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On the origins and extent of mechanical variation among cells JOHN MALONEY<sup>1</sup>, Massachusetts Institute of Technology, ERIC LEHNHARDT<sup>2</sup>, Arizona State University, KRYSTYN VAN VLIET<sup>3</sup>, Massachusetts Institute of Technology — Why would any one biological cell be mechanically different from another from the same population? Prompted by findings of broad distributions of cell stiffness within populations, we investigate possible origins of intrinsic mechanical heterogeneity among single cells. Through optical stretching, a non-contact technique for deforming cells in the suspended state, we obtain the creep compliance and complex modulus of single cells. Measurements of hundreds of human mesenchymal stem cells and murine fibroblasts in the time and frequency domains reveal that mechanical heterogeneity is not detectably dependent on cell lineage, cell cycle, cytoskeletal crosslinking, or repeated loading. However, adenosine triphosphate (ATP) depletion reduces heterogeneity of both stiffness and fluidity values. We explore the connection between these two parameters by positing that mechanical variation predominantly arises from Gaussian fluctuations in cell fluidity, which can be interpreted as emergent agitation in the energy landscape of soft glassy materials. Our findings ultimately link relatively small structural variations within cytoskeletal networks to large mechanical differences among cells and cell populations.

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