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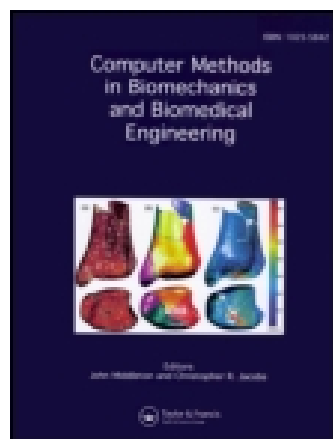
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A 3D model of trabecular bone remodelling based on cellular activities

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Keywords: bone; remodelling; strain; mechanobiology

1. Introduction

Prediction and treatment of bone diseases such as osteoporosis, which weakens bone and increases fracture risk, are major issues. Micro-architecture of trabecular bone contributes importantly to overall mechanical properties of bone and the goal here is to predict its evolution. A model of bone remodelling was developed and allows describing where, when and how the remodelling occurs in a physiological and in a low-loading case (for instance, bedridden and microgravity).

2. Methods

2.1 Mechano-regulation theory

The model is based on two physiological observations: bone adapts to mechanical strain and microdamage targets the remodelling sites.

To explain the loss of bone in a low-loading case, we put forward that remodelling is multiplied by 2.5 (Frost 1990) and as the remodelling has a negative balance (even in a healthy case apposed volume is always smaller than the resorbed volume (Cowin 2001)), the loss of bone is greater in a low-loading case.

2.2 Mechano-regulation algorithm

Algorithm is based on activities of osteocytes which act as mechano-sensors (they can feel changes in mechanical stimuli) and drive the remodelling. The density of osteocytes, which decreases with age, is represented in the model as a function of age.

Osteocytes send a biochemical signal to bone-lining cells which are placed on the trabecular bone surface. The algorithm computes the signal perceived by each bone-lining cell (density of 19 cells/mm², Cowin 2001), depending on the local strain, the position of each

osteocyte inside the bone (an influent area is introduced (Mullender and Huiskes 1995)) and on local micro-damage. When signal is under a threshold, osteoclasts are recruited to resorb a targeted area and then remodelling cycle begins (resorption, reversion, apposition/mineralisation).

2.3 Remodelling kinetics

The kinetic of remodelling has been studied (Eriksen, Gundersen, et al. 1984; Eriksen, Melsen, et al. 1984) and it follows these steps (Figure 1):

- Quiescent phase.
- Osteoclasts resorb the mature bone quickly and dig a hole, the geometry of which is predefined (duration of 9 days).
- Osteoclasts die but mononuclear cells come and resorb mature bone with a smaller kinetic (duration of 20 days).
- Reversal phase (duration of 10 days).

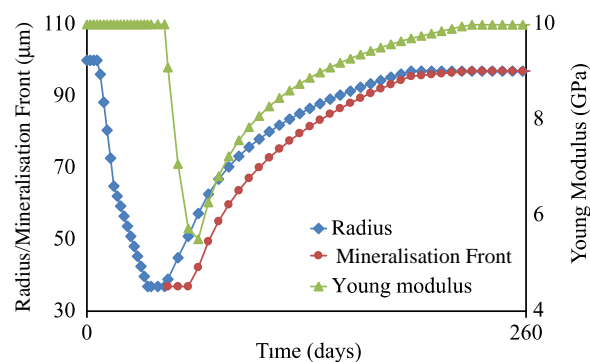


Figure 1. Evolution of the geometry, stiffness and mineralisation of a cylindrical trabeculae on a remodelling site (initial radius 100 μm, final radius 95 μm).

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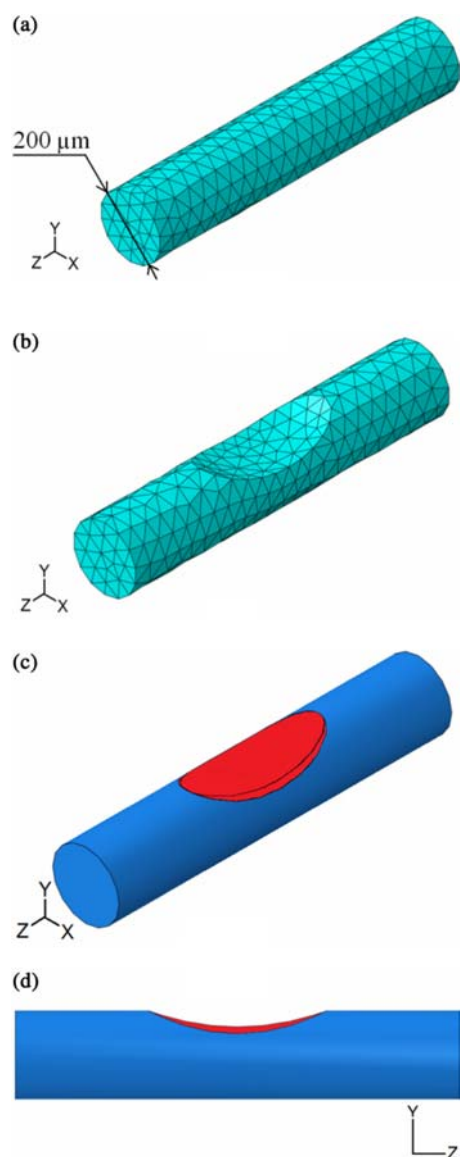


Figure 2. (a) mesh of the initial trabeculae (2150 elements), (b) mesh of the resorbed trabeculae (2344 elements), (c) and (d) trabeculae before apposition (blue) and after 20 days (red) (the red part represents osteoid tissue).

- (e) Apposition phase is linear while 15 days ($1 \mu\text{m}/\text{day}$). The deposited tissue, the osteoid, is composed of a majority of collagen.
- (f) Mineralisation starts when the osteoid thickness is about $15 \mu\text{m}$.
- (g) Apposition goes until 19/20 of the resorbed volume is apposed (duration of 146 days).
- (h) Mineralisation front moves until it reaches the osteoid surface (duration of 163 days).

3. Results and discussion

Linear-elastic, 4-nodal tetrahedral elements were used to model a cylindrical trabecula of radius $100 \mu\text{m}$ (Abaqus). Mature bone tissue was assigned an elastic modulus of 10 GPa, 1 GPa for the osteoid tissue, and Poisson's ratio of 0.3.

The mesh of the initial and the resorbed trabeculae is represented in Figure 2(a) and (b). Figure 2(c) and (d) describes a remodelling site.

As the mineralisation front does not correspond to the osteoid surface, in the numerical model the non-mineralised osteoid is considered impacted in the trabeculae.

In a low-loading case, there are many more remodelling sites. Instead of considering only a single trabecula which is not representative, we consider at the moment a network of trabeculae (3D reconstruction of real trabecular structure).

4. Conclusions

The 3D model of bone remodelling presented here allows to estimate the loss of bone in a healthy case and especially in a low-loading case. The developed model is based on osteocytes, osteoclasts and osteoblasts activity. So we can easily integrate a cell dysfunction and predict the evolution of bone micro-architecture. Indeed, this model allows to predict remodelling over a long time period.

In future works, we will integrate the over-loading case: bone volume increases to reduce strain field. The model will also simulate the effect of a treatment on bone disease. The purpose is to predict from a bone micro-architecture, how would evolve the geometry and how to heal efficiently.

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