MODELING TRABECULAR BONE FRACTURE HEALING

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Introduction
Trabecular bone fractures heal differently than diaphyseal bone fractures. In particular, trabecular bone heals through the process of intramembraneous ossification where bone forms directly on existing bone without formation of a callus or a cartilage intermediate. In addition, the marrow in trabecular bone provides a rich vascular environment compared to the limited vascularity of diaphyseal bone. During trabecular bone fracture healing, woven bone fills the fracture gap. Once woven bone bridges the fracture gap, it is remodeled to form trabecular bone with a load dependent structure.

Previous studies have modeled diaphyseal fracture healing or trabecular remodeling. However, no study has combined the two in a single model. The objective of this study was to simulate trabecular fracture healing, including tissue differentiation during the healing process and remodeling of the woven bone to form trabecular bone.

Methods
We modeled a fracture in trabecular bone using an idealized micro-scale cube (600 µm) through the fracture gap. The cube had trabecular spicules lining the fracture gap (E=10,000 MPa, v=0.3) with soft tissue (E=3 MPa, v=0.3) representing the marrow and hematoma both in the gap and surrounding the trabeculae (Figure 1). The loading was applied as pressure to the trabecular spicules in two stages: (1) 0.05 MPa until the fracture gap was stabilized by woven bone; (2) 3 MPa as the woven bone remodeled to form trabecular bone.

The model combined finite element analysis and fuzzy logic in an iterative healing simulation. Loads were applied to the model, and deviatoric and dilatational strains were determined. The resulting strains and current material properties were used as input to a fuzzy logic controller that determined tissue differentiation based on the tissue differentiation diagram of Claes and Heigele (1). The output from the fuzzy controller determined changes in the amount of bone, cartilage, and soft tissue for each element. Material properties were updated using a rule of mixtures before the next iteration began. Bone resorption was not allowed in the first stage of healing as woven bone formed in the gap. After the fracture was stabilized, however, bone resorption was permitted to occur where the strains were small (< 100 µm). Iterations continued until the model reached a homeostatic stable state.

Results
The simulation predicted formation of woven bone starting from both sides of the gap and proceeding towards the middle of the gap. As the load increased and remodeling was allowed in stage 2, the homogeneous woven bone was remodeled to form trabecular bone with an oriented trabecular structure (Figure 2).

Figure 1: Idealized model of a fracture gap in trabecular bone.

Figure 2: During the first stage (iterations 0-20) woven bone formed in the gap. In stage 2, the woven bone was remodeled to form a trabecular structure.

Additional models were used to determine the effects of loading direction on formation of the trabeculae. It was shown that with altered loading direction, the trabecular orientation changed. Additionally, with increased load magnitude, the trabeculae thickness increased.

Discussion
The predicted trabecular structure was simplified compared to a real trabecular structure because the applied loading was idealized. In vivo trabecular bone, the loading is multi-directional and varies over time, creating a more complex structure. The model was sensitive to the boundaries that determined the strain state resulted in the different tissue types. We approximated these boundaries using in-vivo and in-vitro data (2,3).

This model is the first numerical simulation that combines both fracture healing and bone remodeling in a single model. The method was adapted from a previous study that used finite element analysis and fuzzy logic to predict diaphyseal fracture healing, and therefore can predict both endochondral and intramembraneous ossification as well as bone remodeling. A consistent model that explains bone formation, maintenance, regeneration, and healing could help to determine how bone metabolism and tissue differentiation are influenced by mechanical environments.

References
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