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IN BRIEF

POST-TRANSLATIONAL MODIFICATIONS

ERα activation through methylation

Histone methylation by the methyltransferase G9a causes gene silencing, whereas the interaction of G9a with nuclear receptors such as oestrogen receptor-α (ERα) leads to gene activation through a poorly understood mechanism. Zhang *et al.* showed that the depletion of G9a in ER-expressing breast cancer cell lines reduced oestrogen-induced activation of hundreds of genes, as well as cell proliferation and transformation. These effects were at least partially dependent on the methyltransferase activity of G9a that dimethylated ERα at Lys235, leading to the binding of ERα by PHF20, which is a subunit of the histone acetyltransferase complex MOF. Following oestrogen treatment, G9a-dependent interaction of MOF with ERα at ERα target gene promoters induced histone H4 Lys16 acetylation and enhanced transcription.

ORIGINAL ARTICLE Zhang, X. *et al.* G9a-mediated methylation of ERα links the PHF20/MOF histone acetyltransferase complex to hormonal gene expression. *Nat. Commun.* **7**, 10810 (2016)

TRANSCRIPTION

Pol II and topoisomerase 1 hand-in-hand

The RNA polymerase II (Pol II) machinery generates DNA torsional stress, which, if not relieved by topoisomerase 1 (TOP1) through DNA swivelling, can impede transcription. Baranello *et al.* developed TOP1-seq to map the position of catalytically active TOP1 and found that it is enriched at sites of Pol II pausing, just downstream of transcription start sites. Pol II was found to interact through its carboxy-terminal domain (CTD) with TOP1 and stimulate its activity. CTD phosphorylation at Ser2 by BRD4 promotes Pol II pause–release and transcription elongation, and the authors found that it is also required for TOP1 stimulation. Thus, Pol II and TOP1 constitute a positive feedback loop where Pol II stimulates TOP1, which in turn facilitates Pol II pause–release and transcription elongation.

ORIGINAL ARTICLE Baranello, L. *et al.* RNA polymerase II regulates topoisomerase 1 activity to favor efficient transcription. *Cell* **165**, 357–371 (2016)

PLANT DEVELOPMENT

Non-cell-autonomous retrotransposon silencing

To avoid mutagenesis, the expression of retrotransposons in plant germ cells is repressed by various mechanisms, including post-transcriptionally by retrotransposon-derived short interfering RNAs (siRNAs). Martínez *et al.* investigated whether *Arabidopsis thaliana* sperm cell retrotransposons can be silenced by siRNAs that are produced in the pollen vegetative (non-reproductive) cell that surrounds sperm cells. They found that in RNAi-competent vegetative cells, a pollen retrotransposon-derived siRNA that can target a GFP transcript containing the siRNA target site caused its degradation into GFP-targeting secondary siRNAs, which in turn silenced the expression of another GFP transgene that was expressed in sperm cells. Furthermore, the expression of a GFP transgene containing the retrotransposon-derived siRNA target site was enabled in sperm cells upon the suppression of RNAi in the vegetative cell, demonstrating that endogenous siRNAs expressed in vegetative cells can repress retrotransposons in sperm cells.

ORIGINAL ARTICLE Martínez, G. *et al.* Silencing in sperm cells is directed by RNA movement from the surrounding nurse cell. *Nat. Plants* **2**, 16030 (2016)