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Inflammatory environments disrupt both bone formation and bone resorption

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Abstract

Inflammation has been associated with bone diseases such as osteoporosis and osteoarthritis. Bone loss were reported in the patients of several inflammatory diseases, such as rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease. However, how inflammation influence bone metabolism remains elusive. The bone loss in inflammatory environments are widely considered as the results of osteoclast overactivation which leads to excessive bone resorption. We previously discovered that osteoclasts induced from RAW macrophage treated with RANKL exhibited different cell properties and gene expression profile with undifferentiated macrophage. In this research we examined the excessive bone resorption hypothesis in *in vitro* systems. RANKL stimulated differentiation of RAW cells into bone-resorptive osteoclasts, and induction of pre-osteoblasts (MC-3T3 E1) into mature osteoblasts are utilized in this research. Inflammatory environments are mimic by treating cultured osteoclast or osteoblast with conditioned medium collected from bone marrow derived macrophage primed with LPS or interferon-γ. The pro-inflammatory cytokines inhibit the proliferation and disrupt the expression of genes that are needed for bone formation, such as osteocalcin and collagen. On the other hand, inflammatory environments did not activate osteoclast, nor promote bone resorption. Instead, pro-inflammatory cytokines inhibit osteoclastogenesis and bone resorption, induce mitochondrial dysfunctions and lead to apoptosis of osteoclast. These results indicated that the bone loss developed in the inflammatory environments might be due to the disruption of both bone formation and bone resorption.

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