IN BRIEF

CELL ADHESION

Cdc42, Par6, and aPKC regulate Arp2/3-mediated endocytosis to control local adherens junction stability.

Georgiou, M. *et al. Curr. Biol.* 30 Oct 2008 (doi:10.1016/j.cub.2008.09.029)

Adherens junctions (AJs) link adjacent cells in an epithelial monolayer, and require the actin cytoskeleton for their stability and plasticity. The authors show that the Rho GTPase CDC42, together with the junctional polarity proteins partitioning defective-6 (PAR6) and atypical protein kinase C (aPKC), is required for maintaining the stability of AJs in epithelial tissue of *Drosophila melanogaster*. The actin-related protein-2/3 (ARP2/3) complex and Wiskott–Aldrich syndrome protein (WASP), which are regulators of actin-filament nucleation, and dynamin were also required. Endocytosis assays confirmed that actin-mediated, dynamin-dependent endocytosis is needed for the efficient turnover of AJ components, such as E-cadherin, which is, in turn, essential for AJ stability and plasticity.

CYTOSKELETON

Detection of GTP-tubulin conformation *in vivo* reveals a role for GTP remnants in microtubule rescues.

Dimitrov, A. *et al. Science* 16 Oct 2008 (doi:10.1126/science.1165401)

Microtubules consist of highly dynamic tubulin polymers that undergo alternating phases of growth and shrinkage, which are separated by catastrophe and rescue events. The authors selected a recombinant antibody that specifically recognizes GTP-bound tubulin, which enabled them to visualize GTP–tubulin at the plus ends of growing microtubules *in vivo* for the first time. Surprisingly, the antibody also revealed 'GTP remnants' along older parts of the microtubules, which suggests that GTP hydrolysis during polymerization is sometimes incomplete. GTP remnants coincided with domains of microtubule rescue, which implies that GTP remnants might have a role in rescue events, during which microtubules recover from catastrophe.

DNA REPAIR

53BP1 promotes non-homologous end joining of telomeres by increasing chromatin mobility.

Dimitrova, N. *et al. Nature* 19 Oct 2008 (doi:10.1038/nature07433)

53BP1 facilitates long-range DNA end-joining during V(D)J recombination.

Difilippantonio, S. *et al. Nature* 19 Oct 2008 (doi:10.1038/nature07476)

Double-strand breaks (DSBs) initiate the formation of foci that contain DNA-damage response proteins, including 53BP1, around the chromatin. Using unprotected telomeres to mimic DSBs, Dimitrova and colleagues show that the binding of 53BP1 is required for non-homologous end-joining (NHEJ) of DSBs, as demonstrated by the fusion of telomere ends. Time-lapse microscopy revealed that unprotected telomeres are more mobile and that this mobility depends on 53BP1 and ATM kinase, but not on a functional NHEJ pathway. The authors propose that the increased dynamics of the local chromatin facilitates NHEJ repair that involves distant sites. In fact, this is shown in a second paper, which reports a decreased efficiency in long-range V(D)J recombination in 53BP1-deficient lymphocytes. Difilippantonio and colleagues propose that the initiating lesions in V(D)J recombination induce high-affinity binding of 53BP1, which promotes the formation of 53BP1 homo-oligomers and thereby efficient long-range synapsis and V(D)J recombination.