American Journal of
Oral Medicine and Radiology

ISSN - 2394-7721

www.mcmed.us/journal/ajomr

Review Article

BONE REMODELLING – A REVIEW

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ABSTRACT
Comparable to other bone tissues in the body, alveolar bone is subjected to continual and rapid remodeling associated with tooth eruption and subsequently the functional demands of mastication. The ability of alveolar bone to undergo rapid remodeling is also important for positional adaptation of the teeth.

Key words:- Osteoblast, Osteoclastic activity, Prostoglandin.

Access this article online	
Home page: http://www.mcmed.us/journal/ajomr DOI: http://dx.doi.org/10.21276/ajomr.2020.7.2.9	**Quick Response code**

Received:25.07.20	**Revised**:12.08.20	**Accepted**:25.09.20

INTRODUCTION

Bone is constantly undergoing the process of remodeling. In normal adults, there is a balance between the amount of bone resorbed by osteoclasts and the amount of bone formed by osteoblasts. The current concept of bone remodeling is based on the hypothesis that osteoclastic precursors become activated and differentiate into osteoclasts, and this begins the process of bone resorption. On a bone surface targeted for resorption, a 10-day osteoclastic resorptive phase will be followed by a repair phase that lasts about 3 months.[1]

The termination of bone resorption and the initiation of bone formation in the resorption lacunae occur through a coupling mechanism. The coupling process ensures that the amount of bone removed is equivalent to the amount of bone laid down during the subsequent bone formation phase. The detailed nature of the activation and coupling mechanism is still unknown, although some growth factors have been proposed.

During resorption, the osteoclasts release local factors from the bone. These factors have two effects: inhibition of osteoblast activity and stimulation of osteoclast activity. Moreover, *osteo*clasts themselves produce and release factors that have a negative regulatory effect on themselves and enhance osteoblast function. Finally, when the osteoclasts complete the resorptive process, they secrete proteins that later serve as a substrate for osteoblast attachment. The active osteoblasts also produce local factors, enhancing autocrine regulation.

Ten Cate described the sequence of events in the resorptive process as follows:

1. Attachment of osteoclasts to the mineralized surface of bone.

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2. Creation of a sealed acidic environment through action of the proton pump, which demineralizes bone and exposes the organic matrix.
3. Degradation of exposed organic matrix to its constituent amino acids by the action of released enzymes, such as acid phosphatase and Cathepsin.
4. Sequestering of mineral ions and amino acids within the osteoclasts.

During aging, bone undergoes changes in its three-dimensional structure. This process starts at approximately 25-30 years of age, when maximal bone formation is achieved. From that age, a steady decline in bone mass begins for both men and women. The decrease in bone mass leads to thinning of cortical bone due to tunneling, or trabeculation, of the endosteal cortical envelope, with expansion of the marrow cavity accompanied by some gain in bone diameter.

Factors Regulating Bone Resorption

In recent years, it has become increasingly clear that many of the cellular events involved in bone resorption are modulated by a group of local factors (osteotropic cytokines) that have extremely potent effects on bone cells in both *in vitro* and *in vivo* systems. Bone cell activity, as well as regulation of the remodeling process, is also controlled by circulating systemic factors.

Interleukin-1 (IL- 1)

It is a powerful and potent bone-resorbing cytokine. IL-lα and IL-lβ are equally potent in stimulating bone resorption. The effects of IL-1 probably occur by two mechanisms. One mechanism is by stimulating the production and release of prostaglandin E_2, thereby stimulating bone resorption[3]. The second mechanism involves the direct action of IL-1 on the osteoclast independently of prostaglandin synthesis, through an 80 kDa receptor.

IL-6

In some experimental models, IL-6 appears to have no effects on bone resorption, but in others, it stimulates bone resorption. IL-6 is also responsible for the formation of cells with an osteoclastic phenotype.

Tumor necrosis factor *α* and lymphotoxin

These are multifunctional cytokines produced by activated lymphocytes. Their major effect on bone is to stimulate osteoclastic bone resorption. It is suggested that the effect of TNF α is mediated by prostaglandin E_2 as well as IL-6. TNF α also affects cells with osteoblast phenotypes inhibits differentiated function and stimulates cell proliferation.

Gamma interferon

It is more effective in inhibiting bone resorption induced by IL-1 or TNF α than systemic hormones like parathyroid hormone or 1,25-$(OH)_2D_3$.

Colony-stimulating factors

They can stimulate differentiation of osteoclast precursors into mature osteoclasts. They may mediate their effects on osteoclast formation indirectly. For example, by stimulating IL-1 production, this in turn stimulates PG synthesis.

Prostaglandins and other arachidonic acid metabolites

Prostaglandins of the E series are slow-acting, but powerful, mediators of bone resorption and affect both active mature osteoclasts as well as differentiated osteoclast precursors. Arachidonic acid can be metabolized by an alternative enzyme system, 5-Lipoxygenase, which also produces metabolites capable of stimulating bone resorption.[1]

Sex steroids

They exert an overall anabolic effect on bones by stimulating the proliferation and differentiation of osteoblasts. They also decrease the transcription of IL-6 gene. The combination of osteoclastic bone resorption and decreased osteoblast proliferation, caused by low estrogen levels, is a common cause of osteoporosis in postmenopausal women.

Factors Regulating Bone Formation

Agents that regulate bone formation act on the osteoblast to either increase or decrease the replication of cells in the osteoblastic lineage or to modify the differentiated function of the osteoblast. Bone formation is controlled by systemic hormones and local factors. For the most part, the local regulators of bone formation are growth factors that act directly on cells of the osteoblastic lineage, either in autocrine or paracrine manner.

Platelet-derived growth factor

It exists in three isoforms: AA, AB and BB. The most biologically active form in skeletal tissues is BB. It stimulates DNA synthesis and cell replication in osteoblasts and increases bone collagen synthesis and the rate of bone matrix apposition. In addition, it has also been reported to increase bone resorption and collagen degradation.[2]

Heparin-binding growth factors

Acidic fibroblast growth factor and basic fibroblast growth factor are the two better known forms of the heparin-binding growth factors. Both fibroblast growth factors have been shown to be mitogenic for bone cells and to enhance collagen and noncollagenous protein synthesis in bone culture.

Insulin-like growth factors

They are synthesized by most cell types present in skeletal tissue, including bone fibroblasts and osteoblasts. They probably act as either paracrine or autocrine regulators of bone formation. They increase preosteoblastic cell replication and have a stimulatory effect on osteoblastic collagen synthesis and bone matrix apposition and decrease the degradation of collagen.[3]

Transforming growth factor β

They stimulate pre-osteoblastic cell replication, osteoblastic collagen synthesis, bone matrix apposition and alkaline phosphatase activity [4]. However, it also appears to retard terminal differentiation of osteoblast.

Bone morphogenetic proteins

They directly affect osteoblasts by stimulating the differentiation of osteoblast precursor cells into more mature osteoblasts. They have also been shown to stimulate collagen production by mature osteoblast [5,6].

CONCLUSION

Based on the biochemical marker and bone density studies the alveolar bone has the higher quality of remodeling than other types of bones. Human genes has the role in the osteoblat and ostocytic activity and by this nature the bone remodeling occur. The alveolar and basal bones have different resorption rate.

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Cite this article:
Tamil Selvan D, Balasiddharth S, Sabarigirinathan C, Srinidhi L, Vinayagavel K, Kailash Krishnan. Bone Remodelling – A Review. *American Journal of Oral Medicine and Radiology*, 7(2), 2020, 49-51.
DOI: **http://dx.doi.org/10.21276/ajomr.2020.7.2.9**