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In vivo perturbation of active forces in the actomyosin cortex of C. elegans embryo

Morphogenesis is one of the fundamental processes during development. Critically, it depends on the generation of forces by the actomyosin cortex: a dynamic contractile apparatus that emerges from the cooperative interaction between actin networks and non-muscle myosin II (Alberts, B. et al., 2008, Philips, R. et al., 2012, Salbreux, G. et al., 2012). The past decades have brought a detailed understanding of how myosin generates force at the molecular level (Howard, J., 2001., Ishii, Y. et al., 2004). However, very little is known about the mechanisms by which myosin force generation leads to larger scale responses in cells and tissues.
To address this question, we genetically modify the myosin responsible for morphogenesis in *C. elegans*, NMY-2, with the aim to perturb the naturally occurring forces that drive the actomyosin cortex. NMY-2 is composed of a "head" domain at its N-terminus and a "tail" domain at its C-terminus. The head is the motor domain that hydrolises ATP and binds actin. The tail is required for dimerization and formation of myofilaments. The region between head and tail, called the "lever arm", is responsible for the conversion of the chemical energy into mechanical action. The length of the lever arm determines the distance myosin makes walking on actin filament per ATP hydrolyses, so called "step size". We hypothesize that altering the length of the lever arm would cause a change in the step size which in turn would alter the "power stroke" of myosin and the generated force.
To test this hypothesis, we have generated mutants of myosin that have a longer lever arm and expressed them in *C. elegans* embryos. Using this method, we can modulate active actomyosin tension and torque generation and assess the influence of myosin force generation on large scale response.

Primary author(s) : POLIC, Dora; Dr PERINI, Enrico D.; Prof. GRILL, Stephan

Presenter(s) : POLIC, Dora

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