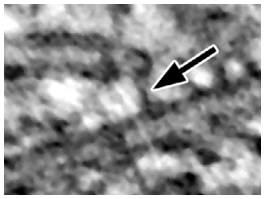
In This Issue

Text by Mitch Leslie
mitchleslie@comcast.net

Catching augmin in the act



A rod-shaped connector (arrow) that could be the augmin complex fastens a microtubule to its growing neighbor.

Kamasaki et al. observe links between microtubules within the mitotic spindle that might allow these microtubules to grow in the right direction.

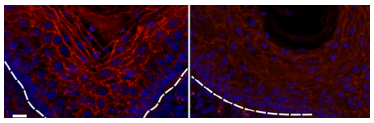
Most spindle microtubules extend from the centrosomes, and some sprout near the chromosomes. But researchers have discovered a third category of microtubules that grow within the spindle. The augmin complex and the γ-tubulin ring complex (γ-TuRC) team up to spur the formation of these microtubules, but the mechanism remains uncertain. In the cell's crowded middle, microtubules within the spindle are difficult to distinguish by fluorescence microscopy.

Kamasaki et al. used electron tomography to visualize part of the spindle in human cells. The researchers then painstakingly identified and traced every microtubule. The team detected microtubules whose ends were distributed within the spindle, with their minus ends pointing toward the spindle pole. However, these microtubules were less than half as abundant if the cell lacked augmin.

Augmin could potentially recruit γ-TuRC to the wall of an existing "mother" microtubule, leading to the initiation of a new "daughter" microtubule within the spindle. In control cells, the tomographs revealed examples of connections between a mother microtubule and the end of a daughter microtubule, but these links were rare in cells missing augmin. Kamasaki et al. showed that the microtubule connections often contained a rod-shaped structure that could be the augmin complex, although the researchers were unable to confirm its identity. Mother and daughter microtubules tended to be almost parallel, potentially explaining why the fibers adopt the correct orientation within the spindle.

Kamasaki, T., et al. 2013. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201304031.

β-Catenin solidifies tight junctions in the skin



Tight junctions (red) are sturdy in skin from control mice (left) but break down in mice lacking β-catenin (right).

Forget about lotions and facial creams. The secret to good—or at least strong—skin is β-catenin, Ray et al. reveal.

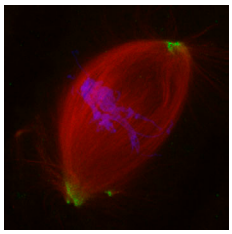
β-Catenin bolsters the adherens junctions that fasten epithelial cells together. But previous work suggested that the protein wasn't important in the skin. Mice lacking β-catenin in the epidermis have normal skin if β-catenin is eliminated after birth, although the protein's function during embryonic development remains uncertain.

Ray et al. deleted the protein from the epidermis of developing mouse embryos. These animals died shortly after birth because their skin was porous. The researchers found that, in their paw skin, embryos lacking β-catenin lost many of the tight junctions that interlock cells and make the skin watertight. The skin on an embryo's paws is likely to be under mechanical stress because the paws are still growing and start moving before birth. The flaws in the knockout animals suggest that β-catenin strengthens the skin against mechanical stress.

The researchers tested this idea in cultured skin cells. Pulling on cells that lack β-catenin spurred their tight junctions to break down. Cells under tension typically bolster their adherens junctions with vinculin, which connects the junction to the actin cytoskeleton. But Ray et al. discovered that cells lacking β-catenin didn't direct vinculin to their adherens junctions. This suggests that β-catenin helps fortify adherens junctions under mechanical stress, which, in turn, leads to the strengthening of tight junctions. How changes to the adherens junctions alter the tight junctions is a question for future research.

Ray, S., et al. 2013. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201212140.

SSX2IP helps the centrosome grow up



In a *Xenopus* cell-free extract, SSX2IP (green) amasses at the spindle poles (red marks the microtubules).

SSX2IP helps the centrosome prepare for mitosis by delivering essential proteins, Bärenz et al. report.

Constructing the mitotic spindle is a logistical challenge. Before mitosis begins, the cell has to synthesize and position a host of proteins, including centrosome proteins that arrange microtubules at the spindle poles and control their growth. One of these arrivals is the γ-tubulin ring complex (γ-TuRC), which gathers in the pericentriolar material of the centrosome to spur microtubule nucleation, but additional microtubule-binding proteins are required for spindle assembly.

Bärenz et al. tallied the microtubule-binding proteins during interphase and metaphase in unfertilized *Xenopus* eggs. One protein, synovial sarcoma X breakpoint 2 interacting protein (SSX2IP), stood out because researchers hadn't previously identified it as a spindle protein. However, SSX2IP was essential for centrosome maturation. In *Xenopus* egg extracts, for example, removal of SSX2IP hindered γ-TuRC accumulation at the centrosome, reducing microtubule nucleation and hampering spindle assembly. The pericentriolar material broke apart and mitotic progression slowed in human somatic cells with reduced SSX2IP levels.

SSX2IP promotes the assembly of a functional centrosome by ferrying γ-TuRC and other proteins. In human somatic cells, SSX2IP rides in centriolar satellites, which carry pericentriolar material to the centrosome. SSX2IP shows an unusual pattern of expression. A cell typically synthesizes spindle proteins before mitosis and destroys them afterward. But SSX2IP remains throughout interphase, suggesting that the organism sets the levels of the protein only once in its lifetime, during egg maturation.

Bärenz, F., et al. 2013. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201302122.