IN BRIEF

CELL POLARITY

Actin-driven chromosomal motility leads to symmetry breaking in mammalian meiotic oocytes.

Li, H. *et al. Nature Cell Biol.* 5 Oct 2008 (doi:10.1038/ncb1788)

Oocyte meiotic divisions are highly asymmetric, and symmetry breaking initiates when chromosomes move from the oocyte centre towards the cortex. Rong Li and colleagues used four-dimensional (4D) tracking, and genetic and pharmacological manipulations to determine the force-generating mechanism that underlies this chromosome movement. They found that chromosomes move towards the cortex in a pulsatile manner along a meandering path. Rather than being propelled by myosin-II-driven cortical flow, this movement was associated with a cloud of dynamic filaments trailing behind the chromosomes. Formation of these filaments depends on the actin nucleation activity of the formin protein FMN2, which concentrates around the chromosomes. These findings do not favour the model that chromosomes are moved as cargo along a pre-existing actin track or through an actin–myosin-II-based contractile network. Instead, they are consistent with a model in which chromosome movement is driven by actin polymerization.

CELL POLARITY

The keratin-binding protein Albatross regulates polarization of epithelial cells.

Sugimoto, M. *et al. J. Cell Biol.* **183**, 19–28 (2008)

Sugimoto and colleagues now shed light onto the role of intermediate filaments in the establishment of cell polarity by showing that the keratin-binding protein Albatross forms a complex with partitioning defective-3 (PAR3), and regulates epithelial polarization. In nonpolarized epithelial cells, Albatross localizes with keratin filaments whereas, in polarized epithelial cells, it is primarily localized in the vicinity of the apical junctional complex (AJC). Knockdown of Albatross in polarized cells causes keratin filament reorganization and a disappearance of AJC components at cell–cell borders. Although Albatross promotes localization of PAR3 to the AJC, PAR3 was still retained at the apical surface in Albatross knockdown cells, which had intact microvilli. Analysis of keratin-deficient epithelial cells revealed that keratins are required to stabilize the Albatross protein, thus promoting AJC formation.

PROTEIN DEGRADATION

Ubiquitin-like protein involved in the proteasome pathway of *Mycobacterium tuberculosis*.

Pearce, M. J. *et al. Science* 2 Oct 2008 (doi:10.1126/science.1163885)

The protein modifier ubiquitin is a signal for proteasome-mediated degradation in eukaryotes. By contrast, proteasome-bearing prokaryotes have been thought to degrade proteins through a ubiquitin-independent pathway. Pearce *et al.* have now identified a prokaryotic ubiquitin-like protein, Pup (also known as Rv2111c), which was specifically conjugated to proteasome substrates in the pathogen *Mycobacterium tuberculosis*. Similar to ubiquitylation, pupylation occurs on Lys residues and requires proteasome accessory factor A (PafA). In a *pafA* mutant, pupylated proteins were absent and substrates accumulated, thereby connecting pupylation with protein degradation. So, like eukaryotes, bacteria might use a small protein modifier to control protein stability.