New mechanism controlling proper organization of the muscle contractile units indentified

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Muscle-specific protein cofilin-2 controls the length of actin filaments in muscle cells.

Sliding of myosin and actin filaments past each other provides the force for muscle contraction. In contrast to most non-muscle cells, the actin filaments in muscle sarcomeres are of precise length and relatively stable. Defects in the organization of these actin filament arrays result in various heart and muscle disorders, such as myopathies.

The research group of professor Pekka Lappalainen at Institute of Biotechnology, University of Helsinki, has now revealed a new mechanism that is essential for the correct organization of sarcomeres.

"We discovered that a muscle-specific protein cofilin-2 'trims' the ends of sarcomeric actin filaments to control their precise length. To this end, cofilin-2 is specific in that it can also disassemble ATP-actin segments, which are expected to be present in the ends of sarcomeric actin filaments," Pekka Lappalainen explains.

These findings also explain why mutations in cofilin-2 gene result in nemaline myopathy.

Provided by University of Helsinki