

IN BRIEF

POST-TRANSLATIONAL MODIFICATION**A new histone mark**

The role of known histone modifications, such as Lys acetylation and methylation, in transcriptional control and epigenetics is well-established. Now, Dai *et al.* identify a novel histone mark, Lys 2-hydroxyisobutyrylation (Khib), and find that this modification is highly abundant and evolutionarily conserved in eukaryotic cells. The authors went on to show that Khib and Lys acetylation marks are located at different histone residues and that Khib is found at amino-terminal histone domains, where most Lys acetylation marks exist, but it also occurs more widely throughout the core histone. Interestingly, the distribution of 2-hydroxyisobutyrylated histone 4 at Lys8 (H4K8hib) is associated with transcriptionally active genes during male germ cell differentiation. Together, these results suggest that Khib is a novel regulator of chromatin function.

ORIGINAL RESEARCH PAPER Dai, L. *et al.* Lysine 2-hydroxyisobutyrylation is a widely distributed active histone mark. *Nature Chem. Biol.* <http://dx.doi.org/10.1038/nchembio.1497> (2014)

PROTEIN FOLDING**Protein aggregation inhibits endocytosis**

The molecular chaperone HSC70 is required for multiple steps of clathrin-mediated endocytosis (CME). Yu *et al.* now show that CME is inhibited in prostate cancer cells and neuronal cells by aggregates of neurodegenerative disease-related proteins, including those formed by polyglutamine (polyQ) expansions such as Q82. Aggregates in the cytoplasm and in the nucleus inhibited CME by sequestering HSC70. Importantly, depletion of HSC70 using RNAi phenocopied the aggregate-induced inhibition of CME, and expressing HSC70 in cells that contain Q82 aggregates increased CME. This mechanism is relevant to neuropathologies, as CME of the neuron-specific cargo AMPA (α -amino-3-hydroxy-5-methyl-4-isoxazole propionic acid) receptor was compromised in the presence of Q82, and CME was reduced in neurons containing aggregates of mutant huntingtin, which recruited HSC70.

ORIGINAL RESEARCH PAPER Yu, A. *et al.* Protein aggregation can inhibit clathrin-mediated endocytosis by chaperone competition. *Proc. Natl Acad. Sci. USA* <http://dx.doi.org/10.1073/pnas.1321811111> (2014)

CELL CYCLE**Cnn as a scaffold for centrosome maturation**

Centrosomes comprise two centrioles that are surrounded by pericentriolar material (PCM). The PCM increases in size during mitosis, as centrioles recruit new PCM from the cytosol — a process known as centrosome maturation. Raff and colleagues studied PCM assembly in mitotic centrosomes of *Drosophila melanogaster* embryos. Using photoactivation and three-dimensional-structured illumination super-resolution microscopy, they found that Centrosomin (Cnn) is incorporated into the PCM at the centrosome centre and then moves towards the periphery, where it detaches from the PCM. In this way, Cnn forms a scaffold-like structure that emanates from centrioles. Centrosome-specific phosphorylation of Cnn by Plk1 (Polo-like kinase 1) was required for this outward movement and for centrosome maturation. This suggests a model in which a Cnn scaffold assembles at the onset of mitosis, when Plk1 is activate, to recruit other PCM proteins.

ORIGINAL RESEARCH PAPER Conduit, P. T. *et al.* The centrosome-specific phosphorylation of Cnn by Polo/Plk1 drives Cnn scaffold assembly and centrosome maturation. *Dev. Cell* **28**, 659–669 (2014)