

P003 Moesin controls cortical rigidity, cell rounding and spindle morphogenesis during mitosis

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Animal cells undergo a complex sequence of morphological changes as they pass through mitosis, including retraction of the cell margin, cell rounding, axial elongation at anaphase and cytokinesis. Here we identify DMoesin, the sole *Drosophila* member of the ERM family of proteins, which crosslink actin filaments to the cytoplasmic tails of plasma membrane proteins, as a key regulator of these changes in mitotic cell shape. We show that DMoesin becomes activated and cortically localized following Slik-dependent phosphorylation at mitotic onset. This cortical pool of active Moesin induces cortical stiffening and cell rounding, and is sufficient to induce both processes in interphase cells, independently of Myosin II. Thus, in the absence of Moesin, cells remain soft and flat throughout mitosis. Strikingly, these soft Moesin RNAi cells exhibit profound defects in spindle morphogenesis and chromosome alignment. Finally, we show that these defects can be rescued by re-establishing cortical tension from the outside the cell. These observations reveal the importance of cortical stiffening and cell rounding as a prelude to mitosis in animal cells, and in doing so help to explain its universality.