Citation: IBMS BoneKEy **9,** Article number: 141 (2012) | doi:10.1038/bonekey.2012.141

www.nature.com/bonekey

MEETING REPORT

8th International Workshop for Musculoskeletal and Neuronal Interactions: ISMNI 2012

Peng-Fei Yang and Jörn Rittweger

Division Space Physiology, Institute of Aerospace Medicine, German Aerospace Center, Cologne, Germany.

IBMS BoneKEy **9**, Article number: 141 (2012) | doi:10.1038/ibmsbonekey.2012.141; published online 25 July 2012

Meeting Report from the 8th International Workshop for Musculoskeletal and Neuronal Interactions: ISMNI 2012, Ipswich, UK. 4–7 May, 2012

About ISMNI Meeting

This meeting is hosted biannually by the 'International Society for Musculoskeletal and Neuronal Interactions' (ISMNI). ISMNI meetings and the journal issued by this society (http://www.ismni.org/jmni/) are a forum for multidisciplinary research on the interface between nerves, muscles and bone. This Meeting Report summarizes some highlights of the 2012 meeting.

Participates and Presentations in 2012 ISMNI Meeting

This year's meeting attracted around 200 delegates, with a total of 29 invited presentations and 44 oral communications. The topics covered were basic muscle biology and physiology, bone biology, interactions between nerves, muscle and bone, tendon and cartilage, physical and pharmacological intervention, vibration therapy, and methodology development.

Conference Highlights

Muscle function and adaption. All skeletal muscles have the capability of modifying their structure by responding to the stimuli, for example training exercises. However, more and more evidence suggests that there are several factors, which might limit this ability of muscles (muscle plasticity). Hans Degens[1] (Manchester Metropolitan University, UK) summarized six main limiting factors, including modification of the myosin molecule, joint size, fibre-type transition, which all are likely to limit muscle plasticity. Further on, Marco Toigo (University of Zurich, Switzerland) reviewed the molecular mechanisms behind the muscles adaption to the environment changes and nutrition, especially during the aging process.

Bone growth. Given the fact of high incidence of distal radius fracture during puberty, Frank Rauch (Shriners Hospital for Children, Montreal, Canada) demonstrated a bone growth model based on the bone structure changes at metaphysis and diaphysis of peripheral long bone as an explanation. A large body of evidence suggests that the metaphyseal cortex of the distal radius remains thin and porous during the bone growth due to the rapid longitudinal bone growth. On the other hand, the required load resistance for the bone keeps increasing. The discrepancy between above two factors during puberty is very likely to be one of the most important reasons of high distal fracture risk in children.[2]

Muscle and bone loss induced by immobilization and the countermeasures. Daniel Belavý (Charité Universitätsmedizin Berlin, Germany) reviewed previous results in the adaptation of bone and muscle to the immobilisation in bed rest study. Evidence shows that the short duration high load-resistive exercise countermeasures, with or without vibration (3 days/week) is not able to completely prevent calf muscle atrophy in 60 days bed rest. Interestingly, their study also shows that the muscles adaptation to the countermeasures is site-specified, which means that muscle-specific exercises should be considered during the development of countermeasures.[3]

Besides the vibration training and the resistive training, the daily physical activities level was believed to be another factor, which affects bone health state. Rebecca M Meiring (University of Witwatersrand, South Africa) studied the ethnic difference and the effects of physical activity level on bone in early puberty. The results showed that high-level activities group did have greater lumbar vertebrae bone mineral content and whole-body bone mineral content. The ethnic difference in high-load group was not significant.

Mechanostat and mechanography. Mechanostat theory suggests that the bone development is supposed to be derived by related muscle growth. In the study of Elmar Anliker (University of Zurich, Switzerland), single leg hopping ground reaction force was used as an indicator of intrinsic muscle force of the lower leg. Furthermore, the relationship between the changes of maximum voluntary ground reaction force and the bone strength (volumetric bone mineral content at 14% site of lower leg) was assessed in 8–12-year-old children after a training protocol. Surprisingly, no correlations were found between the changes in maximum ground reaction force and the bone strength, which suggests that the adaptation process of the maximum muscle force and the bone strength might follow different time courses.[4] However, in a big population investigation, strong correlation does exist ($R^2 = 0.84$) between the maximum ground reaction force and the bone strength in 323 healthy 8–82-year-old males and females.[5]

To assess the role of muscle force on maintaining bone health, the upper limb seems to be the best site, as the bones in upper limb are not negotiating body mass. Alex Ireland (Manchester Metropolitan University, UK) studied the side differences in bone strength parameters of arms in elite junior tennis players. Side differences in total bone mineral content of striking magnitude were found at 4 and 60% distal–proximal radial length and 35% distal–proximal humeral length, respectively. These differences were entirely related to the larger cortical area. The biggest difference occurred at 35% sites in racquet arm humerus, up to a 'whacking' 39.8%. There were also side differences in muscle cross-section area, and a strong relationship existed between muscle and bone.

Jörn Rittweger (German Aerospace Center, Cologne, Germany) presented an old, but still highly controversial topic with new evidence concerning the effects of muscle strength training on the benefits of the bone. On the basis of observations made in bed rest studies and in training studies, respectively, muscle strength training seems to be effective in preventing immobilization-induced bone loss in legs.[6,7] However, training is not effective to change the leg bones in ambulatory people.[8] It seems clearly that there is an upper limit for bone strength, even though the mechanism behind is still not clear.[9,10]

Rainer Rawer (Novotec Medical GmbH, Pforzheim, Germany) highlighted the importance of appropriate corrections, for example for body weight, when reporting jumping mechanography results. For example, over the age, the ground reaction force during a single-legged jump and body mass change significantly, but strongly correlated. However, the ground reaction force normalized to body weight is almost constant and can be used as reference data.

Methodology in the study of bone microstructure. It has been well accepted that osteocytes, one of the most important and abundant cells in bone tissue, are responsible for mechanosensition and mechanotransduction of bone turnover. Recently, some evidence also showed that bone-lining cells is crucial for bone-remodelling process by helping the deposition of the first layer of collagenous in bone resorption pit.[11] However, Alan Boyde (Queen Mary, University of London, UK) challenged this view by drawing a surprising conclusion in his talk. By investigating the different methods of sampling processing for the scanning electron microscopical imaging, he was challenging the current notion of osteocytes being the central element in bone mechanotransduction. Even more critical, he highlighted that the bone-lining cells does not exist *in vivo*. What is observed in previous studies are artifacts induced by the adopted methodology. By comparing three different 3D imaging approach adopted widely in the study of microscopic level bone structure, T. Zikmund (Queen Mary, University of London, UK) demonstrated that scanning electron microscope is able to determine more details on trabecular bone structure than micro-computed tomography (μCT). To remedy that, a mathematical approach has been developed to improve the way of utilising μCT data to study the group differences of bone microstructure. This could be a new possibility to utilise μCT data.

Whole-body vibration. Botox-induced muscle paralysis model has been adopted to assess the effects of attenuated muscle force on bone health. Previous results showed that bone degradation was resulted by the muscle paralysis. With this model, Ralf Beccard (Children's Hospital, University of Cologne, Germany) demonstrated that whole-body vibration, with and without IGF-1 injection, which is essential for bone formation, was able to compensate the trabecular loss, but no effects on the cortical bone loss.

Conflict of Interest

The authors declare no conflict of interest.

References

1. Degens H. Age-related skeletal muscle dysfunction: causes and mechanisms. *J Musculoskelet Neuronal Interact* 2007;**7**:246–252.
2. Rauch F. The dynamics of bone structure development during pubertal growth. *J Musculoskelet Neuronal Interact* 2012;**12**:1–6.
3. Belavy DL, Miokovic T, Armbrecht G, Rittweger J, Felsenberg D. Resistive vibration exercise reduces lower limb muscle atrophy during 56-day bed-rest. *J Musculoskelet Neuronal Interact* 2009;**9**:225–235.
4. Anliker E, Dick C, Rawer R, Toigo M. Effects of jumping exercise on maximum ground reaction force and bone in 8- to 12-year-old boys and girls: a 9-month randomized controlled trial. *J Musculoskelet Neuronal Interact* 2012;**12**:56–67.
5. Anliker E, Rawer R, Boutellier U, Toigo M. Maximum ground reaction force in relation to tibial bone mass in children and adults. *Med Sci Sports Exerc* 2011;**43**:2102–2109.
6. Rittweger J, Beller G, Armbrecht G, Mulder E, Buehring B, Gast U *et al.* Prevention of bone loss during 56 days of strict bed rest by side-alternating resistive vibration exercise. *Bone* 2010;**46**:137–147.
7. Armbrecht G, Belavy DL, Gast U, Bongrazio M, Touby F, Beller G *et al.* Resistive vibration exercise attenuates bone and muscle atrophy in 56 days of bed rest: biochemical markers of bone metabolism. *Osteoporos Int* 2010;**21**:597–607.
8. Wilks DC, Winwood K, Gilliver SF, Kwiet A, Chatfield M, Michaelis I *et al.* Bone mass and geometry of the tibia and the radius of master sprinters, middle and long distance runners, race-walkers and sedentary control participants: a pQCT study. *Bone* 2009;**45**:91–97.
9. Robling AG. Is bone's response to mechanical signals dominated by muscle forces? *Med Sci Sports Exerc* 2009;**41**:2044–2049.
10. Judex S, Carlson KJ. Is bone's response to mechanical signals dominated by gravitational loading? *Med Sci Sports Exerc* 2009;**41**:2037–2043.
11. Everts V, Delaisse JM, Korper W, Jansen DC, Tigchelaar-Gutter W, Saftig P *et al.* The bone lining cell: its role in cleaning Howship's lacunae and initiating bone formation. *J Bone Miner Res* 2002;**17**:77–90.