How cells break the bones that power their movement.

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

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3440 Molecular Sciences
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Please contact Marla Gonzalez, marla@chem.ucla.edu, x57071 for additional information.