

ModelingCafe

Wir möchten alle Interessenten recht herzlich zu unserem nächsten ModelingCafe einladen!

Spatial simulation of bone remodeling using a mathematical model

TOBIAS KORLUß¹, GERNOT SCHALLER², HAGEN DOMASCHKE³, ROBERT MÜLLER⁴,
HOLGER NEUBERT⁵

¹ Institut für Maschinenelemente und Maschinenkonstruktion, TU Dresden

² Institut für Theoretische Physik, TU Berlin

³ Klinik und Poliklinik für Kinder- und Jugendmedizin, TU Dresden

⁴ Institut für Werkstoffwissenschaft, TU Dresden

⁵ Institut für Feinwerktechnik und Elektronik-Design, TU Dresden

Bone consists of mineralized extracellular matrix and bone cells. Bone architecture is permanently optimized regarding to mechanical loads. This process is called bone remodeling and appears in multicellular structures called "basic multicellular units" (BMU). Two cell types are actively involved: Osteoclasts remove bone matrix in a concerted fashion, leaving lacunae and tunnels behind. Osteoblasts enter these resorption areas and produce new bone matrix. Initiation, progress and termination of BMU activity depends very much on communication between bone cells including autocrine and paracrine mechanisms. In a previous publication, a mathematical model of communication between osteoclasts and osteoblasts and the resulting activity of a single BMU was introduced [1]. The model used a system of ordinary differential equations to predict the population dynamics of the different cells.



We have adapted this model to a spatially extended simulation of bone remodeling by using a coupled set of partial differential equations describing the population dynamics and the elastic properties of the bone material. In contrast to the previous model [1], we also consider the back-action of a modified bone-density to the cellular dynamics. We have numerically solved the system by using the finite-difference method in combination with an implicit solver. The model reproduces some basic features of bone remodeling.

[1] Komarova SV, Smith RJ, Dixon SJ, Sims SM, Wahl LM.

Mathematical model predicts a critical role for osteoclast autocrine regulation in the control of bone remodeling. *Bone*. 2003 Aug;33(2):206-15.

18. April 2008, 14:00 Uhr

Informatik-Neubau, Nöthnitzer Str. 46, Kleiner Ratssaal 1005

Kontakt: Dr. Andreas Deutsch, andreas.deutsch@tu-dresden.de