Trabecular Microfracture Precedes Cortical Bone Failure During Cyclic Loading of Rat Vertebrae

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Introduction: Almost half of all vertebral fractures are not associated with a discrete loading event [4]. A vertebral fracture that is not caused immediately by a loading event must be the result of damage caused by multiple or prolonged loading events. Although microscopic tissue damage in the form of microscopic cracks, diffuse damage and trabecular microfractures has been observed in otherwise normal human vertebral cancellous bone, very little is known regarding the loading mechanisms that cause the damage and how such damage is repaired [1].

Existing in vivo models used to study the formation and repair of microscopic tissue damage in cancellous bone are limited in that they require compromising the periosteum and cortical shell, potentially confounding studies of the cancellous bone repair process [7,8]. The rat tail loading model developed by Chambers and colleagues [3] can be used to provide mechanical loads to a region of cancellous bone without compromising the bone being studied, but has so far only been applied to study bone adaptation. It is currently unclear if loads necessary to generate microscopic tissue damage in cancellous bone can be applied without causing an overt fracture (fracture in the cortical shell) in the rat tail. The long-term goal of this project is to understand the mechanical consequences of microscopic tissue damage and repair in cancellous bone. In the current study we determine the relationship between applied cyclic loads and microscopic tissue damage in an isolated rat caudal vertebra.

Methods: This study utilizes caudal vertebrae (7-9) from adult female Sprague Dawley rats (weights range: 375 g - 425 g). A total of 22 caudal vertebrae from 8 animals were examined. Animal use was approved by the IACUC. After being dissected free of soft tissues the distal and proximal ends of the bone were aligned within loading cups and potted with bone cement. Bone cement covered more than 0.8 mm of each end of the vertebra. Drops of saline solution were applied to the vertebral body while the bone cement cured to maintain hydration. Once the bone was secured, a thin strip of saline soaked gauze was wrapped around the bone to ensure hydration. The loading cups were then mounted in a material testing device (Bose Enduratech, ELF 3400) using custom fixtures.

Once mounted in the testing device, a sinusoidal cyclic load ranging from 0N to 260N was applied at 2Hz. Cyclic loading in this fashion provides a combination of creep and fatigue damage to a specimen that can be divided into primary, secondary and tertiary phases (Fig. 1) [6]. Loading was stopped prior to failure based on the rate of change of compliance. Two stopping criteria were used to capture specimens in the tertiary phase: a late tertiary phase criterion (1.6 micron/260N loading cycle) and an early-tertiary phase criterion (0.02 micron/260N loading cycle). If the criteria was not met within 5 hours of loading, the test was stopped and the specimen was classified as being in the early secondary phase (rate of change of compliance near zero) or in the late secondary phase (rate of change in compliance greater than zero) based on inspection of the compliance v. time curve. An additional three experiments were stopped after 30 min of loading to characterize the early secondary phase. These specimens were divided into primary, secondary and tertiary phases of loading. The authors thank David Burr and Matthew Allen for advice regarding microscopic tissue damage identification.

Results: Four specimens were stopped at the 1.6 micron/260 N load cycle criterion, six were stopped at the 1.0 micron/260N load cycle criterion and five run-out specimens were characterized as being in the late secondary phase. All loaded specimens displayed considerable diffuse damage in the epiphyseal regions. Microfractures were observed in nine out of 10 specimens loaded into the tertiary phase (Fig 1). Microfractures were present in specimens loaded into the secondary phase but were fewer in number (Fig 2). The number of trabecular microfractures was not related to bone volume fraction but was correlated with the percent change in compliance of the vertebra (p = 0.02, r²=0.27). Very few microscopic cracks or diffuse damage were observed in the metaphyseal regions or in the cortical shell (6 microcracks total in 3 specimens). No macroscopic or microscopic cracks were observed in the cortical bone of any of the specimens.

Discussion: The current study demonstrates that cyclic loading generates microscopic tissue damage in cancellous bone of the rat tail vertebra in the form of trabecular microfracture. That microscopic cracks were rarely observed suggests that, under these loading conditions, microscopic cracks rapidly propagate to cause trabecular microfracture. Our finding that trabecular microfracture is the primary form of microscopic tissue damage under this loading mode is surprising because previous analyses have suggested that trabecular microfracture is rare in human vertebral trabecular bone [9]. It is possible that trabecular microfracture is more common in the rat tail vertebrae because the bone volume fraction in the rat tail vertebra is larger than in human trabecular bone. However, trabecular microcallus formation (presumably a repair mechanism of trabecular microfracture) has been observed in human vertebral bone although its etiology and relevance to fracture risk remain poorly understood[5]. The current study suggests that the rat tail loading model is an attractive technique for studying repair processes in cancellous bone in vivo.


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Figure 1. (Left) Compressive displacement of the rat tail vertebra during cyclic loading (0-260N) is shown. Displacement components are divided into creep and cyclic damage portions. The primary, secondary and tertiary phases of loading are indicated. (Right) A trabecular microcallus associated with cyclic loading.

Figure 2. There was an increase in the number of microfractures observed in specimens cyclically loaded into the tertiary phase.