



Epigenomics in Immune Oncology and Cancer Therapy

Zing Hie*

Department of Biology, Jeju National University, Jeju, Republic of Korea

DESCRIPTION

Cancer is a complex and multifaceted disease, is driven by a combination of genetic and epigenetic changes. While genetic mutations have long been recognized as key contributors to cancer, recent advances in the field of epigenomics have clarified on the significant role that epigenetic alterations play in tumor genesis. Epigenomics, the study of the complete set of epigenetic modifications on the genetic material of a cell, offers profound insights into how these changes can influence cancer development, progression, and treatment. Heritable modifications in gene expression that do not involve changes to the underlying DNA sequence are referred to as epigenetics. These changes are mediated by several mechanisms, including DNA methylation, histone modification, and non-coding RNA molecules. Each of these mechanisms can modify the chromatin structure, thereby regulating the accessibility of the genetic material to the transcriptional machinery and ultimately influencing gene expression.

DNA Methylation is the addition of a methyl group to the cytosine bases of DNA, typically leading to gene silencing. This process for normal development and cellular differentiation, but aberrant methylation patterns can contribute to cancer by silencing tumor suppressor genes. Histone modifications involve the addition or removal of chemical groups to histone proteins around which DNA is wrapped. Deregulation of histone modification patterns is a common feature in various cancers and can lead to inappropriate activation or repression of genes involved in cell growth and division. Non-coding RNAs play a regulatory role in gene expression at the transcriptional and post-transcriptional levels. They can act as oncogenes or tumor suppressors, and their deregulation is associated with cancer development and progression.

Cancer cells exhibit widespread epigenetic deregulation. These changes can contribute to each stage of cancer development, from initial transformation to metastasis. The epigenetic landscape of cancer cells is characterized by global hypo methylation, leading to genomic instability, and localized hyper

methylation of tumor suppressor genes, resulting in their silencing. Global hypo methylation can activate oncogenes and promote chromosomal instability, a sign of cancer. This global loss of methylation can lead to the reactivation of transposable elements and the expression of normally silenced sequences, contributing to genomic instability. Promoter Hyper methylation of tumor suppressor genes is a well-documented phenomenon in cancer. This hyper methylation leads to the silencing of critical genes involved in cell cycle regulation, DNA repair, apoptosis, and other cellular processes, thereby facilitating uncontrolled cell growth and survival. Histone Modifications also play a critical role in cancer epigenomics. Abnormal patterns of histone acetylation and methylation are frequently observed in cancer cells. For instance, increased histone acetylation can lead to the overexpression of oncogenes, while decreased acetylation can silence tumor suppressor genes. Similarly, aberrant histone methylation patterns can disrupt normal gene regulation and promote tumor genesis. Dysregulated expression of miRNAs can lead to the repression of tumor suppressor genes or the activation of oncogenes. LncRNAs can also influence gene expression and chromatin structure, contributing to cancer development through various mechanisms.

The unique epigenetic alterations associated with cancer provide valuable biomarkers for diagnosis, prognosis, and treatment response. DNA methylation patterns, in particular, are stable and easily detectable in bodily fluids, making them ideal candidates for non-invasive cancer screening tests. For example, the methylation status of specific genes can serve as biomarkers for early detection of colorectal, breast, and lung cancers. Epigenetic markers can also provide prognostic information. For instance, the methylation status of certain genes can predict the aggressiveness of a tumor and the likelihood of recurrence, helping to guide treatment decisions. Furthermore, monitoring changes in epigenetic markers during treatment can provide insights into treatment efficacy and the development of resistance. Understanding the epigenetic basis of cancer has significant therapeutic implications. Epigenetic alterations are potentially reversible, making them attractive targets for cancer

Correspondence to: Zing Hie, Department of Biology, Jeju National University, Jeju, Republic of Korea, E-mail: hie_zing@email.com

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therapy. Several epigenetic drugs, known as epidrugs, have been developed and are in clinical use or trials.

DNA Methyl Transferase Inhibitors (DNMTis), such as azacitidine and decitabine, can reverse abnormal DNA methylation patterns and reactivate silenced tumor suppressor genes. These drugs have shown efficacy in treating hematological malignancies like myelodysplastic syndromes and acute myeloid leukemia. Histone Deacetylase Inhibitors (HDACis), such as vorinostat and romidepsin, can alter histone acetylation patterns and restore normal gene expression. HDAC inhibitors are being used to treat certain lymphomas and are being explored in combination with other therapies for solid tumors. Targeting Non-coding RNAs is an emerging area of cancer therapy.

Additionally, targeting lncRNAs involved in cancer progression holds potential for novel therapeutic approaches.

Epigenomics provides a comprehensive understanding of the epigenetic alterations that contribute to cancer. The insights gained from studying the epigenetic landscape of tumors have significant implications for cancer diagnosis, prognosis, and therapy. As research in this field continues to advance, it holds potential of developing more effective and personalized approaches to cancer treatment, ultimately improving outcomes for patients. The reversibility of epigenetic changes offers a unique therapeutic opportunity to combat this devastating disease, prepare for innovative treatments that can alter the course of cancer progression.