Poromicromechanics reveals that physiological bone strain induces osteocyte-stimulating lacunar pressure

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ABSTRACT

The mechanical loading that bone organs are subjected to is known to influence the activities of cells located in the pore spaces of bone. This concerns in particular the signalling and production processes mediated by osteocytes. The exact mechanism(s) by which osteocytes are actually able to "feel" the mechanical loading and changes thereof has been the subject of numerous studies, and while several hypotheses have been brought forth over time [1], has remained a matter of debate.

A recent experimental study by Gardinier and co-workers [2] revealed that due to the very narrow transport pathways in the lacunae-canaliculi system, when also taking into account that mechanical stimuli occur in transient fashion, substantial convective fluid flow, which has been suspected as prime candidate by a large part of the scientific community for causing osteocyte stimulation, is unlikely to take place. Thus, revisiting the role of the pore pressure building up in the lacunar pore space, whose occurrence is at the same time confirmed in Gardinier's study, for osteocyte stimulation, is required. For this purpose, a thorough multiscale modeling approach is pursued. In particular, the proposed model is based on multiscale poroelasticity theory, able to account for a multiporous material such as bone, and combined with micromechanics-based homogenization. First, the model response is studied qualitatively, and distinctly non-linear dependencies of the resulting lacunar (and vascular) pore pressures on the underlying bone composition are revealed, highlighting the necessity of using a rigorous multiscale approach for computation of these pore pressures. Additionally, the pore pressures vary significantly between different load directions. Then, the derived equations are evaluated for macroscopic physiological strains. The resulting pore pressures agree well with the pressures that have been shown in *in vitro* studies to be of adequate magnitude for modulating cell responses, see e.g. [3]. Further numerical studies show how the lacunar pressure develops in the course of aging.

Thus, the important role of the hydrostatic pressure building up in the bone pore spaces in response to macroscopically applied mechanical loading is corroborated, providing the incentive to further look into the mechanoregulatory role of the lacunar (and vascular) pore pressures in future research.

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