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Variability of tissue mineral density can determine physiological creep of human vertebral cancellous bone

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ABSTRACT

Creep is a time-dependent viscoelastic deformation observed under a constant prolonged load. It has been indicated that progressive vertebral deformation due to creep may increase the risk of vertebral fracture in the long-term. The objective of this study was to examine the relationships of creep with trabecular architecture and tissue mineral density (TMD) parameters in human vertebral cancellous bone at a physiological static strain level. Architecture and TMD parameters of cancellous bone were analyzed using microcomputerized tomography (micro-CT) in specimens cored out of human vertebrae. Then, creep and residual strains of the specimens were measured after a two-hour physiological compressive constant static loading and unloading cycle. Creep developed (3877 ± 2158 με) resulting in substantial levels of non-recoverable post-creep residual strain (1797 ± 1391 με). A strong positive linear correlation was found between creep and residual strain ($r=0.94$, $p<0.001$). The current results showed that smaller thickness, larger surface area, greater connectivity of trabeculae, less mean tissue mineral density (TMD, represented by gray levels) and higher variability of TMD are associated with increasing logarithmic creep rate. The TMD variability (GL_{COV}) was the strongest correlate of creep rate ($r=0.79$, $p<0.001$). This result suggests that TMD variability may be a useful parameter for estimating the long-term deformation of a whole vertebral body. The results further suggest that the changes in TMD variability resulting from bone remodeling are of importance and may provide an insight into the understanding of the mechanisms underlying progressive failure of vertebral bodies and development of a clinical fracture.

1. Introduction

Many clinical studies have indicated that progressive vertebral deformation of elderly patients results in long-term vertebral height loss and back pain (Briggs et al., 2004; Fechtenbaum et al., 2005; Keller et al., 2003; Melton III and Kallmes, 2006) and can eventually lead to a clinically established vertebral fracture (Keller et al., 2003; Sone et al., 1997). This loss of height implies that permanent deformations during prolonged mechanical loading of vertebral bodies are relevant to the tendency of a vertebra to collapse.

Creep is a continuous, time-dependent deformation observed in viscoelastic materials under a constant load (Lakes, 1999). Bone is a viscoelastic material in which mechanical properties change over the duration of loading (Currey, 1965; George and Vashishth, 2005; Kim et al., 2004b; Lynch and Silva, 2008; Rimnac et al., 1993; Sasaki and Enyo, 1995). Yamamoto et al. (2006) applied a physiological static creep (1500 με) on human vertebral cancellous bone and found a substantial creep development up to approximately 180% of the applied initial elastic strain. Of post-creep deformation, about half was not recovered, remaining as a residual strain. Furthermore, similar levels of creep and residual strain were measured during a physiological creep loading (1 kN, corresponding to a range of strains between 1246 and 2018 με)-unloading cycle on whole human vertebrae (Pollintine et al., 2009). These findings suggested that progressive vertebral deformation would develop even at the physiological loading level over years, which may increase the risk of vertebral failure. However, very little is known about the factors that determine creep and recovery behavior of cancellous bone.

Bone mass or bone mineral density (BMD) is the strongest single determinant of cancellous bone mechanical properties (Keaveny et al., 2001; Kopperdahl and Keaveny, 1998). Thus, it is not surprising that less bone mass is associated with higher fracture risk of bone. However, it was indicated that bone mass alone cannot fully explain bone fragility (Heaney, 2003; McCreadie and Goldstein, 2000) and creep of trabecular bone (Yamamoto et al., 2006). While BMD is defined to be the mineral content within an apparent volume of bone (including porosity and bone marrow, as well as bone matrix), the tissue mineral density (TMD) represents mineral content contained only in the matrix of bone (Tassani et al., 2011). Previous studies showed that TMD distribution of trabecular bone is altered after antiresorptive treatment with bisphosphonates in

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