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Actin binding proteins govern the range of polarizing cortical flows in *C. elegans* zygotes

Establishment of polarity is essential for conferring different developmental fates to the dividing cells of an embryo. In *Caenorhabditis elegans* one cell embryos, antero-posterior polarization is facilitated by long-ranged flow of the actomyosin cortex. Even though the flowing cortex contains many actin binding proteins (ABPs) that contribute to its structure and dynamics, there are only a limited number of mechanical properties that are important at large length and time scales relevant for polarization, for example contractility and cortical viscosity (Mayer, Bois, Depken, Jülicher, Grill, 2010). Importantly, this suggests that there is only a reduced spectrum of cortical flow phenotypes that one might expect to obtain by modulating these few mechanical properties through different molecular mechanisms. To bridge the gap between molecular and cellular scales, we here sought to investigate which cell-scale mechanical properties are controlled by which ABPs. We devised a candidate RNAi screen of ABPs and found that several ABPs affect cortical flow. This was achieved by analyzing myosin foci size and density and several flow characteristics, such as peak velocities and spatio-temporal velocity-velocity correlations, for each ABP knockdown. The velocity-velocity correlations provided us with an estimation of the characteristic hydrodynamic length of cortical flow, which describes the extent to which flows are long-ranged. Interestingly, all those ABPs that displayed a detectable cortical flow phenotype did so through affecting this hydrodynamic length. RNAi either resulted in short-ranged flows, indicative of a less viscous cortex, or it resulted in flows that were longer-ranged than wild type, indicative of a cortex that is more viscous that under wild-type conditions. Our results suggest that the characteristic hydrodynamic length is a central physical property subject to precise regulation. They also point towards a type of "mechanical redundancy" in animal development, with many molecular mechanisms affecting the same cell-scale physical property.