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MODELING MECHANOCHEMICAL COUPLINGS IN TRABECULAR AND OSTEONAL BONE REMODELING

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Mechanochemical couplings at cellular level regulate coordinated cycles of osteoclastic bone resorption and osteoblastic bone formation on the microscopic surfaces such as trabecular surface in cancellous bone and on osteonal surface in cortical bone [1]. In addition, mechanosensing by osteocytes and communication among bone cells result in a complex spatiotemporal regulation of bone remodeling. To clarify fundamental mechanisms of the complex bone adaptation phenomenon, we newly proposed a mathematical model of trabecular and osteonal bone remodeling, and conducted computational simulations.

One of the important regulation factors of osteoclast differentiation in bone metabolism, RANKL/RANK/OPG system, was introduced in the mechanoregulation model of trabecular and osteonal remodeling. RANKL activates osteoclastogenesis and OPG prevents RANKL from binding to its receptor, RANK [2], both of which are known to be produced by osteoblasts and/or osteocytes. Therefore, the RANKL/RANK/OPG system provides positive and negative regulations of osteoclast differentiation that make the bone remodeling cycles more complicated.

To investigate spatiotemporal regulations of trabecular and osteonal bone remodeling based on the proposed model, we conducted computer simulations for a single trabecula and a single osteon under uniaxial compressive loading, using voxel finite element models [3]. Effects of the RANKL expression level on the remodeling turnover rate were investigated, and the role of mechanical loading on the trabecular and osteonal orientations was discussed. Distribution of mechanical stimulus in both trabecular and osteonal surfaces was regulated to become uniform with morphological changes to align along their axes to the loading direction. Thus, modeling and simulation of bone signaling and their interactions will be a good framework to investigate the spatiotemporal regulation in complex bone adaptation by remodeling.

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