Challenging our understanding of the musculoskeletal system

Citius, altius, fortius – performance of elite athletics – has always been the promoter for research in sports sciences. At the moment a great deal of attention has focused on ageing in the field of musculoskeletal system research because of elevated expected lifespan. Maintaining muscle mass seems to be critical for the elderly, because muscle atrophy is related to declined force production and fatigue resistance. Therefore, muscle strength is an important factor for the elderly to get through their daily activities. However, the present lifestyle does not necessarily favour elevated expected lifespan in the future, but rather increases the number of people who deal with various types of problems in their musculoskeletal system because of lack of physical activity or diseases.

The adaptability of skeletal muscle to changes in physical activity is remarkable, also in old age, as is reviewed by Narici et al. (2005) in this issue containing the second part of the review articles from the 6th Bispebjerg Symposium on Sports Medicine that convened at the Carlsberg Academy of Science on August 19–21, 2004, in Copenhagen. New evidence shows marked alterations of muscle architecture with ageing together with concomitant changes in tendon mechanical properties. These results suggest that not only sarcopenia, which has long been recognized as a major cause of muscle strength loss in old age, but also the changes in tendons are likely to affect the mechanical behavior of the muscle as a whole.

Mechanotransduction is cellular signal transduction in response to mechanical stimuli. Mechanical stress modulates almost all aspects of cell function, including growth, differentiation, migration, gene expression, protein synthesis, and apoptosis. Mechanical forces also directly affect the form and function of tissues. Soleus muscle is a good example of physiological tissue adaptation because of mechanical loading in the musculoskeletal system. Soleus muscle is a slow type of muscle, which has good capacity to maintain posture and to perform prolonged dynamic work. In soleus, collagen concentration and collagen cross-linking are higher than in fast contracting muscles mainly used during voluntary movements. Therefore, maintaining the balance between intracellular compounds and extracellular matrix is critical in order to meet the requirements in skeletal muscle because of altered physical loading. Both Huijing and Jaspers (2005) and Grounds et al. (2005) review their aspects of force transmission in skeletal muscle in the present issue.

Basement membrane is a specialized extracellular matrix, which is integrated within the connective tissue and contributes to force transmission together with the muscle cell membrane (sarcolemma) in order for force to be transferred to tendons. Connections between the key molecules in the internal structures of the myofibre (cytoskeleton and sarcomeres) and within the sarcolemma as well as outside the myofibre in the basement membrane are critical for successful force transmission. In exercised induced skeletal muscle fibre injury, this connection is partly disrupted. In this quite extreme case of tissue remodelling, type IV collagen immunostaining is still visible in the contour of basement membranes of injured fibres, as well as the endomysial staining for types I and III collagen. These injured muscle fibres are dystrophin negative, intracellularly fibronectin positive, and infiltrated by inflammatory cells. This is compatible with the view that collagen in skeletal muscle could potentially play an important role during the process of fibre damage when the cytoskeleton is disrupted, contractile proteins disorganized and extracellular fibronectin deposited intracellularly. In myopathies like Duchenne muscular dystrophy, a similar interruption in molecular force transmission chain as in exercised induced skeletal muscle fibre injury predisposes the cell membrane of skeletal muscle fibres to breakdown, and this may lead to necrosis. Overexpression of insulin-like growth factor-1 within dystrophic skeletal muscles of mdx mice or blocking activity of the pro-inflammatory cytokine tumor necrosis factor-α helps to protect the myofibres from necrosis (Grounds et al., 2005). Making muscles and tendons stronger during the whole lifespan is a highly relevant and challenging task in sports sciences.

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References

