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Optimal bone density distributions: Numerical analysis of the osteocyte spatial influence in bone remodeling

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ABSTRACT

In this paper a control and optimization procedure for bone remodeling simulations was adopted to study the effect of the osteocyte influence range on the predicted density distribution. In order to reach this goal, the osteocyte network regulating bone remodeling process in a 2-D bone sample was numerically simulated. The assumed proportional-integral-derivative (PID) bone remodeling rule was related to the error signal between the strain energy density and a selected target. Furthermore the control parameters and the target were optimally determined minimizing a suitable cost index: the goal was to minimize the final mass and the energy thus maximizing the stiffness. The continuum model results show that the developed and adapted trabecular structure was consistent with the applied loads and only depended on the external forces, the value of the cost index, the maximum attainable elastic modulus value (hence, the maximum density value) and the value of the energy target. The remodeling phenomenon determined the number and thickness of the trabeculae which are formed from a uniform distribution of mass density in the considered domain; this number and these thicknesses are controlled by the values assigned to the parameters of the model. In particular, the osteocyte decay distance (D) of the influence range affected the trabecular patterns formation, showing an important effect in the adaptive capacity of the optimization numerical model.

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1. Introduction

In bone remodeling investigations, the concept of sensor influence range for the interconnections between osteocytes and osteoblasts/osteoclasts, was introduced in [1]. Osteocytes are the most numerous cells in bone and sensitive to mechanical loading and fluid flow, as demonstrated in cell culture studies [2,3]. At the cell scale, bone tissue is resorbed by osteoclasts, and new bone tissue is formed by osteoblasts. It is hypothesized that bone adaptation regulated by osteoblast and osteoclast activities is controlled by osteocytes, which act as mechanosensors based on local loading conditions [1,4,5]. At the organ level the process of functional adaptation allows

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bone to perform its mechanical functions and to support mechanical loads for daily activities with a minimum of mass and to protect internal organs. Hence, bone remodeling could be designed by a local control process in which sensor cells in the bone matrix measure a mechanical signal and stimulate actor cells to add or remove bone in their neighbourhoods. It is also thought that the intensity of this signal fades with the distance [6]. As more deeply explained in [7–10], all mechanical and biological modes by which the signal is conveyed and transmitted from sensor cells (osteocytes) to actuator cells (osteoclasts and osteoblasts) are still not known with satisfactory accuracy and completeness. Therefore, theoretical models are in part based on simplifying assumptions, such as the conjecture according to which the actuator cells performing the destructive task (osteoclasts) are recruited by the formation of localized damage or micro-cracks. As a consequence, the concentration of stresses in the neighbourhood of the resorption cavities could affect the coupling between mechanical phenomena and chemical phenomena in the balance between the osteoclasts and the osteoblasts activities.

The objective of a theory for investigating bone remodeling is to provide numerical models or the basis for boundary value problems that will predict the adaptive behaviour of the tissue under altered environmental load, e.g. after implantation of prosthetic devices [11–15]. It has been suggested that femoral resorption could be the predominant factor in implant stem fracture of a total hip replacement [16].

Relationships between local bone apparent density and loading history are developed by assuming that bone mass is adjusted in response to energy considerations [17,18]. In fact, the strain energy density (SED) is used as a feedback control variable to determine shape or bone density adaptation to alternative functional requirements, whereby homeostatic SED distribution is assumed as the remodeling objective [18].

To account for resorption and synthesis phenomena, Tovar et al. [19] used the hybrid cellular automaton algorithm to incorporate a distributed control loop within a structure where ideally localized sensor cells activate local processes of bone formation and resorption. In [20,21] the existence of a physiological control process is implied by the functional adaptation of bone to mechanical usage; in the former paper, the assumption is to consider bone adaptation as an optimal control problem: the bone reacts to variation of mechanical loads in an optimal way aiming at the minimization of a suitable cost index that takes into account the rate of remodeling. In the latter the Author made use of the hypothesis of optimal response of bone and derived (instead of postulating) the remodeling rules: the derivative with respect to time of the total strain energy stored in the whole domain was globally minimized, with additional global and local constraints applied on the density of material.

Madeo et al. [22] introduced suitable evolution equations for Lagrangian mass densities of the mixture constituents based on an integrodifferential operator defined on deformation fields.

In Andreaus et al. [23–25] the assumed bone remodeling rule was represented by the classical proportional–integral–derivative (PID) control, applied to the error signal between the strain energy density and a target to be optimized; the underlying premise was that the mass should be distributed in an efficient way, using a minimum amount of material to accomplish the mechanical function. Therefore suitable cost indices were considered, namely the linear combination of weighted mass and strain energy, and the product of mass and energy. Two local evolution rules were compared, revealing the update of mass density and of Young's modulus, respectively.

Interesting analogous studies can be found in [26–28]. In [26], a mathematical model is introduced using a mechanobiological approach describing the process of osseointegration at the bone–dental implant interface in terms of biological and mechanical factors and the implant surface. The model is able to reproduce features of the wound healing process such as blood clotting, osteogenic cell migration, granulation tissue formation, collagen-like matrix displacements and new osteoid formation. In [27], two stages are assumed in the formation of the bones in the calvaria, the first one takes into account the formation of the primary centers of ossification. This step counts on the differentiation from mesenchymal cells into osteoblasts. A molecular mechanism is used based on a system of reaction–diffusion between two antagonistic molecules. In the second step of the model a molecule that is expressed by osteoblasts is used, and that is expressed from the osteoblasts of each flat bone. This molecule allows bone growth through its borders through cell differentiation adjacent to each bone of the skull. In [28], the identification of a monitoring procedure for the bone demineralization in microgravity is based on a mathematical model of the interaction of the most relevant blood and urine indicators of bone demineralization; a model to foresee the evolution of these parameters in the space, depending on the therapy chosen, is provided.

In the present study a control and optimization procedure was adopted to study the effect of the sensor influence range on the predicted bone density distribution, consistent with the applied boundary conditions and bone micro-architecture. In order to reach this goal, the osteocyte network regulating bone remodeling process in a 2-D bone sample, subjected to an in-plane linear loading distribution, was numerically simulated. The tested sample is identical to the one tested by Weinans et al. [7] and Mullender and Huiskes [8]. The present analysis allowed for obtaining additional information about optimal values for control gains, target, and mutual importance of mass and energy in determining the final distribution of mass density of bone in terms of influence distance. This is demonstrated by the quantitative evaluation of mass loss (with respect to the available initial value) and stiffness gain conducted in both optimal and suboptimal trabecular configurations.

It was assumed that osteocytes act as sensors by detecting a mechanical signal and in turn produce a stimulus for bone mass growth/resorption. The reference values of mass density and elastic modulus were typical of bone tissues and the load magnitude was in accordance with physiological values [29]. The assumed PID bone remodeling rule was related to the error signal between the strain energy density and a selected target. Furthermore the control parameters and the target were optimally determined minimizing a suitable cost index: the goal was to minimize the final mass and the energy thus maximizing the stiffness. Two different choices for the cost indices were used. The optimal selection of the weight for one of the proposed cost indices was also assumed. In Section 2

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