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The Incidence and Role of Diffuse Damage in Human Cortical Bone

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THE INCIDENCE AND ROLE OF DIFFUSE DAMAGE IN HUMAN CORTICAL BONE

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Introduction

Microdamage accumulation processes have been proposed to act as a stimulus to bone remodeling¹ and serve as one of the major factors contributing to increased skeletal fragility.² Recent in-vitro studies have demonstrated that bone exhibits stiffness losses prior to the appearance of microscopic cracks.³ Diffuse areas of staining (or diffuse damage) at the submicroscopic level has supported the idea that damage initiates at the ultrastructural level.³⁻⁵ Even though diffuse damage has been implicated in the initiation of microcracks and ultimately bone remodeling, the incidence of in-vivo diffuse damage and the mechanics of its origin and propagation are not known. The objective of this investigation was to determine the incidence of in-vivo diffuse and microscopic damage from the same cadaver bone specimens with ages ranging over eight decades. Additional objectives were to determine if diffuse and microstructural damage change with age and if a relationship exists between them. If the role of diffuse damage were to be a precursor for microcrack formation, then we would expect a relationship to exist between the two levels of damage. This question is examined and the role of diffuse damage with regard to the mechanical behavior of bone is discussed.

Materials and Methods

Thirty-three fresh human femurs were harvested from 16 male and 17 female cadavers. The male donors' ages ranged from 22 to 91 years (average age = 55.8 ± 23.1 yrs.) while female donors' ages ranged from 24 to 92 years (average age = 56.8 ± 19.6 yrs.). The right femurs were cleaned of soft tissue, wrapped in gauze moistened with physiological saline and stored in a freezer at -15°C until they could be prepared for damage evaluation. Once thawed, parallel cross-sectional cuts were made 1 cm apart at the proximal femur (1 cm down from the base of the lesser trochanter). The bulk sections were basic fuchsin (BF) stained according to published procedures.⁶ The stained bulk sections were cut into four quadrants with a metallurgical saw with a diamond blade. The sections were then embedded in plastic, an $80\ \mu\text{m}$ thick transverse slice was removed from the center of each quadrant with a wire saw and then mounted for examination using a fluorescence microscope. Five fields were randomly chosen from each slide for damage measurements. Diffuse damage areas were identified as the areas containing a network of small bright cracks surrounded with pooled blurry stained regions (Fig. 1). Those areas were subsequently circumscribed and the surface areas of circumscribed regions were measured. 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.) ($\text{Df.Dm.Ar.} = \text{Dm.Ar.}/\text{B.Ar.}$, mm^2/mm^2) and was computed for each randomly chosen field and averaged per specimen. Microcracks were identified as having sharp borders with a halo of increased BF stain surrounding them.⁶ The cracks were classified as either matrix cracks or osteonal cracks (cracks within or around the osteon). Linear regression statistical analysis was performed using JMP (SAS Institute, Cary, NC). The data for males and females were pooled.

Results

There was no relationship found between Df.Dm.Ar and age (Fig. 2). The average Df.Dm.Ar. from the pooled data was 2.08% (SD = $\pm 1.31\%$). Microdamage density significantly increased ($p < 0.0001$) with age (Fig. 3). The morphology of the microdamage revealed that 73% of the cracks were matrix cracks and 27% of the cracks were osteonal type cracks. There was no relationship between microdamage density and Df.Dm.Ar. (Fig 4).

Discussion

This study showed that in-vivo diffuse damage occurrence is small, averaging approximately 2.08% of total bone area over the age range of 22-92 years. A previous study showed diffuse damage density equal to approximately 3% in bone specimens from cadavers over the age of 50 years.⁷ Results of this study also showed that microcrack density increases with age, consistent with other studies.^{8,9} In addition, there was no relationship between diffuse damage area and microscopic damage. This finding has certain implications with regard to the mechanics of damage initiation and propagation in bone and suggests that

the two levels of damage are independent events or quantities. However, the lack of a significant relationship does not preclude diffuse damage from propagating or coalescing to form microcracks. Rather, these data suggest that diffuse damage does not necessarily progress to larger cracks, that may become clinically relevant at some point. Taking these results into consideration with a previous report that showed in vivo diffuse damage does not alter bone toughness⁷, the role of in-vivo diffuse damage in the mechanical behavior of cortical bone is unclear. It remains to be seen if its role is primarily biological, acting as a stimulus for bone remodeling.

It is concluded that in-vivo diffuse damage in human cortical bone is small and appears to remain constant at a low level over a lifetime. Microcrack density increases with age. There is also no relationship between submicroscopic and microstructural levels of damage.

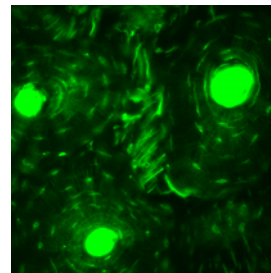


Fig. 1. Diffuse damage shown in the center of the image.

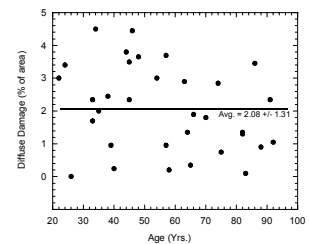


Fig. 2. Diffuse damage does not change with age.

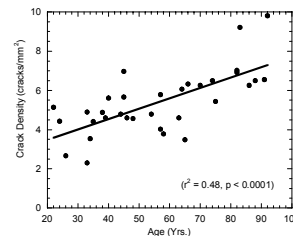


Fig. 3. Microdamage increases with age.

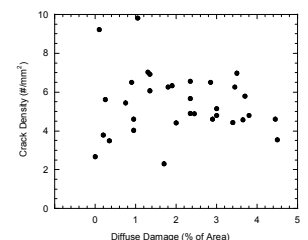


Fig. 4. There is no relationship between damage types.

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Acknowledgement

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