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Perspective

Bone Resorption and Bone Remodeling Cycle Mechanism

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DESCRIPTION

Bone remodeling is a lifelong process that renews the bone matrix. Osteoclasts resorbing existing bone and osteoblasts creating new bone are generally thought to be the two basic processes involved. Any process of renewal, though, unavoidably calls for coordination between the event of destruction and repair. This cooperation in bone remodeling is frequently attributed to the grouping of osteoclasts and osteoblasts into small groups known as Basic Multicellular Units (BMUs). Resorption and formation are thought to be provided by BMUs as two interrelated processes, allowing for the preservation of bone mass and structure. Because of this, conventional bone morphometry assumes that formation and resorption are always "linked" and views the absence of formation is impossible. The imbalance between resorption and creation in the BMUs is thus blamed for the absence of bone restoration, or bone loss. Therefore, whether in drug design, bone morphometry, or study into pathophysiological causes, resorption and formation are the main research focuses.

Any process of regeneration carries the risk of improper coordination between destruction and reconstruction. Additionally, evidence of uncoupling and bone loss has been shown in a variety of contexts, including ageing, unloading, periodontitis, and glucocorticoid- and menopause-induced osteoporosis. However, due to a lack of understanding of the coupling process, these observations could not be fully comprehended. Molecular biologists have published intriguing lists of possible coupling factors that the osteoclast may release. The present bone remodeling models, which imply that bone forming osteoblasts arise many weeks after the osteoclasts have left the resorption site, have been difficult to reconcile with the role of these substances in osteoclast-osteoblast communication. It would be more logical if they took direct action against "reversal" efforts. Figuring out the sequence of events that take place within BMUs after the start of resorption and before the start of formation. Due to a lack of suitable instruments or models, it has been impossible to identify these cellular activities for a long time. The role of reversal in pathophysiology could not be understood without knowledge of the coupling process and was typically ignored. In the meantime, evaluations of remodeling became merely based on the better known resorption and formation events. The disadvantage is a fragmented view of remodeling that ignores other potential key players in bone loss.

Either limiting resorption or encouraging creation is the traditional approach for managing bone loss. The present information makes one think of the possible benefit of treatment strategies based on the combination of osteoclast and osteoblast modulators. Treatments should, in theory, work toward protecting local osteoprogenitor reservoirs, achieving the threshold cell density of osteoprogenitors for the start of bone formation as quickly as possible, and ensuring the presence of enough osteoclasts with pro-osteogenic activity, but with reduced resorption activity (since reduced resorption appears to increase the potential for osteoprogenitor recruitment). Knowing more about the interactions between osteoclasts and osteoblasts can help us employ PTH and PTHrP treatments more effectively. The well-known positive effects of intermittent PTH and PTHrP treatments may be related to such mixed effects on osteoclasts and osteoblasts. Other interesting agents fitting this technique would be CatK inhibitors, which alter the osteoclasts' ratio of resorptive to osteogenic activity.

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