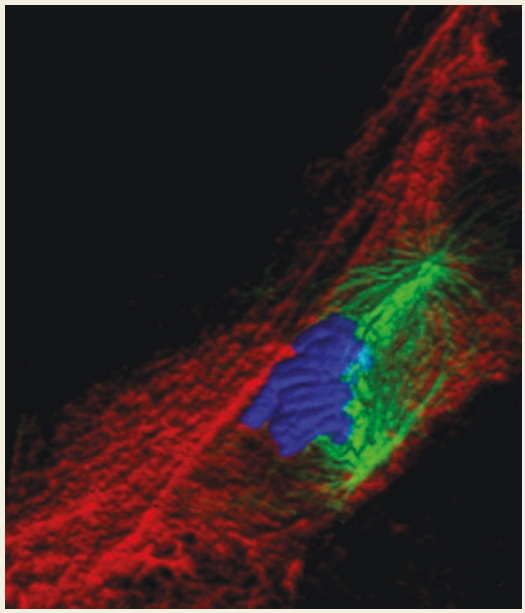
Centrosome migration: until myosin drags us apart



Myosin-dependent cortical flow directs spindle assembly. Blocking cortical movement by crosslinking the cell cortex with the tetravalent lectin WGA results in formation of a 'lopsided' spindle. Image kindly provided by Jody Rosenblatt, UCL, London.

A previously unappreciated requirement for cortical actin and myosin II in spindle assembly has been discovered by Jody Rosenblatt and colleagues (*Cell* **117**, 361–372; 2004). While studies so far have emphasized the function of microtubules and their associated motors in setting up the spindle — relegating myosin II to the subsequent task of cytokinesis — the new work shows that myosin II acting in the cell cortex drives the completion of centrosome separation after breakdown of the nuclear envelope.

The astral microtubules emanating from the centrosomes are known to contact the cortex at the time when centrosomes migrate apart, a key step in spindle assembly. Rosenblatt and colleagues set out to re-examine the significance of this attachment. They found that if they disrupted actin polymerization, or more specifically the activity of myosin II, centrosome separation halted just at the time when the nuclear envelope — known to guide migrating centrosomes — breaks down.

Although myosin II is found predominantly at the cortex during prophase, a smaller amount also lurks in the cytoplasm. So, to verify whether centrosomes require myosin II in the cortex, the authors asked whether they could recapitulate these defects in spindle assembly by crosslinking the cell cortex with lectins. They could, and again the requirement for cortical mobility seemed to be restricted to the period after nuclear envelope breakdown.

So how might cortical movement aid centrosome separation? To address this, Rosenblatt and colleagues used fluorescent latex beads to visualize cortical movement directly. They saw that there is a striking correlation between movement of the cortex during mitosis and the direction of centrosome migration. This cortical flow also depended on myosin II activity. Because the cortex appears to contract on the opposite side of the cell to where the centrosomes are, they speculate that myosin II activity might be required to drive centrosome separation by exerting a pulling force on the cortex.

It is surprising that this role for cortical myosin not been realized before. However, many previous studies were performed in organisms such as yeast and slime mould, in which the nuclear envelope does not break down during mitosis. As the guidance cue provided by the nuclear envelope is never lost in these systems, the spindle can form correctly in the absence of cortical instruction.

One challenge now will be to work out the mechanism by which cortical flow drives centrosome separation, and how the cell knows which direction to move the centrosomes in and when to stop. On the basis of the evidence so far, the authors speculate that astral microtubules might suppress myosin-based contraction at the cortex upon contact, so that contraction occurring at the other side of the cell then triggers cortical flow in that direction. Testing this should provide new insights, not only into the mechanisms driving spindle assembly but also the molecular basis for crosstalk between actin and microtubule networks.

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