THE EFFECT OF R-RATIO ON THE FATIGUE CRACK PROPAGATION RESISTANCE OF HUMAN CORTICAL BONE

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ABSTRACT

Osteopenic and stress-related fractures are a serious health concern affecting patients of all ages and are believed to occur through the formation and accumulation of cracks due to cyclic loading. Understanding the kinetics of fatigue crack propagation (FCP) in human cortical bone is important in understanding fatigue-related bone fractures. The objective of this study was to determine the effect of loading conditions (R-ratio = P_{min}/P_{max}) on the FCP resistance of human bone. Four pairs of human femora (Female, 33, 71 years; Male, 31, 61 years) were used. Compact tension specimens were machined from the mid-diaphysis with the notch for crack growth parallel to the long axis of the bone. Four to 5 compact tension FCP tests were conducted for each group at R= 0.1 and R= 0.5. FCP specimens were tested wet at 37˚C and 2 Hz, with a sinusoidal waveform. Crack growth was followed with a traveling microscope. The fatigue crack growth rate, da/dN, vs. the cyclic stress intensity, ΔK, was determined for each group. For each group, $da/dN = C\Delta K^m$ was determined and statistical comparison between groups were conducted. In all groups, increasing the R-ratio led to a significant decrease in FCP resistance. There was a significant difference in FCP resistance between the Younger Female and the Younger Male bone groups at R = 0.1 but not at R = 0.5. The Older Female bone had significantly decreased FCP resistance compared with the Older Male at both R-ratios. Interestingly, the differences between age/gender groups were not as great at the higher R-ratio. It was observed that cracks grew through diffuse microdamage at the crack tip during cyclic loading. The FCP resistance of human cortical bone was significantly reduced by an increase in R-ratio, consistent with many engineering metals and plastics.

1 INTRODUCTION

Skeletal fragility has for many years been a major public health concern in the United States and throughout the world [1,2]. Osteoporosis is responsible for more than 1.5 million fractures annually, including 300,000 hip fractures, approximately 700,000 vertebral fractures and more than 500,000 fractures at other sites. One out of every two women and one in eight men over the age of 50 will have an osteoporosis-related fracture in his or her lifetime. The burden of health care costs due to osteoporotic fractures is estimated to be $10 to $15 billion per year [1]. Most of these fractures occur as a result of non-traumatic loads applied during regular daily activities. Bone stress fractures are believed to occur through the formation and accumulation of microcracks due to cyclic loading experienced during activities of daily living [3,4].

Understanding how human cortical bone resists crack initiation and growth (or propagation) under cyclic loading is integral to our understanding of skeletal fragility and stress fractures. Previous work has demonstrated that the fatigue crack propagation (FCP) resistance of bone in the longitudinal direction (along the axis of osteons) is reduced with radiation sterilization and possibly also with age [5.6]. These studies were conducted at a single cyclic load ratio ($R = P_{min}/P_{max} = 0.1$). Since bone is exposed to a variety of cyclic load ratios during every day activities, it is important to understand the effect of cyclic load ratio on FCP resistance of bone.

The objective of this study was to determine if the FCP resistance of human cortical bone is reduced by an increase in R-ratio. For this purpose, the FCP resistance at R = 0.1 and R = 0.5 of younger (< 40

years old) and older (>60 years old) male and female human donor femoral donor bone was determined.

2 MATERIALS AND METHODS

Four pairs of human femora with no known skeletal pathology were obtained from the Musculoskeletal Transplant Foundation. The femurs composed four age/gender groups: Younger Female (33yrs), Younger Male (31yrs), Older Female (71yrs), and Older Male (61yrs).

The femurs were cleaned by removing all soft tissue. The anatomic position on each femur was noted based on surface landmarks as anterior, posterior, medial and lateral. The femurs were then sectioned into 30 mm rings taken from the mid-diaiphysis. Each ring was numbered to identify their proximal to distal sequence. Compact tension specimens (width=14mm, thickness=3mm, initial crack length=3.75mm) were wet-machined using a computer numerically-controlled tabletop micromill (Micromill 2000, Denford Corp., Medina, OH) from the rings of bone such that the orientation of crack growth was directed longitudinal to the long axis of the diaphysis (in the primary direction of osteons) [5-7]. The notch was cut into each specimen with a low speed diamond saw and razor sharpened with a microtome 250 microns beyond the machined notch to serve as the initiating stress riser for fatigue crack propagation. To better aid with visualization of the crack, one side of each compact tension specimen was polished using a progression of alumina polishing compounds (finishing with a 0.05 micron solution) to result in a near mirror finish. After each specimen was completed, it was kept wet frozen at -20°C until testing

The specimens from each age/gender group were randomly assigned to one of two cyclic load ratios: R = 0.1 and R = 0.5. Four to 5 compact tension FCP tests were conducted for each treatment group at each R-ratio.

The FCP tests were carried out using a servohydraulic test system (Instron, Canton, MA) at 2Hz [8]. A heated water drip that was monitored with a thermocouple was used to keep the compact tension specimen moist at 37°C during testing. Crack growth was followed using a traveling microscope (Gaertner, Skokie, IL) mounted on an x-y stage. The crack length (a) was measured in 0.01mm increments in both the x and y. Due to the nature of the crack growth, the crack at times branched or there were multiple cracks present at the same time. In these cases, each crack was followed individually. Eventually, a single, dominant crack would that would progress to failure. To aid with visualization of the crack, a tissue marking dye was applied prior to each measurement. The number of cycles (N) associated with crack growth (a) was also recorded.

The cyclic stress intensity factor (ΔK, MPa$\sqrt{m}$) and the rate of crack growth (da/dN, m/cycle) were calculated for each specimen and the data was pooled for each age/gender/R-ratio group. The FCP behavior was evaluated by examining the pooled data plotted as log da/dN vs. log ΔK. The Paris relationship $da/dN = C\Delta K^m$ was determined for each pooled age/gender/R-ratio group. Linear regression was conducted for each pooled group to determine the Paris relationship**.** Relative FCP behavior between groups was considered with respect to differences in C and m. Differences in the coefficient C between groups were examined using the linear test method on the regressions for each group ($p \leq 0.05$ as significant) [9]. A significant increase in C from one group to another was taken as indicative of a decrease in FCP resistance.

Following testing, a portion of each specimen was used to determine wet density, dry density, water content, organic content, and ash content according to established methods [10]. After the specimens were defatted and rehydrated, the submerged and hydrated weights were measured. Dry weight was measured after 48 hours in an 80^{o}C, 20mm Hg oven. The ashed weight (used to determine ash content) was measured following 18 hours in a 600^{o}C furnace. Student's t-test was used for group comparisons ($p \leq 0.05$).

3 RESULTS

In all specimens, stable fatigue crack growth occurred and was observed to proceed through a zone of microdamage at the crack tip. As has been reported previously [5,6], the fatigue crack growth rate, da/dN, was documented to decelerate when there was crack branching or when a crack encountered obstacles such as a vascular channel or a pore. At both R-ratios, as higher crack growth rates were reached, multiple cracks would eventually combine before catastrophic failure. In addition, lamellar bridges were observed behind the crack tip. These bridges were qualitatively observed less frequently at the higher R-ratio of 0.5 than at R= 0.1 in all treatment groups. As the crack would grow, these bridges would eventually break.

In all groups, increasing the R-ratio from 0.1 to 0.5 led to a significant decrease in FCP resistance, as determined by a significant increase in C (Figure 1). The Older Female bone had the lowest FCP resistance compared to the other bone groups at both R-ratios (Figure 2). FCP resistance decreased with increase in age between the two female bone groups at both R-ratios. This difference, however, was not seen between the two male bone groups. There was a significant difference in FCP resistance between the Younger Female and the Younger Male bone groups at R = 0.1 but not at R = 0.5. In the older groups, the Older Female bone had significantly decreased FCP resistance compared with the Older Male at both R-ratios, although the difference was smaller at R = 0.5 (Figure 2).

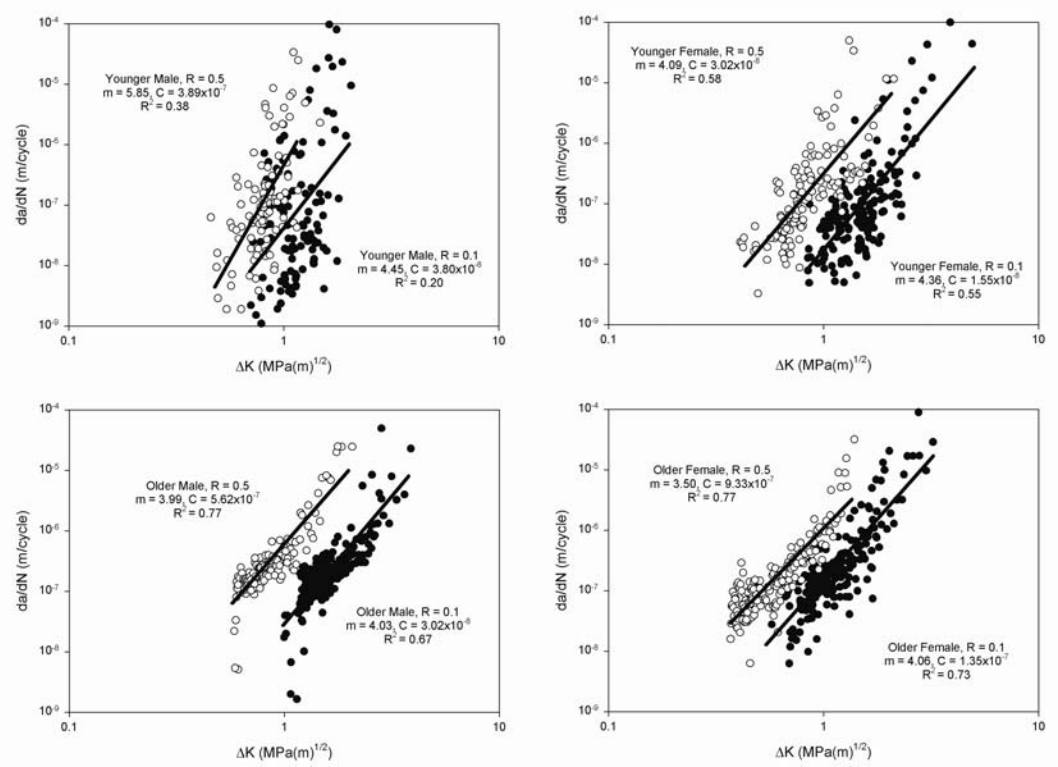

Figure 1. FCP resistance for Younger Male, Younger Female, Older Male, and Older Female bone.

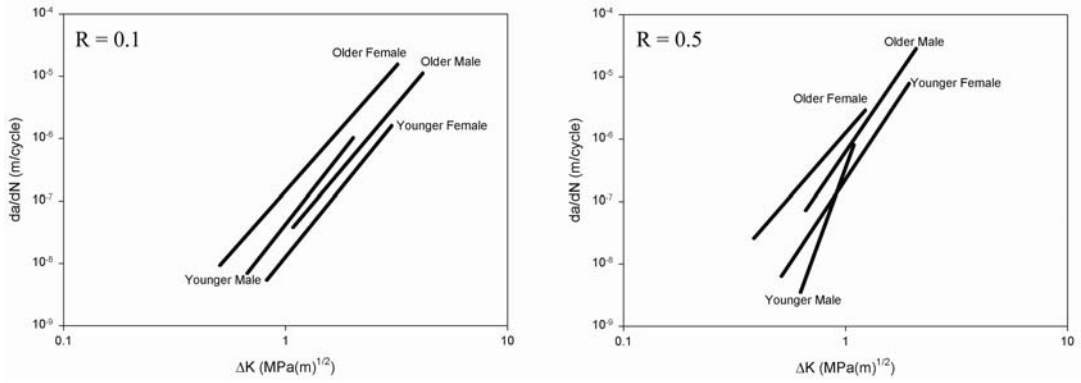

Figure 2. FCP resistance for all four age/gender groups at R = 0.1 and R = 0.5.

Wet density was not significantly different between age/gender groups (Table 1). Dry density was not significantly different except for Younger Female compared to Older Female bone. Water content was significantly different between Younger Female and Older Female bone and between Older Male and Older Female Bone. Organic content and ash content were both significantly different between the age/gender groups with the exception of Younger Female compared with Older Female bone.

	Wet Density ($g\text{-}cm^{-3}$)	**Dry Density** ($g\text{-}cm^{-3}$)	**Water Content** (fraction dry weight)	**Organic Content** (fraction dry weight)	**Ash Content** (fraction dry weight)
Younger Male	1.98 (0.04)	2.26 (0.05)	0.111 (0.006)	0.297 (0.010)	0.666 (0.012)
Younger Female	2.00 (0.02)	2.27 (0.02)	0.105 (0.008)	0.274 (0.007)	0.694 (0.008)
Older Male	2.01 (0.14)	2.30 (0.20)	0.109 (0.004)	0.286 (0.004)	0.680 (0.004)
Older Female	2.00 (0.02)	2.24 (0.02)	0.097 (0.003)	0.270 (0.006)	0.701 (0.006)

Table 1. Bone compositional data.

4 DISCUSSION

An increase in R-ratio from 0.1 to 0.5 resulted in a decrease in FCP resistance for the Younger and Older Male and Female donor femoral bone examined in this study. The decrease in FCP resistance is likely due to the overall increase in mean stress from R = 0.1 to R = 0.5. A loss in FCP resistance with increase in R-ratio is consistent with the FCP behavior of many engineering metals and polymers. The results from this study also support that there may be a loss in FCP resistance with age, particularly for female bone, as we suggested in a previous study [5].

A limitation of this study is that only one pair of femora was studied for each age/gender group; thus, the results regarding age and gender differences on fatigue crack propagation resistance can only be considered as suggestive at this time. Another limitation of this study is that it was an in vitro, not an in vivo study; the FCP behavior of bone tissue in vivo may be different than that in vitro.

Compositionally, there were significant differences in the organic content and ash content between age/gender groups, though we were not able to determine how or if these differences affected relative FCP resistance between the groups. Other ultrastructural and microstructural features, such as collagen quality, osteon density and porosity, also likely affect the relative FCP resistance of the different age/gender groups. For example, in a previous study [6], we found that gamma radiation sterilization of human bone tissue reduced FCP resistance; radiation sterilization is known to affect the

ultrastructure of the bone collagen matrix in the form of crosslinking and chain scission. Alterations in the collagen matrix also occur with age.

Interestingly, in this study, the differences between age/gender groups were not as great at the higher R-ratio. It may be that an increased mean stress may overwhelm compositional and microstructural differences from different age/gender femoral donor bone tissues on FCP resistance of human cortical bone.

The exponents for the Paris relationship varied little between the age/gender groups, ranging from m = 3.99 to 5.85. This finding may be reflective of similar fatigue crack growth mechanisms in the age/gender groups. The range of the exponent m in this study is somewhat higher than that which has been reported for FCP behavior of bovine bone (m = 2.8 to 5.1) and somewhat lower than that reported for equine bone (m = 10.4) [11,12]. In the equine bone study, the fatigue cracks were grown transversely, as opposed to longitudinally, which was the crack growth orientation in this study and in the bovine bone study. Crack growth orientation relative to the bone microstructure likely will affect FCP behavior. In addition, there are microstructural differences in bovine, human and equine bone which also likely influence FCP behavior.

The observation of the mechanism for fatigue crack growth was similar to previous studies in both human and bovine bone [5,6,11]. Visual and histological examinations of damage about the crack plane suggest that fatigue crack growth occurs through a zone of microdamage (microcracks and diffuse damage) ahead of the crack tip. The formation of microdamage at a crack tip in bone tissue results in energy dissipation and has been shown to be an important mechanism by which bone tissue inhibits crack growth under static loading conditions [13-15]. During fatigue, as damage accumulates during successive cycles, the material ahead of the crack tip weakens and the crack eventually grows through the zone of damage. In our previous studies, the zone of microdamage was found to increase in size with increase in ΔK during cyclic loading [5,6].

Human cortical bone is heterogeneous and anisotropic, with a microstructure of osteons, oriented longitudinally in long bones, surrounded by interstitial matrix. In addition, human cortical bone is porous and contains vascular channels. In this study, visual observation of the crack growth showed a deceleration of crack growth when a vascular channel or pore was encountered. In vivo, these types of "crack tip blunters" in the microstructure likely result in sufficient crack growth delay that bone remodeling and repair can occur under normal loading conditions. In circumstances such as stress fractures, the high amount of mechanical cyclic loading in a short time period overwhelms the biological repair process and crack growth can continue, sometimes reaching the stage of unstable fracture [3,4].

The observation of lamellar bridges behind the crack tip [5,6] suggests that human bone also has an extrinsic mechanism by it can resist fatigue crack growth [16]. In this study, lamellar bridges behind the crack tip were also observed. At R = 0.5, there were qualitatively fewer lamellar bridges observed, suggesting that this extrinsic fatigue crack growth resistance mechanism is less effective with an increase in mean stress.

5 CONCLUSIONS

The FCP resistance of human cortical bone is reduced with an increase in R-ratio from 0.1 to 0.5 and may also be reduced with age. Understanding the effect of cyclic load ratio on the FCP resistance of human cortical bone may ultimately help to evaluate the relative contribution of different phases of the gait cycle to fatigue crack formation in vivo. Cyclic loading during gait is variable and is composed of loading segments of different R-ratios due to the variety of muscles involved.

6 REFERENCES

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