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"The role of misregulated epigenetic mechanisms in cellular transformation and colorectal carcinoma"

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Genetic mutations are traditionally thought of as the primary culprit in the onset and development of cancer. However, it has become increasingly apparent that alterations in the epigenetic controls of gene expression that include DNA methylation, posttranslational modifications of histones, and microRNAs also contribute significantly to oncogenesis. While aberrant DNA hypermethylation is known to result in the epigenetic silencing of tumor suppressors in various cancers, the relationship between alterations in histone modifications and the pathogenesis of cancer are significantly less understood.

The proposed research is designed to advance the analysis of how misregulated epigenetic mechanisms lead to cellular transformation and oncogenesis. The primary goal is to examine the prominent histone modification, histone H3 trimethylated at lysine 4 (H3K4me3) and investigate how the deregulation of effector proteins that interpret this histone mark, actively contribute to aberrant gene expression and the transformation of colorectal carcinoma cells.