Osteoporotic fractures have become a prominent health problem, causing a 10% increase in morbidity following a fracture and costing at least $14 billion annually in the United States. Currently, the gold standard for diagnosing osteoporosis uses dual X-ray absorptiometry (DEXA) to quantify bone mineral density (BMD), but changes in BMD explain only 4-17% of the fracture risk. Osteoporosis is treated clinically with anti-resorptive agents, which affect or inhibit the cellular pathways responsible for remodeling damaged bone. Microdamage accumulation can result from the progression of osteoporosis, long-term anti-resorptive use, and overloading, resulting in degraded mechanical properties. Therefore, understanding the balance between increased BMD and improved microarchitecture in the presence of increased microdamage accumulation is important for understanding fracture susceptibility.

To elucidate the role of both damage burden and architecture on the mechanical behavior of trabecular bone, the microarchitecture and volume fraction was quantified and the compressive and shear mechanical properties were determined in both young bovine trabecular bone and in an animal model of osteoporosis. The microarchitecture and volume fraction of trabecular bone played a larger role in the mechanical properties than damage level. Increasing slenderness ratio (trabecular spacing/trabecular thickness) and structure model index (SMI) resulted in decreased toughness, independent of damage level. In addition, small changes in volume fraction resulted in large changes in toughness. In contrast, large amounts of damage were necessary to produce the same effect in both young and diseased trabecular bone.

Both mechanical properties and microdamage accumulation are sensitive to microarchitecture and stress state in diseased trabecular bone. Crack densities increased when the bone is more rod-like. In addition, combined loading modes resulted in longer crack lengths than single overloads, indicating that propagation occurs due to varying stress states. Taken together, the energy-dissipating mechanism in bone relies on the initiation of new cracks, or increasing crack densities, as opposed to the propagation of existing cracks, unless the loading mode changes.

In conclusion, osteoporosis is a major clinical concern that affects the microarchitecture of trabecular bone. Since clinical measures of bone mineral density do not accurately predict fracture susceptibility, other factors play a role. These factors include microdamage accumulation and microarchitecture. Microarchitecture provides insight into the mechanical behavior in both compression and shear. However, the same microarchitectural parameters also predict damage accumulation. Indeed, anti-resorptive agents improve the architecture and volume fraction but also increase microdamage accumulation. In both bovine and ovariectomized ovine trabecular bone, small increases in volume fraction resulted in substantial increases in mechanical competence compared to increased damage accumulation. In the case of bisphosphonate use, the risk of increased microdamage accumulation may be counteracted by higher volume fraction, thereby providing would provide better fracture resistance than no treatment.