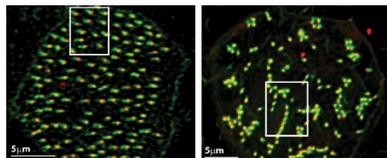


Fibers keep cilia regular



Cilia align in a control cell (left) but not in one with disrupted actin fibers (right).

the musicians in a garage band, each doing its own thing. But over time the filaments turn so that they all beat in the same direction. They also coordinate their timing, so that the cilia at the front of the cell beat first and the ones at the back of the cell stroke last. The planar cell polarity signaling pathway and hydrodynamic forces on the cilia help set up this polarity. Using the cilia-coated epithelial cells of *Xenopus* embryos,

Actin fibers and microtubules help cilia coordinate the direction and sequence of their beating, [Werner et al.](#) show.

At first, the multiple cilia on an embryonic cell are a bit like

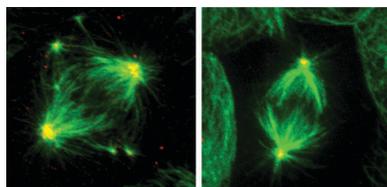
Werner et al. investigated the roles of the actin and microtubule cytoskeletons in the process.

Werner et al. observed actin fibers connecting neighboring cilia. These filaments were absent in the youngest cells that hadn't yet polarized. To determine the fibers' function, the researchers disassembled them using the drug cytochalasin D. Without actin fibers, the cilia couldn't reorient to beat in the same direction. They also couldn't coordinate their timing. However, neighboring clusters of cilia could still synchronize. This local coordination disappeared in cells lacking microtubules, the team found.

These results indicate that two components of the cytoskeleton perform different functions: actin fibers establish cell-wide coordination of cilia orientation and timing, whereas microtubules ensure that cilia are in harmony with their neighbors.

Werner, M.E., et al. 2011. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201106110>.

TPX2 is a drag on Eg5



Multiple spindle poles form in a cell that produces a version of TPX2 that can't interact with Eg5 (left), but a control cell shows a normal spindle (right).

Aurora A, a kinase necessary for spindle formation. TPX2 also latches onto the molecular motor Eg5, which crosslinks microtubules and pulls them past each other. Early in mitosis, Eg5 produces outward forces that push the spindle poles apart. Ma et al. wanted to determine how the interaction between TPX2 and Eg5 affects mitosis.

Putting the brakes on a molecular motor might help cells fashion the mitotic spindle, [Ma et al.](#) report.

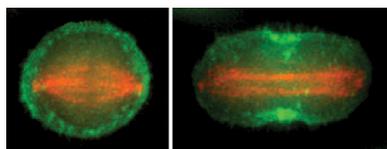
The protein TPX2 performs several jobs during spindle construction. For instance, it activates and positions

The researchers outfitted cells with a bacterial artificial chromosome encoding a version of TPX2 lacking the region that connects to Eg5. Then they used RNAi to deplete the cells' own version of TPX2. The cells made jumbled spindles with multiple poles and bowed microtubules. Thwarting the interaction between Eg5 and TPX2 caused another problem—the cells didn't make the kinetochore microtubules that attach to chromosomes and tug them apart.

Ma et al. found that TPX2 helps Eg5 to settle on the spindle microtubules between the poles. TPX2 also reduces the motor's ability to slide microtubules past one another in the spindle. The researchers hypothesize that, by idling the motor protein, TPX2 prevents it from generating too much outward force, which would lead to multiple poles or distorted fibers. One mystery, the researchers say, is how the interaction between TPX2 and Eg5 promotes stable connections between kinetochores and the spindle.

Ma, N., et al. 2011. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201106149>.

Mobile moesin helps mitotic cells stretch out



Moesin (green) initially disperses around the cortex of a mitotic cell (left) but then shifts to the cell's equator (right).

round up. During anaphase, the poles relax, and the cell equator contracts, allowing the cell to stretch and eventually split. Moesin, which belongs to a family of proteins that link the actin cytoskeleton to the plasma membrane, helps control the cell's shape. Roubinet et al. investigated what regulates the activity and location of moesin.

A mitotic cell reshapes itself twice. [Roubinet et al.](#) identify two molecular networks that help govern these transformations.

Early in mitosis, the cortex tightens, causing the cell to

Moesin's position reflects the cell's form, the researchers found. Until early anaphase, the protein is spread around the cortex, helping to keep the cell spherical. But then moesin begins to disappear from the poles and build up at the equator. Previous work showed that the enzyme Slik switches moesin on early in mitosis. Roubinet et al. discovered that the phosphatase Pp1-87B turns moesin off. Pp1-87B settles on segregating chromosomes during anaphase, which puts it in position to shut down moesin at the cell poles.

Two other enzymes, Pten and Skittles, boost the amounts of the phospholipid PI(4,5)P₂ at the cell's midsection, attracting moesin to that part of the cell. The researchers think that the two networks help ensure that moesin's activity surges all around the cortex before concentrating at the equator, allowing the cell to start out spherical and then to elongate and divide.

Roubinet, C., et al. 2011. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201106048>.