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Effects of filament rigidity in myosin II-induced actin network contractility and dynamics KIMBERLY WEIRICH, MARGARET GARDEL, James Franck Institute, University of Chicago — Cells change shape, deforming to move and divide. The dynamic protein scaffold that shapes the cell is the cortex, a disordered, thin network of actin filaments. Random, local stresses generated by myosin II in the network create cellular-scale deformations. Myosin induced buckling and severing of actin filaments has been shown to underlie the contractility of two-dimensional disordered actin networks. This non-linear elastic response of actin filaments is thought to be an essential symmetry breaking mechanism to produce robust contractility in disordered actomyosin networks. To test this idea, we explore the effects of an actin bundling protein fascin, a crosslinker which induces polarity specific bundling of actin filaments, to create a network of F-actin bundles. We investigate myosin-induced stresses in a network of randomly oriented actin filaments, confined to a thin sheet at a supported lipid bilayer surface through a crowding agent. We find fascin-bundled filaments are less prone to filament buckling and show increased filament sliding, causing the myosin activity to induce network reorganization rather than contraction. Thus, changes in the filament bending rigidity in motor-filament systems can drive the system between distinct states with unique dynamic and mechanical signatures.

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