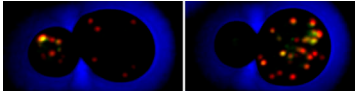


## Peroxisome inheritance at the right time and place



When peroxisome transport is blocked (right), Inp2p (green) localizes to every peroxisome (red) in the mother cell rather than just those that are moving into the bud (left).

Every yeast cell has numerous peroxisomes—organelles that metabolize fatty acids—and roughly half are passed on to their daughter cells. Signaling from the bud to the mother cell ensures even peroxisome distribution by

controlling the organelles' transport, say Fagarasanu et al. peroxisome transport, and were surprised that Inp2p levels increased until every peroxisome in the mother cell carried the receptor. This suggests that once enough peroxisomes have accumulated in the bud of wild-type cells, a signal is relayed to the mother cell resulting in Inp2p degradation and a halt to further peroxisome transfer. The group also observed this feedback mechanism within the membrane of a single peroxisome: mutants with a solitary large peroxisome that positions itself halfway into the bud lost Inp2p from the half that remained in the mother cell.

controlling the organelles' transport, say Fagarasanu et al.

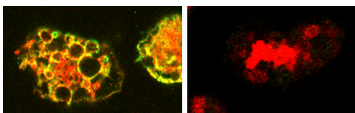
Peroxisomes travel into the bud along actin tracks using the myosin motor Myo2p, which binds to peroxisomes via a specific receptor called Inp2p. Cells ramp up Inp2p expression during mitosis, but the receptor only appears on a subset of peroxisomes, potentially selecting them for Myo2p-mediated movement to the bud.

Fagarasanu et al. made point mutants in Myo2p that blocked

Thus, both the cell cycle and the distribution of peroxisomes control Inp2p expression levels to regulate peroxisome inheritance in time and space. The next challenge, says lead author Andrei Fagarasanu, is to work out how cells sense the presence of peroxisomes in the bud and transmit a signal back to the mother cell to trigger Inp2p's degradation. **BS**

Fagarasanu, A., et al. 2009. *J. Cell Biol.* doi:10.1083/jcb.200904050.

## Myosin drives bladder movements



Contractile vacuoles (red) accumulate in the center of the cell in the absence (right) of MyoJ (green).

A myosin motor dictates the localization and movement of an organelle that expels excess water from *Dictyostelium*, report Jung et al.

To counteract the osmotic stress of an aqueous

environment (when it rains, for example), *Dictyostelia* pump protons into a membranous organelle called the contractile vacuole (CV), generating an osmotic gradient that sucks up extra water from the cytoplasm. The vacuole swells up and fuses with other CVs to form a large bladder that expels the water through brief contact with the plasma membrane. After relieving itself, the bladder rapidly contracts before sending out new CV tubules around the actin-rich cell cortex to begin the cycle again.

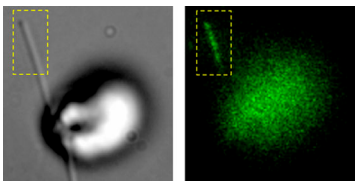
Jung et al. found that a myosin called MyoJ localizes to CV

membranes, and cells lacking the motor no longer accumulated CVs at their edges. Instead, the membranes clumped together in the center of the cell; video microscopy revealed that CVs traveled out to the cell cortex along microtubules but couldn't stay there in the absence of MyoJ. Cells rescued with a version of MyoJ lacking motor activity could capture CV membranes at the cell periphery, but their bladders were unable to spread out into tubules after discharging water at the cell surface. A truncated version of MyoJ that takes shorter steps along actin filaments was able to drive this process, but the tubules spread out more slowly than they did in the presence of full-length MyoJ.

Doubts have been raised about the true function of myosins like MyoJ, says senior author John Hammer III, but the group's results demonstrate that MyoJ acts as a "point-to-point" organelle motor. The next question is how actin filaments are arranged at the cell cortex to facilitate the MyoJ-driven spreading of CV tubules. **BS**

Jung, G., et al. 2009. *J. Cell Biol.* doi:10.1083/jcb.200810147.

## Ubiquitin drives cilia shortening



The ubiquitin-conjugating enzyme CrUbc13 (green) localizes to the alga's flagella.

Huang et al. report that the ubiquitination system has an integral function in disassembling cilia and flagella.

Cilia and flagella are whip-like membrane protrusions that propel cells around and are implicated in many human disorders,

collectively known as ciliopathies. New research demonstrates that they also function as sensory and signal transducers. Cilia and flagella grow and shrink during the cell cycle, becoming completely resorbed before the cell divides. Whereas the mechanism by which cilia assemble has been well studied, how they disassemble remains largely unknown.

Huang et al. isolated flagella from the green alga *Chlamy-*

*domonas reinhardtii* and found that they contained a completely functional ubiquitination system, including free ubiquitin and enzymes necessary for its conjugation. Ubiquitination activity, and the abundance of ubiquitinated proteins increased in response to cues stimulating flagellar disassembly. However, though the group found an active ubiquitination system, there was no evidence of a flagellar-localized proteasome.

The researchers also found that the targets of ubiquitination include  $\alpha$ -tubulin and the cation channel CrPKD2, an algal homologue of a gene mutated in the ciliopathy polycystic kidney disease. The functional consequence of CrPKD2 ubiquitination is not known, but the authors hypothesize that it facilitates recycling. In lieu of degrading flagellar components and making them anew, some proteins may be sent back to the distal tip for reassembly. It seems these algae may be green in more ways than one. **EC**

Huang, K., et al. 2009. *J. Cell Biol.* doi:10.1083/jcb.200903066.