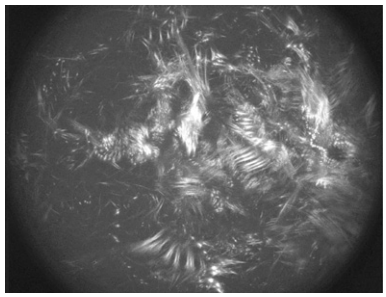
rearrangement processes of the actin cytoskeleton are key requirements. To shed light on the principles underlying these dynamic self organization processes we investigate a minimal reconstituted active system consisting of actin filaments, crosslinking molecules and molecular motor filaments.



By means of quantitative fluorescence microscopy and image analysis, we demonstrate, that these minimal systems exhibit a generic structure formation mechanism. The intricate interplay between force generation by molecular motors and the stabilization of the network by crosslinking proteins is identified to be responsible for the highly dynamic structure formation process. These competing mechanisms result in an anomalous transport dynamics, with a superdiffusive behavior also found in intracellular dynamics.

3511-Pos Board B372

Dynamics and Material Properties in Living Cells

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People have been studying the active motion in living cells for tens of years. Although, it is not well understood since people normally focus on the molecular motor induced directional motion. We show that the motor activity can also induce other type of motion. We observe that microinjected inert particle goes diffusive-like motion in living cells, with an effective diffusion coefficient invertly proportional to the particle size. This is usually thought as a signiture of the thermal induced motion in viscous medium. However, direct active measurement using optical tweezers with the same injected inert particle show that the cytoplasm responses to the extermal force almost elastically in a wide frequency range. The discrepancy suggests that the observed diffusive-like motion is due to the motor based active stress fluctuation in an elastic medium. Through the particle's motion, We quantitatively study the persistence of this diffusive-like motion and the power spectrum of motors in cells effectively.

3512-Pos Board B373

Mechanisms of Contractility and Coarsening of Active Cytoskeletal Networks

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Rapid structural remodeling of the cytoskeleton underlies many crucial processes such as cell polarization, division, and migration. Active remodeling involves the production and dissipation of forces within networks of actin filaments, molecular motors, and passive cross-linkers. However, despite its central importance, the underlying mechanisms remain unclear. A key challenge is to understand how global patterns of force buildup and dissipation during structural remodeling depend on the intrinsic properties of individual cytoskeletal components and their local interactions. To address this challenge, we have developed a three-dimensional agent-based computational model of a cross-linked actomyosin network with minimal components: actin filaments, passive cross-linkers, and active bipolar motors. Our model accounts for several key features neglected by previous studies, despite their likely significance for network remodeling including: volume-exclusion effects, the bending and thermal fluctuation of actin filaments, and the force-dependent unbinding and walking behaviors of the motors. Using the model, we systematically studied the influences of interplay between motors, cross-linkers, and actin dynamics on the contractility and structural dynamics of actomyosin networks. We found three different regimes determined by interplay between the sensitivity of motor unbinding to force and the stall-force level where motors cease walking: i) maximum tension and minimum deformation at low unbinding sensitivity, ii) intermediate tension and maximum deformation at intermediate sensitivity, iii) minimum tension and intermediate deformation at high sensitivity. We show further that passive cross-linkers can effectively tune networks between these regimes by maintaining percolation as well as by precluding large deformation of networks. Towards identifying mechanisms for rapid turnover of contractile foci, as observed in living cells, we are systematically investigating the effects of actin dynamics - assembly, disassembly, severing, capping, and aging - on the active contractile behaviors.

3513-Pos Board B374

Determinants of Fluid-Like Behavior and Effective Viscosity in Cross-Linked Actin Networks

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Actin cortex of animal cells exhibits distinct rheological behaviors at different timescales. At fast timescales (< a few seconds), the cortex exhibits solid-like resistance to internal or external forces, whereas at longer timescales (tens of seconds to minutes), it can behave like fluids, exhibiting long-range cortical flows that play key roles during polarization, motility, and cytokinesis. Origins of these fluid-like behaviors lie in the dynamics of actin networks cross-linked by motors and passive cross-linkers. Long-range flows imply an ability of networks to rapidly remodel while maintaining long-range connectivity, but how this balance is achieved is poorly understood. Previous studies have focused mainly on force-dependent cross-linker unbinding as a mechanism for fluidization, but the significance of network architecture and filament turnover has remained largely unexplored. Here, we use an agent-based computational model to systematically study how the fluid-like behavior depends on the interplay between network architecture and the turnover of cross-linkers and filaments. Our model actin network consists of passive cross-linkers and actin filaments that undergo treadmilling. Local filament disassembly also leads to cross-linker unbinding.

(1) We measured effective viscosity via simulated creep and stress relaxation tests. (2) We decomposed effective viscosity into contributions from ''internal effective viscosity'' due to dissipation of elastic energy within the network and external drag due to interactions with surrounding fluid. (3) We characterized how the internal effective viscosity varies with the density and turnover kinetics of cross-linkers, the length and bending/extensional stiffness of actin filaments, and the concentration and assembly/disassembly rates of actins. Significantly, we found that actin disassembly is much more efficient for fluidization than pure unbinding driven by cross-linkers.

3514-Pos Board B375

Mechanics of Dendritic Actin Networks

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The cytoskeleton is an interconnected network of filamentous polymers and regulatory proteins. The cell's integrity, deformability and shape changing abilities come from the mechanical properties of these organized assemblies. Both the filaments' flexibility and their relative organization give the assembly its mechanical properties.

Our work aims to link the mechanical properties of the actin networks in the cytoskeleton to their architecture, in terms of filament length and relative organization.

We have developed a simple novel experiment using the magnetic dipolar attraction between superparamagnetic colloids to probe the mechanical properties of actin networks.

Compared to previous studies on actin gel, this work has two main advantages. Firstly network growth occurs at the surface of the colloid particles using the actin binding proteins found in the lamellipodium, therefore the network has a structure and a density comparable to ones found in vivo. Secondly, in this technique each pair of colloids is a measurement device by itself allowing large amounts of data collection.

We exploit the capacity of our technique to collect huge amount of statistics to explore different gel architectures by tuning the concentrations of two proteins involved in the growth process, Arp2/3 and gelsolin. Those proteins respectively ensure nucleation of new filaments by branching and regulate branch length by capping growing filaments.

We first demonstrate that the rigidification of filaments themselves by phalloidin (actin filament stabilizing drug) increases the elastic modulus of the whole network by 65%. We then explore the effects of increasing capping and branching proteins and show that they both induce a strong stiffening of the network. We also study the evolution of the network mechanics with the growth time, which reflects the complex mechanical response of actin networks when actin is submitted to internal stress.