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Short Communication

Relation between Osteoporosis and Bone Ageing

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DESCRIPTION

Many cell types control the mineralization, remodeling, and deposition of bone matrix. One such cell type is the osteoclast, a large multinucleated cell that develops from bone marrow monocyte/macrophage progenitors and absorbs bone. By secreting Receptor Activator of Nuclear Factor Kappa-B (RANK) Ligand (RANKL), which binds to the RANK receptor on the osteoclast surface, osteoblasts, osteocytes, and activated T lymphocytes stimulate osteoclast development. Macrophage Colony-Stimulating Factor (M-CSF), which is released by osteoblasts and bone marrow stromal cells, binds to the colony-stimulating factor-1 receptor, also known as c-FMS, on the surface of osteoclasts, increasing the expression of the RANK receptor [1]. Mature osteoclasts break down the bone matrix by secreting acids and proteolytic enzymes, which allows them to absorb bone. Osteoprotegerin (OPG), a decoy receptor that binds RANKL and is released by osteoblasts, osteocytes, B lymphocytes, and the liver, can decrease osteoclast development and activity [2].

Osteoblasts, however, encourage the growth of new bone. WNT signaling causes osteoblasts to diverge from Bone Mesenchymal Stem Cells (BMSCs). When adipogenesis and the CCAAT-enhancer binding protein are inhibited, WNT signaling stabilises β-catenin, which controls the transcription of genes involved in osteoblast development such as Runt-related transcription factor 2 (Runx2) and Osteorix (Osx). Sclerostin and Dickkopf-1 (DKK1) can block WNT signaling in osteoblasts. Type I collagen, which makes up the majority of the matrix, is secreted by mature osteoblasts together with osteocalcin and alkaline phosphatase, which is followed by mineralization with calcium phosphate in the form of hydroxyapatite.

Osteocytes, which are terminally differentiated osteoblasts embedded in the mineralized matrix, play a crucial role in detecting mechanical load and are long-lived. Osteocytes can release a substantial amount of calcium and signaling molecules to maintain mineral homeostasis and control bone remodeling because they are estimated to make up 42 × 109 and occupy 215 m^2 of lacunar-canalicular surface area in an adult skeleton. Many signaling chemicals are secreted by osteocytes, which can either help or hinder bone remodeling. They are a significant source of sclerostin and DKK1, which block WNT signaling and prevent osteoblast bone growth, as well as RANKL, which promotes osteoclastogenesis. Many cell types, including those of the hematopoietic lineages of myeloid (an osteoclast precursor), lymphoid, and erythroid cells, as well as marrow stromal cells and BMSCs that can differentiate into osteoblasts, adipocytes, and chondrocytes, are found in the bone marrow. Bone is not a static structure; it undergoes ongoing remodeling in response to a variety of forces, including mechanical stress, hormonal pressures, and immunologic pressures. Bone is metabolically active in maintaining mineral homeostasis.

The skeleton's normal bone remodeling is a delicate balance between the deposition of new bone and the resorption of old, weak, or diseased bone. It happens in the following order: (i) activation, which happens when osteoclasts are drawn to broken or otherwise ineffective bone; (ii) resorption, which happens when mature osteoclasts remove bone, (iii) reversal, which happens when osteoclasts die and osteoblast progenitors are drawn in; and (iv) formation, which happens when mature osteoblasts deposit new bone [3]. Many substances that osteoclasts and osteoblasts release have been found to be crucial for bone remodeling. Moreover, mediators that are membrane-bound, such as members of the Ephrin family of proteins, are crucial in signaling cascades that are triggered by direct cell-to-cell contact between osteoclasts and osteoblasts. In bone remodeling, matrix-associated proteins link bone production and resorption. Inactive forms of the growth factors Insulin-like Growth Factor type I (IGF-1) and Transforming Growth Factor β1 (TGF-β1) are found in the bone matrix and become active upon osteoclast resorption to induce mesenchymal cell differentiation into mature osteoblasts. The orchestration of bone remodeling happens under the control of these cells and the interaction of the existing secreted substances. The term "bone modelling" refers to instances where bone production and resorption don't occur in the same order. When bone is deposited during growth and periosteal expansion, bone modelling takes place. Pathological skeletal conditions include inflammatory bone loss, and some pharmacological therapies can cause bone remodeling to disappear [4].

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microRNAs (miRNAs) have been shown to play an important role in bone remodeling, controlling post-transcriptional regulation of gene expression by binding to the 3-untranslated region of mRNA to prevent translation or promote destruction. miRNAs have also been demonstrated to affect the development of osteoclasts by reducing the expression of Connective Tissue Growth Factor (CTGF)/CCN family 2, and their miRNA-containing exosomes can affect how bone cells behave. Trabecular bone loss begins in both men and women in their third decade of life and accelerates significantly during perimenopause, and decreases in estrogen and testosterone levels have a direct impact on this process [5]. PTH and corticosteroids are two other hormones that have an impact on bone homeostasis. Age-related bone loss is a process where the balance of bone remodeling is skewed in favor of bone resorption over bone formation.

Age-related changes in Runx2 and Osx expression and an uptick in peroxisome proliferator-activated receptor expression lead to a shift from favoring BMSC adipogenesis to boosting adipogenesis. The number of osteoblasts is further decreased by age-related reports of declining WNT signaling.

CONCLUSION

With age, physical activity declines, causing bone loss and mechanical unloading. Mechanosensory cells in bones are osteoclasts. Long-term immobilized individuals had elevated plasma sclerostin levels. Sclerostin is a known secretory protein by osteoocytes that inhibits WNT signaling and reduces osteoblast quantity and activity. Age-dependent reductions in osteocyte numbers and increases in empty lacunae have been observed in studies of older women, which may explain why they are less sensitive to mechanical stimulation. The mutually exclusive mechanisms of cellular senescence and apoptosis in these cell types, which are prominently present in ageing bone, are significant contributors limiting the number of osteoblasts, osteocytes, and their progenitor cells. Increases in osteoclast development and resorption activity caused by stromal cell/osteoblast activity are significant bone loss potentiators. Age-dependent increases in the expression of RANKL and M-CSF, as well as a decrease in the expression of OPG, are seen in BMSCs from older people. Similarly, osteoblasts from older people display reduced OPG expression and increased IL-6, a pro-osteoclastic cytokine.

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