SHORT COMMUNICATION

BONE REMODELLING IN DENTAL IMPLANTS

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ABSTRACT
The skeleton is a metabolically active organ that is constantly remodeled throughout one's life. Bone remodeling entails the removal of mineralized bone by osteoclasts and the subsequent synthesis of bone matrix by osteoblasts, which thereafter become mineralized. Resorption, when osteoclasts eat old bone; reversal, when mononuclear cells emerge on the bone surface; and production, when osteoblasts lay down new bone until the resorbed bone is completely replaced, are the three steps of the remodeling cycle. Bone remodeling helps to modify bone architecture to meet changing mechanical needs and prevents the accumulation of old bone by repairing micro-damages in the matrix. It is also essential for maintaining plasma calcium homeostasis.

KEYWORDS
Bone remodeling, Osteoclasts, Osteoblasts, Resorption

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Introduction

Skeletal development-bone organization

Bone is a mineralized porous structure made composed of cells, vessels, and calcium compound crystals (hydroxyapatite). Their proportion varies depending on the type of bone and where it is found. Genes control the cellular differentiation processes that give rise to the skeleton, which first establish the pattern of skeletal structure in the form of cartilage and mesenchyme, then replace them with bone through osteoblast differentiation.[1]Extracellular matrix (which is mostly mineralized), collagen, and cells make up the structural components of bone. The normal, adult human skeleton has two forms of bone: cortical and trabecular.Although macroscopically and microscopically distinct, the chemical content of the two forms is identical. Cortical bone, which makes up 80% of the skeleton, is dense and compact, has a moderate turnover rate, and has a strong resistance to bending and torsion. It is found on the outside of all skeletal structures. The cortical bone is mostly calcified, and its primary job is to give mechanical strength and protection, but it can also play a role in metabolic reactions, especially when there is a severe or long-term mineral deficiency.

Trabecular bone makes up 20% of the skeletal mass, but it covers 80% of the surface area of the long bones, including the bodies of the vertebrae, the inner sections of the pelvis, and other big flat bones. Trabecular bone is less dense, more elastic, and has a faster turnover rate than cortical bone, indicating that it has a significant metabolic function. Mechanical support is provided by trabecular

Bone Matrix

Bone matrix is made up primarily of type I collagen fibers (two a1 chains and one a2 chain) and non-collagenous proteins, accounting for roughly 90% of the organic composition of the entire bone tissue. The fibers within lamellar bone create arches, allowing for the maximum density of collagen per unit volume of tissue. The lamellae might be concentric around a channel centered on a blood artery or run parallel to each other (trabecular bone and periosteum) (cortical bone Haversian system). Hydroxyapatite $3Ca_3(PO_4)_2(OH)_2$] crystals are found on, within, and in the matrix of collagen fibers, and are orientated in the same direction as the collagen fibres.The function of various non-collagenous proteins found in the bone matrix is still unknown. The largest non-collagenous protein generated is osteocalcin (Gla protein), which is involved in calcium binding, hydroxyapatite matrix stabilization, and bone formation regulation.[2]Gla protein appears to be a negative regulator of bone development, preventing excessive or premature mineralization.Biglycan, a proteoglycan, is expressed in the bone matrix and influences bone growth positively.[3]

Osteocytes

Osteocytes are osteoblasts that have become stuck in the osteoid. Even though an osteoblast's metabolic activity reduces once it is entirely encased in the bone matrix, these cells continue to create matrix proteins. Osteocytes have multiple lengthy cell processes that are rich in microfilaments and are structured during matrix production and before calcification. They produce a network of thin canaliculi that run throughout the bone matrix.The functional activity and appearance of osteoclasts differ depending on their age. The majority of the structural properties of an osteoblast are present in a juvenile osteocyte, but the cell volume and protein synthesis capacity are reduced. An older osteocyte, found deeper

within the calcified bone, has a smaller cell volume and more glycogen in its cytoplasm. During osteoclastic bone resorption, the osteocytes are finally phagocytosed and consumed.[4]

Despite the osteocytic network's sophisticated structure, the specific function of these cells is unknown. Osteocytes are thought to respond to bone tissue strain by attracting osteoclasts to areas where bone remodeling is necessary enhancing bone remodeling activity.[5] However, no clear evidence of osteocytes signaling to cells on the bone surface in response to bone tension or microdamage has been found thus far.

Osteoblast-Bone Formation

The osteoblast is in charge of producing the components of the bone matrix. Osteoblasts don't work alone; they're found in clusters along the bone's surface, lining the layer of bone matrix they're producing. They are made up of multipotent mesenchymal stem cells that can differentiate into osteoblasts, adipocytes, chondrocytes, myoblasts, and fibroblasts.[6]The lack of runt-related transcription factor 2 (Runx2) or a downstream factor, osterix, is necessary for osteoblast differentiation,[7] according to recent gene deletion experiments. 15% of mature osteoblasts get imprisoned in the new bone matrix and develop into osteocytes near the conclusion of the matrix-secreting cycle. Some cells, on the other hand, remain on the bone surface and develop into flat lining cells.The synthesis and maturation of osteoid matrix, followed by mineralization of the matrix, are the three steps of bone formation. These activities occur at the same rate in normal adult bone, ensuring that the balance between matrix synthesis and mineralization is maintained. Osteoblasts make osteoid by quickly depositing collagen at first. After that, the mineralization rate rises to match the rate of collagen synthesis. The rate of collagen synthesis slows down in the final stage, whereas mineralization continues until the osteoid is entirely mineralized. Insulin-like growth factors (IGF),[8]platelet-derived growth factor (PDGF), basic fibroblast growth factor (bFGF),[9] transforming growth factor-beta (TGF-),[10] and bone morphogenetic proteins (BMPs) are all produced by osteoblasts in response to various stimuli (BMP).[11]

These growth factors, whose receptors have been discovered on osteoblasts, influence osteoblast activity in an autocrine and paracrine way. Osteoblasts also have receptors for classical hormones like parathyroid hormone, parathyroid hormone-related protein, thyroid hormone, growth hormone, insulin, progesterone, and prolactin. Estrogens, androgens, vitamin D3, and retinoids are all receptors found in osteoblastic nuclear steroid hormone receptors.[12]

Osteoclast-Bone Resorption

The osteoclast, a huge multinucleated cell with a diameter of up to 100 mm, is a bone lining cell that originates from mononuclear hematopoietic cells [13] and is responsible for bone resorption. As a result of its inherent resorptive activity, it is generally seen in touch with a calcified bone surface and within a lacuna (Howship's lacunae).

Golgi complexes, mitochondria, and transport vesicles containing lysosomal enzymes are prevalent in osteoclasts. They have deep plasma membrane foldings in the area facing the bone matrix (called ruffled border) and the surrounding zone of attachment (called sealing zone). The osteoclast actively synthesized lysosomal enzymes such tartrate-resistant acid phosphatase and cathepsin K, which are released into the bone-resorbing compartment via the ruffled border.[14]

The binding of integrins expressed in osteoclasts to certain amino acid sequences inside proteins on the surface of the bone matrix is required for osteoclast attachment to the bone surface.[14, 15] Avb3 integrin binding stimulates cytoskeletal reorganization within the osteoclast after attachment to the bone matrix.[16]Podosomes, which are dynamic structures, are commonly used for attachment. They allow osteoclast mobility across the bone surface, which results in bone resorption, because of their constant assembly and disassembly.

A number of adhesion kinases, including the proto-oncogene src, are required for integrin signaling and subsequent podosome formation.[17]

Acidification and proteolysis of the bone matrix and the hydroxyapatite crystals enclosed within the sealing zone are used by osteoclasts to resorb bone. The mobilization of hydroxyapatite crystals by digestion of their collagen connection is the initial step in bone matrix resorption. The remaining collagen fibers are then degraded by cathepsins or active collagenases, with the residues being internalized or transported across the cell and discharged at the basolateral domain. Both locally acting cytokines and systemic hormones affect osteoclast function. Calcitonin, androgens,thyroid hormone, insulin, PTH, IGF-1,interleukin (IL)-1,CSF-1, and PDGF osteoclast receptors have been discovered.[18]

Bone Remodeling

Bone is a living organ that changes throughout one's life. The action of osteoblasts and osteoclasts causes remodeling, and their coupling helps to mend abnormalities like microfractures. Resorption and production are maintained in a homeostatic equilibrium, such that old bone is continuously replaced by new tissue, allowing it to adjust to mechanical load and strain. Frost coined the term "bone remodeling" to describe this occurrence in 1990.[19]

In what is known as a basic multicellular unit, osteoclasts and osteoblasts work together closely in the remodeling process (BMU). The BMUs in cortical and trabecular bone are organized differently, but the distinctions are mostly morphological rather than biological. The BMU burrows through cortical bone at a rate of 20–40 m/day, forming a cylindrical canal that is about 2,000 m long and 150–200 m wide.

During a cycle, ten osteoclasts excavate a circular tunnel in the prevailing loading direction, which is subsequently filled by hundreds of thousands of osteoblasts.Each year, between 2% and 5% of cortical bone is rebuilt in this manner.

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