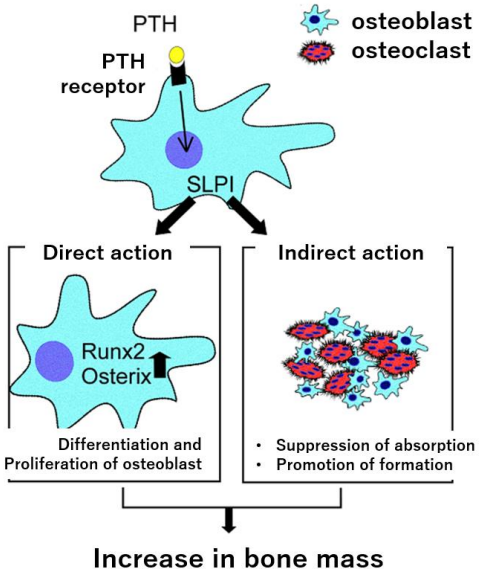
How the bone metabolism is regulated by PTH?

Osteoclastic bone resorption and osteoblastic bone formation/replenishment are closely coupled in bone metabolism. Anabolic parathyroid hormone (PTH), which is commonly used for treating osteoporosis, shifts the balance from osteoclastic to osteoblastic, although it is unclear how these cells are coordinately regulated by PTH. The research group of Masaru Ishii (Immunology and Cell Biology, IFReC/Graduate School of Medicine/Graduate School of Frontier Bio Science, Osaka University) identified a serine protease inhibitor, secretory leukocyte protease inhibitor (SLPI), as a critical mediator that is involved in the PTH-mediated shift to the osteoblastic phase. *Slpi* is highly upregulated in osteoblasts by PTH, while genetic ablation of *Slpi* severely impairs PTH-induced bone formation. *Slpi* induction in osteoblasts enhances its differentiation, and increases osteoblast–osteoclast contact, thereby suppressing osteoclastic function. Intravital bone imaging reveals that the PTH-mediated association between osteoblasts and osteoclasts is disrupted in the absence of SLPI. Collectively, these results demonstrate that SLPI regulates the communication between osteoblasts and osteoclasts to promote PTH-induced bone anabolism.



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- **Authors:** Akito Morimoto, Junichi Kikuta*, Keizo Nishikawa, Takao Sudo, Maki Uenaka, Masayuki Furuya, Tetsuo Hasegawa, Kunihiko Hashimoto, Hiroyuki Tsukazaki, Shigeto Seno, Akira Nakamura, Daisuke Okuzaki, Fuminori Sugihara, Akinori Ninomiya, Takeshi Yoshimura, Ryoko Takao-Kawabata, Hideo Matsuda, Masaru Ishii* (*corresponding authors)