Nonequilibrium Mechanics of Active Cytoskeletal Networks
— from in vitro model system to cultured living cells —

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Mechanics directly controls many functions of cells: motion, force generation, and mechano-sensing. The cytoskeleton is a network of semiflexible filamentous proteins that is responsible for most of the mechanical functions of cells. One of the principal features of the cytoskeleton in vivo is its non-equilibrium character, due to mechanoenzymes (motor proteins). Prior in vitro studies, however, have focused on passive structures in equilibrium.

Here we show for the first time how non-equilibrium motor activity controls the mechanical properties of in vitro model of the cytoskeleton [1]. We applied both active and passive microrheology techniques [3] to a simple three-component system consisting of myosin II, actin filaments, and crosslinkers. The non-equilibrium origin of this active mechanical control was demonstrated by the violation of a fundamental principle/theorem of equilibrium statistical physics: the fluctuation-dissipation theorem. We show that nonequilibrium stresses arising from motor activity exquisitely controls cytoskeletal network mechanics: both increasing stiffness by nearly 100 times and qualitatively changing the viscoleastic response of the network in an ATP-dependent manner. We present a quantitative theoretical model connecting the large-scale

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properties of this active gel to molecular force generation.

We also present studies on intracellular mechanics of cultured fibroblasts, which show that our physical description of in vitro active cytoskeleton is applicable to in vivo cytoskeletons [2].

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References

