In This Issue

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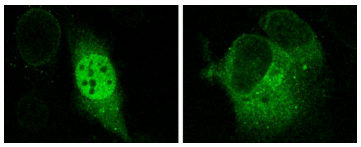
RanBP2 stops importin-β from running away



RanBP2 that carries a specific Ran-binding domain can import a GFP-tagged cargo into the nucleus (left), but RanBP2 lacking the domain can't (right).

Like taxis waiting for a fare, importin-β molecules gather outside nuclear pores, ready to pick up cargoes and haul them into the nucleus. The protein RanBP2 helps corral these importin-β molecules, Hamada et al. reveal.

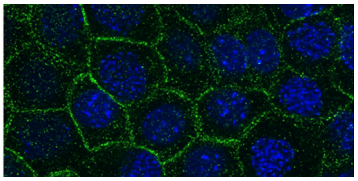
RanBP2 does double duty in the cell. During interphase, it forms the long filaments that protrude from the cytoplasmic side of a nuclear pore. During mitosis, it helps ensure that the chromosomes separate properly. Cells lacking the protein often show faulty spindles and misaligned chromosomes, whereas cells with abnormally low levels of RanBP2 are prone to aneuploidy.

To find out more about how RanBP2 performs its functions, Hamada et al. inactivated the gene in mouse embryonic fibroblasts. Cells lacking the protein died after 8–10 days, the researchers found. But they didn't die during or shortly after mitosis, suggesting that RanBP2's mitotic role isn't essential. Instead, the researchers think that the cause of death is disrupted transport across the nuclear pores. Export of mRNA and other cargoes fell in the RanBP2-lacking cells, as did import of cargoes that use transportin 1 and importin-β as receptors.

Hamada et al. determined that the presence of one Ran-GTPase–binding domain in RanBP2 was crucial for cell survival and cargo transportation by importin-β, which carries a Ran-GTP complex. The researchers think that the Ran-binding domain on RanBP2 helps snare importin-β complexes as they return to the cytoplasm from a trip into the nucleus. This interaction detains importin-β molecules in a location where they can pick up another load.

Hamada, M., et al. 2011. *J. Cell Biol.* doi:10.1083/jcb.201102018.

Lis1 finds new digs on the desmosome



The centrosomal protein Lis1 (green) localizes to the cortex of differentiated epithelial cells.

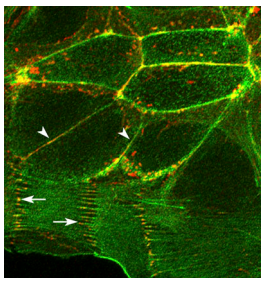
A protein that stabilizes microtubules also helps maintain desmosomes, Sumigray et al. report.

Desmosomes fasten cells together in organs such as the heart and skin that have to withstand intense mechanical stress. Defects in these structures cause problems such as skin blistering and cardiomyopathy. But desmosomes are more than just cellular Velcro. As some cells differentiate, microtubules no longer radiate out from the centrosome and instead localize to the cell cortex. The researchers have previously found that, during this processs in epithelial cells, the desmosome protein desmoplakin lures another protein, ninein, from the centrosome to the desmosome.

The researchers discovered that two other centrosome proteins, Lis1 and Ndel1, also make the jump to desmosomes and that desmoplakin recruits both of them. Lis1 and Ndel1 help organize microtubules in other contexts, making them good candidates for reconnecting microtubules to the cortex. To test that possibility, the researchers created mice that are missing Lis1 in their epidermis. In the animals' epidermal cells, the microtubules did not reorganize, indicating that Lis1 is necessary for this rearrangement. Sumigray et al. also discovered that Lis1 may have an additional desmosomal function. The Lis1-lacking animals died as newborns because their skin was permeable. Desmosomes were faulty in these animals, suggesting that Lis1 helps stabilize the structures. The next question is how Lis1 performs that function.

Sumigray, K.D., et al. 2011. *J. Cell Biol.* doi:10.1083/jcb.201104009.

Switching adherens junctions to seal wounds



In a colony of epithelial cells, arrowheads point to ZAs and arrows indicate pAJs.

Before a wound can heal, epithelial cells on its edges have to swap their adherens junctions (AJs), Taguchi et al. reveal.

A unique type of AJ, the zonula adherens (ZA), generally connects epithelial cells. ZAs are linear, which allows them to zip adjacent cells together without leaving gaps. During development and wound healing, epithelial layers go through an upheaval. Taguchi et al. investigated what happened to ZAs during such changes.

The researchers found that different types of AJs occur in different parts of an epithelial layer. ZAs predominate in the interior, but cells at the tissue margins carry punctate adherens junctions (pAJs), which aren't linear. Scratching a layer of epithelial cells to simulate a wound spurred the cells at the edges to replace their ZAs with punctate junctions, suggesting that pAJs are necessary to bring the two sides together.

pAJs lack EPLIN, a protein that helps organize ZAs, and the researchers discovered that actin fibers perpendicular to the junction keep EPLIN away. Zapping these cables with a laser caused the junctions to convert to ZAs. By contrast, stretching a cell layer boosted the amount of EPLIN at the intercellular junctions. The researchers think that EPLIN serves as a tension sensor in epithelial layers. When the layer is taut, there's plenty of EPLIN in the ZAs. But when the tension eases, such as when the layer is wounded, EPLIN is lost from ZAs, allowing them to convert into the pAJs that will help knit the wound together.

Taguchi, K., et al. 2011. *J. Cell Biol.* doi:10.1083/jcb.201104124.