IN BRIEF

GENE EXPRESSION

CTD Tyr1 gives direction

The carboxy-terminal domain (CTD) of the large subunit of RNA polymerase II (Pol II), RBP1, consists of repeats of the sequence YSPTSPS. Post-transcriptional modifications at specific CTD residues are crucial for transcriptional regulation, and two studies now describe a role for RBP1 Tyr1 phosphorylation in mammalian cells. Substitution of Tyr1 residues in CTD repeats led to the partial degradation of RPB1 to a truncated, 'CTD-less' form, which suggests a role of this residue in RBP1 stability. Furthermore, Descostes *et al.* showed that phosphorylated Tyr1 is associated with the 5′ end of gene promoters and antisense transcription, and Hsin *et al.* reported that Tyr1 phosphorylation has a role in the expression of upstream antisense transcripts. Thus, Tyr1 phosphorylation of the CTD functions in Pol II stability and may control promoter directionality.

ORIGINAL RESEARCH PAPERS Hsin, J.-P. *et al.* RNAP II CTD tyrosine 1 performs diverse functions in vertebrate cells. *eLife* http://dx.doi.org/10.7554/eLife.02112 (2014) | Descostes, N. *et al.* Tyrosine phosphorylation of RNA Polymerase II CTD is associated with antisense promoter transcription and active enhancers in mammalian cells. *eLife* http://dx.doi.org/10.7554/eLife.02105 (2014)

AUTOPHAGY

Structural insight into autophagy initiation

In yeast, the starvation-induced dephosphorylation of Atg13 enables it to form the Atg1 complex (comprising Atg1, Atg13, Atg17, Atg29 and Atg31); this complex is necessary to assemble the pre-autophagosomal structure (PAS) and thus to initiate autophagy. Fujioka *et al.* solved the structure of the Atg1 carboxy-terminal domain in complex with the minimum Atg1-binding region of Atg13, and of Atg17–Atg29–Atg31 in complex with the minimum Atg17-binding region of Atg13, using bacterial homologues of the yeast proteins. The analysis revealed that Atg1 harbours two tandem microtubule-interacting and transport domains that bind to Atg13. In Atg17 a hydrophobic pocket, and its surrounding acidic residues, comprise the Atg13-binding site. Importantly, dephosphorylation of Ser residues in Atg13 increased its interaction with Atg1 and Atg17, which explains how formation of the Atg1 complex, and thus of the PAS, is driven by starvation-induced dephosphorylation of Atg13.

ORIGINAL RESEARCH PAPER Fujioka, Y. *et al.* Structural basis of starvation-induced assembly of the autophagy initiation complex. *Nature Struct. Mol. Biol.* http://dx.doi.org/10.1038/nsmb.2822 (2014)

MECHANOTRANSDUCTION

Vinculin discrimination at adhesions

Cells sense external mechanical forces through cadherins (which form cell–cell adhesions) and integrins (which form cell–matrix adhesions). Vinculin (VCL) accumulates at both types of adhesions, where it may exert distinct functions. Bays *et al.* show that VCL is phosphorylated at Tyr822 in cell–cell adhesions but not in cell–matrix adhesions; applying force to cadherins (but not integrins) increased Tyr822 phosphorylation. Tyr822 phosphorylation has a physiological role, as applying force to cadherins in *VCL*-knockout cells rescued with wild-type VCL, but not with VCL in which Tyr822 was substituted, enabled cells to stiffen. By contrast, both cell types underwent stiffening when force was applied to integrins. Finally, VCL was phosphorylated at Tyr822 by the Tyr kinase ABL, the activity of which was increased when force was applied to cadherins. Thus, ABL-mediated phosphorylation of VCL at Tyr822 is necessary for cadherins, but not integrins, to transmit force.

ORIGINAL RESEARCH PAPER Bays, J. L. *et al.* Vinculin phosphorylation differentially regulates mechanotransduction at cell–cell and cell–matrix adhesions. *J. Cell Biol.* **205**, 251–263 (2014)