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Loss of an actin crosslinker uncouples cell spreading from cell stiffening on gels with a gradient of stiffness QI WEN, Department of Physics, University of Pennsylvania, FITZROY J. BYFIELD, Institute for Medicine and Engineering, University of Pennsyvania, KERSTIN NORDSTROM, Department of Physics, University of Pennsylvania, PAULO E. ARRATIA, Departments of Mechanical Engineering and Applied Mechanics, University of Pennsylvania, R.TYLER MILLER, Departments of Medicine and Physiology, Case-Western Reserve University, PAUL A. JANMEY, Institute for Medicine and Engineering, Departments of Physics, Engineering, University of Pennsylvania — We use microfluidics techniques to produce gels with a gradient of stiffness to show the essential function of the actin crosslinker filamin A in cell responses to mechanical stimuli. M2 melanoma cells null for filamin A do not alter their adherent area in response to increased substrate stiffness when they link to the substrate only through collagen receptors, but change adherent area normally when bound through fibronectin receptors. In contrast, filamin A-replete A7 cells change adherent area on both substrates and respond more strongly to collagen 1-coated gels than to fibronectin-coated gels. A7 cells alter their stiffness, as measured by atomic force microscopy, to match the elastic modulus of the substrate immediately adjacent to them on the gradient. M2 cells, in contrast, maintain a constant stiffness on all substrates that is as low as that of A7 cells on the softest gels achievable (1000 Pa). By contrasting the responses of these cell types to different adhesive substrates, cell spreading can be dissociated from stiffening.

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