Computational Model of Cortical Actomyosin

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***Short Abstract* — Cortical actomyosin contractions have been implicated in a broad range of morphogenetic tissue movements. Actomyosin consists of two cytoskeletal proteins, filamentous actin (f-actin) and non-muscle myosin II. We consider the biomechanics of actomyosin, how force within the cell is produced, and how these forces remodel the actin cytoskeleton. We have constructed a 2D agent-based model representing a patch of cell cortex. We compare experimental actomyosin to our simulated 2D network in order to gain insight into the biophysical origin of pulsatile contractions, how intra-filament forces modulate f-actin array morphologies, and how these arrays drive cell shape and tissue morphogenesis.**

***Keywords* — actomyosin, Monte Carlo, agent-based modeling, cell mechanics, cytoskeleton function & dynamics**

I. Purpose

DYNAMIC actomyosin networks play a critical role in morphogenesis by providing forces to move cells and establishing tissue mechanics. For example, contractile actomyosin networks drive cell shape change, resulting in bending of epithelial sheets during *Drosophila* gastrulation [1], and are responsible for the viscoelasticity and force production in *Xenopus* embryonic tissues [2,3]. It is surprising that we know little about the biophysical connection between active, dynamic pulses in the actomyosin network, which occurs on the molecular level, and the mechanical processes of cell rearrangement and bulk movements on the tissue level.

Actomyosin dynamics have been studied *in vivo* utilizing fluorescently tagged f-actin and actomyosin targeting drug perturbation studies [4,5]. *In vitro* models use reconstituted gels [6] and micropatterned arrays [7] to understand the mechanical properties of cortical actomyosin and characterize biophysical properties. *In silico* models investigate the biophysical principles and processes leading to emergent behaviors of actomyosin arrays [8,9]. In the work presented here, we have built on our previous rotational model [10] to address the gaps of understanding dynamic actomyosin networks from the molecular to the cellular level. We have developed a two-dimensional model that incorporates dynamic aspects of *in vivo* actomyosin interactions, captures the observed behaviors of *in vitro* model system actomyosin, and lays the simplified groundwork for future efforts.

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II. Results

For physiologically relevant parameters, we observe the emergent morphology of actomyosin as a f-actin aster with a punctuated concentration of myosin II at the center. The aster morphology arises from an isotropic contraction within the actomyosin network, but is a stable morphology. In order to investigate ways of making the aster contraction dynamic, we performed a parametric analysis on the actomyosin biophysical parameters to identify key candidates in the regulation of aster emergence and disappearance.

To investigate *in silico* results, we simulated cases where f-actin plus ends were tethered into a bar geometry, and where myosin II were tethered to a specific location within the domain. As a further way of determining the specificity of various actomyosin morphologies as they relate to stress fiber generation, for example, we introduced a population of non-motile motors to serve as actin cross-linking proteins. The crosslinkers were able to interrupt the aster morphology from forming, and instead resulted in a ring of actomyosin.

III. Future Directions

The model has multiple applications, some of which will be discussed in this poster. For example, understanding the role of signaling in actomyosin biophysical properties, and predicting organization of actomyosin based on externally applied forces.

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