Citation: IBMS BoneKEy **9,** Article number: 81 (2012) | doi:10.1038/bonekey.2012.81

www.nature.com/bonekey

MEETING REPORT

Bone and spine biomechanics

Elise F Morgan

Department of Mechanical Engineering, Orthopaedic and Developmental Biomechanics Laboratory, Boston University, Boston, MA, USA.

IBMS BoneKEy **9**, Article number: 81 (2012) | doi:10.1038/bonekey.2012.81; published online 2 May 2012

Meeting report from the 2012 Annual Meeting of the Orthopaedic Research Society, San Francisco, CA, USA, 4–7 February 2012.

The fields of bone mechanics and spine mechanics each involve a diverse set of research foci and are often discussed separately in forums on orthopedic research. However, over the past several years, there has been growing recognition of the interplay, both biological and biomechanical, between different tissues within an organ system or musculoskeletal system such as the spine. Important advances in treatment and prevention of many pathologies and injuries may thus arise through discussion of multiple aspects of these orthopedic systems. At the 2012 meeting of the Orthopaedic Research Society, a combined session on 'Bone and Spine Biomechanics' provided an opportunity for this type of discussion with a collection of presentations that spanned diagnosis and treatment of injuries and disease, identification of comorbidities, and study of mechanisms of failure and of mechanosensing.

The session began with a clinical study that defined the prevalence of what may be a very important risk factor or co-morbidity in osteoporotic vertebral fractures, sarcopenia. Hida *et al.*[1] measured body composition (bone mineral content, fat mass, and lean soft-tissue mass) in a group of 265 elderly patients with acute vertebral fractures and in 2154 non-fracture controls. The skeletal muscle mass index was calculated for the appendicular skeleton as well as for the lower limbs. Sarcopenia was defined as having a skeletal muscle mass index that is two standard deviations below the mean value for young, healthy individuals.[2] After adjusting for age, sex, and vertebral bone mineral density, the prevalence of sarcopenia was higher in the fracture than non-fracture group. Overall, the prevalence of sarcopenia was 49% in the fracture group and 31% in the non-fracture group. These data are important in that they are some of the first to report the higher prevalence of sarcopenia in individuals with vs without acute spine fracture. These data raise the question, as posed by one of the moderators, of whether osteopenia/osteoporosis and sarcopenia develop in concert with one another or if one more often precedes the other. Longitudinal data, such as could be obtained from the cohort of this study and of other studies on the spine[3] and hip,[4,5] are required to answer this question and to determine the contribution of sarcopenia to fracture risk in the spine.

Two other presentations focused specifically on the use of image-based finite element modeling to examine mechanisms of failure in bone and to estimate bone stiffness in the clinical setting. Sanyal *et al.*[6] used micro-finite element models of 54 cubes of trabecular bone from multiple anatomic sites to examine the shear strength and mechanisms of shear failure. As with compression and tension, the strength in shear was highly and non-linearly dependent on bone volume fraction. When the shear data were compared with the corresponding data from compression tests that were simulated on these same specimens, a pronounced difference in strength between the two loading modes was noted. On average, the shear strength was only 44% of the compressive strength. Examination of the predicted distributions of strains within the trabecular tissue indicated that shear loading induced tissue-level tensile failure primarily in obliquely oriented trabeculae, regardless of the volume fraction of the specimen. In comparison, a mixture of tensile and compressive tissue failure occurred under compressive loading, and the relative amounts of these two modes of failure appeared to vary with volume fraction. Overall, these data indicate that shear loading may be a weak link for trabecular bone as a result of the preponderance of tensile, and tissue-level stresses that this loading mode induces (in the tissue that is weaker in tension than compression). These findings further suggest that, if the obliquely oriented trabeculae provide the majority of the resistance to shear loading, loss of these relatively thin structural elements during aging may cause a dramatic drop in shear strength.

Different means by which bone volume fraction can decrease with age, the potential functional consequences of these decreases, and the feasibility of detecting these consequences in the clinical setting, were investigated in the study by Sun *et al*.[7] A 2% loss of bone mass via trabecular thinning or, separately, by perforation of trabeculae was simulated on micro-computed tomography images of the trabecular compartment of a region of human cadaveric distal tibiae ($n = 15$). The resulting images were then processed to resemble those that would be obtained by *in vivo* micro-magnetic resonance imaging (μMRI), and μMRI-based finite element models were created. Simulations of compression tests on these models produced detectable changes in stiffness as a consequence of bone loss, suggesting that *in vivo* μMRI, in combination with finite element analysis, may be able to provide patient-specific estimates of

changes in bone stiffness, even when those changes are relatively subtle. Of note, however, is that the simulation of bone loss via perforation predicted smaller changes in stiffness than those predicted by the simulations of bone loss via thinning. Prior studies on geometrically idealized models of trabecular bone have found the opposite ranking.[8–10] These differences in results may be due to differences in the amount of bone loss that was simulated, differences in the micromechanics of predominantly plate-like vs rod-like trabecular structures, and other effects of the differences in model architecture among the studies.

The session's final paper to use numerical simulation was a parametric study of solute transport through the osteocytic pericellular matrix (PCM) in bone canaliculi. Zhou and Wang[11] represented the PCM by a mesh of randomly oriented fibers that filled a cylindrical annulus representing the canaliculus. Diffusion of spherical solutes through the PCM was simulated using three-dimensional Monte-Carlo methods. Of the four parameters that were varied—solute size, fiber diameter, fiber length, and fiber volume fraction—solute diffusivity was found to be least sensitive to fiber length. Good fits to the simulation results were found by modifying an existing stochastic model[12] to include two additional parameters beyond solute size, fiber diameter, and fiber volume fraction. Development of simulation tools and analytical models to represent accurately and efficiently the transport of nutrients and pericellular fluid within the lacunar-canalicular network will no doubt benefit studies of bone mechanotransduction and osteocyte biology.

The remaining two studies in the session focused on the intervertebral disk. Antoniou *et al.*[13] examined the use of quantitative MRI for the detection of disk degeneration. In this proof-of-concept study, 10 cadaveric segments of the lumbar spine were imaged with MRI to measure the longitudinal magnetization recovery T1, the transverse magnetization decay T2, the magnetization transfer ratio, and the apparent diffusion coefficient. Cylindrical plus of the nucleus pulposis and annulus fibrosis were then harvested and subjected to mechanical testing in compression and shear. Several correlations between mechanical properties and MRI parameters were found. For instance, T2 was inversely correlated ($r = -0.465$) with the dynamic shear modulus in the nucleus pulposis, and T1 was inversely correlated ($r = -0.468$) with permeability in the annulus fibrosis. Although preliminary, these findings and others at the same meeting[14] suggest that quantitative MRI may in the future allow non-invasive assessment of the biomechanical health of the intervertebral disk.

Bookending the above study on diagnosis was an investigation on treatment of one of the most common symptoms of disk degeneration, lower back pain. Motivated by clinical evidence of pain relief following spinal motions and by some *in-vitro* evidence that small torsional movements within the spine column can depressurize the nucleus pulposis and may decrease load transmission across the facet joints, Mammoser *et al.*[15] quantified the effect of torsional movement on disk height *in vivo* in the lumbar spines of 81 human subjects. Disk height in five different zones of each disk (L1/2 through L5/S1) was measured as endplate-to-endplate distance, as quantified using CT scans performed while the spine was in a neutral position and, subsequently, in a 50° rightward rotation. Although rotation increased the overall average disk height, this change was only because of increases in disk height in the right and posterior regions. Disk height in the central region actually decreased. These findings do not support the hypothesis that torsional movement relieves lower back pain via depressurization of the nucleus and suggest that the mechanism of pain relief may instead involve configurational changes at the facet joints.

This collection of six studies served to illustrate the breadth of current research in bone and spine mechanics, and also to identify some key themes in these areas. Imaging, whether for diagnosis or study of mechanisms, was an integral part of nearly all of these studies, and the demand for better resolution and faster acquisition times continues to grow. Modeling and simulation were also well represented as equal partners to experiment in the array of research tools available to orthopedic scientists and engineers. With all of these tools, researchers will better armed for tackling the challenges of studying pathologies and injuries affecting multiple tissues within the bone and spine organ systems.

Conflict of Interest

The author declares no conflict of interest.

References

1. Hida T, Ishiguro N, Sakai Y, Matsui Y, Takemura M, Terabe Y *et al.* High prevalence of sarcopenia and decreased muscle mass in patients with osteoporotic vertebral fracture. Orthopaedic Research Society 2012 (Available at http://www.ors.org/abstracts/ paper 0121).
2. Baumgartner RN, Koehler KM, Gallagher D, Romero L, Heymsfield SB, Ross RR *et al.* Epidemiology of sarcopenia among the elderly in New Mexico. *Am J Epidemiol* 1998;**147**: 755–763.
3. Rodondi A, Chevalley T, Rizzoli R. Prevalence of vertebral fracture in oldest old nursing home residents. *Osteoporos Int* 2012 (e-pub ahead of print 1 February 2012; DOI: 10.1007/s00198-012-1900-6).
4. Fiatarone Singh MA, Singh NA, Hansen RD, Finnegan TP, Allen BJ, Diamond TH *et al.* Methodology and baseline characteristics for the Sarcopenia and Hip Fracture study: a 5-year prospective study. *J Gerontol A Biol Sci Med Sci* 2009;**64**:568–574.
5. Lloyd BD, Williamson DA, Singh NA, Hansen RD, Diamond TH, Finnegan TP *et al.* Recurrent and injurious falls in the year following hip fracture: a prospective study of incidence and risk factors from the Sarcopenia and Hip Fracture study. *J Gerontol A Biol Sci Med Sci* 2009;**64**:599–609.
6. Sanyal A, Gupta A, Bayraktar H, Kwon R, Keaveny T. Shear strength behavior of human trabecular bone. Orthopaedic Research Society 2012 (Available at http://www.ors.org/abstracts/ paper 0125).
7. Sun W, Rajapakse C, Wehrli F. Sensitivity of *in-vitro* micro-MRI-based biomechanics in detecting trabecular bone loss. Orthopaedic Research Society 2012 (Available at http://www.ors.org/ abstracts/ paper 0123).
8. Guo X, Kim C. Mechanical consequence of trabecular bone loss and its treatment: a three-dimensional model simulation. *Bone* 2002;**30**:404–411.
9. Silva M, Gibson L. Modeling the mechanical behavior of vertebral trabecular bone: effects of age-related changes in microstructure. *Bone* 1997;**21**:191–199.
10. Vajihala S, Kraynik A, Gibson L. A cellular solid model for modulus reduction due to resorption of trabeculae in bone. *J Biomech Eng* 2000;**122**:511–515.
11. Zhou X, Wang L. 3-D Monte-Carlo simulation on solute diffusion through pericellular matrix in bone canaliculi. Orthopaedic Research Society 2012 (Available at http://www.ors.org/abstracts/ paper 0124).
12. Ogston AG. On the transport of compact particles through solutions of chain polymers. *Pro R Soc London A* 1973;**333**:297–316.
13. Antoniou J, Epure LM, Michalek AJ, Iatridis J, Mwale F. Correlations between quantitative MRI and mechanical parameters of human intervertebral discs with different grades of degeneration. Orthopaedic Research Society 2012 (Available at http://www.ors.org/abstracts/ paper 0122).
14. Ellingson A, Mehta H, Santos E, Sembrano J, Polly Jr D, Nuckley D. Intervertebral disc MRI T2* mapping correlated with kinematic outcomes. Orthopaedic Research Society 2012 (Available at http://www.ors.org/abstracts/ paper 0066).
15. Mammoser N, Espinoza Orias AA, An H, Andersson GB, Inoue N. Effects of torsion on disc height distribution: an *in vivo* study. Orthopaedic Research Society 2012 (Available at http://www.ors.org/abstracts/ paper 0126).