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Short Communication

Epigenetic Biomarkers for Diagnosis and Prognosis in Hematologic Cancers

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DESCRIPTION

Leukemia represents a heterogeneous group of hematologic malignancies characterized by the uncontrolled proliferation of immature blood cells in the bone marrow and peripheral blood. Although genetic mutations have long been recognized as major drivers of leukemogenesis, it has become increasingly evident that epigenetic mechanisms play a pivotal role in the initiation and progression of leukemia [1]. Epigenetic regulation refers to heritable changes in gene expression that do not involve alterations in the DNA sequence itself but instead are mediated by molecular modifications such as DNA methylation, histone modification, chromatin remodeling and non-coding RNA regulation. These mechanisms determine whether specific genes are turned on or off and maintain proper lineage commitment and differentiation of hematopoietic stem cells. When disrupted, they can lead to abnormal gene expression profiles that favor the expansion of leukemic clones [2].

The hematopoietic system relies heavily on a balanced regulation of gene expression to ensure normal differentiation of stem cells into various blood cell lineages. Epigenetic control mechanisms are crucial for maintaining this balance. DNA methylation, catalyzed by DNA methyltransferases, typically occurs at cytosine residues in CpG islands and leads to transcriptional repression of target genes. In normal hematopoiesis, DNA methylation ensures that lineage-inappropriate genes remain silent, while demethylation allows for the activation of genes required for specific differentiation pathways. Aberrant methylation patterns have been widely documented in leukemia. Conversely, global hypomethylation can promote chromosomal instability and activation of oncogenes, thereby contributing to leukemogenesis [3].

Histone modifications are another central aspect of epigenetic regulation. Histones, the protein components of chromatin, can undergo post-translational modifications such as methylation, acetylation, phosphorylation, ubiquitination and sumoylation. These chemical marks influence chromatin structure and accessibility of transcriptional machinery to DNA [4]. Histone acetylation, mediated by histone acetyltransferases, generally leads to chromatin relaxation and transcriptional activation, while histone deacetylation by Histone Deacetylases (HDACs) induces chromatin condensation and gene silencing. In leukemia, dysregulation of histone-modifying enzymes often results in abnormal activation of oncogenes or repression of differentiation-related genes. For instance, fusion proteins like AML1-ETO in Acute Myeloid Leukemia (AML) recruit HDACs to repress transcription of genes required for myeloid differentiation, locking cells in an immature state. Similarly, Mixed Lineage Leukemia (MLL) gene rearrangements disrupt normal histone methylation patterns and lead to aberrant expression of HOX genes, which are critical regulators of hematopoietic development [5].

Chromatin remodeling complexes also contribute to the epigenetic landscape of leukemia. These multi-protein assemblies use ATP hydrolysis to alter nucleosome positioning and thereby regulate the accessibility of transcription factors to DNA [6,7]. Mutations in components of these complexes, such as the SWI/SNF family, have been identified in several forms of leukemia. These alterations can lead to either inappropriate activation or repression of genes involved in proliferation and differentiation. Importantly, chromatin remodeling interacts dynamically with other epigenetic processes mutations in one mechanism can influence the function of others, producing a cumulative effect that enhances leukemic transformation [8,9].

Non-coding RNAs, including microRNAs and long non-coding RNAs, have emerged as additional layers of epigenetic regulation that influence leukemia development [10]. MicroRNAs function by binding to complementary sequences in target messenger RNAs, leading to mRNA degradation or inhibition of translation. Dysregulation of specific miRNAs has been implicated in leukemogenesis. Long non-coding RNAs can modulate gene expression through diverse mechanisms, including chromatin modification and transcriptional interference. Aberrant expression of lncRNAs such as HOTAIR and MALAT1 has been correlated with poor prognosis and resistance to therapy in leukemia patients.

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