Lipid droplets ensure their own consumption

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.

Loss of protein remolds shape-shifting parasite

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

Paxillin brings peace to microtubules

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.


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