Holding on to peroxisomes

In the field of organelle inheritance, peroxisomes are the forgotten stepchild no more. Fagarasanu et al. (page 765) find that a previously uncharacterized protein, Inp1p, tethers a fraction of the peroxisomes to the cortex of the mother cell during bud formation in *S. cerevisiae*. Cells lacking Inp1p lose almost all peroxisomes to the bud.

Yeast cells are known to actively partition organelles between the mother and bud during division. Mitochondria, for example, are subjected to both retention and ordered movement to ensure that a proper fraction of the organelles end up in each cell after cytokinesis. In the case of peroxisomes, researchers knew that a subset of the organelles moved into the bud in a myosin-dependent manner, but a retention mechanism hadn’t been detected.

Inp1p was putatively localized to the peroxisome based on genome-wide GFP tagging efforts conducted several years ago. The current study confirmed Inp1p’s location and defined it as a peripheral peroxisomal membrane protein. It associated with proteins known to influence peroxisome size and shape.

Video microscopy of wild-type cells showed peroxisomes moving in a directed manner to the bud. Mother cells lacking Inp1p retain few of the peroxisomes, most of which move to the growing bud tip. In cells overexpressing Inp1p, peroxisomes were tightly associated with the cortical regions of mother cells, and buds developed with few or no peroxisomes.

Movement of excess peroxisomes to the bud in cells lacking Inp1p may be due to a decreased affinity of peroxisomes for some cortical anchor in the mother cell. But what that anchor is or how Inp1p attaches to it is unclear.

![Without Inp1p (bottom), all peroxisomes are transported into the bud.](image)

Sperm’s quick calcium response

Some free-swimming sperm detect the presence of an egg through chemotactic signals and respond by altering their swimming trajectory. On page 725, Wood et al. show that intact sea urchin sperm respond not to overall increases in intracellular Ca\(^{2+}\) levels, as previously thought, but to rapid changes in Ca\(^{2+}\) concentration. Ca\(^{2+}\) entry into the flagella is biphasic, with a fast and slow phase, but only the initial fast influx affects sperm trajectory.

Studies on demembranated sperm indicated that the cells change direction by asymmetrical bending of their flagella in response to overall Ca\(^{2+}\) increases and that cGMP signaling was involved. But until now scientists didn’t have the tools to watch the process in real time in intact cells.

Using a novel fast strobe lighting system for fluorescence microscopy, which they devised, Wood et al. attempted to stimulate chemotaxis by releasing caged cGMP in *Strongylocentrotus purpuratus* sperm cells loaded with a fluorescent Ca\(^{2+}\) indicator. As predicted, Ca\(^{2+}\) levels immediately increased in both the head and the flagella. However, the flagella showed a short rapid burst of Ca\(^{2+}\) influx followed by a longer slower uptake. Ca\(^{2+}\) increases induced transient bending in the flagella. Repeated stimuli induced repeated direction changes, even if the overall Ca\(^{2+}\) concentration was already above baseline levels.

Nimodipine, which is used to block voltage-gated Ca\(^{2+}\) channels, blocked the initial fast influx into the flagella, suggesting that such channels mediate the fast uptake, but did not affect the slow portion of the Ca\(^{2+}\) uptake. In the presence of nimodipine, the sperm did not alter their swimming direction, even though the intracellular Ca\(^{2+}\) increased.

The fact that a slow rise in Ca\(^{2+}\) was not sufficient to induce flagellar bending leads the team to suggest two possibilities. Either there is a Ca\(^{2+}\) sensor that detects only a rapid flux in concentration, or the sensor is located so close to the nimodipine-sensitive channel that it only responds to Ca\(^{2+}\) coming in via this port.

![The direction of sperm tail movement (red to black to blue) changes with a sharp pulse of Ca\(^{2+}\) (top).](image)