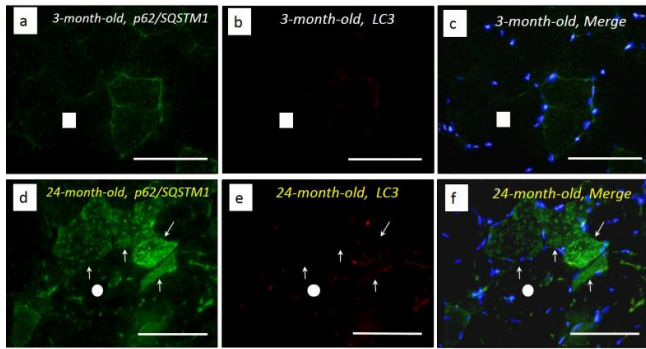


# Autophagy defect causes loss of muscle in aging

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Bar = 50 μm

P62/SQSTM1, but not LC3, is markedly expressed in the cytosol of muscle fibers of sarcopenic mice. Credit: (c) 2015 Toyohashi University Of Technology

Sarcopenia is the aging-related loss of skeletal muscle mass and strength. Preventing sarcopenia is important for maintaining a high quality of life (QOL) in the aged population. However, the molecular mechanism of sarcopenia has not yet been unraveled and is still a matter of debate.

Determining whether the levels of autophagy-related mediators (e.g., p62/SQSTM1, LC3, etc.) in [muscle](#) change with ageing is important to understanding [sarcopenia](#). Such information could enhance the therapeutic strategies for attenuating mammalian sarcopenia.

In previous studies, autophagic defects were detected in the sarcopenic muscle of mice, rats, and humans. However, all these studies involved only western blotting analyses of crude not cell-fractionated muscle homogenates. Thus, these data were insufficient to describe the adaptive changes in autophagy-linked molecules within sarcopenic muscle.

Associate Professor Kunihiro Sakuma and his colleagues at Toyohashi Tech found a marked accumulation of p62/SQSTM1 in the sarcopenic quadriceps muscle of mice using two different methods (western blotting of cell-fractionated homogenates and immunofluorescence). In contrast, the expression level of LC3, a partner of p62/SQSTM1 in autophagy progression, was not modulated.

The found autophagic defect improves our understanding of the mechanism underlying sarcopenia. The researchers would like to further study this mechanism with an aim to attenuate sarcopenia by improving this autophagic defect using nutrient- and pharmaceutical-based treatments.

**More information:** Kunihiro Sakuma<sup>1</sup>, Masakazu Kinoshita<sup>1</sup>, Yoshinori Ito<sup>1</sup>, Miki Aizawa<sup>1</sup>, Wataru Aoi<sup>2</sup>, and Akihiko Yamaguchi, p62/SQSTM1 but not LC3 is accumulated in sarcopenic muscle of mice, *Journal of Cachexia, Sarcopenia, and Muscle*, vol. 6, 2015 (in press)

Provided by Toyohashi University of Technology

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