Skeletons dynamically cycle between bone formation and bone resorption through coordinated efforts of osteocytes, osteoclasts, and osteoblasts. The protein sclerostin plays an important role in regulating these processes.

**Healthy Bone Structure**

Lower levels of sclerostin are associated with increased bone formation:
- Sclerostin is a negative regulator of bone formation and limits the accumulation of bone mass.
- With more exercise or increased levels of estrogen, osteocytes secrete less sclerostin, and bone formation increases.

**Compromised Bone Structure**

Higher levels of sclerostin are also associated with reduced bone formation:
- With less exercise and lower levels of estrogen, more secretion of sclerostin by osteocytes indirectly results in reduced bone formation.

**Osteoporotic Bone**

Higher levels of sclerostin are also associated with increased bone resorption:
- Higher levels of sclerostin decrease the expression of osteoprotegerin (OPG) and increase the expression of RANKL, indirectly resulting in an increased amount of bone resorbed by osteoclasts.
Sclerostin plays a key role in bone formation

- Natural genetic deficiencies of sclerostin (known as sclerosteosis) are associated with higher bone mass and stronger, more fracture-resistant bone\textsuperscript{13,14}
- Sclerostin also indirectly affects bone resorption\textsuperscript{12}

Amgen is committed to deepening our understanding about the role of sclerostin in bone formation.

Learn more about the role of sclerostin in bone formation at www.notjustafracture.com.
References


