2006 IOF+ANZBMS Combined Meeting

Combined Meeting of the
3rd IOF Asia-Pacific Regional Conference on Osteoporosis *and the*
16th Annual Meeting of the ANZ Bone & Mineral Society
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www.anzbms.org.au/asm/asm2006

SESSION TIME: 1500 - 1630, Tuesday 24 Oct 2006

Workshop Abstracts

Workshop C - Bone Quality: what is it, can it be measured and applied clinically?

W7

Bone microdamage and bone quality

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Low bone mass is not the only factor contributing to deceased bone strength. The accumulation of microdamage has been proposed as one factor that contributes to increased bone fragility with age and that may increase the risk for fracture in aging population. Microdamage is a manifestation of fatigue, which has been well recognized mode of material failure since long ago in the engineering field. In the fatigue process of materials, repetition of small load can cause microscopic physical damage, including cracks. The accumulation of microdamage increases fragility of the materials and finally leads to the material failure. However, in living skeleton, fractures never occurr in usual situation although repetitive load is applied to the bone as well. This is because fatigue is self repaired by physiological bone remodeling. The microdamage burden in bone is a function both of damage that is produced, and the amount that is repaired through normal physiologic remodeling process. Either increased production of damage, or suppressed repair, can elevate the level of microdamage in bone.

The fact that increased microdamage accumulation causes reduction of bone mechanical properties makes a bad impression for bone metabolic mechanism. However, bone microdamage plays an important role in accelerating bone turnover by being detected and repaired. Therefore, production or accumulation of microdamage is indispensable for fundamental bone metabolism. The other important role of microdamage is to avoid sudden stress concentration and to elongate fatigue life of the bone. Thus, microdamage in bone is not an independent factor related to bone quality but closely related to bone remodeling or mechanical environment.

In this session, the following data regarding dog studies will be mainly presented.

1. Relationship between microdamage accumulation and bone remodeling in association with their localizations in transverse section of rib
2. Effects of suppressed bone remodeling by bisphosphonates on microdamage accumulation and mechniacal properties of bone
3. Relationships between bone remodeling at ilium and bone rmodleing or microdamage accumulation at distant skeletal sites

W8

Material and structural determinants of bone strength

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The most important clinical manifestation of osteoporosis is a fracture, which occurs when the loads applied to a bone exceed its strength. To develop effective diagnostic tools and fracture prevention strategies, it is critical to understand both the loads that lead to fracture as well as the determinants of bone strength itself. Bone mineral density (BMD) measurements are very strongly associated with fracture risk and thus are presently the gold standard for diagnosing osteoporosis and predicting the risk of fracture. Yet, evidence is mounting to support the concept that characteristics in addition to, and perhaps independent of BMD, may be important in the pathophysiology of osteoporosis and in the mechanisms that underlie the antifracture effects of osteoporosis therapies. These observations show that 1) many fractures occur among patients with BMD values that are not "osteoporotic" by WHO criteria and 2) changes in BMD following antiresorptive therapy explain a small proportion of the variance in fracture risk reduction. This presentation will interpret some of these clinical observations in the context of basic principles of bone biomechanics. The ability of a bone to resist fracture is governed by its morphology (ie, its size and shape and microarchitecture) and by the intrinsic properties of the bone material itself (i.e., the extent to which the bone matrix is mineralized, and the amount and type of collagen). Bone remodeling is the process that affects changes in these structural and material characteristics. Thus, age-related changes, diseases, or therapies that influence any of these components, or that influence bone remodeling, will ultimately influence fracture risk. New techniques to assess bone strength non-invasively will also be discussed.

W9

The skeleton's tissue level response to mechanical demands

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The causes of many disorders not due to injury, cancer or infection are at the tissue level. Biologists separate anatomy, pathology and physiology according to the levels of biologic organisation. They speak of cell, tissue and organ levels, of organ systems and of the intact subject. The tissue level includes the physiology between cells through to organs, which takes part in organ function, properties, health and disorders including all the mechanisms, activities and functions that exist or arise in the tissue.

The most primary function of bone is to bear the mechanical load of everyday movement with subsidiary functions in plasma calcium homeostasis and supporting hematopoiesis. This paradigm has emerged from studies of the morphology and dynamics of bone cells and tissue modelling and remodelling, microdamage and biomechanical influence on bone adaptation. In simple biomechanical terms a bone will fracture if the load applied exceeds its strength.

Currently, it is clear that physiological strain continually produces fatigue induced microdamage in bone. This damage weakens bone and is associated with both the activation of remodelling and osteocyte apoptosis. Remodelling is the only known means by which this damage can be removed and repaired.

Bone is able to sense and adapt to its mechanical environment. The biological adaptive machinery includes modelling-dependent bone gain, remodelling-dependent bone loss and the detection of effective strain thresholds necessary to activate bone modelling or remodelling. Finally, changes in bone microstructure and geometry interact with each other in determining the overall tissue level mechanical properties.