Lipid droplets fatten up with Fsp27

A membrane protein promotes the growth of lipid droplets (LDs) by facilitating lipid transfer from smaller to larger droplets, Gong et al. report. Consisting of a neutral lipid core surrounded by a monolayer of phospholipids and associated proteins, LDs serve as the cell’s fat storage depot, particularly in adipocytes where they grow to extra large sizes. How LDs grow is unknown, but adipocytes lacking the LD-associated proteins Acb1, Vps23, and Grh1, a homologue of a mammalian Golgi protein, don’t pass through the endoplasmic reticulum (ER) and Golgi on its way to the plasma membrane.

Several proteins mediate its journey. Components required for Acb1 secretion include autophagy proteins (such as Atg8 and Atg9), proteins that deliver cargo to multivesicular bodies (for example, Atg8), and the phosphoinositide PI(3)P synthase. Blocking PI(3)P synthesis or deleting Grh1 prevented the formation of these compartments in starving yeast.

By electron microscopy, the Grh1-containing membranes appeared cup-shaped, leading the authors to name them compartments for unconventional protein secretion or CUPS. Their shape and the presence of Atg8 and Atg9 are reminiscent of autophagosome precursors, but Bruns et al. found that CUPS weren’t formed in response to the autophagy-inducing drug rapamycin, suggesting that CUPS are a novel, albeit related, compartment. Senior author Vivek Malhotra says that the identification of CUPS gives researchers a handle to uncover other steps in Acb1 secretion. CUPS may engulf Acb1 in the cytoplasm and deliver it to the plasma membrane, either directly or via fusion with secretory endosomes.

Bruns, C., et al. 2011. J. Cell Biol. http://dx.doi.org/10.1083/jcb.201106098.

Cell death helps give closure

By visualizing the dynamics of apoptosis in living mouse embryos, Yamaguchi et al. reveal that cell death helps drive the morphogenetic movements required for neural tube closure (NTC).

Yamaguchi et al. generated transgenic mice expressing a FRET-based apoptotic reporter whose signal is decreased when activated caspases cleave the link between two fluorescent proteins. Using this reporter, the researchers identified two types of apoptotic cells in embryonic brains undergoing NTC. Some cells died and fragmented rapidly, whereas others—particularly near the tips of the folding neural plate—persisted for longer without breaking apart.

Both types of apoptotic cells were absent from mouse embryos lacking Apaf-1 and Caspase-3. In these animals, neural plate bending was reduced, thereby delaying NTC. Senior authors Yoshifumi Yamaguchi and Masayuki Miura think that the death, and subsequent extrusion, of cells from the tips of the neural plate may generate forces that help to shape the developing tissue and facilitate cranial NTC. Alternatively, the two types of apoptotic cells may direct this developmental process by sending different signals to their surviving neighbors.

Yamaguchi, Y., et al. 2011. J. Cell Biol. http://dx.doi.org/10.1083/jcb.201104057.