that endurance exercise training results in a cardiac phenotype that protected against ischemia-reperfusion induced injury. The mechanism(s) to explain this exercise-induced cardioprotection appear to be multifactorial. Also, new evidence reveals that exercise training results in an altered skeletal muscle phenotype that is protected against disuse-induced muscle atrophy. Studies to explain the mechanism(s) responsible for this type of exercise-induced protection are in their infancy but emerging evidence indicates that exercise-induced increases in both heat shock protein 72 and superoxide dismutase 2 play an important role in protecting against inactivity-induced muscleatrophy.

Investigations into the mechanism(s) responsible for exercise-induced protection of skeletal muscles will assist in the identification of pharmacological targets to protect the heart against ischemia-reperfusion injury and to defend skeletal muscle fibers against muscle wasting.

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Exercise-induced biological effects of myokines in muscle: role of mitochondrial homeostasis

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Physical exercise improves skeletal muscle health benefits but also positively impacts many other tissues. These distal effects are largely mediated by numerous metabolites and peptides (myokines) released from skeletal muscle. Although, it has been described that myokines, for example IL-6 can exert both pro-and anti-inflammatory effects depending on the intensity and the manner of exercise. However, the mechanisms involved in regulation biological effects of IL-6 induced by exercise remain unclear. Our preliminary data have shown that exercise-induced mitochondrial homeostasis is actually required for optimal performance of skeletal muscle. In this presentation, we will introduce our recently works about the relationship between mitochondrial homeostasis and IL-6 biological effects in sarcopenia and exercise. In addition, we will describe the different effects of concentric and eccentric training programs on skeletal muscle mitochondrial homeostasis associated with different biological effects of IL-6. Furthermore, we will show cross-talk between PGC-1α and NFκB, and the potential handoff mechanism between the pro- and anti-inflammatory properties of the myokine IL-6.

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Role of redox signaling in muscle mitochondrial homeostasis

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Mitochondrion is an organelle that undergoes constant biogenesis and degradation controlled by signal transduction pathways in response to intracellular and extracellular signals. Mitochondrial biogenesis is regulated primarily by PGC-1α pathway under the influence of hormonal and neural inputs, as well as intracellular redox status. In skeletal muscle, denervation, gene knockout, immobilization and aging have been shown to severely impair PGC-1α signaling and hence mitochondrial density and function. Mitochondrial degradation is controlled by FoxO family transcription factors and mitochondrial fusion and fission dynamics. FoxO3a activation promotes mitochondrial autophagic-lysosomal pathway (mitophagy) and ubiquitin-proteosome pathway, whereas mitochondrial fragmentation due to imbalance of fusion and fission renders the organelle more vulnerable to mitophagy. It is noteworthy that a discord of mitochondrial dynamics and mitophagy may activate apoptosis, important for the maintenance of muscle mass and function. Finally, mitochondria participate in the regulation of reactive oxygen species (ROS) production and antioxidant gene expression and thus the intracellular redox status, providing a feedback control for redox signaling. Muscle immobilization activates all mitochondrial degradation pathways and imposes oxidative stress resulting in myocyte atrophy and functional deterioration, whereas exercise with proper mode and intensity promotes a healthy mitochondrial population. Finally, over-expression of PGC-1α via transgenic model or in vivo transfection has been shown to effectively promote mitochondrial biosynthesis and inhibit mitochondrial degradation thus ameliorating structural and functional integrity in atrophying muscle.

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