MICRODAMAGE ACCUMULATIONS AT STRESS FRACTURE SITES IN HUMAN METATARSALS

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INTRODUCTION

Bone microdamage accumulation has been implicated in increasing skeletal fragility, and in the pathomechanics of osteoporotic and stress fractures (Burr et al., 1997). In vitro, bone fatigue life exponentially decreases with increasing strains, this behavior is also attributed to microdamage accumulation. However, the roles of bone microdamage and strain in the development of stress fractures is still unclear. It has been postulated that strain, damage, and remodeling interact in a positive feedback mechanism that can become unstable and result in a stress fracture (Martin, 1995).

In spinal trabecular bone, damage was not significantly related to age (Wenzel et al., 1996). Microcrack density in femoral neck trabecular bone was significantly (p = 0.032) higher in women over 70 years (Mori et al., 1997).

Both fatigue and insufficiency type stress fractures occur more frequently in second than fifth metatarsals. The goals of this work were to assess microdamage accumulations in regions where stress fractures are common (second metatarsal diaphysis) and rare (fifth metatarsal diaphysis), and investigate how damage accumulation is influenced by age and strain.

PROCEDURES

A dynamic gait simulator loaded nine cadaver feet with physiologic muscle and ground reaction forces over the entire stance phase of gait for conditions simulating normal walking (Sharkey and Hamel, 1998). Diaphyseal strains were measured on the second and fifth metatarsals using axial strain gauges. The contralateral second and fifth metatarsals were assessed histologically at the same location the strain gages were mounted on the experimental bones.

Metatarsals were stained en bloc in basic fuchsine to label microcracks and embedded in methacrylate. Three 120 micron thick cross-sections were taken from the strain gage location of each control metatarsal; cross-sectional areas were measured at 23x magnification using a light microscope video imaging system. Linear microcracks were counted and measured at 200x magnification. The crack density (Cr.Dn: #/mm²) and crack length density (Cr.Le.Dn: µm/mm²) were averaged for the three cross-sections. ANOVA’s were used to assess differences in Cr.Dn and Cr.Le.Dn between second and fifth metatarsals. Regressions were used to explore relationships between the damage parameters, age, and peak metatarsal strain.

RESULTS AND DISCUSSION

Cr.Dn and Cr.Le.Dn were higher in the fifth metatarsal than in the second, but the differences were not significant (p > 0.1), (Figure 1). Both Cr.Dn and Cr.Le.Dn significantly (p < 0.023) increased with age in the second metatarsal, but not in the fifth
(p > 0.2), (Figure 2). Neither Cr.Dn nor Cr.Le.Dn were significantly (p > 0.3) related to peak strain in either metatarsal.

Figure 1: Second vs. fifth metatarsal crack length density.

Cr.Dn and Cr.Le.Dn were expected to be higher in second than in fifth metatarsals since stress fractures are more common in the second. A homeostatic level of damage may have been maintained in these bones by the removal of microcracks by bone remodeling, explaining why there were no significant differences in the damage parameters between metatarsals.

Over a wide range of ages, 41 to 85 years, strain alone was not a good predictor of in vivo metatarsal microdamage accumulation. Although higher bone strains are likely to result in higher microdamage accumulations, it is also likely that remodeling is activated at higher rates to repair the damaged bone (Burr and Mori, 1993). An increased number of resorption spaces will reduce the stiffness of a bone causing strains to be higher under habitual loading conditions. If this feedback mechanism becomes unstable (i.e. damage exceeds repair) a stress fracture may occur.

SUMMARY

Cr.Dn and Cr.Le.Dn were unable to explain the greater incidence of second metatarsal stress fractures, supporting the theory that resorption spaces caused by remodeling play a critical role in stress fracture etiology. Second metatarsal Cr.Dn and Cr.Le.Dn significantly increased with age which may explain why insufficiency stress fractures are more common in the second metatarsal. Contrary to laboratory studies, bone strain may not be a good predictor of in vivo fatigue damage accumulation or fatigue life because of the repair capabilities of bone remodeling.

REFERENCES


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