MECHANOTRANSDUCTION AND STRAIN AMPLIFICATION IN BONE CELL PROCESSES

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A paradox in bone tissue is that tissue-level strains due to animal and human locomotion are too small to initiate intracellular chemical responses directly. A model recently was proposed to resolve this paradox, which predicts that the fluid flow through the pericellular matrix in the lacunar-canalicular porosity due to mechanical loading can induce strains in the actin filament bundles of the cytoskeleton that are more than an order of magnitude larger than tissue level strains. In this study, we greatly refine this model by using the latest ultrastructural data for the cell process cytoskeleton, the tethering elements that attach the process to the canalicular wall and their finite flexural rigidity *EI*. We construct a much more realistic 3D model for the osteocyte process and then use large-deformation ‘‘elastica’’ theory for finite *EI* to predict the deformed shape of the tethering elements and the hoop strain on the central actin bundle. Our model predicts a cell process that is 3 times stiffer than in a previous study but hoop strain of >0.5% for tissue-level strains of >1,000 microstrain at 1 Hz and >250 microstrain at frequencies >10 Hz. We propose that this strain amplification model provides a more likely hypothesis for the excitation of osteocytes than the previously proposed fluid-shear hypothesis.

Keywords: osteocytes, actin cytoskeleton