8th. World Congress on Computational Mechanics (WCCM8)
5th European Congress on Computational Methods in Applied Sciences and Engineeering (ECCOMAS 2008)
June 30 –July 5, 2008
Venice, Italy

BMU steering and its influence on bone anisotropy

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Key Words: *BMU, targeted bone remodelling, anisotropy, damage, femur.*

ABSTRACT

A bone remodelling model has recently been developed [1] to take into account the effect of BMUs' progression direction in bone anisotropy. This model assumed that, once activated, BMUs progress parallel to the strain principal directions until apoptosis of their osteoclasts. Burger et al. [2] explained this hypothesis in the following way: apoptotic osteocytes and bone line cells generate signals for the nucleation and attraction of osteoclasts [3]. If BMUs are progressing parallel to the strain principal directions, osteocytes ahead of the resorption cavity sense no strain and receive no flow of nutrients through their canalicular network. This fact induces their apoptosis by the expression of Bax [4], a molecule that attracts osteoclasts to resorb the tissue in front of the resorption cavity, thus keeping the progression's direction constant.

Microstructural damage can also disrupt that flow of nutrients and cause the apoptosis of osteocytes. In a recent paper, Martin [5] has shown that microdamage is able to steer and attract existing BMUs as they continue to tunnel through the bone matrix. This way, damage repairing is more efficient than it would be if BMUs progress along the strain principal direction.

The model presented here establishes two competing mechanisms for BMU steering: following the strain principal direction and pointing to the damaged areas of bone. In both cases BMUs are driven by the signals expressed by apoptotic osteocytes. So, BMUs' progression direction in cortical bone is assumed to be a weighted average of the damage gradient, ∇d, and the maximum principal strain direction, $\mathbf{e}_{max}$, at the point where the BMUs is progressing

$$\mathbf{e} = k\,\frac{\nabla d}{\|\nabla d\|} + (1-k)\,\frac{\mathbf{e}_{max}}{\|\mathbf{e}_{max}\|} \tag{1}$$

Both directions have been normalized and weighted with the parameter k, which is defined as a function of the damage level and the modulus of the damage gradient:

$$k(d, \|\nabla d\|) = f_1(d) \cdot f_2(\|\nabla d\|) \tag{2}$$

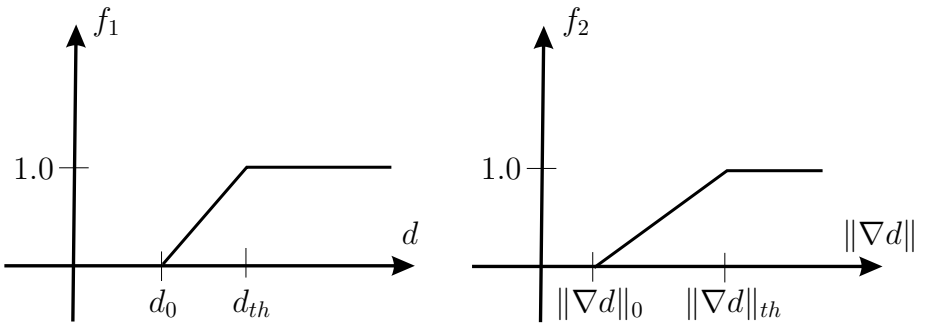

Figure 1: Functions f_1 and f_2 which together provide parameter k.

The functions f_1 and f_2 have been assumed piecewise linear as figure 1 shows. With that definition of k, only areas with a notable damage level will attract BMUs to repair it and only the BMUs closer to the damaged area will steer toward it.

The damage level is obtained in the previously cited bone remodelling model [1], through a balance of damage accumulation by fatigue and damage repairing, carried out by BMUs in the resorption phase.

The formulation presented in this work has been included in the previous model [1] to modifiy BMUs' progression direction in cortical bone. BMUs in trabecular bone do not tunnel through the bone matrix, but progress onto its surface and can not steer like osteons. So, no change has been made in the formulation regarding with trabecular bone.

The new model has been used to estimate the elastic constants of a human femur. Starting from a density distribution obtained numerically by Doblaré and García [6], assuming bone to be initially isotropic and applying loads corresponding to a one-legged stance, the temporal evolution of damage, anisotropy, elastic constants and progression's direction of BMUs were obtained.

REFERENCES

[1] J. Martínez-Reina, J. M. García-Aznar, J. Domínguez and M. Doblaré. "A bone remodelling model including the directional activity of BMUs". *Biomech. Model. Mechanobiol.*, (in press). DOI:10.1007/s10237-008-0122-5

[2] E. H. Burger, J. Klein-Nulend and T. H. Smit. "Strain-derived canalicular fluid flow regulates osteoclast activity in a remodelling osteon - a proposal". *J. Biomech.*, Vol. **36**, 1453–1459, 2003.

[3] W. D. Clark, E. L. Smith, K. A. Link, J. R. Paul-Murphy, P. Muir and M. E. Cook. "Osteocyte apoptosis and osteoclast presence in chicken radii 0-4 days following osteotomy". *Calcif. Tissue Int.*, Vol. **77**, 327–336, 2005.

[4] O. Verbogt, N. A. Tatton, R. J. Majeska and M. B. Schaffler. "Spatial distribution of Bax and Bcl-2 in osteocytes after bone fatigue: complementary roles in bone remodeling regulation?". *J. Bone Miner. Res.*, Vol. **17**, 907–914, 2002.

[5] R. B. Martin. "Targeted bone remodeling involves BMU steering as well as activation". *Bone*, Vol. **40**, 1574–1580, 2007.

[6] M. Doblaré and J. M. García. "Anisotropic bone remodelling model based on a continuum damage-repair theory". *J. Biomech.*, Vol. **35**, 1–17, 2002.