Research Roundup

Wingless and the argosomes

Signaling proteins may set up gradients by moving in membrane fragments, dubbed argosomes by Suzanne Eaton and colleagues (Max Planck Institute, Dresden, Germany).

Eaton first observed the structures when she was studying apical–basal sorting of proteins that have glycosylphosphatidylinositol (gpi) tails. “It was this completely random observation several years ago,” she says. “When we saw this we just dropped working on apical–basal sorting.”

What she saw were clusters of her labeled protein, a fusion of GFP to a gpi tail, in nonexpressing tissue. The signaling molecule wingless was found in a similar pattern, with both molecules forming gradients that drop with increasing distance from the domain of expression (although the gradient is steeper for wingless).

Eaton believes that lipid carriers are responsible because GFP lacks a surface receptor that could concentrate any protein that was somehow solubilized. She also found that a fluorescent fusion protein GFPgpi (green) spreads from the expressing region (right) attached to argosomes. Eaton/Elsevier

Argosomes might allow the cell to cluster signaling molecules into concentrated packages, and to pair them with other regulatory molecules. In addition, argosomes move proteins rapidly inside cells, although this movement appears to be undirected. The failure to see argosomes outside of cells may reflect an inability to detect smaller protein clusters.

Eaton and colleagues suggest two potential mechanisms for creating argosomes. One possibility involves the engulfment by one cell of protrusions formed by another cell, as seen during the spread of Listeria bacteria. Alternatively, proteins such as wingless may first be exported from the cell, bind to a gpi-linked heparan sulfate proteoglycan, and be brought back into the cell by endocytosis. Then membrane vesicles would bud internally into the endosomes to form multivesicular bodies, which have been noted before as a site of wingless localization. Fusion of the multi-vesicular bodies with the plasma membrane would release the argosomes.

Reference: Greco, V., et al. 2001. Cell. 106:633–645.

Cold-induced dwarfism

A n increase in the rate of exocytosis has been put forth as one hypothesis to explain how plants maintain their growth rate in cold weather, in work from Jian Hua (now at Cornell University, New York, NY), Gerald Fink (Whitehead Institute, Cambridge, MA) and colleagues. They base their suspicion on a plant mutant that is a midget only in the cold.

The mutant, bonzai1 (bon1), was isolated by Hua. “In the beginning I was just looking for something that would affect cell division and expansion,” she says. But she ended up with bon1, which at 2°C is almost normal but at 22°C is a real shrimp, with epidermal cells and stems that are seven and eight times shorter than normal, respectively. The mutant also has fewer cells when grown in the cold, although this may be secondary to the reduction in cell volume.

BON1 gives plants a boost in the cold.

BON1 belongs to a poorly characterized family of proteins called the copines. Like other members of this family it has a calcium-stimulated phospholipid binding activity, and can enhance vesicle aggregation in vitro.

Little else is known about copines, but the binding of one to secretory vesicles, and the localization of BON1 to the plasma membrane have led to suggestions that copines participate in exocytosis.

The expression of BON1 in growing tissues, and its increased expression at low temperatures, suggest that BON1 could compensate for otherwise inefficient membrane fusion at low temperature. “It makes sense having this hypothesis,” says Hua, “but there is no direct evidence.”

Reference: Hua, J., et al. 2001. Genes Dev. 15:2263–2272.