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Linear Microcracks and Diffuse Damage Accumulate Differently with Age

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INTRODUCTION:

Microdamage accumulation processes are believed to act as a stimulus to bone remodeling and serve as one of the major factors contributing to increased skeletal fragility and stress fractures. Bone microdamage has been classified into two general categories: discrete linear microcracks (cracks on the order of 30-100 μm in length[1-3]) and diffuse damage. Diffuse damage is believed to consist of damage at the sublamellar or ultrastructural level. Recent studies suggest that young bone develops fatigue damage differently than old bone[4,5]. Diab et al.[5] found that during fatigue loading, younger bone formed diffuse damage whereas older bone formed linear microcracks. They concluded that the propensity of aging human bone to form more linear microcracks than diffuse damage may be a significant contributor to bone quality and age related fragility. Although relationships between linear microcracks and age are well established, the relationships between diffuse damage and age and diffuse damage and linear microcracks has not been established. The objective of this study was to develop such relationships to test the hypothesis that linear microcracks and diffuse damage accumulate damage at different rates.

METHODS:

Bulk sections were cut from the proximal femurs of 31 males (22 to 91 yrs.) and 28 females (24 to 94 yrs.). The sections were stained with basic fuchsin from which 80μm thick transverse slices were removed and mounted for examination using a brightfield and fluorescence microscope at a magnification of 125x. Five fields were randomly chosen from each quadrant for damage measurements yielding a total of 20 fields per bone. Microcracks were identified as linear type morphology, typically on the order of 30-100um in length.[2,3] Crack density parameter (Cr.Dn.) was defined as the ratio of the total number of discrete linear cracks (#cracks) and the bone area (B.Ar.)(Cr.Dn.= #cracks/B.Ar., #cracks/mm^2). Diffuse damage areas were identified as focal areas of diffuse staining. Diffuse damage area density parameter (Df.Dm.Ar.) was defined as the ratio of the total damaged area (Dm.Ar.) and bone area (B.Ar.) (Df.Dm.Ar.=Dm.Ar./B.Ar., mm^2/mm^2). Correlations were made between crack density, diffuse damage area and age. Simple regression analysis using the statistical package JMP^{TM} (SAS Institute, Cary, NC) was used in the statistical analysis. Significance was set at $p<0.05$.

RESULTS:

Cr.Dn. (avg. = 5.84 cracks/mm,^{2} SD= ±1.51 cracks/mm^2) significantly increased with age ($p<0.0001$) (Figure 1) whereas Df.Dm.Ar. (avg.= 2.78%, SD= ±1.52%) did not increase with age (Figure 2). Cr.Dn. was also linearly ($p<0.0011$) related to Df.Dm.Ar (Figure 3).

DISCUSSION:

The results of the current study are consistent with previous work which shows that young and old bone develops damage differently; linear microcracks accumulate with age, whereas diffuse damage is sustained at a constant level throughout life. Therefore, older bone would have a relatively higher incidence of linear microcracks compared to diffuse damage than younger bone .Our results also revealed a significant relationship between crack density and diffuse damage density that has not previously defined. The relationship between Cr.Dn. and diffuse damage density supports the idea that microdamage begins at the ultrastructural level, consistent with previous reports.[6,7] It is reasonable that microcracks can result from coalescencing of the smaller, submicroscopic cracks that occur in a damage process zone, a region with increased localized crack density[8]. Such localized regions occur in areas of high stress concentration, e.g. notches, voids and other microstructural features, where cracks tend to initiate[6]. Although bone is anisotropic and nonhomogeneous, cracks resident in planes of weakness (i.e. in the longitudinal directions along osteons) may be able to coalescence as they propagate along the weak planes. However, this does not preclude that discrete linear cracks also initiate at the microscopic level as well. In summary, we found that linear microcracks and diffuse damage accumulate differently with age and that diffuse damage area was significantly related to incidence of linear microcracks.

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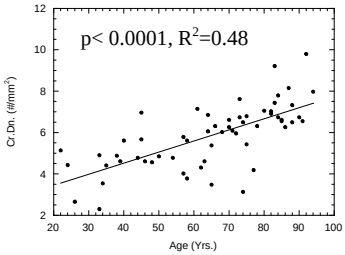

Figure 1. Cr.Dn. vs. age

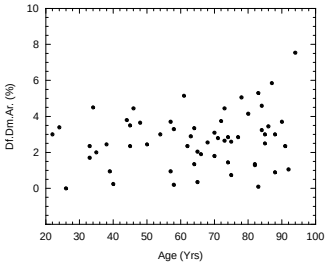

Figure 2. Diffuse damage vs. age.

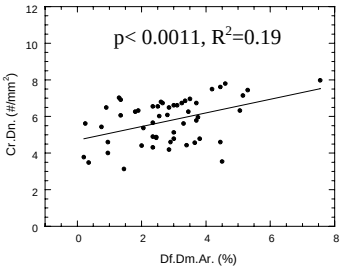

Figure 3. Crack density vs. diffuse damage density.