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Opinion

Skeletal Muscle Adaptations to Exercise and Aging

Mehmet Arslan*

Department of Anatomy and Neuroscience, Anatolia Medical University, Ankara, Ankara Province, Türkiye

DESCRIPTION

Skeletal muscle is a highly dynamic and adaptable tissue that plays essential roles in movement, posture maintenance, energy metabolism and overall physiological health. Throughout life, skeletal muscle continuously responds to internal and external stimuli, including physical activity, nutrition, hormonal changes and aging related processes. Exercise represents one of the most powerful interventions capable of improving muscle structure and function, whereas aging is associated with progressive declines in muscle mass, strength and regenerative capacity. In my opinion, understanding the complex interaction between exercise-induced adaptations and age related changes in skeletal muscle is critical for developing effective strategies to promote healthy aging, prevent functional decline and enhance quality of life. Skeletal muscle adaptation to exercise occurs through a coordinated series of cellular and physiological responses. Resistance training, endurance exercise and high intensity interval training stimulate distinct but overlapping mechanisms that improve muscle performance. Resistance exercise primarily promotes muscle hypertrophy by increasing mechanical tension, activating anabolic signaling pathways, and stimulating muscle protein synthesis. The mammalian Target Of Rapamycin (mTOR) pathway plays a central role in regulating protein translation and muscle growth following exercise stimulation. Increased recruitment and activation of satellite cells, the resident muscle stem cells, further support muscle repair and remodeling by contributing new nuclei to growing muscle fibers.

Endurance exercise induces different but equally important adaptations. Repeated aerobic activity enhances mitochondrial biogenesis, oxidative enzyme activity, capillary density and fatty acid utilization, thereby improving muscle endurance and metabolic efficiency. These adaptations are largely regulated by signaling molecules such as peroxisome Proliferator Activated Receptor Gamma Coactivator 1-alpha (PGC-1α), which promotes mitochondrial development and energy metabolism. Aging, however, introduces significant challenges to skeletal muscle maintenance. One of the most prominent age related changes is sarcopenia, characterized by progressive loss of muscle mass, strength and functional capacity. Sarcopenia results from multiple interacting mechanisms, including reduced satellite cell activity, impaired protein synthesis, mitochondrial dysfunction, chronic inflammation, hormonal alterations and changes in neuromuscular communication. These processes collectively reduce muscle quality and increase vulnerability to falls, disability, metabolic disorders and reduced independence among older adults.

Aging related mitochondrial dysfunction is a major contributor to declining muscle performance. Older muscle fibers often exhibit reduced mitochondrial number, impaired energy production, increased oxidative stress and decreased ability to repair mitochondrial damage. Since skeletal muscle requires substantial energy for contraction and regeneration, mitochondrial impairment directly affects physical capacity and recovery following exercise. Emerging evidence suggests that exercise, particularly endurance and combined training approaches, can partially reverse mitochondrial decline by stimulating mitochondrial turnover and improving metabolic efficiency. Another important factor in age related muscle decline is impaired anabolic responsiveness, often referred to as anabolic resistance. Older individuals may require stronger nutritional and mechanical stimuli to achieve the same level of muscle protein synthesis observed in younger adults. Reduced sensitivity to amino acids, altered hormonal signaling and chronic low grade inflammation contribute to this phenomenon. Combining resistance exercise with adequate protein intake, particularly high quality sources rich in essential amino acids, represents an effective strategy for maintaining muscle health during aging. The interaction between exercise and immune function has also become an important area of skeletal muscle research. Contracting muscles release myokines, signaling molecules that influence inflammation, metabolism and communication between tissues. Exercise induced myokine production contributes to improved insulin sensitivity, reduced chronic inflammation and enhanced tissue repair. In contrast, aging is associated with increased inflammatory activity, often described as “inflammaging,” which can negatively affect muscle regeneration. Understanding this balance may provide new approaches for protecting muscle function in older populations.

Correspondence to: Mehmet Arslan, Department of Anatomy and Neuroscience, Anatolia Medical University, Ankara, Ankara Province, Türkiye, E-mail: mehmet.arslan@amu.edu.tr

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