Simulation of Microcrack Growth and Repair in Living Bone

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Cracks form in bone due to daily loading activities, especially cyclic loading which causes fatigue cracking. Normally, cracks remain small (less than 1 mm long) thanks to a process of continual repair orchestrated by living cells. This balance between damage and repair is a vital element in maintaining the mechanical integrity of bone [1]. The published literature contains much experimental data on the numbers and lengths of these microcracks in bones examined ex vivo and after laboratory fatigue tests. However, there have been few attempts to model and describe fatigue crack growth in bone using fracture mechanics principles. The mechanism by which cracks are detected and repaired by cells is still poorly understood. The objective of the present work was to create a computer simulation incorporating two elements: (i) a model of microcrack initiation and growth, and; (ii) a model of crack detection and repair by cells.

In order to estimate crack initiation rates and the lengths of initiated cracks we analysed our own data and data obtained from the literature (e.g.[2,3]). We found that crack lengths conformed to a Weibull distribution, with values which are consistent with the spacings of microstructural barriers to growth, especially osteon cement lines. Figure 1 shows an example of this distribution. The number density of cracks per unit volume of bone was found to vary in predictable ways with cyclic stress level, number of cycles and other factors such as animal species and the existence of osteoporosis in the subject.

![](image)

Figure 1: An example of experimental data showing the distribution of crack lengths (a) in the form of a cumulative probability function P. The data can be accurately modeled as a Weibull distribution, using the equation shown.
This approach was incorporated into a computer simulation created using the Matlab Simulink software. Combining this information with a fracture mechanics model of crack growth, and introducing stochastic variables, allowed us to predict the number of cracks, and the length of each crack in a volume of bone subjected to cyclic stress, as a function of time, thus simulating the development of fatigue damage under normal and extreme loading conditions.

We also incorporated into the simulation a model whereby cracks are detected, and their lengths measured, by nearby osteocyte cells. This part of the simulation was based on our recently-proposed “scissors” model whereby the osteocyte network is disrupted by fracture of cellular processes which cross the crack faces [4,5]. This is envisaged to generate an inter-cell signal (via RANKL and other cytokines) to simulate the differentiation of osteoclast and osteoblast cells which subsequently act in concert to repair the cracked volume of bone.

The model can be used to investigate regimes of stability and instability as a function of various parameters, including the applied cyclic stress, material quality, microdamage burden and the responses of the cell signaling system. This allowed us to develop a model which is physically reasonable in that it remains stable under normal physiological conditions, and which demonstrates instability in crack length and density leading to the clinically observed phenomena of stress fractures (under extreme loading) and fragility fractures (when material quality is poor). Using this approach we can also investigate the effects of disease states such as osteoporosis and the effect of drug treatments, both of which tend to affect cell signaling levels and the balance of osteoclast/osteoblast activity.

References