Introduction: Periosteal woven bone forms in response to stress fractures and pathological overloading. The mechanical factors that regulate the formation of this woven bone are not well understood. Recent in vivo fatigue loading studies used peak displacement to control the level of bone damage and reported that woven bone formation occurred in proportion to the level of displacement damage [1,2]. However, fatigue produces damage by application of cyclic loading it is unclear if the osteogenic response is due to bone damage (injury response) or dynamic strain (adaptive response). Creep loading, in contrast to fatigue, involves application of a static force. It is known that bone sustains progressive displacement under creep loading, and thus static creep is a method that can be used to produce bone damage without dynamic loading. Our objectives were to use the rat forelimb loading model to produce various damage levels using static creep and to determine the bone response to creep-induced damage. We hypothesized that: 1) increasing creep displacement produces ulnae with increased cracks and decreased mechanical properties at time-zero; 2) in vivo creep damage stimulates periosteal woven bone formation.

Methods: The right forelimbs of 92 anesthetized, adult (5 mo) male Fischer rats were loaded in axial compression at a constant force as the (creep) displacement was monitored. The applied force magnitude ranged between 17.3-19.8 N (although constant for each rat), with constant loading times less than 1 hour. Contralateral left forelimbs served as non-loaded controls. Time-Zero Creep Damage: First, creep to fracture tests indicated that forelimbs fractured reproducibly (independent of force magnitude) when the displacement increased by an average of 2.32 ± 0.46 mm (n=29) compared to the displacement at 5 sec. We then loaded forelimbs to sub-fracture displacements, creating eight groups (n=7-11/group) at 20-90% of fracture displacement. Rats were sacrificed on day 7. Ulnae were scanned with microCT to determine in vivo survival study approved by our Animal Studies Committee. Analyses were performed 2 mm proximal to the ulnar midpoint. Data were analyzed using analysis of variance (ANOVA). This study was in part, to the effects of bone damage.

Results: Time-Zero Creep Damage: Increasing creep displacements produced increasing levels of ulnar damage. After creep loading cracks were detected by microCT in some, but not all ulnae from each displacement group. Crack length increased from 0.08 ±0.17 to 0.84 ±0.68 mm in the same displacement group and 100% of ulnae from the high displacement group significantly increased in the high displacement group as compared to both placement groups (p<0.05). Three-point bending tests revealed degradation in ulnar mechanical properties. There was a significant loss of stiffness in displacement groups higher than 20%, but beyond 60% displacement there were no further reductions in stiffness (Figure 1). There were similar reductions in ultimate force although of lesser magnitude. In Vivo Response to Creep: Damaging creep loading stimulated a woven bone response. Woven bone was formed in 66% of ulnae from the low displacement group and 100% of ulnae from the high displacement group (Figure 2). Visible cracks were seen in 10% and 80% of the low and high displacement groups, respectively. BMC and B.Ar were significantly increased in the high displacement group as compared to both controls and low displacement groups, while there were no significant differences in the low displacement group from controls (Table). LS/BS was significantly increased between loaded and control limbs.

Discussion: In support of our first hypothesis we found increased crack length and crack extent, and decreased structural properties at higher creep displacements. There was a nonlinear relationship between displacement and damage, but generally increased displacement led to increased damage. Interestingly, even in bones with no visible cracks there were significant reductions in whole-bone properties, which may be attributed to diffuse damage (not visible at 16 μm resolution on microCT). In support of our second hypothesis, the in vivo survival study found periosteal woven bone forms as a result of static creep loading. Furthermore, visible bone cracks were not required for woven bone formation. In contrast to the findings of Lanyon and Rubin, who reported no increase in bone area using physiologically relevant static loads [3], we find that supraphysiologic, damaging static loads caused periosteal woven bone formation. This finding suggests that the woven bone bone reported after fatigue loading and in stress fractures is due, at least in part, to the effects of bone damage.


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