Experimental and numerical simulation of microdamage and failure of thoracic vertebral trabecular bone

*Kosmopoulos, V; +*Keller, T S; **Baroud, G; **Steffen, T
+Musculoskeletal Research Laboratory, Department of Mechanical Engineering, The University of Vermont, Burlington, VT 05405-0156.

**Introduction** Microdamage within trabeculae has been postulated to contribute to decreased strength and stiffness of trabecular bone [1]. Recent investigators have shown that damage accumulation in vertebral structures can be modeled using plasticity theory [2]. The overall goal of this study was to predict the non-linear stress-strain response and failure of human vertebral trabecular bone. Results of an anatomically precise, structural finite element (FE) model incorporating plasticity theory are compared to experimental data. The combined experimental and numerical results provide insight into mechanisms of damage accumulation in within trabeculae.

Methods A thoracic (T7) vertebral body was harvested from a 79-year-old female, potted in polymethylmethacrylate (PMMA) and mechanical preconditioned for 20 uniaxial compression load-unload cycles using an MTS 858 Bionix testing system (50N to 1000N at a loading rate of 50N/sec). The VB was then load cycled to a post-yield load of 1500N and then to failure at the same loading rate. Apparent stress and strain were calculated from the VB cross-sectional area (mm²) and the height of the VB and PMMA (44.2 mm), respectively. From the pre-conditioning cycles an equilibrium apparent modulus (E₀ = 0.444 GPa) was determined as the slope of the stress-strain curve at the region spanning 1% of the maximum strain. Following mechanical testing, a 2.25 mm mid-sagittal section slab was cut from the vertebral body. The slab was defatted and digitally imaged at a resolution of 160 μm/pixel.

An anatomically precise, two-dimensional, structural finite element model was prepared from the thresholded digital image of the T7 VB, and a linear stress-strain FE simulation, mimicking the experimental conditions, was performed to determine the numerical apparent modulus Eₐ(FEA). The effective tissue modulus was then obtained as Eₑ = Eₐ(FEA)Eₑ/ Eₑ(FEA) = 1.588 GPa, where Eₑ(FEA) was initially 10 GPa. In the FEA model, the trabecular bone tissue (Eₑ =1.588 GPa), PMMA (Eₑ =0 GPa), and bone marrow tissues (Eₑ =10 kPa), were assumed to be isotropic, linear elastic materials. A Poisson’s ratio value of 0.3 was assumed for all 57,812 (194 x 298), 4-node isoparametric bone tissue, marrow and PMMA elements.

The structural FE model was coupled with a non-linear, stress-strain (σ-ε) function and an implicit modulus reduction algorithm derived from plasticity theory [2]:

\[ σ = σ₀ [\tanh(E₀/σ₀)]^m \]
\[ E_{\text{strain}} = \frac{dσ}{de} = -β[\text{tanh}^m \frac{σ}{σ₀}] + \text{tanh}^m \] (1)
\[ E_{\text{strain}} = \frac{dσ}{de} = -β[\text{tanh}^m \frac{σ}{σ₀}] + \text{tanh}^m \] (2)

where \( β = \frac{E₀}{σ₀}\), \( σ₀ \) is the tissue stress asymptote, \( m \) is an empirically-derived exponent, and \( E_{\text{strain}} \) is the reduced or microdamaged tissue modulus [2]. An additional simulation was then performed using the plasticity-based FE model, which was incrementally loaded (16 increments of -0.188 MPa) to a maximum compressive stress of 3 MPa (experimental value). After each increment of load, the principle strain difference \( ε = ε_1-ε_2 \) was used to determine the modulus reduction (if any) for each bone tissue element using equation (2).

**Results** Compared to the equilibrium experimental modulus, the post-yield experimental compression loading regimen resulted in a 15.8% overall reduction in the compressive apparent modulus. Optimization of equation (1) using the experimental data resulted in an exponent \( m = 4 \), and a stress-asymptote \( σ₀ = 12 \) MPa. The yield stress \( σ₁ \) according to the 0.2% strain offset method was 2.92 MPa. The plasticity-based, structural FE simulation closely predicted the experimental σ-ε behavior of the T7 VB (Fig. 1) and mirrored the tangent modulus reduction, which showed an abrupt decrease for stresses greater than 2.5 MPa (Fig. 2). Closer examination of the FE microdamage simulation results revealed that areas of increased stress (25.5% elements > \( σ_1 \)) corresponded well with diffuse damage microfractures that were observed in relatively confined regions below the T7 VB endplates, and also agree with experimental findings for human vertebrae subjected to compressive fatigue loading [3]. The reduced modulus areas corresponded to the highly stressed trabeculae (961 elements having a stress concentration: \( σ/σ₁ > 3 \)).

**Discussion** The objective of this study was to numerically reproduce the non-linear stress-strain behavior of trabecular bone. The microdamage simulation procedure is a systematic means to accomplish this goal and was very effective at predicting the mechanical behavior of a single thoracic vertebrae test specimen. An exponent \( m = 4 \) in equations (1) and (2) appears to be appropriate for predictions of the complex post-yield behavior of vertebrae. Our findings, and the findings of others [4,5] indicate that appreciable local tissue stress and strain levels are present during compressive loading of vertebrae and trabecular tissues. The FE-based plasticity model accurately predicted the regions of microdamage, which were confined to a relatively small subset of trabeculae. Rigorous validation of the model, including tests of normal and osteoporotic bone is needed, as is FE simulations of repeated loading. Other considerations affecting the mechanical response of trabecular bone include tissue anisotropy and multi-axial loading and can be accommodated by the model.

**Figure 1:** Experimental T7 VB stress-strain behavior and that determined from equation (1) and the non-linear, iterative FE analysis.

**Figure 2:** Experimental T7 VB tangent modulus and that determined from equation (2) and the non-linear, iterative FE analysis.


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