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Two distinct actin networks mediate traction oscillations to confer mechanosensitivity of focal adhesions ZHANGHAN WU, Natl Inst of Health - NIH, SERGEY PLOTNIKOV, Univ of Toronto, CLARE WATERMAN, JIAN LIU, Natl Inst of Health - NIH — Cells sense the mechanical stiffness of their extracellular matrix (ECM) by exerting traction force through focal adhesions (FAs), which are integrin-based protein assemblies. Strikingly, FA-mediated traction forces oscillate in time and space and govern durotaxis – the tendency of most cell types to migrate toward stiffer ECM. The underlying mechanism of this intriguing oscillation of FA traction force is unknown. Combing theory and experiment, we develop a model of FA growth, which integrates coordinated contributions of a branched actin network and stress fibers in the process. We show that retrograde flux of branched actin network contributes to a traction peak near the FA distal tip and that stress fiber-mediated actomyosin contractility generates a second traction peak near the FA center. Formin-mediated stress fiber elongation negatively feeds back with actomyosin contractility, resulting in the central traction peak oscillation. This underpins observed spatio-temporal patterns of the FA traction, and broadens the ECM stiffness range, over which FAs could accurately adapt with traction force generation. Our findings shed light on the fundamental mechanism of FA mechanosensing and hence durotaxis.

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