

Impact of different exercise modalities on mitophagy in human skeletal muscle

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Abstract

Exercise induces profound mitochondrial adaptations in skeletal muscle, with different modalities uniquely influencing different branches of mitochondrial quality control (MQC). This review examines how endurance, resistance, and high-intensity interval training (HIIT) regulate mitophagy, the selective degradation of damaged mitochondria, in skeletal muscle (SkM). Research in rodents has shown that endurance exercise upregulates mitophagy primarily through the AMPK/PGC-1 α signaling axis, promoting mitochondrial turnover and ensuring metabolic efficiency. In humans, high-intensity exercise increases mitophagy to a larger extent when compared to traditional endurance exercises. On the other hand, resistance exercise triggers alternative MQC mechanisms, including potential mitochondrial ejection. Collectively, these results suggest that mitophagy and MQC pathways are regulated in human