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Opinion Article

Interactions between Immune Cells and Osteoclasts

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DESCRIPTION

Osteoclasts are specialized bone-resorbing cells that play an important role in bone remodelling, a continuous process where old bone is replaced by new bone tissue. The formation and activity of osteoclasts are tightly regulated to maintain bone health. An important aspect of this regulation involves the immune system. Immune cells and their signalling molecules significantly influence osteoclast differentiation and function. This article delves into the interactions between immune cells and osteoclasts and their implications for bone health.

Immune cells involved in *osteoclast* regulation

T cells: T cells, particularly T helper cells (Th1, Th2, and Th17), are key modulators of osteoclastogenesis. Th1 cells produce Interferon-Gamma (IFN-γ), which can inhibit osteoclast formation by reducing the expression of Receptor Activator of Nuclear Factor Kappa-B Ligand (RANKL) on osteoblasts and stromal cells. RANKL is an important factor for osteoclast differentiation. Th2 cells secrete cytokines like Interleukin-4 (IL-4) and Interleukin-10 (IL-10), which also inhibit osteoclastogenesis by downregulating RANKL expression and upregulating Osteoprotegerin (OPG), a decoy receptor that binds RANKL. Th17 cells produce Interleukin-17 (IL-17), which promotes osteoclast differentiation by increasing RANKL expression on osteoblasts and stromal cells, thus enhancing bone resorption.

B cells: B cells can both promote and inhibit osteoclast formation. They produce RANKL, which can stimulate osteoclastogenesis. However, they also secrete OPG, which inhibits RANKL-RANK interactions, thereby reducing osteoclast activity.

Macrophages: Macrophages are precursor cells that can differentiate into osteoclasts under the influence of RANKL and Macrophage Colony-Stimulating Factor (M-CSF). Additionally, macrophages secrete various cytokines that can either promote or inhibit osteoclast differentiation.

Dendritic cells: Dendritic cells can express RANKL and thus participate in osteoclastogenesis. They can present antigens and secrete cytokines that influence the local bone environment and osteoclast activity.

Regulatory T cells (Tregs): Tregs play an inhibitory role in osteoclast formation by producing anti-inflammatory cytokines such as IL-10 and Transforming Growth Factor-Beta (TGF-β), which suppress osteoclast differentiation and activity.

Cytokines and signalling pathways

Cytokines are signalling molecules that mediate the communication between immune cells and osteoclasts. The balance between pro-inflammatory and anti-inflammatory cytokines is critical for regulating osteoclast genesis. Pro-inflammatory cytokines like Tumor Necrosis Factor-Alpha (TNF-α), Interleukin-1 (IL-1), and IL-6 are potent stimulators of osteoclast differentiation and activity. They enhance RANKL expression and support the maturation of osteoclast precursors.

Anti-inflammatory cytokines such as IL-4, IL-10, and TGF-β inhibit osteoclastogenesis. They reduce RANKL expression and promote the production of OPG, thereby limiting osteoclast formation and bone resorption. The RANK/RANKL/OPG pathway is central to osteoclast regulation. RANKL binds to its receptor RANK on osteoclast precursors, promoting their differentiation into mature osteoclasts.OPG, produced by osteoblasts and immune cells, acts as a decoy receptor, binding to RANKL and preventing it from interacting with RANK, thus inhibiting osteoclast formation.

The interplay between immune cells and osteoclasts has significant implications for various bone diseases, such as, Osteoporosis an imbalance favouring pro-inflammatory cytokines can lead to increased osteoclast activity and bone loss, contributing to osteoporosis. Chronic inflammation and elevated levels of pro-inflammatory cytokines in rheumatoid arthritis enhance osteoclastogenesis, leading to bone erosion and joint damage. Immune responses to bacterial infections in periodontitis result in increased RANKL expression and osteoclast activity, causing bone loss around teeth.

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CONCLUSION

Immune cells play a pivotal role in modulating osteoclast formation and activity through complex signaling networks involving cytokines and the RANK/RANKL/OPG pathway. Understanding these interactions provides insights into the mechanisms underlying bone diseases and opens up potential therapeutic avenues for modulating immune responses to maintain bone health and treat bone-related disorders.