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Opinion Article

Epigenetic Dynamics: *Tet2*-Mediated DNA Demethylation Modulates the Analysis of Leukemia K562 Cells

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DESCRIPTION

Mutations of *Tet2* often result in hematological malignancies. *Tet2* is one of the TET protein family members that are involved for active DNA demethylation. It is unclear, therefore, how *Tet2*-mediated demethylation and the incidence of hematological malignancies are related. As an *in vitro* model of erythroleukemia, the immortalized human leukemia K562 cell line was utilized. In this study, impact of *Tet2*-mediated demethylation on the proliferation and apoptosis of human leukemia K562 cells has been studied. The *Tet2* knockdown increased K562 cell proliferation and inhibited apoptosis, while TET2 enzymatic activity enhancement with α-KG decreased K562 cell proliferation and increased apoptosis. Consequently, the *Tet2* gene functions as a viable target for leukemia treatment, and small compounds that target the *Tet2* gene may be used in hematological malignancies to search for anti-tumor medicines. A well-studied chromatin modification, DNA methylation frequently takes place at the 5-carbon on cytosine residues in CpG dinucleotides. It is essential for many vital biological processes, including the development of mammals, the maintenance of stem cells, and the proliferation and differentiation of cells. It also had an impact on cancer, a topic of much inquiry because aberrant methylation is frequently connected to the development of tumors. DNA Methyltransferases (DNMTs) catalyze the methylation of DNA. The maintenance methyltransferase DNMT1 consistently preserves the parental DNA strands' methylation patterns during DNA replication, while the *de novo* DNA methyltransferases DNMT3A and DNMT3B create the first DNA patterns of methylation. While the mechanism of DNA demethylation remains unclear, the mechanism of DNA methylation has been well-characterized up to this point. The iron(II)/α-ketoglutarate (Fe(II)/α-KG)-dependent Ten-Eleven Translocation (TET) family of methyldioxygenases (*TET1*, *TET2*, and *TET3*) has recently been demonstrated to be involved in DNA demethylation by converting 5-methylcytosine (5mC) of DNA to 5-hydroxymethylcytosine (5hmC). Subsequent research revealed that TET proteins are also involved in the oxidation of 5hmC to 5-formyl and 5-carboxylcytosines (5fC and 5caC). Actually, the *MLL* gene's fusion partner from the breakpoint of chromosomal translocation t(10;11) (q22; q23) in Acute Myeloid Leukemia (AML) was found to be *TET1*, the earliest member of the TET family. In mammalian development, retrotransposon silencing, genomic imprinting, X chromosome inactivation, and cancer, DNA methylation is a critical regulatory factor. Global hypomethylation and hypermethylation of the CpG islands in the promoter characterize the highly dysregulated DNA methylation profiles seen in cancer cells. Genomic instability and dysregulated expression of tumor suppressor genes are frequently linked to aberrant methylation. DNA demethylation has been linked to the TET family (*Tet1*, *Tet2*, and *Tet3*), which is responsible for changing DNA's 5-methylcytosine (5mC) to 5-hydroxymethylcytosine (5hmC). Furthermore, somatic *TET2* mutations have been found to occur in a variety of hematological diseases, including lymphoid and myeloid cancers, to differing degrees. In patients with MDS, *TET2* mutations are found in 6%-26% of cases, CMML in 20%-58% of cases, and primary and secondary AML in 12%–32% of cases, blastic plasmacytoid dendritic neoplasm in 25%-54% of cases, and Myeloproliferative Neoplasms (MPNs) including polycythemia vera, primary myelofibrosis, and essential thrombocytosis in 2%-20% of cases. The landscape of mutations indicates that these changes may have a role in the development of hematological diseases. However, the exact method by which *TET2* proteins cause various cancers is still unknown, and it is yet unknown if *TET2*-mediated DNA demethylation contributes to these illnesses.

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