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OSTEOCYTE STIMULATION THROUGH PORE PRESSURIZATION INDUCED BY PHYSIOLOGICAL MACROSCOPIC BONE STRAINS: INSIGHTS FROM A MICROPOROMECHANICAL MODEL

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Bone tissue is a living biological entity. In particular, it is able to renew itself, which is key for maintaining its mechanical integrity necessary for fulfilling fundamental tasks such as providing protection of organs and facilitating locomotion. Moreover, bone is also able to accommodate to an enduringly changing mechanical loading environment (e.g. due to exercise regimes, bedrest, or exposure to microgravity). *Bone remodeling* is the term summarizing the underlying cellular activities, leading to removal of mature bone tissue and subsequent formation of new bone tissue, driven by complex actions of biological cells and biochemical factors. Importantly, bone remodeling is also strongly influenced by the mechanical loads applied onto the skeleton [1].

State-of-the-art mathematical models of bone remodeling include multiscale aspects regarding the significant difference between the characteristic lengths of the macroscopic application of mechanical loads (on the observation scale of whole bone organs), on the one hand, and the microscopic sensing of mechanical loads (on the observation scale of cells), on the other hand, see e.g. [2]. However, the exact mechanism by which cells are actually able to sense mechanical signals is still a matter of debate. While in mathematical models of bone remodeling it is an often pursued strategy to consider the changes of the strain energy density as representative (scalar) measure for the temporal changes of the mechanical loading, the time has come to bring mathematical models of bone remodeling closer to the “real” bone physiology.

Experimental evidence suggests that the hydrostatic pressure acting onto osteocytes in the lacunar pore space, related to the macroscopically applied physiological loading, is a potential stimulus for modulating mechanobiological regulation of bone remodeling. Here, we aim at investigating this mechanism, by adapting the micromechanics-based poroelasticity model of Hellmich et al. [3], allowing us to accurately compute the lacunar pore pressures in response to macroscopic mechanical loading. In particular, the model takes into account the composition of bone on the considered length scales, the interactions of the lacunar and vascular pore spaces with the respectively surrounding extracellular and extravascular bone matrices, and the interactions between the pore spaces across several orders of magnitude of length scale.

Computation of the lacunar pore pressures, arising due to physiological macroscopic strains, shows that the hydrostatic pressure identified *in vivo* as optimum mechanoregulatory cell stimulus (i.e. several tens, up to approximately 100 kPa [4]) indeed occurs in the physiological environment of the involved cells. This result provides the incentive to further invest into studying the effects of changing lacunar pore pressures on the related bone remodeling events, thus to combine the presented model with aforementioned integrated approaches to computational simulation of the bone remodeling process [2].

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