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Mechanism and therapeutic application against skeletal muscle atrophy in aging and diseases.

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Musculoskeletal tissues undergo dramatic morphological and functional changes by aging, obesity, and muscular dystrophies. Atrophy of type II myofibers and ectopic fat formation are observed in aged muscles. Properties of satellite cells change by aging as well. Protein degradation also plays an important role in muscle strophy.

Recently, blocking myostatin, which is a skeletal muscle-specific TGF- β superfamily, was found to be effective to prevent various types of muscle atrophy including sarcopenia. Controlling of the activities of myostatin and/or activin is promising for treatment not only of muscle atrophy but also of obesity, fatty liver and even cachexia. Ectopic fat formation in skeletal muscle and other tissues is observed in muscular diseases, metabolic syndrome and aging. Origins of several types of adipogenic progenitors including intramuscular adipocytes, are clarified by sophisticated cell isolation technology. In this review, we will summarize the pathophysiology of muscle atrophy by aging and muscular diseases, and discuss about the origins and functions of adipogenic progenitors.

Key words: sarcopenia, muscle atrophy, myostatin, muscular disease, ectopic fat formation

