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Regulation of myosin II activity by actin architecture KIMBERLY WEIRICH, James Franck Institute, SAMANTHA STAM, Biophysical Sciences, PATRICK MCCALL, Physics, EDWIN MUNRO, Molecular Genetics and Cell Biology, MARGARET GARDEL, Physics and James Franck Institute, University of Chicago — Networks of actin filaments containing myosin II motors generate forces and motions that promote biological processes such as cell division, motility, and cargo transport. In cells, actin filaments are arranged in various structures from disordered meshworks to tight bundles. Clusters of myosin II motors, known as myosin filaments, crosslink and generate force on neighboring actin filaments. We hypothesized that the local actin architecture controls the magnitude and duration of force generated by myosin II motors. We used fluorescence imaging to directly measure the mobility of myosin II filaments on actin networks and bundles with varying actin filament polarity, orientation, spacing, and length. On unipolar bundles, myosin exhibits fast, unidirectional motion consistent with their unloaded gliding speed. On mixed polarity bundles, myosin speed is reduced by one order of magnitude and marked by direction switching and trapping. Increasing filament spacing and bundle flexibility reduces the duration of trapping and enhances the mobility of motors. Simulations indicate that stable trapping is a signature of large generated forces while increased mobility indicates force release. Our data underscore that the efficiency of force generation by myosin motors in an actin network depends sensitively on its architecture and suggests actin crosslinking proteins are tuned to optimize actomyosin contractility.

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