INTRODUCTION

Microdamage formation occurs in trabecular bone in vivo and is considered an important aspect of bone quality. Microcrack length is self-limiting under monotonic loading, but cracks propagated when the applied loading mode was changed. Clinically, such changes in loading mode can be interpreted as loading due to a fall or other abnormal load. In addition, microcracks are more common in bone with deteriorated architecture, suggesting that damage formation may be enhanced in diseased trabecular bone. If damage initiation and propagation are enhanced in diseased bone, it could further exacerbate the decrease in toughness with architectural deterioration.

Ovariectomized sheep have been used as a model of osteoporosis and provide a means to investigate osteopenic vs. normal trabecular bone while eliminating many confounding effects of lifestyle, genetics, and age. Ovariectomy results in compromised architecture and mechanical properties, which may enhance microdamage susceptibility.

The goal of this study was to determine the effects of architectural deterioration from osteopenia on microdamage initiation and propagation. Specifically, the aims were to: 1) induce damage in trabecular bone samples in compression; 2) propagate the damage by loading in shear; and 3) quantify the microdamage formation and propagation using fluorescence microscopy to determine their dependence on disease state and architectural measures.

METHODS

Three skeletally mature ewes, approximately seven years old, underwent bilateral ovariectomy under general anesthesia, were allowed to recover, and then turned out to pasture for 2 years (OVX-2). Three age-matched (CTL) ewes served as controls.

Six aligned cylindrical specimens from each group were prepared for mechanical testing. Micro-CT (Scanco µCT-80, Brüttschellen, Switzerland) images were obtained of each sample at 20 μm resolution for architectural assessment.

The samples were subjected to compressive followed by torsional overloading according to the protocol in Figure 1. Microdamage due to each loading phase was differentially labeled by fluorescent stains, which were applied prior to testing and after each overload.

RESULTS

On average, the length of cracks due to compression and shear were 38.6 ± 12.7 μm and 53.1 ± 11.1 μm, respectively (Fig. 2a, p = 0.008, paired t-test). Propagating cracks were longer than those due to either compression or shear alone (p < 0.065). The length of cracks formed in compression increased with increasing structure model index (SMI; Fig. 2b, p = 0.03), while Cr.Dn. and Cr.S.Dn. both decreased with trabecular thickness (p < 0.10). In contrast, damage formed in torsion was independent of architecture (p > 0.17).

Damage measures did not differ between groups (p > 0.14).

FIGURE 2: a) Cracks due to shear loading were longer than those due to compression. b) CrLn. in compression depended on SMI.

DISCUSSION

The goal of this study was to quantify differences in microdamage initiation and propagation in diseased vs. normal trabecular bone. Damage due to on-axis loading was enhanced in samples with higher SMI and lower trabecular thickness, both of which are associated with osteopenia. In addition, the length of microcracks initiated by shear overloading was longer than those initiated by compressive overloading at the same or lower strain level. Taken together, the results indicate that compromised architecture and off-axis loading both result in increased microdamage formation and may lead to further degradation in strength.

The overloading resulted in clinically relevant damage levels. The density of damage induced by shear overloading was similar to in vivo damage levels, and, as found in vivo, microcrack length was significantly and positively correlated with SMI. Although differences due to OVX status were not detected, this is likely due to the small number of samples that have been studied to this point.

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REFERENCES