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## Editorial

**Special Issue: Metabolic diseases and injuries of skeletal muscles: Current diagnosis, treatment and prevention**


Skeletal muscle is the largest organ in our body and essential for our daily functions. There are around 600 muscles in the human, where muscle mass constitutes 60–86% of body composition depending on the age. Therefore, any kind of injuries or problems in skeletal muscles will readily lead to functional impairment. Muscle injury can be caused by contusion, laceration, strain or degenerative problems. In general, muscle regeneration will last for 3–4 weeks depending on the injury severity. There is always a need to seek for interventions to accelerate the muscle healing.

Rotator cuff tear is a very common shoulder problem with pain and decreased motion that may occur due to both traumatic injuries in young patients as well as degenerative disease in older people. In this special issue, Liu et al. reviewed preclinical animal models for muscle regeneration after rotator cuff injury [5]. In addition, Peng et al. studied to apply a novel biodegradable high-purity magnesium suture for rotator cuff repair in sheep model [8]. These would give a new insight on the research of rotator cuff injuries and repair.

Ageing has become a global public concern, whereas musculoskeletal ageing is also a major health condition in orthopaedics. Musculoskeletal ageing may cause many muscle disorders, where sarcopenia is one of them. Sarcopenia is recognized as a disease and a new ICD-10-CM (M62.84) code has been assigned for it. A few organizations announced new diagnostic definitions for sarcopenia, such as European Working Group on Sarcopenia in Older People (EWGSOP), Asian Working Group for Sarcopenia (AWGS), etc., which catch much attention of researchers to study sarcopenia in recent decade [6]. The etiology of sarcopenia is multifactorial, whereas mitochondrial dysfunction is one of the recognized factors that needs more evidence to understand the mechanisms. Long et al. reviewed the changes of mitochondria in sarcopenia, including mitochondrial biogenesis, fusion, fission and mitophagy, as well as their dynamic equilibrium modulated by physical exercise [7]. Interestingly, Stephenson et al. explored the use of new settings of magnetic field to enhance mitochondrial bioenergetics in skeletal muscles clinically that showed encouraging results [10]. In addition, another study by Leucharusmee et al. also reported that ischemic preconditioning could upregulate mitofusin 2, a mitochondrial membrane protein, to preserve muscle strength [3].

Currently, there is no promising intervention to treat sarcopenia. The current gold standard interventions remain to be physical exercise and nutritional supplementation, yet the effects are not satisfactory due to

poor compliance. There is a pressing need to seek for new interventions. In this special issue, Dai et al. reported an innovative approach to use muscle satellite cells to inhibit uptake of extracellular vesicles derived from senescent bone marrow mesenchymal stem cells in order to attenuate sarcopenia [1]. Also, Yang et al. investigated new application of a phytomolecule, puerarin, to improve skeletal strength by regulating gut microbiota [11], which opens up a new area of research in management of sarcopenia.

In the meantime, there are more concerns on the relationship between sarcopenia and osteoporosis recently [2]. Muscle–bone relationship becomes a keen research area. Shi et al. showed that exercise accelerated the production of muscle-derived kynurenic acid in skeletal muscle to alleviate postmenopausal osteoporosis through Gpr35/NFκB p65 pathway [9]. Liang et al. also conducted a multi-center randomized, double-blind, double-dummy, positive-controlled clinical trial to demonstrate that a combination treatment of Jintiang (biomimetic drug made of artificial tiger bone) and alfacalcidol improved muscle strength and balance in primary osteoporosis [4]. Through these studies, it is expected to understand more about muscle–bone relationship.

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