

Epigenetic Reprogramming in Cancer: Tumour Cells Exploit Cellular Mechanisms

Jihoon Kim*

Department of Molecular Genetics, Seoul National University, Seoul, Korea

DESCRIPTION

Cancer, a complex and multifactorial disease, arises from the accumulation of genetic mutations that disrupt normal cellular functions. However, increasingly, it is becoming clear that genetic mutations alone are not enough to explain the full spectrum of cancer development. Epigenetics, the study of changes in gene expression that do not involve alterations to the underlying Deoxyribonucleic Acid (DNA) sequence, plays a pivotal role in tumorigenesis. In particular, cancer cells hijack normal epigenetic mechanisms to promote uncontrolled growth, evade cell death, and enhance their ability to metastasize. Understanding how cancer cells exploit these processes provides new insights into cancer biology and opens up potential avenues for targeted therapies.

Epigenetic changes in cancer cells

Cancer cells often exhibit widespread epigenetic reprogramming, primarily through global hypo methylation and localized hyper methylation.

Global hypo methylation: Many cancers show a reduction in DNA methylation across the genome, leading to the activation of oncogenes that promote uncontrolled cell growth and survival. This also destabilizes the genome, contributing to chromosomal instability, a sign of cancer.

Localized hyper methylation: Specific tumor suppressor genes, such as *p16INK4a*, *p53* and *BRCA1*, are often silenced by hyper methylation at their promoter regions. This prevents the regulation of cell growth and apoptosis, aiding tumor progression [1-3].

Histone modifications and chromatin remodelling

Histones undergo various modifications, such as acetylation and methylation, to regulate gene expression. In cancer cells:

- Decreased histone acetylation leads to a more condensed chromatin structure, silencing tumor suppressor genes.

- Dysregulated histone methylation can either silence tumor suppressor genes or activate oncogenes, promoting tumorigenesis. For example, the *H3K27me3* mark silences key genes involved in tumor suppression [4-6].

Epigenetic mechanisms for tumorigenesis

Cancer cells exploit epigenetic mechanisms in several ways to promote their survival and tumorigenic potential:

Increased cell proliferation: By silencing tumor suppressor genes and activating oncogenes through DNA methylation and histone modifications, cancer cells gain the ability to proliferate uncontrollably.

Resistance to apoptosis: Epigenetic changes often result in the silencing of genes that regulate apoptosis, such as *p53*, allowing cancer cells to evade programmed cell death and persist in the tumor microenvironment.

Genomic instability: Hypo methylation and other epigenetic alterations lead to genomic instability, which provides a selective advantage to cancer cells by accelerating the acquisition of mutations that further promote tumorigenesis.

Metastasis: Epigenetic modifications can also influence the ability of cancer cells to metastasize by regulating genes involved in cell migration and invasion. For example, altered expression of E-cadherin and matrix Metalloproteinases (MMPs) can facilitate the detachment of cancer cells from the primary tumor and their ability to invade surrounding tissues [7-9].

Targeting epigenetics in cancer therapy

The recognition of epigenetic alterations in cancer has led to the development of therapies targeting these changes. Drugs like DNA methyltransferase inhibitors and histone deacetylase inhibitors aim to reverse epigenetic modifications, restore normal gene expression, and inhibit tumor growth. These therapies offer potential new approaches in cancer treatment [10].

Correspondence to: Jihoon Kim, Department of Molecular Genetics, Seoul National University, Seoul, Korea, E-mail: hoon.0999ji@kim.kr

Received: 26-Nov-2024, Manuscript No. EROA-24-36017; **Editor assigned:** 28-Nov-2024, PreQC No. EROA-24-36017 (PQ); **Reviewed:** 12-Dec-2024, QC No. EROA-24-36017; **Revised:** 19-Dec-2024, Manuscript No. EROA-24-36017 (R); **Published:** 26-Dec-2024, DOI:10.35248/EROA.24.6.192

Citation: Kim J (2024). Epigenetic Reprogramming in Cancer: Tumour Cells Exploit Cellular Mechanisms. J Epigenetics Res. 6:192.

Copyright: © 2024 Kim J. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

CONCLUSION

Epigenetic alterations play a central role in the development and progression of cancer. By hijacking normal epigenetic mechanisms, cancer cells can drive their own tumorigenesis, evade immune surveillance, and acquire the ability to metastasize. As our understanding of the epigenetic landscape of cancer improves, it is likely that epigenetic therapies will become an important part of the clinical management of cancer, offering new hope for patients with difficult-to-treat cancers.

REFERENCES

1. Schiliro C, Firestein BL. Mechanisms of metabolic reprogramming in cancer cells supporting enhanced growth and proliferation. *Cells*. 2021;10(5):1056.
2. Dai E, Zhu Z, Wahed S, Qu Z, Storkus WJ, Guo ZS. Epigenetic modulation of antitumor immunity for improved cancer immunotherapy. *Mol Cancer*. 2021;20:1-27.
3. Gagliano T, Brancolini C. Epigenetic mechanisms beyond tumour-stroma crosstalk. *Cancers*. 2021;13(4):914.
4. Ohshima K, Morii E. Metabolic reprogramming of cancer cells during tumor progression and metastasis. *Metabolites*. 2021;11(1):28.
5. Gouirand V, Guillaumond F, Vasseur S. Influence of the tumor microenvironment on cancer cells metabolic reprogramming. *Front Oncol*. 2018;8:117.
6. Jiang H, Jedoui M, Ye J. The Warburg effect drives dedifferentiation through epigenetic reprogramming. *Cancer Biol Med*. 2024;20(12):891.
7. Elia I, Haigis MC. Metabolites and the tumour microenvironment: From cellular mechanisms to systemic metabolism. *Nat Metabol*. 2021;3(1):21-32.
8. Rodriguez R, Schreiber SL, Conrad M. Persister cancer cells: Iron addiction and vulnerability to ferroptosis. *Mol Cell*. 2022;82(4):728-740.
9. Hendrix MJ, Seftor EA, Seftor RE, Kasemeier-Kulesa J, Kulesa PM, Postovit LM. Reprogramming metastatic tumour cells with embryonic microenvironments. *Nat Rev Cancer*. 2007;7(4):246-255.
10. Haase S, Carney S, Varela ML, Mukherji D, Zhu Z, Li Y, et al. Epigenetic reprogramming in pediatric gliomas: From molecular mechanisms to therapeutic implications. *Trends Cancer*. 2024.