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A coupled systems biology-micromechanical model for mechanostat-type regulation of bone remodeling

The capacity of bone tissue to alter its mass and structure in response to mechanical demands was recognized more than a century ago and Frost formulated the so-called mechanostat theory for capturing this phenomenon mathematically. This theory proposes that bone responds to changes from a loading relating to an equilibrated bone turnover by triggering either increased bone resorption or formation as response to decreased or increased loading. While this conceptual theory is useful for a qualitative understanding of bone tissue level responses to mechanical loading no quantitative estimates of bone volume/mass changes can be made. Also incorporation of the underlying cellular mechanisms is still outstanding. Over the last several years significant progress has been made to identify the cells and signaling molecules involved in the mechanical adaptation of bone. It is now well accepted that osteocytes act as mechanosensory cells in bone which express several signaling molecules able to trigger bone adaptation responses. Here we present an extended bone cell population model incorporating a simplified osteocyte-feedback to simulate bone remodeling events corresponding to the actual mechanical loading. The mechanical feedback to bone biology is achieved by employing continuum micromechanics-based homogenization of bone stiffness, allowing for estimation of the deformation osteocytes are subjected to. This methodology allows for monitoring effects of mechanical load changes on the composition, and thus on the load-carrying capacity of bone. To the authors knowledge, this is the first model which incorporates the mechanostat theory based on cellular feedback mechanisms.