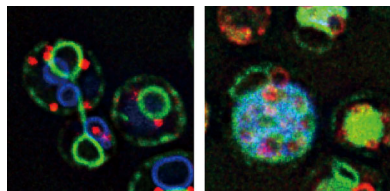


Lipid droplets ensure their own consumption



In well-fed yeast cells (left) lipid droplets (red) adhere to the ER (green), but in hungry cells (right) the vacuole (blue) has absorbed several of the droplets.

Hungry yeast cells use a clever form of recycling to ensure that they can keep consuming stored lipids, Wang et al. show.

Lean times spur yeast cells to enter a quiescent state known as the stationary phase.

They break down cached lipids by delivering lipid droplets to the vacuole and resort to autophagy, in which they digest a portion of their own contents. Wang et al. discovered that, to transfer lipid droplets into the vacuole, yeast in the stationary phase rely on a selective form of this process known as microautophagy.

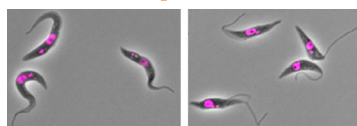
In mutant yeast cells that lacked proteins necessary for microautophagy, lipid droplets were locked out of the vacuole.

The vacuole's membrane contains sterol-rich liquid-ordered domains and more disorganized liquid-disordered domains. Wang et al. determined that lipid droplets can't cross into vacuoles lacking these domains, and when lipid droplets do enter the vacuole it is specifically through the sterol-rich liquid-ordered domains.

These domains disperse in cells unable to perform autophagy, explaining the loss of lipid droplet transfer under these conditions. Wang et al.'s results further suggest that cells return some of the sterol already inside the vacuole to the liquid-ordered domains. By recycling some of the contents of the lipid droplets already in the vacuole, a yeast cell might ensure an ample supply of liquid-ordered domains that allow the vacuole to absorb and digest additional droplets.

Wang, C.-W., et al. 2014. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201404115>.

Loss of protein remodels shape-shifting parasite



T. brucei trypomastigotes (left) contrast with the epimastigote-like cells induced by suppression of ClpGM6 (right).

Parasites frequently undergo dramatic shape changes during their life cycles, but these transformations might not be as difficult as they appear, Hayes et al. suggest.

Trypanosoma brucei

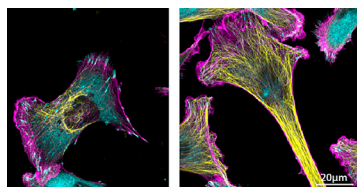
parasites cause African sleeping sickness. In their trypomastigote stage, the kinetoplast, which houses the cell's mitochondrial DNA, is posterior to the nucleus, and almost all of the flagellum is connected to the cell. In the epimastigote stage, on the other hand, the kinetoplast is anterior to the nucleus, and only part of the flagellum is fastened to the cell. *T. brucei*'s close relatives come in many different shapes, indicating that the parasites have also altered their morphology during evolution.

When Hayes et al. used RNAi to quash a protein called ClpGM6 in *T. brucei* trypomastigotes, the cells switched to an epimastigote-like morphology. The kinetoplast was close to the nucleus or anterior to it, and a long section of the flagellum extended beyond the cell. The parasites weren't identical to epimastigotes—they lacked a distinctive surface protein found at this life stage—but they were able to survive and reproduce for more than 40 generations.

ClpGM6 resides in the flagellar attachment zone (FAZ) and likely helps fasten the flagellum to the cell body. Loss of ClpGM6 shortened the FAZ, which defines the position of the cytokinetic furrow during division and hence determines cell size and shape. The study suggests that the dramatic morphological changes during the life cycle and during parasite evolution may result from adjustments in the levels of a few key proteins, rather than from wholesale changes in protein expression or in the genome.

Hayes, P., et al. 2014. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201312067>.

Paxillin brings peace to microtubules



A control cell (left) shows a modest amount of microtubule acetylation (yellow), but the amount increases after removal of HDAC6 (right).

Paxillin spurs cell migration and polarization by suppressing an enzyme that destabilizes microtubules, Deakin and Turner show.

Before a cell can crawl in a particular direction, it has to polarize. One cellular change that

makes polarization and migration possible is stabilization of the microtubule cytoskeleton. In addition, the microtubule cytoskeleton helps the Golgi apparatus move in front of the nucleus. Deakin and Turner discovered that both alterations depend on paxillin, a protein that normally controls the structure and dynamics of the focal adhesions where a cell attaches to its substrate.

The researchers found that paxillin blocks the enzyme HDAC6,

which disrupts microtubules by removing the acetyl groups that stabilize them. Depleting HDAC6 increased microtubule acetylation, whereas knocking down paxillin in a variety of cell types reduced acetylation. Loss of paxillin also prevented relocation of the Golgi. Deakin and Turner determined that paxillin controls cell migration and invasion through its effects on HDAC6.

Paxillin is a scaffold protein—it provides a platform where other proteins can interact and communicate. The researchers discovered that paxillin and HDAC6 stick together, although it is unclear whether paxillin directly inhibits HDAC6 or relies on other proteins. Paxillin normally operates at cell adhesions, but the researchers also found complexes containing paxillin and HDAC6 scattered around the cell. The authors suggest these far-flung complexes control Golgi reorganization and microtubule stability throughout the cell.

Deakin, N.O., and C.E. Turner. 2014. *J. Cell Biol.* <http://dx.doi.org/10.1083/jcb.201403039>.