3D Simulation of damage repair along a trabecular strut

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Introduction

Trabecular remodelling consists of the continuous resorption and formation of bone along the surface of a trabecular strut. It is carried out by the coupled action of osteoclasts and osteoblasts which function together as a Bone Multicellular Unit (BMU), see Fig 1. Experiments have shown that changes in mechanical stimuli, such as damage and strain, can effect the biomechanical behaviour of the remodelling activity [1,2]. Therefore, various theories have been generated postulating the mechanics by which these stimuli regulate cellular activity [3,4]. Generally computer programs implementing these theories have only considered a single stimulus. However, experimental evidence suggests that bone cells may respond to both stimuli [5] .

Indeed, a previous computational algorithm which hypothesised that remodelling is regulated by a combination of stain and microdamage, with damage being prioritised, successfully simulated BMU progression [6]. However, the hypothesis was only tested in 2D and the complex development of a BMU in 3D has not yet been examined.

Fig 1: **Bone Remodelling Cells [7]**

Objectives

This project investigates the hypothesis that bone remodelling is regulated by a combination of strain and microdamage, with damage being prioritised once a threshold is exceeded. It is hoped that by incorporating this hypothesis into a computer model, the 3D activity of a BMU along a single trabecular strut can be simulated.

Materials & Methods

To test the validity of the hypothesis, a 3D finite element model representing a single trabecular strut was constructed in MSC.marc, as shown in Fig 2. Two linear elastic materials were modelled, an outer marrow layer surrounding a rectangular bone strut. Material properties used were as follows: Trabecular bone, E=1800MPa, υ=0.3 and ρ=0.67g/cm3; Marrow E=2MPa, υ=0.3 and ρ=0.01g/cm3 [6].

To examine the repair of microdamage by a BMU, an initial region of bone was defined to have a pre-existing damage level. A physiological strain of 1500με was applied to one end of the strut while the other end was fully constrained.

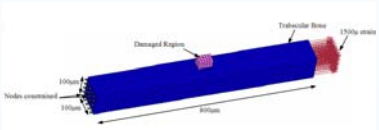

Fig 2: **Finite element model of trabecular strut with damaged region (surrounding marrow layer not shown)**

Following previous methods [8], two different mechano-sensors were considered: osteocytes and bone-lining cells. The stimulus received at each of these locations was dependant on the level of stress and strain obtained from the finite element analysis.

Material properties were then adjusted according to the stimulus level and the applied hypothesis, see Fig 3. Changes were confined to surface elements.

As damage levels are dependant on the loading history of bone, damage was allowed to accumulate throughout the loading cycle.

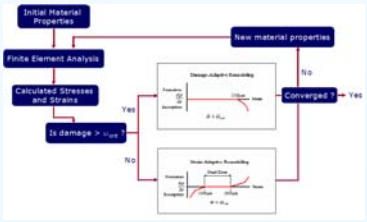

Fig 3: Flow chart of computational algorithm

Results

Using bone-lining cells as the mechano-sensors, it was found that all the damaged region was removed, however, the resorption cavity was not successfully refilled. Instead a bone layer was formed across the top of the lacuna, encompassing a marrow space, see Fig 4(a). Further examination of the results showed that high stresses arise at the top of the cavity, see Fig 5, resulting in more bone formation at this location than at the base leading to this enclosure.

Examining the effect of using osteocytes as the sensors, complete removal of the damaged tissue was not predicted and refilling of the resorption cavity did not occur, see Fig 4(b).

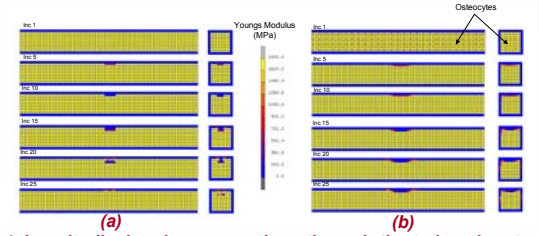

Fig 4: **Longitudinal and cross-sections through the trabecular strut with (a) bone-lining cells and (b) osteocytes as mechano-sensors**

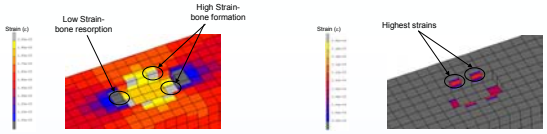

Fig 5: **Distribution of strains in trabecular bone at Inc 21 using bone-lining cells as mechano-sensors**

Discussion & Conclusion

It was found that by using the remodelling hypothesis alone, the process could not be completely simulated in 3D, either by using bone-lining cells or osteocytes as the mechano-sensors. Due to the number of biological processes involved in the process, it may be necessary to implement rule-based algorithms [9] in conjunction with the remodelling hypothesis in order to successfully simulate BMU progression. One rule may be to restrict the location osteoclasts are permitted to attach and form bone, irrespective of the stimulus received.

To develop the project further, the algorithm will be applied to in vitro trabecular bone samples similar to that shown in Fig 6. Voxel images from micro-CT scans are directly converted into elements which can then be imported into finite element packages. By using the real architecture of trabecular bone, the true advancement of a BMU along the surface of the strut can be examined.

Fig 6: Voxel-based finite element model of in vivo trabecular strut

References

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