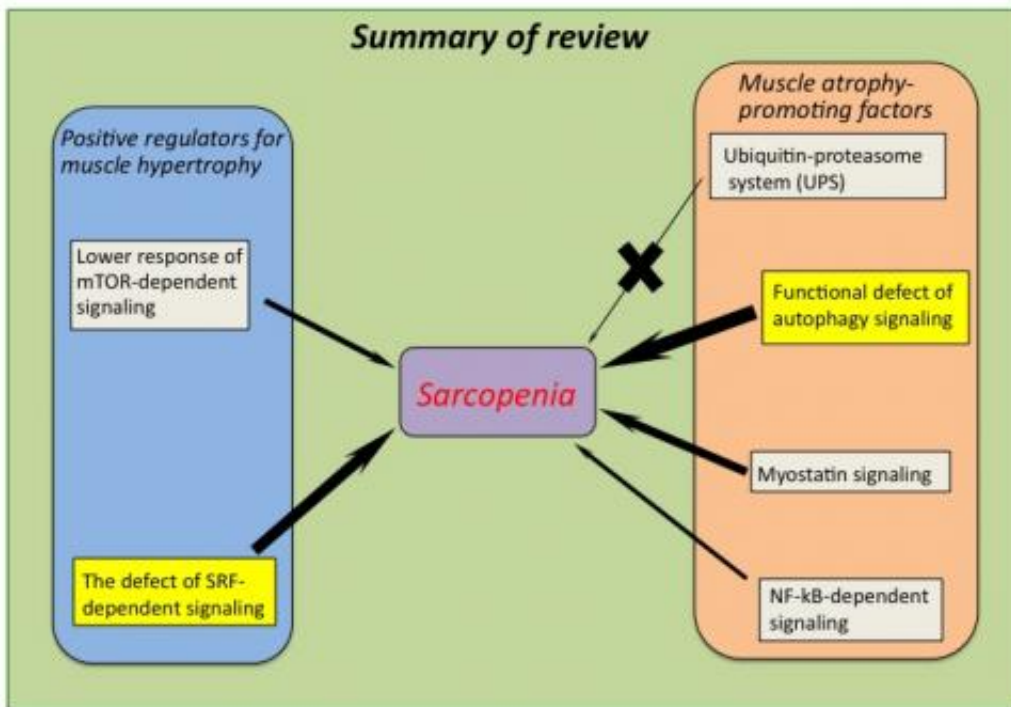


Insights into physiological mechanisms underlying symptoms of aging

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

Toyohashi Tech researchers have proposed a new perspective on possible candidates of muscle aging, which contributes to develop effective supplements or pharmaceuticals to attenuate aging.

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 on the age-related adaptation of positive and negative factors regulating sarcopenia in *Pflügers Archiv - European Journal of Physiology*, which is the oldest physiological journal in the world.

This review describes the positive regulators such as mTOR- and SRF-dependent signaling, which modulate protein synthesis and mRNA transcription 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](#).

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 pharmaceuticals, to attenuate the symptoms.

More information: Kunihiro Sakuma, Wataru Aoi, and Akihiko Yamaguchi. "Current understanding of sarcopenia: possible candidates

modulating muscle mass." *European Journal of Physiology*, Electrically published ahead of print (2014 May 7). [DOI: 10.1007/s00424-014-1527-x](https://doi.org/10.1007/s00424-014-1527-x)

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