

RELATING OSTEON DIAMETER TO STRAIN

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INTRODUCTION

Bone is continuously remodeled by bone-resorbing osteoclasts and bone-forming osteoblasts, combined in Basic Multicellular Units (BMUs). In cortical bone a cutting cone of osteoclasts excavates a tunnel, closely followed by a closing cone of osteoblasts that fills the tunnel with new bone [1]. Thus, the BMU creates a new bone structure called osteon. Osteon diameter is determined by the size of the cutting cone, but it is not known what determines cutting cone size. Frost [2] suggested that osteon diameter decreases with strain, since osteons in the endosteal side of the cortex are wider than osteons in the more strained periosteal side. Skedros et al. [3] also observed smaller osteon diameter in bone regions that experience larger strains. A mechanism relating osteon diameter to strain is as yet unknown.

A relation between strain and osteon diameter implies a relation between strain and osteoclastic bone resorption. These are related in our bone adaptation model [4], which was previously used to explain why osteons are aligned to the main loading direction [5]. Our model is based on a mechanosensory function of osteocytes [6,7,8]. Upon sensing a mechanical stimulus, osteocytes are assumed to emit a biochemical signal that inhibits osteoclastic bone resorption [9] and activates osteoblastic bone formation [6]. In the present work we investigate how this model relates osteon diameter to loading magnitude.

METHODS

Osteon development is simulated in a 2D finite-element model, using a $10\ \mu\text{m}$ element size. The initial configuration consists of a $4 \times 4\ \text{mm}^2$ piece of compact bone, subjected to compressive loads in the vertical direction. Osteoclasts start from an initial resorption cavity of $180\ \mu\text{m}$ diameter. Osteocytes are positioned in the bone tissue at a density of $1600\ \text{mm}^{-2}$. Depending on the local strain-energy density (SED) experienced, the osteocytes emit a signal. This signal weakens with increasing distance from the osteocyte, up to a maximum influence distance of $150\ \mu\text{m}$. Each element receives signals from the nearby osteocytes. A high concentration of this signal inhibits the adhesion of osteoclasts to the bone surface, thus inhibiting resorption. Osteoclasts are explicitly modeled using the Glazier & Graner model of differential cell adhesion [10]. Where the concentration of osteocyte signal is low, osteoclasts adhere to the bone surface and start to resorb bone. New osteoclasts can originate on such bone surfaces. A high signal concentration causes osteoclasts to detach from the bone surface. Osteoclasts are assumed to die after an extended period of detachment. We perform three simulations at different loading magnitudes: 17.0, 18.5 and 20.0 MPa. These run for 100 model increments, representing a 25 day remodeling period.

RESULTS

Around the cavity, strains are low in loading direction and high in transverse directions. Since the osteocyte signal reflects this mechanical environment, there is little inhibition to resorption in the loading direction, whereas lateral resorption is inhibited. Osteoclasts resorb a tunnel in loading direction (Fig. 1). Osteoblasts are recruited to the eroded surface where they form a closing cone of new bone.

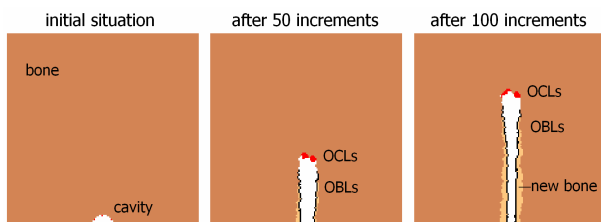


Figure 1: Development of the 20 MPa simulation.

The three simulations show similar osteon development, but with different osteon diameters (Fig. 2, above). The osteocyte signal (Fig. 2, below) is weaker at the lower loads, allowing more lateral resorption from the initial cavity and thus a larger cutting cone size.

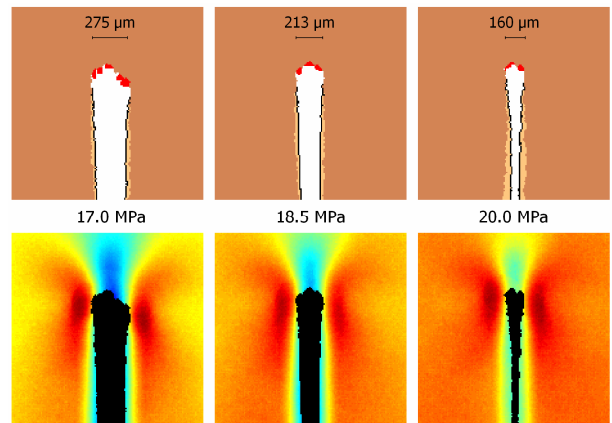


Figure 2: Final configurations (above) and corresponding signal distributions (below) for simulations at different loading magnitudes.

DISCUSSION

The proposed mechanism explains why osteon diameter is usually smaller in bone regions that experience larger strains. This mechanism, the inhibition of resorption by mechanically induced osteocyte signals, is not really controversial. It is widely believed that osteocytes serve as sensors of mechanical stimuli within bone tissue and there are also indications that osteocytic signals can inhibit bone resorption [9,11].

Osteoclast origination is included in the model, on bone surfaces where osteoclast adhesion is possible [12]. The theory also incorporates osteoclast death, assuming that detachment from the bone surface induces apoptosis. This assumption is based on the observation [13] that pre-osteoclasts only fuse with attached osteoclasts. The fusion of pre-osteoclasts is needed for the survival of an osteoclast, as its nuclei have only a short life-span [14]. Thus, an osteoclast would not survive an extended period of detachment. This may explain the observation [15] that osteoclast apoptosis occurs mostly at the sides of the cutting cone, because it is there that high osteocyte signals promote the detachment of osteoclasts.

In sum, we show how our theory, previously introduced to explain osteonal load alignment, also explains the inverse relation between osteon diameter and strain magnitude.

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