OPEN ACCESS Freely available online

Epigenetics Research: Open Access

Perspective

Overview of Epigenetics in Cancer Development and Progression

Khaled Gao*

Department of Cancer Biology, Bioterra University of Bucharest, Bucharest, Romania

DESCRIPTION

Cancer is a complex and heterogeneous disease, characterized by uncontrolled cell growth and invasion of surrounding tissues. The development of cancer is influenced by a combination of genetic and environmental factors. In recent years, there has been increasing recognition of the role of epigenetic modifications in the pathogenesis of cancer. Epigenetics refers to the regulation of gene expression through modifications to the DNA molecule and its associated proteins, rather than changes to the DNA sequence itself.

Epigenetic modifications are dynamic and reversible, allowing cells to respond to environmental stimuli and maintain cellular identity. The two most common types of epigenetic modifications are DNA methylation and histone modification. DNA methylation involves the addition of a methyl group to cytosine residues in DNA, usually at CpG dinucleotides. This modification is associated with gene silencing and is frequently observed in cancer cells. Histone modification refers to the addition or removal of chemical groups to histone proteins, which package and organize DNA in the nucleus. This modification can alter chromatin structure and accessibility, leading to changes in gene expression.

Aberrant epigenetic modifications have been implicated in various aspects of cancer development and progression, including tumor initiation, maintenance, and metastasis. For example, global hypomethylation, or a reduction in DNA methylation levels, can lead to genomic instability and chromosomal abnormalities, which can promote cancer development. Conversely, hypermethylation of specific tumor suppressor genes can lead to their silencing, allowing for uncontrolled cell growth and division.

Epigenetic modifications also play a critical role in the Epithelial-Mesenchymal Transition (EMT), a process in which cells lose their epithelial characteristics and acquire mesenchymal properties, allowing for increased motility and invasiveness. EMT is a critical step in cancer metastasis, and several epigenetic modifications have been identified as regulators of this process. For example, the transcription factor snail, which is a key inducer of EMT, can be upregulated through histone acetylation and downregulated through histone deacetylation.

In addition to their role in cancer development and progression, epigenetic modifications have also been implicated in the response to cancer therapy. Epigenetic alterations can mediate resistance to chemotherapy and radiation therapy, as well as targeted therapies such as tyrosine kinase inhibitors and immune checkpoint inhibitors. For example, the silencing of tumor suppressor genes through DNA methylation can lead to resistance to chemotherapy and targeted therapies. Additionally, the overexpression of Histone Deacetylases (HDACs), enzymes that remove acetyl groups from histones, has been associated with resistance to radiation therapy and immune checkpoint inhibitors.

Given the importance of epigenetic modifications in cancer biology, there has been increasing interest in developing epigenetic therapies for cancer treatment. Several epigenetic drugs have been approved by the FDA for the treatment of hematologic malignancies, including azacitidine and decitabine, which are DNA methyltransferase inhibitors, and vorinostat and romidepsin, which are HDAC inhibitors. These drugs have shown promising results in clinical trials and have been incorporated into standard treatment regimens for certain cancers.

CONCLUSION

However, the development of epigenetic therapies for solid tumors has proven to be more challenging. Solid tumors are often characterized by a heterogeneous cell population, which can lead to differences in drug sensitivity and resistance. Additionally, epigenetic modifications can be context-specific, meaning that the same modification may have different effects depending on the cellular context. This complexity has made it difficult to identify effective epigenetic targets for solid tumors and develop drugs that can effectively modulate epigenetic modifications in these tumors.

Correspondence to: Khaled Gao, Department of Cancer Biology, Bioterra University of Bucharest, Bucharest, Romania, E-mail: gaokhaled@hotmail.com

Received: 31-Jan-2023, Manuscript No. EROA-23-23242; **Editor assigned:** 02-Feb-2023, PreQC No. EROA-23-23242 (PQ); **Reviewed:** 16-Feb-2023, QC No. EROA-23-23242; **Revised:** 23-Feb-2023, Manuscript No. EROA-23-23242 (R); **Published:** 02-Mar-2023, DOI: 10.35248/EROA.23.5.132

Citation: Gao K (2023) Overview of Epigenetics in Cancer Development and Progression. J Epigenetics Res. 5:132.