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Perspective

Endocrine Regulation of Bone Remodeling: Emerging Hormonal Players

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ABOVE THE STUDY

Bone remodeling has long been understood as a hormonally regulated process, classically controlled by well-known endocrine factors such as Parathyroid Hormone (PTH), vitamin D, calcitonin, and sex steroids. However, recent advances in endocrinology and bone biology have revealed a far more complex network of hormonal regulation, involving signals from multiple systems including adipose tissue, the gut, kidneys, and even skeletal muscle. In my view, this expanding endocrine landscape is redefining how we understand bone remodeling and opening new possibilities for therapeutic intervention.

Traditionally, PTH and vitamin D have been central to maintaining calcium homeostasis and skeletal integrity. Intermittent PTH stimulates osteoblast activity and bone formation, whereas continuous exposure promotes bone resorption. Vitamin D enhances calcium absorption and supports mineralization. While these hormones remain critical, they represent only part of a broader endocrine network that integrates systemic metabolism with bone turnover.

One of the most intriguing developments is the recognition of bone as an endocrine organ itself. Osteoblasts and osteocytes produce hormones such as osteocalcin and Fibroblast Growth Factor 23 (FGF23), which exert systemic effects. Osteocalcin has been implicated in glucose metabolism and insulin sensitivity, suggesting a bidirectional relationship between bone and energy metabolism. FGF23, on the other hand, regulates phosphate balance and vitamin D metabolism through its actions on the kidneys. These findings highlight how bone participates in whole-body homeostasis, rather than functioning as an isolated structural tissue.

Adipose tissue has emerged as another endocrine contributor to bone remodeling. Adipokines such as leptin and adiponectin influence bone mass through both central and peripheral mechanisms. Leptin, for instance, can regulate bone formation via the central nervous system, while also having direct effects on osteoblasts. In obesity, altered adipokine profiles and chronic low-grade inflammation can disrupt normal bone remodeling, often leading to compromised bone quality despite increased body weight. In my opinion, the interplay between fat and bone represents a critical area for future research, particularly given the global rise in metabolic disorders.

The gut–bone axis is another rapidly evolving field. Hormones such as Glucagon-Like Peptide-1 (GLP-1), peptide YY (PYY), and ghrelin, traditionally associated with appetite regulation, have been shown to influence bone metabolism. GLP-1, for example, appears to have anabolic effects on bone, while PYY may inhibit bone formation. Additionally, gut-derived serotonin has been identified as a negative regulator of osteoblast activity. These findings suggest that nutrient intake and gastrointestinal signaling are closely linked to skeletal health.

Skeletal muscle also contributes to endocrine regulation through the secretion of myokines. Factors such as irisin and myostatin influence bone remodeling by modulating osteoblast and osteoclast activity. Irisin, released during physical activity, has been shown to promote osteogenesis, providing a molecular link between exercise and bone health. This crosstalk between muscle and bone underscores the of physical activity in maintaining skeletal integrity.

Emerging evidence also points to the role of stress-related and circadian hormones in bone remodeling. Cortisol, a glucocorticoid hormone, is well known for its catabolic effects on bone when chronically elevated. Meanwhile, circadian regulators such as melatonin influence bone formation and resorption, suggesting that disruptions in sleep patterns and biological rhythms may negatively impact skeletal health. In my view, these insights highlight the importance of considering lifestyle and environmental factors in endocrine regulation.

The integration of these diverse hormonal signals occurs through complex intracellular pathways involving receptors, transcription factors, and signaling cascades such as Wnt/β-catenin and RANKL/OPG. These pathways act as convergence points where multiple endocrine inputs are processed to regulate bone cell activity. Understanding how these signals interact is essential for developing targeted therapies.

From a clinical perspective, the identification of new hormonal players offers opportunities for innovative treatments. Drugs targeting incretin pathways, adipokines, or myokines may have dual benefits for metabolic and skeletal health. However, the

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systemic nature of these hormones presents challenges in achieving tissue-specific effects without unintended consequences.

In conclusion, the endocrine regulation of bone remodeling is far more intricate than previously appreciated, involving a network of hormones from multiple organs that collectively influence skeletal health. In my opinion, embracing this integrated perspective will be key to advancing both our understanding and treatment of bone diseases, particularly in the context of aging and metabolic disorders.