Shortening actin filaments cause force generation in actomyosin network to change from contractile to extensile. Nitin Kumar, Margaret Garidel, James Franck Institute, University of Chicago — Motor proteins in conjunction with filamentous proteins convert biochemical energy into mechanical energy which serves a number of cellular processes including cell motility, force generation and intracellular cargo transport. In-vitro experiments suggest that the forces generated by kinesin motors on microtubule bundles are extensile in nature whereas myosin motors on actin filaments are contractile. It is not clear how qualitatively similar systems can show completely different behaviors in terms of the nature of force generation. In order to answer this question, we carry out in vitro experiments where we form quasi 2D filamentous actomyosin networks and vary the length of actin filaments by adding capping protein. We show that when filaments are much shorter than their typical persistence length (approximately 10 microns), the forces generated are extensile and we see active nematic defect propagation, as seen in the microtubule-kinesin system. Based on this observation, we claim that the rigidity of rods plays an important role in dictating the nature of force generation in such systems. In order to understand this transition, we selectively label individual filaments and find that longer filaments show considerable bending and buckling, making them difficult to slide and extend along their length.

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