

Abstract

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Abstract

Cytokines play important roles in bone remodeling by modulating the balance between bone formation and resorption. In mouse model, Interleukin-4(IL-4) has been shown to inhibit osteoclast formation. However, the effect of IL-4 on human osteoblast is still unknown. Our study showed osteoblast-like cells expressed both IL-4 receptor and STAT6, the main intermediate signaling molecules in the IL-4 pathway. The results prompt us to investigate the influence of IL-4 on osteoclastogenesis by determining the expression of RANKL/OPG in human osteoblast-like cells. Mandible-derived osteoblast-like cells were established. The expression of RANKL/OPG mRNA was investigated using real-time RT-PCR. The results indicated IL-4 suppressed the expression of RANKL/OPG dose-dependently. The mechanism of IL-4- modulated RANKL/OPG expression was studied by means of inhibitors including STAT6 inhibitor (AS1517499), NF- κ B inhibitor and PI3K inhibitor. The addition of STAT6 inhibitor as well as NF- κ B inhibitor, but not PI3K inhibitor could abolish the effect of IL-4. Interestingly, addition of CoCl₂, an activator of HIF-1 α , abolished the inductive effect of IL-4 on OPG expression indicating the influence of oxygen level. These findings suggested the role of IL-4 on osteoclastogenesis by suppressing RANKL/OPG ratio via STAT6 and NF- κ B pathways and might be influenced by the oxygen level in the tissue.

Keywords : Interleukin-4, Osteoblasts, Osteoclastogenesis, RANKL/OPG expression