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# Theories and computational models for strain-related bone remodeling and adaptation : validation and application

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# THEORIES AND COMPUTATIONAL MODELS FOR STRAIN-RELATED BONE REMODELING AND ADAPTATION: VALIDATION AND APPLICATION

**Rik Huiskes (1), Ronald Ruimerman (1), Esther Tanck (2)**

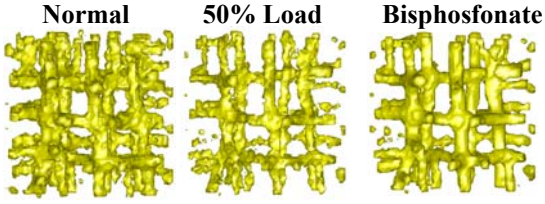
1. Dept. Biomedical Engineering, Eindhoven University of Technology, The Netherlands.
2. Orthopaedic Research Lab, University Nijmegen, The Netherlands.

After cartilaginous bone tissue has mineralized in the embryo, the tissue is modeled to bone by invading bone-forming osteoblasts and bone-resorbing osteoclasts. This *modeling* process continues during growth, producing trabecular patterns that seem mechanically optimized in density and directionality, relative to the external forces. In maturity, the trabeculae are continuously *remodeled* through resorption and apposition of bone tissue at free surfaces, repairing micro-cracks, while maintaining structural optimality relative to external loads. Later in life trabecular number and thickness gradually decrease, 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 link strain-related loading variables to loss or gain of bone density were proposed and used in computational models [e.g. 1,2]. Later it was found, in serendipity, that the recursive formulas used in these models inherently caused trabeculation [3]. We put it 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].

Later, we refined and unified the theory to include the separate activities of osteoclasts and osteoblasts in both *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].

This work will be discussed, also in relation to work from other groups [9,10,et al.] Important aspects are the mathematical representations used for local signaling pathways. A theory may produce realistic results, but that by itself does not proof validity. A second issue of importance is how these theoretical models can be made useful for clinical and basic research. As it is now, they predict the 'past' – that what we already know (validation.) What we want are revelations about the 'future' – that what we don't know.




**1**. Carter, 1987, *J Biomech;* **2**. Huiskes et al., 1987, *J Biomech*; **3**. Weinans et al., 1992, *J Biomech;* **4**. Mullender & Huiskes, 1995, *J Orthop Res*; **5**. Huiskes et al., 2000, *Nature* 405; **6**. Ruimerman et al., 2001, *Comp Meth in Biomech & Biomed Engr*; **7**. Ruimerman et al., 2004, *ESB2004 Poster*; **8**. Tanck et al., 2001, *Bone.* **9**. Langton et al., 2000, *Bone*; **10**. Adachi et al., 2001, *J Biomech Engr*.