Actin filament severing by cofilin.

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Keywords: actin, cytoskeleton, severing.

Abstract

The polymerization of the protein actin into helical filaments powers many eukaryotic cell movements and provides cells with mechanical strength and integrity. The actin regulatory protein, cofilin, promotes actin assembly dynamics by severing filaments and increasing the number of ends from which subunits add and dissociate. I will present results from biochemical and biophysical studies focused on defining in chemical and physical terms (thermodynamics, structure, mechanics and kinetics) how cofilin binds and fragments actin filaments. The experimental data are well described by a model in which discontinuities in filament topology and compliance promote fracture preferentially at junctions of bare and cofilin-decorated segments along filaments. The changes in filament mechanics and structure that drive severing originate from the linked dissociation of a single, site-specific cation. This work reveals that site-specific interactions with cations serve a key regulatory function in actin filament fragmentation and dynamics.