Flow to the front

Cortical actomyosin flow carries polarity proteins to the front of the worm embryo, according to Edwin Munro (Center for Cell Dynamics, Friday Harbor, WA), Jeremy Nance, and James Priess (Fred Hutchinson Cancer Research Center, Seattle, WA). Similar flows may set up polarity in many other systems.

The idea that asymmetrical contraction drives cortical flow and the segregation of cell fate determinants has a long and controversial history. Earlier efforts were dogged by the transience of the flow and different results after the use of different fixation methods.

But when Munro finally had GFP-labeled myosin to work with, "the whole story unfolded in front of me," he says. Contractile, coupled foci of cortical myosin gave way at the posterior when the sperm centrosomes approached the posterior cortex. The actomyosin network then contracted toward the anterior, taking a host of cytoskeletal proteins and anterior determinants with it.

Absence of these anterior determinants allows the PAR-2 determinant to attach to the posterior cortex, where it was needed to prevent a reverse flow of actomyosin back to the posterior. Both the PAR-2 and centrosome signal may somehow weaken or degrade parts of the actomyosin system, although the specific mechanism is a mystery.

Similar flows were seen at the 8-cell stage when cells set up PAR-based apicobasal polarity. In this case, the cue that weakens the actomyosin cortex may be basolateral contact with surrounding cells. "Any time you get differences in contractility you'll see flows in the cortex," says Munro. "The mystery is not why do you have flows but how you prevent flows." JCB

Reference: Munro, E., et al. 2004. Dev. Cell. 7:413–424.

Endocytosis gets squeezed

A squeeze from myosin may separate incoming endocytic vesicles from the plasma membrane, according to Gudrun Jonsdottir and Rong Li (Harvard Medical School, Boston, MA).

The myosin in question, Myo5, is a class I myosin from budding yeast. The Boston group found that Myo5 had unusual dynamics: it was stationary at cortical actin patches, the presumptive endocytic sites in yeast, and departed after only a brief stay. The peak of Myo5 localization came immediately before the actin polymerization factor Arp2 switched from slow to fast movement away from the membrane. This switch was delayed (although the fast phase, once started, was normal) when Myo5 was mutant.

Previous workers have defined a sequence of comings and goings of different components at the actin patches. Some of these components are thought to move away from the plasma membrane with the endocytic vesicles in a stereotypical slow-then-fast progression. In the model based on these results, slow movement is proposed to involve invagination, and subsequent fast movement initiates only once a vesicle is pinched off and more explosive actin polymerization takes over.

Myo5 was not previously put into this sequence, but the new results suggest that Myo5 may drive vesicle pinching off (scission). The driving together of two membranes during scission could occur by directed myosin movement or contraction of an actomyosin mesh around the neck of an invaginating vesicle. Li now hopes to determine whether the motor activity of Myo5 is needed for endocytosis and to investigate Myo5 activity in an in vitro endocytosis system. JCB

Reference: Jonsdottir, G.A., and R. Li. 2004. Curr. Biol. 14:1604–1609.

Immune synapses make a choice

The immunological synapse between T cells and the antigen-presenting dendritic cells acts as a locus for T cell activation. Now, Roberto Maldonado, Laurie Glimcher (Harvard School of Public Health, Boston, MA), and colleagues find that this synapse also helps the T cells decide between two different activated, differentiated fates based on the extent of colocalization of receptors at the synapse.

The end products of this decision are the bacteria-fighting Th1 cells and the parasite-fighting Th2 cells. Activation of the interferon-γ receptor (IFNGR) or interleukin 4 receptor (IL-4R) is known to favor Th1 production or Th2 production, respectively.

Now, Glimcher's group shows that the IFNGR but not IL-4R colocalizes with the T cell receptor (TCR) at the immunological synapse. The extent of this colocalization is greatest in mice that tend to generate more Th1 cells. IL-4, which favors production of Th2 cells, inhibits the colocalization. Cross-linking of the IFNGR and TCR might set up a positive feedback between activation and differentiation pathways.

Reference: Maldonado, R.A., et al. 2004. Nature. doi:10.1038/nature02916.