

LACUNOCANALICULAR FLUID FLOW AND REGULATION OF BASIC MULTICELLULAR UNIT ACTIVITY

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INTRODUCTION

Human bone is continuously regenerated through remodelling, in which discrete packets of bone are replaced while maintaining a constant geometry (Parfitt, 1994). The process of remodelling is carried out by a complex mediator mechanism called the Basic Multicellular Unit (BMU) (Frost, 1990). In cortical bone, BMUs proceed by osteonal tunneling, during which osteoclasts excavate a canal that is subsequently refilled by osteoblasts (Smit and Burger, 2000). Current theories suggest that coordinated cellular activities associated with BMU remodelling are strain and fluid-flow regulated (Burger et al., 2003; Smit and Burger, 2000; Smit et al., 2002). Theoretical models to examine cortical remodelling have focused on idealized views of BMU forms. In reality, BMU morphology varies and includes unidirectional, bidirectional, and branched forms (Cooper et al., 2006) (Figure 1).

PURPOSE

The purpose of this study was to examine remodelling theories in relation to complex BMU forms observed in secondary bone.

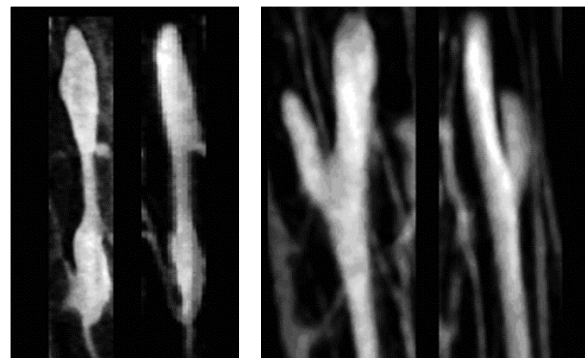


Figure 1. Micro-computed tomography images of bidirectional (left) and branched (right) BMU morphologies (Cooper et al., 2006).

METHODS

Computational models were developed in STARS, a coupled finite difference fluid flow and finite element mechanics simulator (Computer Modelling Group, Ltd., Calgary, AB, Canada). One idealized model and two geometrically accurate (unidirectional and branched canal forms) generated from three-dimensional micro-computed tomography image data were analyzed. Models were subjected to physiologically relevant axial compressive loads, which induced pressure-driven fluid flow through the simulated lacunocanalicular porosity.

RESULTS

Upon loading, fluid flowed from the high-pressure bone matrix into the low-pressure resorption space. Consistent with previous models, the idealized form had reduced longitudinal normal strain directly in front of the cutting cone where osteoclasts are activated, and increased strain behind the cutting cone where osteoclasts are inhibited. The unidirectional canal model also displayed decreased strain in front of the cutting cone, although strain behind the cone increased minimally, relative to the idealized case. Similarly, the geometrically accurate branched model demonstrated strain reduction in front of both cutting cones and minimally increased strain behind the cones. Fluid stasis has been implicated as a regulating mechanism in BMU activity, whereby decreased osteocytic stimulation results in reduced production of nitric oxide, inducing apoptosis, which subsequently signals the recruitment of osteoclasts. In all three BMU models, regions of relative fluid stasis were observed in front of the cutting cones (Figure 2). Branched morphology also displayed significant strain reduction and fluid stasis above the fork in the canal, where it divided into two distinct resorption spaces.

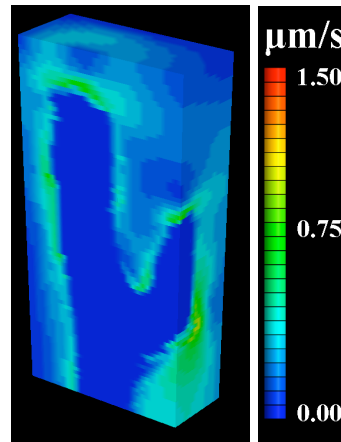
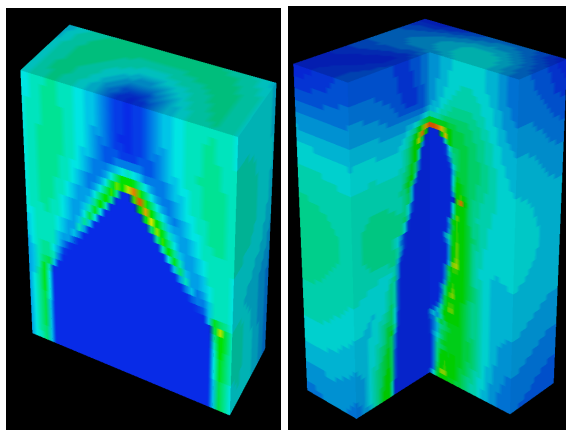


Figure 2. Three-dimensional sectioned views of idealized (lower left), unidirectional (lower right), and branched (left) BMU fluid velocity magnitude distributions.

CONCLUSIONS

If strain and fluid flow play a central role in coordinating BMU activity, inter-branch strain shielding and reduced fluid flow suggested that the region between resorption spaces would also be prone to osteocyte apoptosis and osteoclastic activity, effectively eliminating the branched structure. Therefore, strain and related fluid flow may not explain complex canal geometries. Consequently, further investigation is required to elucidate the underlying mechanisms involved in cortical bone remodelling.

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