BONE: THE ENGINEER'S ULTIMATE DREAM MATERIAL

Rik Huiskes
Department of Biomedical Engineering, Eindhoven University of Technology, The Netherlands

After cartilaginous bone tissue mineralizes in the embryo, the tissue is modeled to bone by bone-forming osteoblasts and bone-resorbing osteoclasts. This modeling process continues during growth, producing cortical shells and trabecular patterns that seem mechanically optimized in form, density and directionality, relative to the external forces. In maturity, bone is continuously remodeled through resorption and apposition of bone tissue at free trabecular surfaces and within the cortical shells, while maintaining structural optimality relative to external loads. Later in life trabeculae reduce in number and thickness, while cortices become thinner, when musculo-skeletal activities reduce.

The evident – though implicit – question for the scientific biomechanics community is whether effects of external forces on bones can be linked to local metabolism, so that they could be made predictable in a quantitative sense. From the mid-Eighties, several empiric theories to this end were proposed and used in computational models [1,2]. It was found, in serendipity, that the recursive formulas used in these models inherently cause trabeculation [3]. We put this to use in a new theory, by separation of 'actor' and 'sensor' functions [4]. Osteocytes within the trabeculae were considered as strain-energy-density (SED) sensors, signaling Basic Multicellular Units (BMU's) of osteoclasts and osteoblasts at trabecular surfaces to add or remove net bone mass [4].

We refined and unified the theory to include the separate activities of osteoclasts and osteoblasts in modeling & adaptation and in remodeling of trabecular structure [5,6]. We use dynamic loading variables (SED-rate) that activate osteocytes in the bone matrix to transfer osteoblast bone-formation stimuli to trabecular surfaces, through the canalicular network. The stimulus received at the surface depends on osteocyte density, mechano-sensitivity and signal decay by distance. Bone is formed at the surface where and while the stimulus exceeds a threshold value. Concurrently, osteoclasts are assumed to resorb bone which is (micro)damaged, the sites of which are determined at random per iteration. Coupling between osteoclast and osteoblast activities in remodeling is governed implicitly by the mechanics, through SED concentrations around resorption cavities, due to a notching effect. Applied in an FEA computer simulation of a bone cube, the theory produces a mature trabecular structure, aligned to the external loads [6,7], with morphological parameters similar to reality [8].

Hence, the strain-related signaling processes producing trabecular morphology in accordance with optimal mechanical resistance can now be understood. We have discovered recently that this trabecular computational theory can be unified to also describe remodeling processes in the cortical shells. These theories can now be applied for the investigation of osteoporotic processes, in search for preventive and curative agents. A complication is still that only small bone cubes can be investigated, due to limitations in computational capacities, but we expect that these limitations can be reduced with time.

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