IN BRIEF

CYTOSKELETON

Controlling microtubules with light

The activity of traditional small-molecule inhibitors of microtubules cannot be restricted to target cells in research and chemotherapy. Borowiak *et al.* have developed optically controlled photostatins against microtubules by chemically modifying the microtubule inhibitor combretastatin A4 so that it can be reversibly switched between its *trans*, inactive form and its *cis*, toxic form in response to light; a series of photostatins were generated. The toxicity of photostatins is highly specific and reversible; photostatins were non-toxic to cancer cell lines in the dark but highly toxic in the light (maximum toxicity was achieved with 390 nm light). Photostatins were shown to inhibit tubulin polymerization, and their biological importance was demonstrated on several levels. Photostatins induced cell death and mitotic arrest in several cell lines. Moreover, they reversibly controlled microtubule dynamics and induced mitotic arrest in a single cell within a developing *Caenorhabditis elegans* embryo.

ORIGINAL RESEARCH PAPER Borowiak, M. *et al.* Photoswitchable inhibitors of microtubule dynamics optically control mitosis and cell death. *Cell* http://dx.doi.org/10.1016/j.cell.2015.06.049 (2015)

POST-TRANSLATIONAL MODIFICATIONS

Resisting heat stress with SUMO

Heat shock or other proteotoxic stresses that cause the accumulation of misfolded proteins promote the conjugation of small ubiquitin-like modifier 2 (SUMO2) to nuclear proteins; the chromatin-binding profile of SUMO2 during heat shock is not fully characterized. By combining data from CHIP–seq and RNA-seq with previously published proteomics data, Hay and colleagues now find that, following heat shock, SUMO2 is conjugated to large protein complexes; these complexes are associated with the regulatory elements of active genes that encode regulators of gene expression and the post-transcriptional modification of RNA. SUMO2 did not directly activate or inhibit transcription, but its conjugation was required to maintain the maximal expression of target genes. The authors propose that sumoylation is an integral component of the proteotoxic stress response that helps to maintain the integrity of transcription regulatory protein complexes.

ORIGINAL RESEARCH PAPER Seifert, A. *et al.* Proteotoxic stress reprograms the chromatin landscape of SUMO modification. *Sci. Signal.* **8**, rs7 (2015)

DNA DAMAGE RESPONSE

'enABLing' the Microprocessor

Studying the role of the Tyr kinase ABL in promoting apoptosis through DNA damage-mediated activation of microRNAs, Wang and colleagues identified the pro-apoptotic microRNA miR-34c as an effector of ABL in human kidney cells. The processing of functional miR-34c from a primary miR-34c (pri-miR-34c) minigene (but not of the closely related miR-34a from a pri-miR-34a minigene) increased following the expression of constitutively active ABL and was reduced by inhibiting the Microprocessor complex subunit Drosha. Conversely, the other Microprocessor subunit, DGCR8, bound to pri-miR-34c and blocked its processing; this effect was reduced by the phosphorylation of DGCR8 at Tyr267 by ABL. This phosphorylation probably stimulated recruitment of Drosha to DGCR8 and microRNA processing. Additionally, in mouse kidneys, nuclear ABL was required for the proper expression of miR-34c (but not that of miR-34a).

ORIGINAL RESEARCH PAPER Tu, C.-C. *et al.* The kinase ABL phosphorylates the microprocessor subunit DGCR8 to stimulate primary microRNA processing in response to DNA damage. *Sci. Signal.* **8**, ra64 (2015)