Playing FtsZ before sporulation

*Bacillus subtilis* puts its division plane in the right place with the help of cytoskeletal spirals, according to new results from Sigal Ben-Yehuda and Richard Losick (Harvard University, Cambridge, MA).

In a growing *B. subtilis* cell, the tubulin-like protein FtsZ assembles into a ring structure at the division plane in the middle of the cell. Upon sporulation, cell division is asymmetric, and FtsZ rings form at the poles. Previous models suggested that the shift occurred when FtsZ assembly was blocked at the midcell and activated at the poles.

Ben-Yehuda and Losick examined a GFP fusion of FtsZ during sporulation and discovered that this model was inadequate. The fusions revealed a spiral-like intermediate of FtsZ, which over time extended from the midcell toward both poles. The spirals eventually gave way to polar rings, one of which became the division plane. Thus, generation of polarity requires that the ring still form at the midcell, with the spirals causing its relocation. The spirals were also seen moving from the poles to the midcell when bacteria entering sporulation were returned to growth medium.

The spirals may result from elevated levels of FtsZ present during sporulation, as overexpression of *ftsZ* in *Escherichia coli* has also been shown to cause the formation of spirals. “Possibly, it is an intrinsic property of FtsZ that high concentrations cause it to form spirals,” says Losick. “Or maybe the ring structure is actually a tight spiral, and only the periodicity changes.”

Reference: Ben-Yehuda, S., and R. Losick. 2002. Cell. 109:257–266.

Getting a GRIP on kinesin movement

Kinesin can transport vesicles along microtubules to the axon by binding to the scaffolding protein JSAP1. But now it appears that the motor is not always partial to the axon. Using the glutamate receptor subunit GluR2 as cargo, Hirokawa’s group demonstrated that kinesin could also transport its cargo to dendrites, where the receptors are required.

Upon this discovery, Hirokawa says, “we were then interested in understanding how the same motor could determine the direction of transport.” They found their answer in a screen for kinesin binding partners, which identified a glutamate receptor–interacting protein, GRIP-1. Whereas kinesin bound to GRIP-1 was recruited to dendrites, kinesin bound to JSAP1 moved to axons. It is thus the scaffolding proteins that direct kinesin and its cargo toward their destination.

Previously identified scaffolding proteins, such as JSAP1, have shown a preference for binding to kinesin light chain. Hirokawa points out that fungal kinesin lacks a light chain but still transports cargo. Sure enough, GRIP-1 binds the heavy chain. Thus, the choice of subunit bound may be important in determining the direction kinesin travels.

Reference: Setou, M., et al. 2002. *Nature*. 10.1038/nature743.

Sorting it out, without clathrin

Clathrin-independent endocytosis is revealing itself at last. Intermediary organelles in this pathway have been difficult to identify, due in part to a dearth of markers and, until recently, difficulties in blocking the clathrin-dependent process. But now, Benjamin Nichols (MRC Laboratory of Molecular Biology, Cambridge, UK) has identified a set of endosomes that are uniquely involved in clathrin-independent trafficking.

Nichols’ results demonstrate that vesicles containing caveolin-1 define a set of early endosomes that are distinct from those that form from clathrin-coated pits. Proteins that were endocytosed independently of clathrin, including GPI-anchored proteins and the cholera toxin B subunit, were found within the caveolin-1–positive endosomes. Even in the absence of clathrin-mediated endocytosis, these proteins were delivered from the plasma membrane to the Golgi.

Although caveolin-1 provides a useful marker for the pathway, the protein was not important for endocytosis. Nichols found that caveolin-1 was sorted away from Golgi-bound vesicles, and diminished caveolin-1 levels did not inhibit clathrin-independent endocytosis. Caveolin-1–containing endosomes have previously been shown to transport SV40 virus to the ER. Nichols believes these may be the same organelles, although this is not yet proven.

The function of the clathrin-independent pathway will be better understood when specific inhibitors can be identified. For now, Nichols hypothesizes that clathrin-independent endocytosis is important for delivery of certain plasma membrane lipids to the trans face of the Golgi, the site of lipid raft formation and Golgi cargo sorting.

Reference: Nichols, B. 2002. *Nat. Cell Biol*. 10.1038/ncb787.