IN BRIEF

CELL MIGRATION

Putting a brake for cells to turn

The actin-related protein 2/3 (ARP2/3) complex nucleates branched actin networks. During cell migration, it is activated at the leading edge of cells to power the protrusion of the plasma membrane in lamellipodia. The small GTPase RAC directly activates the WAVE complex, which in turn activates the ARP2/3 complex. As proteins that contain an acidic motif inhibit ARP2/3 at other locations in the cell, Dang *et al.* performed a bioinformatics search for proteins with such a motif that might counteract WAVE at lamellipodia. They identified Arpin, which binds to ARP2/3 via the acidic motif to inhibit actin polymerization and is also activated by RAC. Thus, RAC signalling induces both activation and repression of ARP2/3 at lamellipodia. The inhibitory activity of Arpin has a steering function: cells depleted of Arpin migrated faster and straighter, whereas microinjection of Arpin in cells that normally move with straight trajectories led them to turn.

ORIGINAL RESEARCH PAPER Dang, I. *et al.* Inhibitory signalling to the Arp2/3 complex steers cell migration. *Nature* http://dx.doi.org/10.1038/nature12611 (2013)

MORPHOGENESIS

New action in the lumen

During kidney development, branching morphogenesis occurs in the ureteric bud epithelial tube. Here, using time-lapse microscopy in mouse kidneys, the authors observe that this involves a unique behaviour of dividing cells in the branching bud that they term 'mitosis-associated cell dispersal'. Specifically, they see that many cells delaminate into the lumen before division and that the daughter cell then reinserts into the original site of the epithelium by virtue of a thin basal process that it inherits. The other cell, by contrast, reinserts at varying positions (1–3 cell diameters away), and this contributes to cell rearrangements during development of this tissue. Thus, cell division does not seem to be restricted to the epithelial plane during bud morphogenesis.

ORIGINAL RESEARCH PAPER Packard, A. *et al.* Luminal mitosis drives epithelial cell dispersal within the branching ureteric bud. *Dev. Cell* http://dx.doi.org/10.1016/j.devcel.2013.09.001 (2013)

CELL DIVISION

Nuclear pore inheritance

In contrast to vertebrate cells, which dismantle their nuclear pore complexes (NPCs) during mitosis, there has been a strong interest in whether budding yeast might directly inherit NPCs during their closed cell division. Two groups now cement this, finding that NPC inheritance is driven by the nucleoporin Nsp1 that allows NPC uptake in daughter cells. Makio *et al.* show that NPC movement into daughter cells is disrupted when Nsp1 is depleted and that this requirement extends to the nucleoporins that Nsp1 interacts with. If this nucleoporin subcomplex is lacking, NPCs remain in the mother cell. Colombi *et al.* use a labelling technique to track both new and old nucleoporins during time-lapse analysis of dividing yeast cells and observe that newly synthesized Nsp1 accumulates in the daughter cell specifically, whereas other nucleoporins are distributed between both cells. They also show that a cytoplasmic pool of Nsp1 (and its interacting nucleoporins) are important for NPC inheritance by daughter cells. Together, these studies indicate that Nsp1 may promote access of NPCs to daughter cells.

ORIGINAL RESEARCH PAPERS Colombi, P. *et al.* The transmission of nuclear pore complexes to daughter cells requires a cytoplasmic pool of Nsp1. *J. Cell Biol.* **203**, 215–232 (2013) | Makio, T. *et al.* Inheritance of yeast nuclear pore complexes requires the Nsp1p subcomplex. *J. Cell Biol.* **203**, 187–196 (2013)