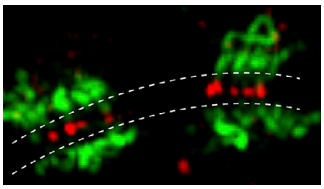
In This Issue

Text by Ben Short
bshort@rockefeller.edu

A potato virus makes its move



TGB2 (green) localizes to membranous caps at the entrance to plasmodesmata, whereas TGB1 (red) is found inside the channels. Dashed lines indicate cell walls.

Tilsner et al. describe how three viral proteins combine to help newly synthesized viruses spread to neighboring plant cells.

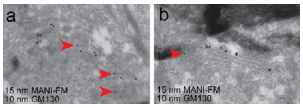
Plant viruses move between cells through narrow channels in the cell wall called plasmodesmata. Potato virus X, which replicates on the surface of the endoplasmic reticulum (ER), encodes three proteins—TGB1, 2, and 3—that promote the virus' transport into neighboring cells, but the precise function of each of these "movement proteins" is unclear.

Tilsner et al. found that TGB2 and TGB3 established clusters of ER membranes that capped the entrances to plasmodesmata. These caps appeared to be sites of viral replication because they contained the virus' RNA polymerase and RNA that was not yet encased by viral coat proteins. In addition, TGB2 and TGB3 recruited TGB1 to plasmodesmata, where it helped insert viral coat proteins into the intercellular channel.

The authors think that newly synthesized viral RNAs are encased by coat proteins and quickly trafficked into neighboring cells. Thus, by compartmentalizing viral replication to plasmodesmata, the movement proteins allow the potato virus to rapidly spread before the plant's defense mechanisms can shut the pathogen down. Lead author Jens Tilsner now wants to investigate how TGB1 interacts with the viral coat protein in order to insert it into the plasmodesmata channel.

Tilsner, J., et al. 2013. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201304003.

A mature view of the Golgi



MANI (large gold particles) localizes near the cis-Golgi (arrowheads) when monomeric (left) but shifts to the trans-Golgi upon polymerization (right).

Rizzo et al. demonstrate that Golgi-resident enzymes recycle backwards to promote the maturation of cargo-containing Golgi cisternae.

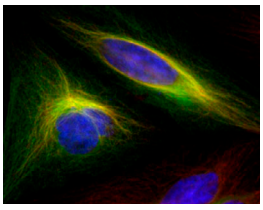
In mammalian cells, the Golgi apparatus consists of stacks of flat, membrane-bound compartments called cisternae. How secretory proteins move through the stack—from the cis side near the ER to the trans side where they are sorted to their final destination—is unclear. In one model, secretory cargo is transported in vesicles from one cisterna to the next, with each Golgi compartment stably maintaining its own composition. An alternative view is that secretory proteins remain in place while the composition of the cisterna changes around them; Golgi resident enzymes are recycled back to earlier cisternae so that each compartment gradually matures and acquires the composition of the trans-Golgi.

To distinguish between these possibilities, Rizzo et al. generated a version of the cis-/medial Golgi enzyme mannosidase I (MANI) that could reversibly polymerize into a large network incapable of being incorporated into recycling vesicles or tubules. Just like the endogenous enzyme, monomeric MANI localized in cis and medial cisternae and in transport vesicles and tubules around the Golgi stack. But when MANI polymerized, it shifted to the trans-Golgi and was no longer found inside vesicles and tubules. Disrupting the MANI network prompted the enzyme's incorporation into vesicles and its rapid return to the cis-/medial Golgi.

This suggests that MANI and other Golgi enzymes maintain their localization by recycling backwards as individual cisternae carrying protein cargo mature. Senior author Alberto Luini says that other intra-Golgi transport mechanisms may exist and that some secretory proteins may move forwards through the Golgi in transport vesicles. He now wants to investigate how Golgi enzymes are recycled back to earlier compartments.

Rizzo, R., et al. 2013. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201211147.

Sequestration puts Drp1 on furlough



Drp1-x01 (green) localizes to microtubules (red).

Strack et al. identify a splice variant of the mitochondrial fission protein Drp1 that can be sequestered away on microtubules.

Cells regulate their mitochondria by fusing them into an interconnected network or separating them into smaller, discrete structures. The dynamin-related GTPase Drp1 forms spiral-shaped oligomers on the surface of mitochondria that constrict and break the organelle apart. The Drp1 mRNA can be alternatively spliced with up to three additional exons, but the significance of this diversity is unclear.

Strack et al. found that Drp1 variants encoded by mRNAs containing the third alternative exon but excluding the second localized to microtubules. These variants—collectively known as Drp1-x01—appeared to directly bind microtubules via two arginine residues encoded by the third alternative exon, forming oligomeric spirals around the cytoskeletal filaments in vivo. Cells expressing Drp1-x01 had elongated mitochondria because this form of the GTPase showed a reduced association with the organelle. Mitochondrial fission is an important step in apoptosis, so these cells were more resistant to the apoptosis-inducing drug staurosporine.

Mitochondria also fragment during mitosis, a process promoted by the phosphorylation of a serine residue near the microtubule-binding domain of Drp1-x01. Strack et al. found that the cyclin-dependent kinase Cdk1 phosphorylated this serine during mitosis to promote Drp-x01's release from microtubules. Cdk5 performed the same function during interphase.

Microtubule-bound Drp1-x01 therefore forms an inactive pool of the GTPase that can be mobilized to fragment mitochondria when needed. Lead author Stefan Strack now wants to investigate whether the expression of Drp1 splice variants is altered during disease.

Strack, S., et al. 2013. *J. Cell Biol.* http://dx.doi.org/10.1083/jcb.201210045.