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Antibodies to Sclerostin or G-CSF Receptor Partially Eliminate Bone or Marrow Adipocyte Loss

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Description

The mineralized bone matrix is embedded with the most abundant bone cell, osteocytes. While they extend their dendritic processes through tiny channels known as canaliculi, their cell bodies are contained within pores known as osteocyte lacunae. They combine to form an extensive communication network that is referred to as the osteocyte lacuno-canalicular network. This network is comparable to the neural network. As a result, osteocytes are essential to maintaining bone homeostasis. They are particularly capable of converting mechanical signals into chemical signals, such as lacunar strain and/or shear stress of the surrounding fluid flow. By directing the activities of osteoblasts and osteoclasts, they conceal regulatory factors that control calcium and phosphate homeostasis and orchestrate bone remodeling. Additionally, therapeutics may influence OL characteristics, as was demonstrated for the treatment of early-onset osteoporosis with teriparatide. In addition, it was discovered that the frequency of highly mineralized, or micropetrotic, OLs increased with age, indicating a progressive loss of mechanosensitivity.

Lacuno-Canalicular Network

In healthy young adults, consuming glucose had an immediate anti-resorptive effect on the bones. Changes in biomarkers of bone metabolism and bone microarchitecture were linked to glucagon-like peptide 1, an incretin hormone involved in the production of post-prandial insulin. A potential mechanism for influencing bone accumulation in the years preceding peak bone mass is the gut-bone axis. Around the third decade of life, peak bone mass is reached, laying the groundwork for healthy bones throughout life. Endocrine mediators of nutrient metabolism are said to play a role in the effects of nutrition, which is one of the main modifiable factors in reaching peak bone mass. These signals instruct the pancreatic beta cells to increase insulin secretion and the pancreatic alpha cells to decrease glucagon production in a glucose-dependent manner. These hormones, which are derived from the gut and are referred to as incretins, also control bone turnover and have well-defined functions in glucose control. Osteoblasts and osteoclasts, the essential components of the cellular machinery involved in the metabolism of bone, undergo dynamic changes in activity during sleep and wake periods to control bone formation and resorption.

An anti-resorptive effect on bone is achieved through intravenous infusion and subcutaneous injection of incretin hormones. All of these findings point to a gut-mediated mechanism for how nutrition affects bone. The adult skeleton's bone biology during the years leading up to reaching peak bone mass is unique. Bone remodeling, a process that involves the coordinated action of the osteoblasts and osteoclasts to maintain mineral homeostasis, is predominant in the aged skeleton, whereas bone modeling, a process involving the independent action of the osteoblasts and osteoclasts to enhance bone size, mass, and strength, is predominant in adolescence. Clinical studies on incretin hormones and bone metabolism have primarily focused on adults up until this point. To confirm that the gut-bone axis is active during the crucial life stage of peak bone mass and peak bone strength attainment, studies on people going through the adolescent-to-adult transition are necessary. Bone has a wide range of mechanical, chemical, and biological properties due to the intricate arrangement of materials at various length scales.

Mineral Homeostasis

The fact that the stress–strain behavior that was obtained for the various models that were introduced was significantly different attests to the significance of the microstructure of the bone tissue for its failure behavior. The results highlighted the effect of cement lines on the crack deflection path and global fracture behavior of the bone microstructure when considering the role of interfaces. The behavior of bone fractures was also influenced by bone micromorphology and the area fraction of cortical bone tissue components like osteons, cement lines, and pores; particularly, increased porosity decreased the maximum stress required to initiate crack propagation, thereby altering the path that cracks take. In bone fracture, therefore, cement line structure, mineralization, and areal fraction are significant parameters. Using cryo-3D printing and dipping, a hierarchical scaffold with a two-stage drug release strategy is made. The scaffold has antibacterial and osteogenic properties thanks to

the combination of osteogenic peptide and chlorhexidine@graphene oxide. *In vitro*, the multifunctional scaffold has synergistic antibacterial and osteogenic effects. In a critical-sized, infected mandibular bone defect, the scaffold successfully regenerates the bone tissue *in vivo*. Sadly, bariatric surgeries are also linked to negative side effects like bone loss and an increased risk of fracture. Because these insights have the potential to uncover novel therapeutic targets, it is essential to investigate the mechanisms underlying the bone loss caused by bariatric surgery. Vertical sleeve gastrectomy, which removes a significant portion of the stomach along the greater curvature, is the most common type of bariatric surgery. Our previous research used a mouse model to demonstrate that VSG impairs osteoid mineralization and bone formation, which results in the loss of trabecular and cortical bone.

Breast cancer kills more women than any other cancer, affecting approximately one in twelve women. Osteolytic bone metastases and excessive bone resorption are associated with a number of complications, including severe pain, hypercalcemia, bone fractures, spinal cord compression, and a significant decrease in the patient's quality of life. It is becoming increasingly clear that bone metastasis requires tumor cells to express bone-compatible matrix proteins and adhesion molecules, as well as interactions between tumor and stromal cells in the bone microenvironment, despite the fact that the processes controlling breast cancer metastasis prefer bone. Several local growth factors that play a role in bone turnover and metabolism are secreted by breast cancer cells, which then release growth factors stored in the bone matrix to encourage tumor growth.