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Epigenetic Programming via DNA Deamination

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Methylation of DNA has evolved across many organisms as an epigenetic marker typically associated with transcriptional repression. As cells develop, their epigenetic profile changes to reflect their differentiated state, with the promoters of genes to be silenced getting methylated, and the promoters of genes whose expression is required losing their methylation. Although a significant amount of insight has been gained in the mechanisms of DNA methylation, those of active DNA demethylation are yet to be determined. It has long been postulated that replication-dependent "passive" DNA demethylation is one route of transcriptional regulation. In addition, recent studies suggest that the DNA deamination activity of Apo-lipoprotein B (APOBEC) family member Activation Induced cytidine Deaminase (AID) determines genomic DNA methylation patterns. Here, we discuss the evidence and the arguments regarding AID function in DNA demethylation. Understanding how methylation and demethylation are established and regulated is crucial for at least two reasons. First, some diseases, exhibit a change in their epigenetic profile due to DNA methylation dysregulation. Second, considerable efforts have been expended in the reprogramming of somatic cells to give rise to other cell types. The substantial therapeutic advantages that would result from such technology are invaluable, but they have been hampered in part by our inability to fully understand how DNA demethylation occurs.

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