IN BRIEF

SIGNALLING

PKA-dependent regulation of the histone lysine demethylase complex PHF2–ARID5B

Baba, A. *et al. Nature Cell Biol.* 1 May 2011 (doi:10.1038/ncb2228)

The idea that signalling pathways can directly regulate chromatin-modifying factors and thus provide rapid control over gene expression is gaining ground. Baba *et al.* show here that the PHF2 histone Lys demethylase of the Jumonji C family is directly regulated by protein kinase A (PKA). They show that PHF2 alone is enzymatically inactive but that direct phosphorylation by PKA triggers its association with the DNA-binding protein ARID5B, promoting PHF2-mediated demethylation of ARID5B. This allows the PHF2–ARID5B complex to associate with target promoters and to remove repressive histone dimethylation on Lys9 of histone 3 (H3K9Me2). This seems to be physiologically relevant: under conditions of fasting in mice, which activate PKA, PHF2–ARID5B localizes to target promoters in the liver, and this correlates with reduced levels of the H3K9Me2 modification.

NUCLEAR ORGANIZATION

Targeting of the SUN protein Mps3 to the inner nuclear membrane by the histone variant H2A.Z

Gardner, J. M. *et al. J. Cell Biol.* **193**, 489–507 (2011)

SUN proteins are conserved inner nuclear membrane (INM) proteins that have essential roles in organizing nuclear architecture by positioning specific chromosomal domains at the nuclear periphery. Gardner *et al.* find that the histone variant H2A.Z has a chromatin-independent function in the localization of the sole SUN protein in *Saccharomyces cerevisae*, monopolar spindle protein 3 (Mps3), to the INM. Using a series of point and deletion mutants, they find that Mps3 binds to regions of the amino-terminal domain and histone fold that are unique to H2A.Z. The H2A.Z–Mps3 interaction is not essential for the previously known roles of H2A.Z, but is required for Mps3 to localize to the INM. These results reveal a novel chromatin-independent function for H2A.Z in the regulation of nuclear architecture.

GENE EXPRESSION

Nuclear actin polymerization is required for transcriptional reprogramming of *Oct4* by oocytes

Miyamoto, K. *et al. Genes Dev.* **25**, 946–958 (2011)

Amphibian oocytes contain an abundance of nuclear actin; they can also reprogramme transplanted somatic cells. A role for nuclear actin in gene reactivation has not previously been investigated, but Miyamoto *et al.* now report that the polymerization of nuclear actin is required for reactivating the pluripotency gene *oct4* (also known as *pou5f1*). Inhibiting nuclear β-actin (using a specific antibody) or actin polymerization (using actin polymerization mutants or cytochalasin) strongly reduced *oct4* reactivation. The authors identify toca1 (also known as fnbp1l), an actin-signalling protein, as a positive regulator of nuclear actin polymerization and *oct4* transcriptional reactivation. Chromatin immunoprecipitation analysis showed that toca1 facilitates the binding of nuclear actin and chromatin factors to *oct4*, presumably contributing to its reactivation. Nuclear actin, but not toca1, was also found to be important for continuous *oct4* expression. So, endogenous nuclear actin — most likely both filamentous and globular forms — is involved in transcriptional reprogramming.

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