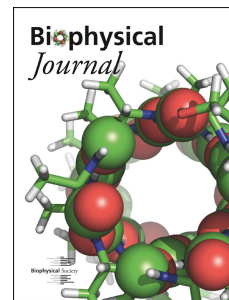


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Rethinking Contractility in Active Cytoskeletal Matter

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Introduction

Disordered actomyosin networks, such as those found in the cell cortex, generate contractile stresses without the crystalline, sarcomeric organization characteristic of muscle. These networks are active biological materials that operate far from thermodynamic equilibrium: molecular motors inject energy, crosslinkers transmit stresses and reorganize the network, and actin filaments continuously assemble and disassemble, providing dynamic tracks of varying lengths for motor activity. Contractile stresses emerge not simply from motor-driven actin filament sliding but from the complex interplay among these processes[1-4], which also continuously remodel the network architecture, maintaining stress[5-8]. How such a dynamic steady state both selects and maintains a contractile network remains largely unresolved. The turnover of the actin filaments is central to this puzzle. Beyond enabling structural reorganization, turnover is increasingly recognized as essential for stress generation itself[9-12]. However, most previous frameworks have treated turnover as spatially uniform, thereby overlooking the heterogeneous renewal patterns observed in cells, where distinct actin architectures coexist and interact within lamellipodia, lamellae, the cortex, and other regions[12-14]. The study by Tam, Mogilner, and Oelz moves beyond this simplification, demonstrating that the geometry of actin filament turnover, and not only its rate, plays a decisive role in governing how structure and contractility co-emerge in out-of-equilibrium actomyosin networks.

Defining the Bundling–Contractility Trade-Off

A central advance of the study is the quantitative definition of the trade-off between actin filament bundling and contractile stress. Earlier work showed that actomyosin networks lacking turnover initially generate contractile stress but eventually reorganize into bundles that lose contractile capacity[15, 16]. The present study formalizes this observation. Contractile stress peaks at intermediate bundling (Figure 1A): insufficient connectivity limits force transmission, whereas excessive bundling suppresses contraction. This nonlinear relationship reframes actin bundling

as neither inherently beneficial nor detrimental to contractility, but rather as defining a structural state within an active, nonequilibrium phase diagram (Figure 1B). In the language of nonequilibrium physics, disordered actomyosin networks transition between distinct regimes depending on connectivity and motor-driven activity. The bundling–contractility trade-off thus emerges as a structural control axis within this nonequilibrium phase space (Figure 1A).

Turnover Geometry as a Mechanical Control Parameter

The central finding of the study is the demonstration that the geometry of actin filament turnover governs how the bundling–contractility trade-off, and thus the network’s phase behavior, is resolved (Figure 1B). Four turnover modes are compared: uniform, biased, branching-mediated, and treadmilling. In the case of uniform turnover, where new filaments are added at random positions and orientations, filament bundling is disrupted, and long-term stress is sustained (Figure 1C). Biased turnover, characterized by the placement of new filaments near existing ones, reinforces bundling but accelerates contractile decay, ultimately stabilizing a network that is weakly contractile or non-contractile (Figure 1D). Branching-mediated turnover, in which new filaments are introduced at a 70° angle relative to existing ones, occupies an intermediate regime, limiting bundling while maintaining contractility (Figure 1E). Most strikingly, treadmilling, where filaments grow at one end and shrink at the other, reveals that actin filament disassembly can directly contribute to sustained stress generation (Figure 1F). Although bundling increases, contractility is preserved, weakening the negative feedback between filament alignment and contraction.

Integration with Mechanics: Coupled Control of Nonequilibrium Transitions

The study further integrates turnover geometry with mechanical parameters, showing that filament bending drives stress generation, while crosslink-mediated resistance regulates force transmission[17, 18]. Notably, the effects of these mechanical parameters are strongly influenced

by the turnover mode. For example, reduced flexural rigidity promotes filament bundling, but whether this shifts the network toward or away from a contractile state depends on turnover geometry. Similarly, increasing friction generally enhances contractility, yet its effect on bundling varies, especially under treadmilling. Together, turnover and mechanics jointly govern transitions between nonequilibrium network states.

Outlook: Toward a Unified Nonequilibrium Framework

Open questions remain. The model simplifies motor and crosslink kinetics, omitting effects such as motor pausing, force-dependent crosslink binding and unbinding, and heterogeneous stress transmission. Multiple actin filament populations coexist in cells, and distinct actin architectures in lamellipodia, lamellae, and the cortex may interact[12-14]. How combinations of uniform, biased, branching-mediated, and treadmilling turnover reshape active phase behavior remains unexplored. Future work could test these predictions through controlled reconstitution with defined nucleation geometries and by combining turnover modes during cytokinesis, migration, and tissue morphogenesis. On the theoretical side, incorporating turnover geometry into continuum active-gel descriptions would enable construction of a more complete nonequilibrium phase diagram that includes both motor activity and actin filament disassembly.

Conclusion

This study reveals that turnover geometry, rather than turnover rate alone, governs how disordered actomyosin networks balance the aggregation–contractility trade-off. More broadly, it reframes contractile stress generation as an emergent property of a dynamic, out-of-equilibrium steady state, where molecular motors, crosslinkers, and actin filament turnover act cooperatively. By integrating structural renewal into the framework of active phase behavior, the work advances a mechanistic understanding of force generation and opens a path toward a unified nonequilibrium theory of cytoskeletal mechanics.

Figure 1: Turnover geometry modulates the bundling–contractility trade-off in actomyosin networks. (a) Schematic representation of the trade-off between actin filament bundling and contractility. Peak contractility occurs at intermediate bundling levels. Arrows indicate how treadmilling turnover geometry shifts the peak, sustaining contractility despite variations in bundling. (b) Phase diagram showing contractility as a function of turnover mode and bundling. High and low contractile stress regions are highlighted, with markers denoting characteristic turnover modes. (c–f) Representative filament architectures under distinct turnover geometries: (c) Uniform turnover, (d) Biased turnover, (e) Branching-mediated turnover, and (f) Treadmilling turnover. Filament color corresponds to contractility levels as indicated by the heatmap in panel (b). In panel (c), black dots represent the positions of new filaments added at random. In panels (d) and (f), arrows indicate the directions of filament growth (and both growth and shrinkage in panel f). Panel (e) illustrates the Y-shaped branching structure formed by two branches growing from a single filament.

Declaration of Interests

The author declares no competing interests.

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