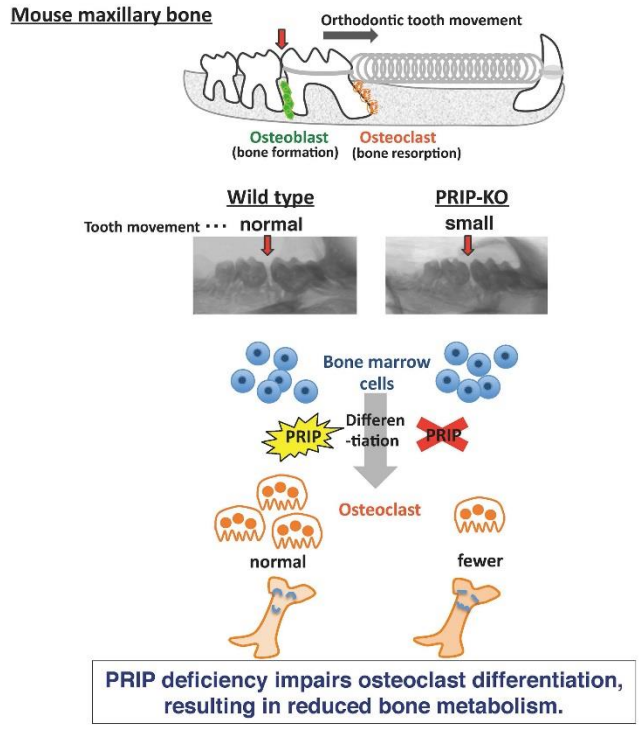
PRESS RELEASE(2017/04/27)

Positive regulation of osteoclastogenesis by PRIP

Bones are important not only because they provide structure and support for an organism's body, but also act as an endocrine organ that regulate a number of metabolic processes. The research group of Associate Professor Miho Matsuda recently showed that PRIP*1 gene knockout (KO) in mice*2 increases bone formation and concomitantly decreases bone resorption, resulting in increased bone mineral density and trabecular bone volume. However, the role of PRIP in osteoclastogenesis has not yet been fully elucidated. Here, the author aimed to identify the effects of PRIP on bone remodeling by investigating the tooth movement in mice fitted with orthodontic devices. The extent of tooth movement was smaller in the PRIP-KO mice than in wild-type mice. Fewer osteoclasts were observed on the bone resorption side in the maxillary bones of PRIP-KO mice. Lower osteoclast differentiation was observed in the bone marrow cells that were isolated from these mice. The expression of genes that are involved in osteoclast differentiation were exhibited to a lesser extent in immature PRIP-KO cells when compared to those of wild-type mice. The PRIP-KO cells also displayed fewer changes in intracellular Ca^{2+} and reduced nuclear localization of a transcription factor NFATc1 which is a master regulator of osteoclastogenesis. Up-regulation of intracellular Ca^{2+} restored osteoclastogenesis of the PRIP-KO cells. These results indicate that PRIP positively regulates osteoclast differentiation through calcium-calcineurin-NFATc1 signaling *via* regulating intracellular Ca^{2+}.

The above result has been published online on March 24th 2017 (US Time) in Journal of Biological Chemistry ('Phospholipase C-related, but catalytically inactive protein (PRIP) upregulates osteoclast differentiation *via* calcium-calcineurin-NFATc1 signaling).

Figure



PRIP*1 :
Phospholipase C-Related but catalytically Inactive Protein

PRIP gene knockout(KO)in mice *2:
the condition of genes in all tissues being knocked out; the PRIP gene-deficient mice

【Contact】 Miho Matsuda, Associate Professor
Molecular and Cellular Biochemistry/ Faculty of Dental Science
Tel：092-642-6321／Fax：092-642-6322 Mail：natural@dent.kyushu-u.ac.jp