Age-related changes in human trabecular bone: Relationship between microstructural stress and strain and damage morphology

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1. Introduction

Peak bone mass is reached at approximately age 30, after which a slow decline in bone mass begins throughout the skeleton at rates that vary among individuals and anatomical sites. For trabecular bone in particular, age-related changes include alterations in extracellular matrix heterogeneity and composition (e.g. collagen and noncollagenous proteins, mineral, and water content), quantity (bone volume fraction), remodeling, and microarchitecture (e.g. trabecular thickness, degree of anisotropy, and structural model index) (Hildebrand et al., 1999; Paschalis et al., 2004; Recker et al., 2004; Busse et al., 2009). Of these factors, reductions in bone quantity with corresponding alterations in microarchitecture have been the most extensively studied. Decreased bone volume fraction with age is typically associated with microstructural deterioration in the form of decreased trabecular thickness and connectivity with increased trabecular spacing and anisotropy (Parfitt et al., 1983; Kimmel et al., 1990).

Alterations in bone mass and microstructure are not the only phenomena that manifest with age. Decreased bone quality is also reflected in an accumulation of unrepaird microdamage. The relationship of microdamage with skeletal fragility has been previously investigated with reports of non-linear increases in crack density with age (Schaffler et al., 1995; Burr et al., 1997; Mori et al., 1997; Fazzalari et al., 1998; Fazzalari et al., 2002). It has been speculated that the accumulation of damage with age may be the result of a breakdown in targeted remodeling of microdamage regions (Burr and Allen, 2008). However, microdamage does not exclusively occur in aging or disease conditions. Damage and repair of trabeculae are part of the normal turnover...