Autophagy and exercise-induced mitochondrial adaptions

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Non-communicable diseases, also known as chronic diseases, such as cardiovascular, metabolic and neurodegenerative diseases and cancer, account for 60% of the death and 75% of health care costs in America. On the other hand, it is undisputed that regular exercise has profound health benefits and is the most effective intervention in prevention and treatment of these chronic diseases. Extensive research has led to improved understanding of the benefits of regular exercise; however, much of the molecular mechanisms remain elusive. Recent development of molecular genetics and animal models of physiological exercise have significantly improved our understanding of mitochondrial adaptation in skeletal muscle. Mitochondria, the power plant of cells, are dynamic organelles that play critical roles in physiology, and the adaptive changes of mitochondria underscores the improved physical performance and the health benefits of exercise. Research in this laboratory has focused on two opposite processes: addition (mitochondrial biogenesis) and removal (mitophagy) of mitochondria in skeletal muscle. In particular, we have recently developed a novel mitochondrial reporter gene, MitoTimer, for mitochondrial quantity and quality assessment *in vivo*. We have found that a single bout of exercise activates mitophagy and exercise training improves mitochondrial quality and enhances mitophagy capacity. With this unique system, we are investigating the functional role mitophagy in exercise training-induced skeletal muscle adaptations. An improved understanding of mitochondrial maintenance and remodeling will facilitate the development of new interventions for numerous medical conditions.