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The actin nucleator CYK-1/mDia drives chirality of actomyosin flows and facilitates left-right symmetry breaking in early C. elegans embryo's.

The emergence of organismal left-right asymmetry has puzzled developmental biologists for decades. In recent years, actomyosin activity has proven to be instrumental in driving the chirality of various cells, tissues and organisms. While several myosin motors and actin nucleators of the Formin family can rotate helical actin filaments in vitro, little is known about how these activities can lead to the chirality of entire cellular actin networks in vivo. Recently, it was shown that the *C. elegans* actomyosin cortex generates active torques that drive chiral cortical flows and organismal left-right symmetry breaking. These chiral flows are dependent on the RhoA GTPase and its target non-muscle myosin II (NMII), but the underlying molecular mechanism remains elusive. By combining the strength of *C. elegans* genetics with quantitative live-imaging, we show that the activity of the Formin CYK-1/mDia is a key determinant for chiral morphogenesis of *C. elegans* embryos. Like NMII, CYK-1/mDia is recruited to active cortical RhoA patches. Moreover, loss of *cyk-1/mDia* prevents proper left-right symmetry breaking and, in sharp contrast to loss of the NMII complex, fully rescues the hyperchiral flow phenotype induced by ectopic RhoA activity. Finally, expression of a constitutively active CYK-1/mDia construct significantly increased the chiral component of the flow. These results imply that RhoA activates NMII to trigger overall actomyosin flow magnitude, while activation of CYK-1/mDia determines the strength of flow chirality. This is consistent with a mechanism in which active tension and torque generation in the actomyosin layer are molecularly distinct and driven by NMII and CYK-1/mDia respectively.

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