IN BRIEF

AUTOPHAGY

Hox times autophagy

Autophagy is regulated by stimuli, including developmental signals such as the steroid hormone ecdysone in flies and environmental cues such as nutrient deprivation. This study shows that in the *Drosophila melanogaster* fat body, autophagy is inhibited by Hox proteins — transcription factors that drive morphogenesis along the anterior–posterior axis. The authors found that Hox genes are downregulated at the onset of developmental autophagy and following starvation, suggesting that Hox proteins inhibit autophagy. Temporal Hox expression was decreased by ecdysone (developmental)-mediated as well as target of rapamycin and insulin receptor (starvation)-mediated downregulation of Pontin (a known component of the Brahma chromatin remodelling complex that maintains Hox gene expression). Finally, Hox proteins repressed *Atg* and other autophagy genes to inhibit autophagy. Thus, in addition to providing spatial information during development, Hox proteins act as temporal negative regulators of autophagy.

ORIGINAL RESEARCH PAPER Banreti, A. *et al.* Hox proteins mediate developmental and environmental control of autophagy. *Dev. Cell* http://dx.doi.org/10.1016/j.devcel.2013.11.024 (2014)

CHROMOSOMES

Histone modifications in mitosis

Wilkins *et al.* now show that, in yeast, a series of histone post-translational modifications promotes the interaction of histone 2A (H2A) and H4 to induce chromosome condensation during mitosis. They devised a system to monitor H2A–H4 interactions, which peaked in mitosis and correlated with the phosphorylation of H3 at Ser10 (H3S10) and a decrease in acetylation of H4 at Lys16 (H4K16). Investigating these relationships further, they found that acetylated H4K16 decreased H2A–H4 interactions *in vivo* during mitosis and that H3 phosphorylated at Thr3 (by Haspin) and then at Ser10 (by the chromosomal passenger complex) promoted the deacetylation of acetylated H4K16 by Hst2p and therefore H2A–H4 interactions. Mutation of Hst2p or H3S10 prevented the condensation of a long compound chromosome during mitosis, confirming that these histone modifications work together to promote the condensation of mitotic chromosomes.

ORIGINAL RESEARCH PAPER Wilkins, B. J. *et al.* A cascade of histone modifications induces chromatin condensation in mitosis. *Science* **343**, 77–80 (2014)

PROTEIN METABOLISM

N-terminal Met can trigger degradation

The N-end rule pathway for regulated protein degradation is crucial for many cellular functions. This pathway recognizes amino-terminal degradation signals (N-degrons) and promotes target protein ubiquitylation and degradation by the proteasome. There are two branches of this pathway in eukaryotes: the Arg/N-end rule pathway, which targets a specific set of unacetylated N-terminal residues that become exposed after co-translational cleavage of Met by specific enzymes, and the Ac/N-end rule pathway, which recognizes proteins with N^{α}-terminally acetylated residues; most proteins are N^{α}-terminally acetylated. Kim *et al.* find that the Arg/N-end rule pathway targets a much larger set of proteins than previously appreciated, as its dedicated E3 ubiquitin ligase, Ubr1, also recognizes unacetylated N-terminal Met followed by a hydrophobic residue.

ORIGINAL RESEARCH PAPER Kim, H.-K. *et al.* The N-terminal methionine of cellular proteins as a degradation signal. *Cell* http://dx.doi.org/10.1016/j.cell.2013.11.031 (2014)