IN BRIEF

CELL CYCLE

A role for BRCA2 in abscission

It had previously been suggested that breast cancer type 2 susceptibility (BRCA2) forms a complex with Aurora B during cytokinesis and may thus have a role in abscission. Here, Couch and colleagues show that, during cytokinesis, BRCA2 is recruited to the midbody by interacting with the actin-binding protein filamin A. There, BRCA2 has a role in recruiting endosomal complexes required for transport (ESCRTs) and ESCRT-interacting proteins such as CEP55, which in turn mediate abscission. Importantly, mutations that inhibit binding of BRCA2 to filamin A disrupt localization of midbody components, and thus abscission, but have no effect on homologous recombination. So, this study confirms that BRCA2 plays a part in abscission that is independent of its role in DNA repair.

ORIGINAL RESEARCH PAPER Mondal, G. *et al.* BRCA2 localization to the midbody by filamin A regulates CEP55 signaling and completion of cytokinesis. *Dev. Cell* **23**, 1–16 (2012)

AUTOPHAGY

BIM doubles its responsibilities

The pro-apoptotic BH3-only protein BIM is inactivated by binding to the dynein light chain protein LC8. However, under conditions of stress BIM is phosphorylated by Jun N-terminal kinase (JNK), which leads to its dissociation from LC8 and localization at mitochondria, where it can induce apoptosis. This study finds that the interaction of BIM with LC8 also regulates autophagy. Under steady-state conditions, LC8 mediates binding of apoptosis-inactive BIM to BECLIN1, leading to the mislocalization of the latter from the endoplasmic reticulum to microtubules, where it cannot promote autophagosome formation and maturation. However, under starvation conditions BIM is phosphorylated by JNK, thereby releasing BECLIN1 and allowing autophagy induction. Consistent with this, loss of BIM leads to enhanced autophagosome induction. Thus, similarly to anti-apoptotic BH3-only proteins, BIM has a key role in inhibiting autophagy under steady-state conditions.

ORIGINAL RESEARCH PAPER Luo, S. *et al.* BIM inhibits autophagy by recruiting BECLIN1 to microtubules. *Mol. Cell* **47**, 1–12 (2012)

CIRCADIAN RHYTHMS

Small molecule time management

A balanced circadian rhythm requires intricate control of transcription regulatory networks, including regulated expression of the clock protein cryptochrome (CRY). By screening a library of ~60,000 small molecules for their effects on circadian rhythm in mammalian cell lines, Hirota *et al.* identify the small molecule KL001 as a specific inhibitor of CRY degradation. They show that KL001 targets an E3 ligase that normally mediates CRY turnover, and KL001 therefore triggers a longer circadian period. By combining mathematical modelling and treatment of *Cry1*- or *Cry2*-knockout cells with KL001, they were able to show that the two CRY isoforms have a common role in regulation of period length. Finally, the effects of KL001 in blocking gluconeogenesis triggered by glucagon in primary hepatocytes showed that this inhibitor may have relevance for diabetes treatment.

ORIGINAL RESEARCH PAPER Hirota, T. *et al.* Identification of small molecule activators of cryptochrome. *Science* 12 Jul 2012 (doi:10.1126/science.1223710)