

Invited Speaker Abstract

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Osteocytes and bone remodeling: They're just dying to do the job

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Healthy bone maintains a balance between wear and tear damage and intrinsic, matrix-level repair. Imbalance in this damage-repair homeostasis, either because of excessively rapid damage accumulation or because of ineffective, inadequate or inappropriate biological responses leads to pathology and ultimately mechanical failure of skeletal elements.

In bone, remodeling serves to remove and replace focal regions of tissue that have reached the end of their life span. How bone remodeling units "target" such effete microscopic areas of bone for replacement remains unclear. This talk will focus on our efforts to understand the cellular controls of targeting remodeling by exploring the remodeling responses that occur in responses to fatigue microdamage in vivo. We have found that fatigue damage leads to osteocyte apoptosis, or programmed cell death, in the immediate regions surrounding bone microcracks. These areas of apoptosis precede and co-localize with the area of subsequent osteoclastic resorption in fatigue bone. This has led to the hypothesis that osteocyte apoptosis may provide the activating or targeting signal for this remodeling. We confirmed this in recent pharmacological studies, which demonstrated that there is a causal relationship between osteocyte apoptosis at microdamage sites and the subsequent activation of bone remodeling. Implications of osteocyte apoptosis for bone remodeling activation in response to other challenges (e.g., estrogen loss, immobilization) will also be discussed.