IN BRIEF

CYTOSKELETON

Coronin switches roles in actin disassembly depending on the nucleotide state of actin

Gandhi, M. *et al. Mol. Cell* **34**, 364–374 (2009)

Cofilin promotes the severing and depolymerization of actin filaments, which is important for actin-based cellular processes, in conjunction with actin-binding proteins such as coronin. Surprisingly, coronin both promotes and inhibits cofilin-mediated actin disassembly. Gandhi *et al.* now shed light on this paradox by showing that coronin differentially influences cofilin activity depending on the nucleotide state of actin. Specifically, coronin blocks the cofilin-mediated severing of newly assembled actin filaments that are bound to ATP, or to the ATP–ADP intermediate, ADP with inorganic phosphate. By contrast, it promotes cofilin-mediated severing of older, ADP-bound actin filaments.

CELL DIVISION

Polo-like kinase 1 directs assembly of the HsCyk-4 RhoGAP/Ect2 RhoGEF complex to initiate cleavage furrow formation

Wolfe, B. A. *et al. PLoS Biol.* **7**, e1000110 (2009)

Plk1 self-organization and priming phosphorylation of HsCYK-4 at the spindle midzone regulate the onset of division in human cells

Burkard, M. E. *et al. PLoS Biol.* **7**, e1000111 (2009)

The centralspindlin complex, which includes the GTP activator protein HsCYK4 (also known as RACGAP1), is crucial for the assembly of the central spindle that forms between segregating chromosomes during anaphase. HsCYK4 also recruits the Rho-family guanine nucleotide-exchange factor ECT2 to the central spindle, which activates the small GTPase RhoA and initiates cytokinesis. Two studies now reveal that polo-like kinase 1 (PLK1) phosphorylates HsCYK4 to stimulate its interaction with ECT2. Wolfe *et al.* show that microtubules and the microtubule-associated protein PRC1 facilitate PLK1-mediated phosphorylation of HsCYK4. Burkard *et al.* show that PLK1 phosphorylates HsCYK4 specifically at Ser157 to enable ECT2 to bind. In short, PLK1 regulates the formation of the HsCYK4–ECT2 complex at the central spindle and the induction of cytokinesis.

DNA DAMAGE RESPONSE

Crystal structure of the Rad9–Rad1–Hus1 DNA damage checkpoint complex — implications for clamp loading and regulation

Doré, A. S. *et al. Mol. Cell* 14 May 2009 (doi:10.1016/j.molcel.2009.04.027)

DNA clamps allow DNA polymerase to achieve high processivity during DNA replication. In response to DNA damage, the RAD9–RAD1–HUS1 (9–1–1) heteromeric DNA clamp is loaded on to DNA, where it activates the checkpoint kinase ataxia telangiectasia and RAD3-related protein (ATR). It also interacts with several DNA repair enzymes. The authors determine the crystal structure of human 9–1–1 at a resolution of 2.9 Å, which reveals a toroidal structure that is similar to that of the DNA clamp proliferating cell nuclear antigen (PCNA). Previous reports suggest that 9–1–1 might substitute for PCNA, when it is inhibited by the cyclin-dependent kinase inhibitor p21, in the DNA damage response. However, Doré *et al.* show that 9–1–1 also binds to p21, which suggests that this might not be the case.