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Research highlights

Insights into physiological mechanisms underlying symptoms of aging

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Sarcopenia refers to age-related loss of skeletal muscle mass characterized by a deterioration of muscle quantity and quality, which leads to a gradual slowing of movement, a decline in strength and power, increased risk of fall-related injury, and often, frailty.

Several possible candidates for modulating sarcopenia have been proposed, however, the precise contribution of each is unknown.

Now, Kunihiro Sakuma and colleagues at Toyohashi Tech have published a review article *Pflügers Archiv* on the age-related adaptation of positive and negative factors regulating sarcopenia.

This review describes the positive regulators such as mTOR- and SRF-dependent signaling, which modulate protein syntesis and mRNA transcripiton to enhance muscle hypertrophy. In addition, the authors discuss major negative signaling (UPS, autophagy, myostatin-Smad, NF-kappaB) to elicit protein breakdown resulting in muscle atrophy.

Fig.1: Functional defect of autophagy- and SRF-dependent signaling regulate sarcopenia.

The report highlights the fact that autophagy-dependent signaling, and not the UPS system, is destroyed in sarcopenic muscle. Although the UPS system, an activator of protein degradation in various catabolic conditions (i.e., immobilization), is believed to elicit the atrophy of muscle fiber during aging, this review concludes there to be no contribution to this.

Advances in our understanding of sarcopenia have led to new approaches, such as supplements, and pharmatheuticals, to attenuate the symptoms.

Reference:

Authors: Kunihiro Sakuma, Wataru Aoi, and Akihiko Yamaguchi.
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