BOTM: February 2009

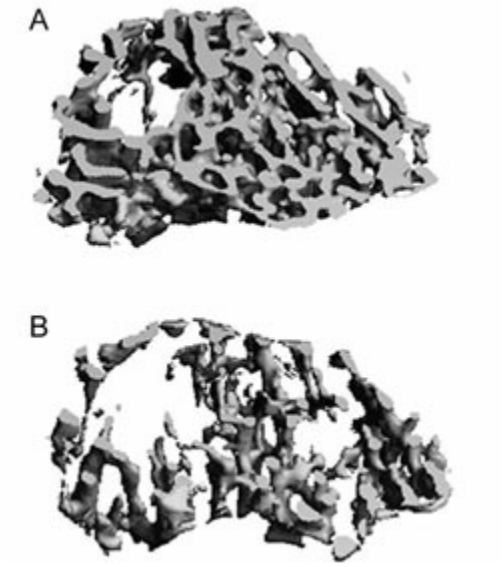
Osteoporotic Bone

Bone is an extremely dynamic tissue with an exceptional ability to self repair. Everyday activities, such as running, walking, and lifting, induce microcracks throughout the skeleton. Bone resorbing cells (osteoclasts) phagocytize the area of bone surrounding these stress-induced cracks. Bone forming cells (osteoblasts) then lay down new bone to replace the erosion cavity created by the osteoclasts. Tightly controlled coupling of osteoclasts and osteoblasts dictate the overall heath, and consequently the mechanical properties, of bone. Imbalances in the activity of osteoclasts and osteoblasts, due to decreased levels of estrogen in post-menopausal women, for example, often lead to osteoporosis.

Currently, greater than fifty percent of Americans over the age of fifty are suffering with osteoporosis.

Patients with this condition experience pathological decreases in bone mineral density (BMD), undesirable increases in trabecular porosity, and heightened risks of catastrophic bone fractures. Fortunately, several FDA approved treatments for osteoporosis are currently available. Bisphosphonates (anti-resorptive therapy), for example, such as Boniva®, Fosamax®, Actonel®, and Reclast®, bind to calcium in bone, become resorbed along with the bone, and cause an inhibition of osteoclast activity. A second treatment modality for osteoporosis is anabolic therapy. Anabolic therapies, such as the recombinant human parathyroid hormone (rhPTH) Forteo®, stimulate bone growth by inducing osteoblast activity.

Thus, by slowing the level of bone resorption with anti-resorptive treatment or by heightening the level of bone formation with anabolic treatment, bone remodeling in osteoporotic patients suffering with pathologically skewed osteoclast/osteoblast activity can be coaxed to a healthier balance of resorption and formation, ultimately restoring its mechanical properties and decreasing the likelihood of fracture. Since the mortality rate of Americans over the age of fifty is twenty-four percent following a major bone fracture, understanding the ability to “grow” new bone in osteoporotic patients through various modalities is a novel step towards discovering a cure for the debilitating disease of osteoporosis.



The trabecular architecture of normal bone (A) is responsive to loads placed upon it and adapts to efficiently distribute the loads. Osteoporotic bone (B) lacks the efficient trabecular network of normal bone. The increased porosity of osteoporotic bone decreases its mechanical properties and increases its risk of fracture. (Images obtained from µCT analysis at Clemson University)