SLPI is a critical mediator that controls PTH-induced bone formation.

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Recommended ★★

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Bone is a highly dynamic tissue. It is remodeled throughout life by the coordinated and tightly regulated activity of osteoclasts and osteoblasts. Many secreted factors and cell-cell contact are involved in the regulation of bone remodeling. The parathyroid hormone (PTH) is one of these factors, and it is known to stimulate both bone formation and bone resorption. However, the final effect, anabolic *vs* catabolic, is strictly dependent on the duration and periodicity of exposure {1}. Pre-clinical {2,3} and clinical {4-6} studies indicate that the anabolic action of PTH requires the presence of osteoclasts. However, the mechanisms involved in the coordinated activity of these two cell types in PTH anabolism are largely unknown. Morimoto et al. identified a serine protease inhibitor (secretory leukocyte protease inhibitor, SLPI) that plays a role in the PTH-dependent shift to osteogenesis. They demonstrated that PTH strongly induced *Slpi* expression in osteoblasts, that genetic ablation of *Slpi* impaired the anabolic effect of PTH and, through intravital bone imaging, that SLPI promoted direct osteoclast-osteoblast contact. As already noted by Bikle {7}, the evidence that a protease inhibitor mediates PTH action introduces a novel concept in bone remodeling. In addition, even though further studies are needed, the work by Morimoto et al. provides new insights into the biology of osteoblasts and their communication with osteoclasts and may be of help for the development/refinement of PTH-based therapeutic protocols.

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Disclosures
None declared

Notes:
This evaluation has been transferred from Alessandro Corsi to Mara Riminucci.