

How cells use chemistry and physics to break the bones that power their movement.



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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 how vertebrate cofilin binds and fragments actin filaments. The experimental data are well described by a model in which the cofilin-linked dissociation of filament-associated cations introduces discontinuities in filament topology and mechanical properties that promote fracture preferentially at junctions of bare and cofilin-decorated segments along filaments. Site-specific actin mutants support a cation-linked mechanism for vertebrate cofilin and demonstrate that filament severing is the essential function of cofilin in cells.