AGE-RELATED CHANGES IN BENDING FATIGUE OF HUMAN CORTICAL BONE

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INTRODUCTION

Age-related non-traumatic fractures are a major health problem. Historically, only bone mass was considered to predict fracture risk but other factors, including accumulation of microdamage, may also affect bone quality and contribute to bone fragility. Previous studies conducted in our laboratory have demonstrated that the predisposition of bone to form linear microcracks and not diffuse damage is a significant contributor to bone fragility [1]. It is, therefore, likely that an age-related increase in bone fragility could be caused by an alteration in mode and magnitude of microdamage formation.

Fatigue tests conducted under four point bending provide a useful means to evaluate the propensity of bone to form different damage morphologies that consequently determine the fatigue life of bone. Linear microcracks form in the region of the beam that is subjected to compressive stresses [2,3]. Diffuse damage, in contrast, form in the areas subjected to tensile stresses as submicroscopic cracks [2,3].

The aim of this study was to identify age-related changes in bone fragility by conducting fatigue tests under four point bending loads. More significantly, since fatigue life of bone is dependent on the mode and magnitude of microdamage, an age-related decrease in the fatigue life would suggest that the age-related increase in fracture incidence is caused by the alteration in mode and magnitude of microdamage formation. It is noteworthy that despite a number of studies on bone fatigue, age-related changes in the fatigue life of bone have never been investigated.

METHODS

Sixteen rectangular parallelepiped beams (4mm × 4mm × 48mm), were wet machined from the anterior and posterior quadrants of 7 donor tibiae [Age/Sex: 46/F, 58/M, 62/F, 65/M, 76/F, 87/F, 89/M] with no history of bone disease. Four-point bending fatigue tests were conducted on a servohydraullic machine (MTS Bionix Model 858) using specially designed fixtures under constant irrigation of calcium buffered physiological saline.

All specimens were loaded in the same anatomical configuration to induce compression on the endocortical side and tension on the periosteal side. As initial bending modulus was expected to vary between individual specimens, the testing protocol included initial cycling (20 cycles) of each specimen to a low load (100N) in order to determine the modulus for each specimen. Specimen modulus (E), was then used to calculate the normalized load required to produce 5000μstrain at the midspan. Normalized load calculation required substituting specimen modulus (E), strain (ε=5000 μstrain), specimen geometry (b=4mm, h=4mm) and inner support length (l=20mm) in the following equation:

$$F = \frac{2Ebh^2\varepsilon}{3l}$$

Each specimen was fatigue loaded under load control at 2Hz upto its normalized load corresponding to 5000 μstrain at the midspan. During each test maximum cyclic load (corresponds to specimen specific normalized load) and displacement (d, variable) were continuously monitored and measured using data acquisition system and dedicated software (MTS TestStar and TestWare Sx) to calculate the change in specimen modulus as a function of loading cycle. The test was automatically stopped when specimen deflection reached a preset point corresponding to 40% modulus. Boyce et al. [2] have demonstrated that loading of human cortical bone specimens in four-point bending load configuration to 5000 μstrain initial strain and 60% modulus loss successfully captures all three characteristic phases of modulus loss and related fatigue damage without catastrophic failure. Following testing, specimens were removed from the fixture and immediately stained en bloc with 1% basic fuschin based in ascending series of ethanol in vacuum for a period of five days. This method of staining is known to stain and conserve all forms of microdamage

present in the specimen at the time of staining. Previous studies have conclusively demonstrated that basic fuschin can be successfully used to identify linear microcracks and diffuse damage [2, 3]. Linear microcracks appear as a sharply defined line and diffuse damage appears as an area of pooled staining.

RESULTS & DISCUSSION

The fatigue life of bone showed a highly correlated decrease with age. The decrease could be best described either with an exponential or power law relationship (Fig. 1) (Table Curve 2D, Jandel Scientific). It is interesting to note that the age-related decrease in the fatigue life of bone coincides with the age-related increase in microdamage, which, like this study, has been described by a power relationship (+ power) [4]. Thus, a cause and effect relationship between the accumulation of microdamage and decreased fatigue life can be expected.

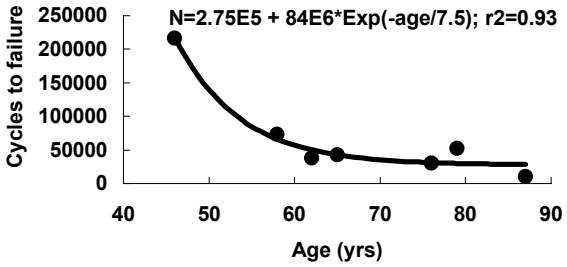

Figure 1: Age-related change in the fatigue life of human cortical bone subjected to bending fatigue. Each point in the figure represents an average of two specimens obtained from the anterior and posterior cortex of tibial diaphysis.

The analysis of modulus loss data provides further insight into the mechanisms of age-related decrease in the fatigue life of bone and clearly indicates that bone fragility under cyclic loading is determined by the processes and consequently the mode by which the stiffness is lost (Fig. 2). Specimens obtained from younger donors (46, 58, 62) lost stiffness gradually in the primary phase and had a longer phase II. This progressive and non-linear loss of stiffness is indicative of controlled damage initiation.

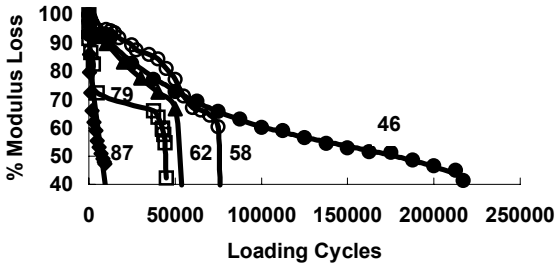

Figure 2: Percent modulus loss versus loading cycles for human cortical bone specimens from 5th to 9th decades.

Previous studies have shown that damage initiates at the ultrastructural level [5] in the form of a frontal process zone [6] or damage process zone [7] and can be readily identified near the site of crack initiation by diffuse areas of basic fuchsin staining [8] or lead-uranyl acetate staining [7]. Fatigue behavior of younger specimens may, therefore, be characterized by the formation of diffuse damage seen in the tensile cortices of fatigued bone [2,3].

In contrast to the above situation, specimens from older donors (79, 87) rapidly lost stiffness in the primary phase and displayed a reduced or absent secondary phase. The accelerated loss of stiffness in fatigue tests is a hallmark of brittle materials and is associated with a rapid growth of linear failure cracks. Fatigue behavior of specimens from older donors may, therefore, be characterized by the formation of linear cracks seen in the compressive cortices of fatigued bone [2, 3].

In summary, the results of this study demonstrate that the damage processes and consequently the mode by which the stiffness is lost determine the age-related loss in the fatigue life of human cortical bone.

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