A Continuum Model for Microscale Bone Plasticity

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ABSTRACT

In [1] it has been demonstrated that bone plastic deformation is due to a tensile stretching of the mineralized collagen fibril and a predominant shear deformation of the interfibrillar matrix. Mineralized collagen fibrils possess specific orientation distributions [2] and are fibrils strongly aligned parallel to the bone surface. In [3] it was demonstrated that such orientation strongly would affect microcrack formation.

It is common practice to use Mises, Hill, Mohr Coulomb models to compute plastically deformed domains in trabecular bone. However, if a shear dominated irreversible mechanism underpins plasticity in bone, and if fibril orientation is specific to bone shape, then what is the elastic-plastic response of a single trabecula under mechanical loading and how is plastic deformation distributed in a core of trabecular bone. In order to implement a model for the analysis of this problem a bone tissue orientation model is implemented such mineralized fibrils are all oriented normal to the bone surface. Then the dominant shear deformation is to occur in planes parallel to the free bone surface.

In the present work, the initiation and progression of shear sliding is modelled by the use of an extended finite element model with the post ignition traction-separation law simulating perfectly plastic behaviour. In addition, bone tissue is elastic anisotropic and heterogeneous, introducing a feature rich heterogeneous tissue.

This contribution reports on simulation outcomes obtained with the model described here both for loading of single trabecula and compression of a bone core. The results are compared to those from classical plasticity models applied to bone using isotropic Mises and anisotropic Hill-type plasticity. The present plasticity model for bone tissue leads to plastic zones distinctly different from those obtained by associated flow plasticity models and the spatial extent of the plastic zones reflects the directionality of the bone tissue and the present stress state. Finally, the model is applied to estimate the effects of bone aging and treatment on plastic deformation.

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REFERENCES