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# Quantification of individual myosin minifilaments in the cortex of the C. elegans zygote

Contractile forces generated in the actomyosin cortex of cells drive many different processes during cellular and tissue morphogenesis. In the cortex, Nonmuscle myosin motors assemble into bipolar minifilaments, crosslink actin filaments and exert forces on them. Physical descriptions of the cortex link the myosin activity to the cortical dynamics at the cellular level and singling pathways that regulate myosin activity, have been described. At the mesoscale, however, the mechanism in which the interaction of the minifilaments and actin filaments results in contractile forces is not clearly understood yet. This is mainly due to the limitations in direct observation of such interactions. Identifying the mesoscale properties of single minifilaments that vary in order to regulate levels of contractile forces at the cortex can lead to insights into the mechanism behind force generation. Thus, we sought for such properties. We utilised the cytokinetic ring of the C. elegans zygote, as a system with high spatiotemporal regulation of contractile forces.

We observed that there is a spacial redistribution of myosin at the cortex that manifests as the variation of both the size of minifilaments and their density. At the ring, this is achieved by nucleation of new minifilaments and their growth. By down-regulation of myosin activity, through let-502 RNAi, we observed a significant decay of cortical contractility which is linked with a decay in the density and size of the minifilaments and a delay in redistribution of myosin at the ring.

These results suggest that recruitment of more minifilaments at the cortex and more myosin motors in the minifilaments increases the levels of contractile force at the cortex. This is most probably by strengthening the ability of minifilaments to cross-link actin filaments.

**Primary author(s) :** RAFIEIAN, Fereshteh

**Presenter(s) :** RAFIEIAN, Fereshteh

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