IN BRIEF

CELL SIGNALLING

Restricting non-canonical NFκB

The transcription factor nuclear factor-κB (NF-κB) is sequestered in the cytoplasm by inhibitor of κB (IκB) and activated by two pathways: canonical signalling involves the degradation of IκB by IKKα, IKKβ and NEMO (which together form the IKK complex) followed by the nuclear translocation of the NF-κB dimer; non-canonical signalling is mediated by NIK (NF-κB-inducing kinase)-dependent activation of IKKα, which results in the release of the mature p52 subunit from the NF-κB precursor p100. This study now shows that the basal levels of NIK (which is rapidly degraded in unstimulated cells) were enhanced in NEMO-deficient cells, suggesting that the presence of NEMO usually decreases NIK levels to prevent non-canonical NF-κB signalling. The authors found that an intact and catalytically active IKK complex, as well as NF-κB transcriptional activity, was required to reduce NIK levels. Thus, classic NF-κB signalling suppresses the accumulation of NIK to inhibit basal non-canonical NF-κB signalling.

ORIGINAL RESEARCH PAPER Gray, C. M. *et al.* Noncanonical NF-κB signaling is limited by classical NF-κB activity. *Sci. Signal.* **7**, ra13 (2014)

CELL CYCLE

Changing lipids

Eggert and colleagues report the first comprehensive analysis of how the lipidome changes from interphase to cytokinesis. They identified 11 lipid species (out of thousands) that accumulated fourfold in dividing cells, including sphingolipids and ceramides. Further experiments revealed that films made from mixtures of lipids from interphase or dividing cells had different physical properties, suggesting that lipids may have a mechanical role in cell division. RNAi-mediated knockdown of 23 lipid biosynthetic enzymes caused cytokinetic failure. Moreover, some of the lipids that were upregulated in dividing cells accumulated in midbodies (all sphingolipid derivatives), which indicates that local changes at the plasma membrane may have crucial functions during cell division, either directly, or by affecting membrane-associated signalling and the cytoskeleton. This study opens new avenues for investigating the role of specific lipids at the subcellular level.

ORIGINAL RESEARCH PAPER Atilla-Gokcumen, G. E. *et al.* Dividing cells regulate their lipid composition and localization. *Cell* http://dx.doi.org/10.1016/j.cell.2013.12.015 (2014)

WOUND HEALING

ESCRTs help repair membranes

A study now shows that the endosomal sorting complex required for transport (ESCRT) machinery plays a part in plasma membrane repair. There are five ESCRT complexes: ESCRT-0, ESCRT-I, ESCRT-II, ESCRT-III and ESCRT-disassembly subcomplex. Jimenez *et al.* observed that CHMP4B — an ESCRT-III accessory protein that is required for all known functions of ESCRTs — localized to the plasma membrane of HeLa cells after they were wounded. Other ESCRT-III subunits (CHMP2A, CHMP2B and CHMP3) colocalized with CHMP4B. Interestingly, depletion of CHMP2A compromised the closure of holes smaller than 100 nm, and ESCRT-III function was found to be necessary for the survival of cells harbouring small wounds. The authors also observed the presence of extracellular buds and the shedding of ESCRT-positive membranes at wounded sites and proposed that "ESCRT-III proteins play a central role in repairing local injuries by ensuring extracellular shedding of damaged areas."

ORIGINAL RESEARCH PAPER Jimenez, A. J. *et al.* ESCRT machinery is required for plasma membrane repair. *Science* http://dx.doi.org/0.1126/science.1247136 (2014)