The Effect of Histone Modifications Induced by Ack sRNAs on Ac Transposable Elements

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Chromosomal rearrangements caused by mobile transposons (TEs) are implicated in human cancers and neurological disorders. The Ac element of the Ac(Activator)/Ds(Dissociation) TE system, found in maize– a gold-standard model organism for TE studies– can alternatively transpose, producing 24-, 22-, and 21-nt Ac-killer (Ack) small-RNAs (sRNAs). While these sRNAs target and silence active Ac/Ds elements through DNA methylation, contributing to phenotypic changes, lethality, and sterility, the relationship between Ack-sRNAs, histone modifications, and the precise silencing mechanisms remains unknown. In this study, RNA, DNA, and chromatin were extracted at three maize developmental and one reproductive stage (embryo, leaf 3, leaf 10, silk) for RT-qPCR, qPCR, sRNA, and bisulfite sequencing in the id1(+Ack sRNAs) and 9d9a(-Ack sRNAs) genotypes. In the id1 genotype, significant H3K9me2 enrichment was observed at the internal Ac element region at all stages ($p<0.01$) and was consistent with decreased id1 Ac expression/activity ($p<0.04$). H3K27me3 enrichment was similarly observed in the internal region of id1 leaf 10 stage ($p=0.00987248$), with Ac expression/activity being the lowest ($p<0.001$). Furthermore, significant enrichments of both histone modifications ($p<0.0106129$) were limited to the Ac internal region (homologous to 21- and 22-nt Ack sRNAs); whereas, conversely, DNA methylation was higher in the Ac TIR (homologous to 24-nt Ack sRNAs). These findings suggest a novel silencing pathway of Ac/Ds elements through 21- and 22-nt Ack sRNA-induced H3K9me2 and H3K27me3, advancing the understanding of dynamic TE silencing and paving the way for future, targeted TE-based genetic therapies for transposition-induced diseases in both humans agriculture.

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