The skeleton is constantly changing. Even in an adult, bone is always being renewed and broken down. The miracle of development that leaves humans with a skeleton strong enough to support their upright posture, but light and flexible enough to allow for a full range of movement, requires a delicate balance of growth and remodeling.

The strength of healthy bone and its mechanical function are the result of a beautiful and intricate structure. The skeleton, despite its apparent solidity, contains a latticework of open spaces where the cells that regulate its function do much of their work. Osteoblasts and osteoclasts are found there, and act in careful concert laying down new bone and removing the old.

If this balance is upset, the human body becomes fragile. In individuals with osteoporosis, the osteoclasts, which eat at the surface of the old bone, resorb it faster than new bone can be laid down. Trabecular, or spongy, bone with its vast surface area is most susceptible to this form of degeneration, and over a period of many years can become weakened and more susceptible to fracture. What causes this increase in the number of osteoclasts? A molecule called RANK-L.

Osteoclasts begin their development as pre-osteoclasts, cells that are harmless except in their potential. On their surface is a receptor known as RANK, a padlock which traps the pre-osteoclasts in their undisruptive state until the key, RANK-L, shows up to unlock the door to their development.

In normal bone, the amount of RANK-L is carefully controlled. Osteoblasts, the cells responsible for laying down new bone, produce both it and another protein called osteoprotegrin (or OPG) the two of which together regulate bone resorption. When their balance is proper, OPG binds to excess RANK-L to prevent the development of too many osteoclasts. The key can no longer fit into the lock. However in some diseases, and in post-menopausal women, there is not enough OPG, and the pre-osteoclasts are unlocked to develop in large numbers.

Through increased understanding of the mechanisms of bone erosion, XXXXX has found a way to intervene in the pathogenesis of osteoporosis and keep those padlocks closed. XXXXX is a human monoclonal antibody that works in an analogous way to OPG – binding to RANK-L and preventing it from inducing the formation of osteoclasts. In contrast to bisphosphonates, which accumulate in the bone matrix and can negatively affect bone formation, the effects of XXXXX are rapid, reversible and limited to reducing bone resorption. Used only once a year, XXXXX can put bone development back in balance.