
At the crossroads of epigenetic pathways: H3K27me3 and transposable elements

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Abstract

The histone modification known as histone 3 lysine 27 trimethylation (H3K27me3) enables transcriptional repression of genes that is both stable across cell divisions and reversible in response to developmental and environmental signals. Despite increasing advances in understanding the mechanisms leading to the establishment of the H3K27me3 mark and its consequences at the gene level, our knowledge of its influence on the inactivation and/or mobilization of transposable elements (TEs) remains extremely limited. In differentiated *Arabidopsis* somatic tissues, H3K27me3 is present on approximately 10%-15% of TEs. The proportion of TEs targeted by H3K27me3 increases in tissues characterized by natural DNA hypomethylation, such as the endosperm, potentially for their repression. Furthermore, relocation of H3K27me3 marks on TEs is observed in mutants exhibiting DNA methylation defects. Constitutive heterochromatin on TEs appears to prevent the deposition of H3K27me3 and the chromatin state of TEs seems to be monitored by unknown factors that initiate the establishment of H3K27me3 marks in certain specific contexts. To shed light on the intricate molecular interplay between DNA methylation pathways and H3K27me3 deposition at the TEs level, I am employing a combination of genetic and genomic methodologies to address the following question: what chromatin landscape prompts the targeting of TEs by H3K27me3? Previous studies have noted a shift in the localization of the H3K27me3 mark on TEs in *met1* mutant plants. To investigate the impact of non-CG methylation on H3K27me3 relocation at TEs, I have disrupted the RdDM pathway in the *met1* background. The resulting *met1 nrpd2a/2b* mutants display severe phenotypes, including total sterility and homeotic defects, suggesting alterations in H3K27me3 profiles. ChIP-seq analysis of these profiles holds the potential to establish a functional connection between the RdDM and H3K27me3 pathways through yet-to-be-identified mediators. Additionally, integration of methylome and transcriptome analyses of these mutants will offer insights into the underlying mechanisms.

Keywords: DNA methylation, H3K27me3, *Arabidopsis thaliana*

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