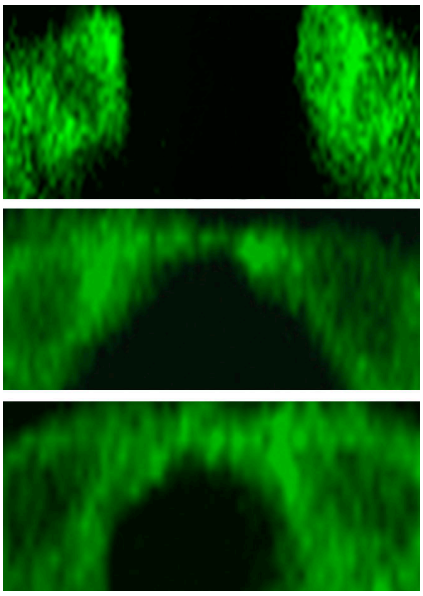
In This Issue



***Drosophila* cardioblasts meet and bend into a tube.**

UNTYING TUBE FORMATION

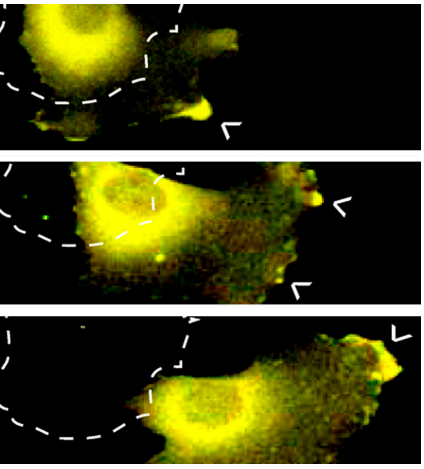
From the trachea to the capillaries of the retina, the body teems with tubes. Even the heart is a glorified tube. Two papers have identified a pathway that helps tube cells create an opening.

Building a tube from solid tissue involves making space between adjacent cells to form a lumen. The two groups recorded similar results about the process, but they started with different goals. Medioni et al. wanted to determine the changes in cell shape and polarity, whereas Santiago-Martínez et al. wanted to know how cells modify their stickiness so they can separate.

Both teams took a close look at the embryonic *Drosophila* heart, which forms when two rows of cardioblasts converge and flex to produce a hollow cylinder. Medioni et al. performed live imaging with confocal microscopy to follow this cellular choreography, and Santiago-Martínez et al. captured three stages of the process with EM. The groups observed the same changes. Cardioblasts in opposite rows first attach at the top. They then bow outward into a sickle shape and connect at the bottom, leaving a doughnut hole in the middle. The two studies also reached similar conclusions about a pathway that involves the extracellular matrix protein Slit and its receptor, Robo. In effect, the pathway creates a non-stick surface on the lumen side of heart cells.

Santiago-Martínez et al. think that the Slit/Robo pathway works by exiling the protein E-cadherin, which hooks neighboring cells together, from the cells' future lumen surface. Medioni et al. found that cardioblasts with mutant Slit remain round and display an expanded cell-to-cell adhesion domain that holds the lumen closed.

Medioni, C., et al. 2008. *J. Cell Biol.* doi:10.1083/jcb.200801100.
Santiago-Martínez, E., et al. 2008. *J. Cell Biol.* doi:10.1083/jcb.200804120.



Gαi3 (yellow) clusters at the leading edge (arrows) of a crawling cell.

DRIVEN TO THE BRINK BY A G PROTEIN

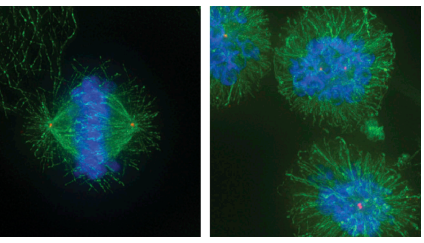
Missing links aren't just for paleontologists. Ghosh et al. report what might be the long-sought connection between cell surface receptors and the direction in which cells crawl.

Nearby food and growth factors galvanize a cell. At the section of the membrane nearest the stimulus, activity of the signaling molecule Akt cranks up and actin elongates into stress fibers essential for crawling. The cell then pushes forward this part of its membrane, the leading edge. Surface receptors first detect the stimulus, and then trigger G proteins, which pass the signal on. What scientists don't know is how cells confine the molecular action to the leading edge. The team suspected it might involve an intermediary, the protein GIV, which can latch onto G proteins and stimulate Akt.

To find out, Ghosh et al. investigated the interaction between GIV and a G protein component known as Gαi3. If Gαi3 is absent, the team found, actin doesn't extend, Akt activity doesn't rev up, and cells are stuck. Gαi3 homes in on the leading edge, and it appears to drag GIV along with it. In cells lacking Gαi3, GIV collects near the Golgi apparatus instead of dispersing to the edge of the cell. Gαi3 might even instigate a positive feedback loop because it presents GIV to Akt to be switched on; GIV can then further amplify Akt activity.

GIV and Gαi3 also help macrophages and tumor cells migrate, the team found. By ferrying GIV to the leading edge, Gαi3 might ensure that only one portion of the membrane undergoes the changes required for movement.

Ghosh, P., et al. 2008. *J. Cell Biol.* doi:10.1083/jcb.200712066.



A normal spindle (left) contrasts with frizzy ones from cells lacking TPX2 (right).

NEW SPIN ON SPINDLE SIZE

Like bridges and extension cords, the spindle that helps separate chromosomes during mitosis has to be the right length. Bird and Hyman show that two interacting proteins help set the spindle's dimensions.

When a cell builds a spindle, microtubules extend from several locations, including the chromosomes and the centrosomes. One mystery about the process is how cells dictate spindle length. A protein that might be involved is Aurora A, which promotes microtubule growth and is necessary for spindle formation. Another protein, TPX2, switches on Aurora A and helps position it on the spindle. By preventing TPX2 from activating Aurora A, Bird and Hyman tested whether this pair helps determine spindle length in human cells.

The standard way to address the question would be to add the gene for a defective version of TPX2, along with a viral promoter that controls its activity. However, previous work has shown that this method impairs mitosis. Instead, the researchers incorporated the mutant gene into a bacterial