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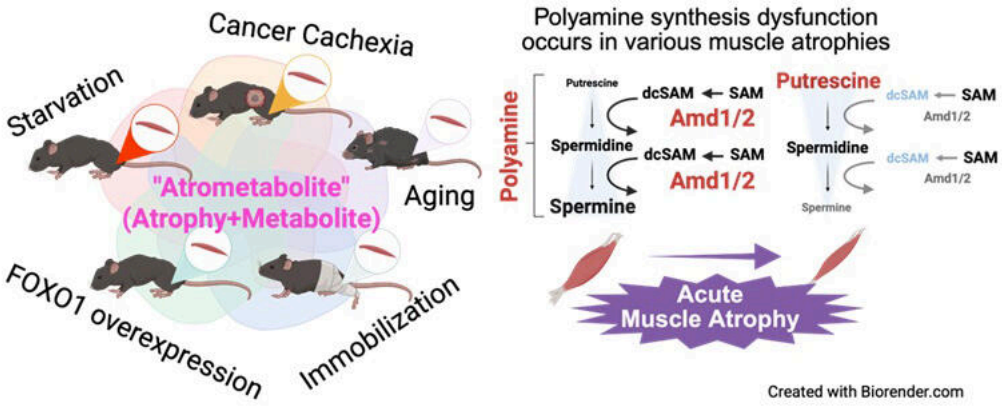
Kyoto Prefectural University hypothesizes that polyamine synthesis dysfunction in skeletal muscle may be a cause of muscle atrophy

2025.10.30

A collaborative research group led by Professor Yasutomi Kamei and Dr. Mamoru Oyabu (currently affiliated with the National Center for Geriatrics and Gerontology) from the Graduate School of Life and Environmental Sciences at Kyoto Prefectural University, along with researchers from the University of Shizuoka, the Institute for Research on Productive Aging (IRPA), Kyushu University, and Kumamoto University, announced the discovery through comprehensive metabolite analysis that metabolism undergoes significant changes in skeletal muscle during muscle atrophy and that dysfunction in the synthesis of a substance called polyamine in skeletal muscle is a characteristic feature of muscle atrophy. This finding is expected to lead to the development of new treatments for sarcopenia. The results were published in *Cell Reports* on August 6.



An overview of the research findings.
Provided by Kyoto Prefectural University

Muscle atrophy is caused by various factors such as aging, cancer, malnutrition, and inactivity, and has become a social issue as it leads to increased care dependency and medical costs. The decline in skeletal muscle mass and function associated with aging is called sarcopenia, and this increases the risk of falls, fractures, and the onset of dementia. However, it was not well understood what metabolic changes such muscle atrophy causes in skeletal muscle.

In this study, the research group conducted comprehensive metabolomic analysis of water-soluble metabolites in skeletal muscle using multiple mouse models including disease-induced muscle atrophy (cancer cachexia, immobilization, starvation, and aging).

The results confirmed that metabolism in skeletal muscle undergoes significant changes during muscle atrophy. Metabolites modulated by muscle atrophy were newly defined as "atrometabolites," and a dataset was

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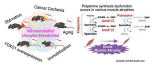
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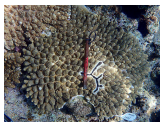
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Conversely, supplementation with polyamines from external sources significantly promoted protein synthesis. It was revealed that polyamines have high bioactivity in regulating muscle protein synthesis.

The study also identified that skeletal muscle cells atrophy and revealed that the FoxO family of transcription factors plays an important role during muscle atrophy.

In mice with skeletal muscle-specific FoxO deficiency, immobilization-induced muscle atrophy was suppressed, and metabolite changes were greatly reduced. This suggested that the FoxO family may be a central regulatory factor in metabolic changes.

Atrometabolites have the potential to function as indicators of muscle atrophy and reflect the state of muscles and functional decline. They are expected to be useful in elucidating the molecular mechanisms of muscle atrophy and developing diagnostic markers.

Kamei commented: "Maintaining the quantity and quality of skeletal muscle is important for health maintenance. However, muscle atrophy occurs due to various factors such as inactivity, malnutrition, diseases like cancer, and aging. In this study, we examined comprehensive metabolite changes in the skeletal muscle of mouse models of muscle atrophy (experimental animals) and found that polyamine metabolism is important for maintaining muscle mass. We will continue our research so that these findings can be utilized for the prevention and improvement of muscle atrophy."

Journal Information

Publication: *Cell Reports*
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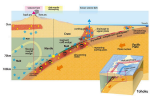

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