Abstract Submitted
for the MAR16 Meeting of
The American Physical Society

Rapid non-equilibrium turnover fluidizes entangled F-actin solutions PATRICK M. MCCALL, Dept of Physics, Univ of Chicago, DAVID R. KOVAR, Depts of MGCB and BMB, Univ of Chicago, MARGARET L. GARDEL, Dept of Physics, Univ of Chicago — The actin cytoskeleton of living cells is a semiflexible polymer network which regulates cell division, motility, and morphogenesis by controlling cell shape. These complex shape-changing processes require both mechanical deformation and remodeling of the actin cytoskeleton. Molecular motors generate internal forces to drive deformation, while cytoskeletal remodeling is regulated by non-equilibrium polymer turnover. Although the mechanical properties of equilibrium actin filament (F-actin) networks are well-described by theories of semiflexible polymers, these theories do not incorporate the effects of non-equilibrium turnover. To address this experimentally, we developed a model system in which both the turnover rate and the length distribution of purified F-actin can be tuned independently at steady-state through the combined action of actin regulatory proteins. Specifically we tune the concentrations of cofilin, profilin, and formin to regulate F-actin severing, recycling, and nucleation, respectively. We find that the actin turnover rate can be tuned by cofilin up to 25-fold (31 +/- 2 subunits/sec/filament). Surprisingly, changes in turnover rate have no effect on the steady-state F-actin length distribution, which is instead set by formin concentration. Passive microrheology measurements show that increased turnover leads to striking fluidization in both entangled and crosslinked networks. Non-equilibrium turnover thus enables modulation of network mechanics, which impacts force transmission and material deformation.

Patrick M. McCall
Department of Physics and the James Franck Institute, University of Chicago

Date submitted: 06 Nov 2015 Electronic form version 1.4