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Stress Enhanced Gelation in α-Actinin-4 Cross-linked Actin Networks NORMAN YAO, Harvard University, CHASE BROEDERSZ, Princeton University, MARTIN DEPKEN, Vrije Universiteit, DANIEL BECKER, Brigham and Women's Hospital and Harvard Medical School, MARTIN POLLAK, Beth Israel Deaconess Medical Center and Harvard Medical School, FREDERICK MACKINTOSH, Vrije Universiteit, DAVID WEITZ, Harvard University — A hallmark of biopolymer networks is their exquisite sensitivity to stress, demonstrated for example, by pronounced nonlinear elastic stiffening. Typically, they also yield under increased static load, providing a mechanism to achieve fluid-like behavior. In this talk, I will demonstrate an unexpected dynamical behavior in biopolymer networks consisting of F-actin cross-linked by a physiological actin binding protein, α-Actinin-4. Applied stress actually enhances gelation of these networks by delaying the onset of structural relaxation and network flow, thereby extending the regime of solid-like behavior to much lower frequencies. By using human kidney disease-associated mutant cross-linkers with varying binding affinities, we propose a molecular origin for this stress-enhanced gelation: It arises from the increased binding affinity of the cross-linker under load, characteristic of catch-bond-like behavior. This property may have important biological implications for intracellular mechanics, representing as it does a qualitatively new class of material behavior.

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