ISSN: 2157-2518
Journal of Carcinogenesis & Mutagenesis

OPEN ACCESS Freely available online

Commentary

Epigenetic Alterations in Carcinogenesis and Cancer Progression

Siyu Kokubun*

Department of Pathology, Tokyo Dental College, Kandamisaki-choTokyo, Japan

DESCRIPTION

Epigenetic alterations are heritable changes in gene expression that occur without changes in the underlying DNA sequence. These modifications regulate key cellular processes including growth, differentiation, and apoptosis. Unlike genetic mutations, epigenetic changes are potentially reversible, making them critical for understanding cancer development and for designing targeted therapies. Epigenetic alterations play a central role in carcinogenesis by disrupting normal regulatory pathways, promoting genomic instability, and enabling cells to acquire malignant characteristics.

The main mechanisms of epigenetic regulation include DNA methylation, histone modifications, and non-coding RNA mediated gene regulation. DNA methylation involves the addition of a methyl group to cytosine residues, typically within CpG islands near gene promoters. Hypermethylation of tumor suppressor gene promoters leads to gene silencing, preventing the expression of proteins that regulate cell cycle, DNA repair, or apoptosis. Conversely, global hypomethylation can destabilize the genome, activate oncogenes, and promote chromosomal rearrangements. Both hypermethylation and hypomethylation contribute to tumor initiation and progression.

Histone modifications, including acetylation, methylation, phosphorylation, and ubiquitination, regulate chromatin structure and accessibility of DNA to transcription machinery. Altered patterns of histone modifications can lead to aberrant gene expression, silencing of tumor suppressor genes, or activation of oncogenes. For example, loss of histone acetylation in specific regions can compact chromatin and prevent transcription of genes critical for controlling cell proliferation. Histone-modifying enzymes such as histone deacetylases and methyltransferases are often dysregulated in cancer, emphasizing the importance of epigenetic control in maintaining normal cellular functions.

Non-coding RNAs, including microRNAs and long non-coding RNAs, also contribute to epigenetic regulation. These molecules can repress or enhance the translation of target mRNAs, thereby influencing pathways involved in cell growth, differentiation, and survival. Dysregulation of non-coding RNAs can disrupt the balance between oncogenes and tumor suppressor genes, facilitating malignant transformation and tumor progression.

Epigenetic alterations can be influenced by environmental and lifestyle factors. Exposure to chemical carcinogens, tobacco smoke, ultraviolet radiation, and chronic inflammation can induce DNA methylation changes and modify histone marks. Dietary factors and metabolic conditions also affect epigenetic patterns, highlighting the dynamic interplay between the environment and gene regulation. This connection explains why cancers often arise from a combination of inherited genetic susceptibility and external exposures.

Epigenetic changes are critical not only for tumor initiation but also for tumor progression, metastasis, and therapy resistance. Altered gene expression patterns can promote angiogenesis, evade immune surveillance, and allow cancer cells to adapt to changing microenvironments. These features underscore the potential of targeting epigenetic alterations as part of cancer treatment strategies. Drugs such as DNA methyltransferase inhibitors and histone deacetylase inhibitors have shown promise in reversing aberrant epigenetic modifications and restoring normal gene expression in cancer cells.

Advances in epigenomics and sequencing technologies have enabled detailed mapping of epigenetic changes across different cancers. Identification of epigenetic biomarkers aids in early detection, prognosis, and predicting response to therapy. Because epigenetic alterations are reversible, therapies targeting these mechanisms offer a unique opportunity to complement conventional treatments such as chemotherapy, radiotherapy, and immunotherapy.

In conclusion, epigenetic alterations are central to the development and progression of cancer. By modulating DNA methylation, histone modifications, and non-coding RNA expression, these changes can silence tumor suppressor genes, activate oncogenes, and promote genomic instability. Environmental factors and cellular stress can further influence epigenetic patterns, highlighting the interplay between genetics,

Correspondence to: Siyu Kokubun, Department of Pathology, Tokyo Dental College, Kandamisaki-choTokyo, Japan. E-mail: siyukokubun@468.jp

Received: 29-Aug-2025, Manuscript No. JCM-25-30671; **Editor assigned:** 01-Sep-2025, Pre QC No. JCM-25-30671; **Reviewed:** 15-Sep-2025, QC No. JCM-25-30671; **Revised:** 22-Sep-2025, Manuscript No. JCM-25-30671; **Published:** 29-Sep-2025, DOI: 10.35248/2157-2518.25.16.002.

Citation: Kokubun S, (2025) Epigenetic Alterations in Carcinogenesis and Cancer Progression. J Carcinog Mutagen. S52:002.

epigenetics, and the environment in carcinogenesis. Understanding these mechanisms provides insights into early detection, prevention, and the development of targeted therapies, making epigenetic research a critical component of modern cancer biology.