IN BRIEF

CELL POLARITY

PAR access control

The *Caenorhabditis elegans* zygote rapidly establishes regional domains of partitioning defective (PAR) polarity factors along the anterior–posterior axis. Microtubules can affect this, and Seydoux and colleagues now show that this is through microtubule-mediated local protection of PAR-2. Contact of the sperm centrosome, a microtubule-organizing centre, with the posterior cell cortex triggers polarization in two ways: by initiating cortical actomyosin flow; and by recruiting PAR-2. Seydoux and colleagues find that PAR-2, which is initially excluded throughout the cortex by uniform atypical protein kinase C (aPKC) distribution, is recruited to the sperm centrosome through direct microtubule binding; this locally protects PAR-2 from aPKC-mediated phosphorylation and initiates a feedback loop in which stabilized cortical PAR-2 directly recruits PAR-1. PAR-1-mediated phosphorylation of PAR-3 then triggers exit of PAR-3–aPKC from the posterior cortex. This mechanism appears to act very early in symmetry breaking, in parallel with the effects of actomyosin flow.

ORIGINAL RESEARCH PAPER Motegi, F. *et al.* Microtubules induce self-organization of polarized PAR domains in *Caenorhabditis elegans* zygotes. *Nature Cell Biol.* 9 Oct 2011 (doi:10.1038/ncb2354)

GENE EXPRESSION

An importin-α stress response

Nuclear import is facilitated by carrier proteins of the importin family, with importin-α acting as an adaptor that mediates the association of cargo with importin β1. In response to stress, importin-α rapidly accumulates in the nucleus, and Yoneda and colleagues have found that it has a direct role in regulating gene expression here. They show that nuclear importin α2 alters the transcription of a specific set of mRNAs, including Ser/Thr kinase 35 (STK35), and that it directly associates with the STK35 promoter region. Moreover, nuclear accumulation of importin α2 in response to oxidative stress also correlates with the induction of caspase-independent cell death. As increased levels of STK35 also trigger this pathway, this supports a model in which nuclear accumulation of importin-α in response to stress promotes STK35-mediated non-apoptotic cell death.

ORIGINAL RESEARCH PAPER Yasuda, Y. *et al.* Nuclear retention of importin α coordinates cell fate through changes in gene expression. *EMBO J.* 30 Sep 2011 (doi:10.1038/emboj.2011.360)

CIRCADIAN RHYTHMS

Modifying the clock

The circadian clock is controlled by a transcription–translation circuit that revolves around the transcription factors CLOCK and BMAL1. Chromatin modifications are important for maintaining circadian rhythms, but their precise role and how they are regulated remains unclear. DiTacchio *et al.* find that the histone demethylase JARID1A is recruited to the promoter of the *Per2* gene (which encodes a clock component) through the action of CLOCK and BMAL1. They show that JARID1A enhances CLOCK–BMAL1-dependent transcription from the *Per2* promoter and that, in its absence, *Per2* transcripts oscillate with a shorter period. This function of JARID1A did not require its demethylase activity. Instead, JARID1A promoted histone acetylation, possibly by inhibiting the activity of the HDAC1 deacetylase. The exact mechanism by which JARID1A mediates these effects on the clock remains to be determined.

ORIGINAL RESEARCH PAPER DiTacchio, L. *et al.* Histone lysine demethylase JARID1a activates CLOCK-BMAL1 and influences the circadian clock. *Science* **333**, 1881–1885 (2011)