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第 370 回松本歯科大学大学院セミナー

日　時: 2018 年 1 月 24 日(水)　18 時 15 分～19 時 00 分

場　所: 実習館 2 階研究所 セミナー室

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タイトル: The HIV co-receptor CCR5 regulates cellular pathways required for osteoclast function

C-C chemokine receptor 5 (CCR5) is a critical co-receptor for macrophage-tropic HIV, and Maraviroc, an inhibitor against CCR5, has significantly increased the lifespan of patients with HIV infection through blocking HIV transmission. Simultaneously age-related comorbidities including bone diseases have been needed to prevent in patients with HIV. Epidemiological and pathological findings in human studies reported that functional loss in CCR5 were correlated with the resistance to bone destruction diseases such as rheumatoid arthritis and osteoporosis. This possible association between loss of CCR5 and resistance to bone loss has been largely interpreted by the changes in the inflammatory and immunomodulatory responses caused by functional loss of CCR5, thus affecting the readout of bone metabolism. However, pathophysiological roles of CCR5 in bone metabolism have not been experimentally well documented. This study demonstrated that the blockade of CCR5 using its specific antibodies impaired in vitro human osteoclastogenesis with disorganized actin rings, but not osteoblastogenesis. Ccr5-deficient mice with dysfunctional osteoclasts were resistant to osteoporotic stimulation via the administration of receptor activator of nuclear factor

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kappa-B ligand (RANKL), which induces osteoporosis independently of the inflammatory and immunomodulatory responses. Furthermore, CCL5, a ligand for CCR5, enhanced the integrin- and chemokine-mediated pathways in osteoclasts. The present study experimentally provides further evidence that CCR5 plays an essential role in bone destructive diseases through the functional regulation of osteoclasts, thus suggesting a skeletal benefit of the CCR5-targeting therapy.

略　歴

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担当:硬組織疾患制御再建学講座　小林泰浩