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EPIGENETICS

Jump-starting transposons

Understanding how transposons contribute to genome diversity and evolution means investigating not only how they move but also which host mechanisms keep them in check. Two genome-wide studies have investigated the epigenetic mechanisms that silence transposon activity in *Arabidopsis thaliana*. Together they have found that transposon families are controlled by a diverse set of epigenetic processes and that regulation can extend beyond transcriptional suppression.

The genome of *A. thaliana* is rife with mobile elements, particularly long terminal repeat (LTR) retrotransposons, potentially making it ideal for studying transposon dynamics. The only problem is that the endogenous mobile elements of this species are largely immobile, probably as a result of host-encoded epigenetic suppression. Two groups have examined plants in which DNA methylation is impaired: in this background, several families of transposon are reawakened, which allows the analysis of transposon regulation.

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In the first study, Tsukahara, Kobayashi and colleagues looked at mutants in the chromatin remodelling gene *DECREASE IN DNA METHYLATION 1* (*DDM1*). These plants have several developmental defects, and the *wavy sepal* phenotype in particular (see figure; the wild type is shown at the top) was found to be caused by the insertion of a *gypsy* class retrotransposon (*ATGP3*) into a developmental gene (*FASCIATA1*). *ATGP3* was mobilized in all 12 lines of *ddm1*, which suggests that such elements are silenced by methylation. Genetic mutant studies then revealed that the silencing requires methylation at both CG and non-CG sequences. *ATGP3* was not the only element to be mobilized: elements from multiple *copia* and *Mutator* families also transposed. Interestingly, the pattern and rate of transposition of the various elements varies widely across *ddm1* lineages and among *Arabidopsis* species in natural populations.

The plants studied by Mirouze, Reinders and colleagues were mutants in the methyltransferase gene *MET1*. Transposons are not normally activated in this background; however, among the recombinant inbred progeny of *met1* and wild-type parents, a *copia* element, *Évadé* (*EVD*), escaped suppression and was used to study the mechanisms that normally keep it silenced. In the *met1* lines and the lines in which *EVD* was activated, the element had unmethylated CGs in its 5′ LTR and produced full-length transcripts. What then was preventing *EVD* movement in the *met1* background? A candidate mechanism was RNA-directed DNA methylation (RdDM), a process in which small RNAs direct DNA methylation and therefore silencing. Indeed, double mutations of *met1* and either of the two components of the RdDM machinery acted synergistically in increasing *EVD* transcript levels and provoked transposition. Double mutations of *met1* and *kryptonite* (which encodes the main histone H3 lysine 9 methyltransferase) showed no effects on *EVD* transcript levels but activated element movement, which indicated that post-transcriptional processes were also operating. Together, RdDM-linked small RNAs and histone H3 lysine 9 methyltransferase are essential to ensure the post-transcriptional suppression of *EVD* — but they do not affect other elements.

Until now, most studies of *A. thaliana* transposons have been restricted to elements that are experimentally introduced from other species. These studies provide the opportunity to unleash transposon activity and use the tools of *A. thaliana* genetics to understand the molecular dynamics and evolutionary history of plant genomes.

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ORIGINAL RESEARCH PAPERS Tsukahara, S. & Kobayashi, A. *et al.* Bursts of retrotransposition reproduced in *Arabidopsis*. *Nature* 6 Sep 2009 (doi:10.1038/nature08351) | Mirouze, M. & Reinders, J. *et al.* Selective epigenetic control of retrotransposition in *Arabidopsis*. *Nature* 6 Sep 2009 (doi:10.1038/nature08328)
FURTHER READING Slotkin, R. K. & Martienssen, R. Transposable elements and the epigenetic regulation of the genome. *Nature Rev. Genet.* **8**, 272–285 (2007)