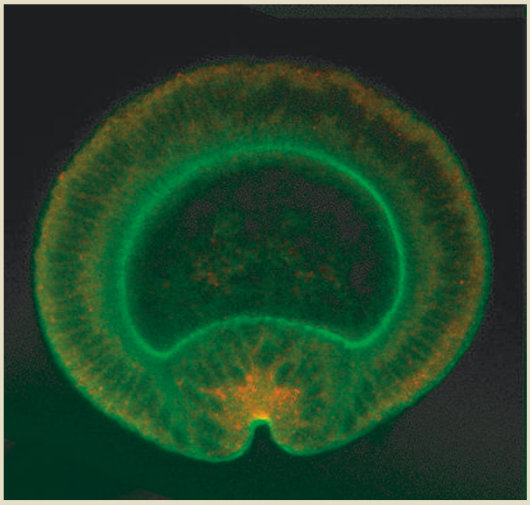
Fog shapes up myosin for gastrulation



Fog (shown in red) localizes to the apical surface of ventral cells, where it coordinates gastrulation through effects on myosin. Image courtesy of Rachel Dawes-Hoang.

The opening act of gastrulation begins with an orchestrated series of cell shape changes at the ventral surface of the embryo, culminating in ventral furrow formation. The *folded gastrulation* (*fog*) gene encodes a secreted protein that choreographs these cell movements, and which is itself under the control of patterning genes. Rachel Dawes-Hoang and colleagues (*Development* **132,** 4165; 2005) now show that *fog* drives localization of myosin II during gastrulation, providing a link between patterning control and the cytoskeletal machinery that controls cell shape.

Fog is needed for the cell shape changes at the ventral surface, but how it affects the cytoskeletal apparatus coordinating this process is unclear. Dawes-Hoang *et al.* reasoned that non-muscle myosin II might be one candidate target for fog, because it undergoes a dynamic relocalization to the apical surface of ventral cells as gastrulation initiates. The first hint that this might indeed be the case was the observation that fog also localizes to the apical surface of ventral cells. They went on to show that in the absence of fog, myosin II does not redistribute to the apical surface of cells. Not only was fog important for myosin II localization but it was sufficient: driving fog expression in more lateral cells also resulted in apical localization of myosin II and delayed ventral furrow formation. Myosin II localization to the apical surface also required its ability to bind actin and contract, suggesting that fog acts on active myosin. In addition, two effectors for cytoskeletal signalling, Rho-GEF2 and ROCK, contribute to myosin II localization.

So what are the consequences of localizing fog and myosin II to the apical surface of cells? Using scanning electron microscopy, the authors found that the apical cell surface becomes flattened when fog is overexpressed. Moreover, ectopic fog expression appears to correlate with an abnormal shift of adherens junctions to the apical surface. Using two genetic approaches to disrupt junction formation, they see that these junctions are not required for initial localization of myosin II to the apical surface but that in their absence, myosin contraction results in an abnormal actin network. So it seems that myosin needs to be tethered by the apical junctions in order to exert force on the plasma membrane and to elicit cell shape changes.

Taken together, it seems that the initial polarizing signal that drives myosin II activation at the apical surface is fog signalling, and not the adhesion junctions. Thus, expression of a secreted protein through the action of patterning genes can control the actin cytoskeleton. But how direct is this effect? The key now is to ask how fog signalling controls myosin II recruitment and how it affects the organization of adhesion junctions to coordinate gastrulation.

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