Perspective
Volume 18:1, 2024

Journal of Molecular and Genetic Medicine

ISSN: 1747-0862 Open Access

Genetic and Epigenetic Drivers of Cancer Progression: Implications for Precision Medicine

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Abstract

Cancer is a complex and heterogeneous disease that results from genetic and epigenetic alterations in the genome. These alterations are known as drivers of cancer progression, which enable the acquisition of the hallmarks of cancer, including sustained proliferative signaling, evasion of apoptosis, and tissue invasion and metastasis. The identification of genetic and epigenetic drivers of cancer has led to the development of precision medicine approaches that aim to personalize cancer treatment based on the specific molecular alterations driving each individual patient's tumor. In this review, we discuss the genetic and epigenetic drivers of cancer progression and their implications for precision medicine.

Keywords: Cancer • Apoptosis • Metastasis • Heterogeneous disease • Genetic and epigenetic

Introduction

Cancer is a leading cause of death worldwide, with an estimated 10 million deaths in 2020. It is a complex and heterogeneous disease that arises from the accumulation of genetic and epigenetic alterations in the genome, leading to the acquisition of hallmarks of cancer, including sustained proliferative signaling, evasion of apoptosis, and tissue invasion and metastasis. Understanding the genetic and epigenetic drivers of cancer is critical for developing effective cancer therapies.

Genetic drivers of cancer progression

Genetic alterations in the genome can drive cancer progression by promoting the acquisition of the hallmarks of cancer. These alterations can be broadly categorized as oncogenes, which promote cell proliferation, and tumor suppressor genes, which inhibit cell proliferation. Oncogenes are activated by gain of function mutations or amplification, while tumor suppressor genes are inactivated by loss of function mutations or deletion.

One of the most well-known oncogenes is the proto-oncogene RAS, which encodes a GTPase involved in cell signaling pathways. Mutations in RAS are found in up to 30% of all human cancers and promote cell proliferation and survival. Another oncogene, MYC, is a transcription factor that regulates the expression of genes involved in cell growth and proliferation. MYC is overexpressed in many cancers, and its amplification is associated with poor prognosis.

Tumor suppressor genes, on the other hand, function to inhibit cell proliferation and promote apoptosis. The tumor suppressor gene *TP53*, also known as p53, is the most commonly mutated gene in human cancers, with mutations found in over 50% of all cancers. Loss of p53 function leads to unchecked cell proliferation and is associated with poor prognosis. Other tumor suppressor genes, such as *PTEN, BRCA1,* and *BRCA2*, are also frequently mutated in cancer and play important roles in regulating cell growth and DNA repair.

Description

Epigenetic drivers of cancer progression

Epigenetic alterations are modifications to the genome that do not involve changes in the DNA sequence but can still alter gene expression and contribute to cancer progression. Epigenetic alterations can be broadly categorized into DNA methylation, histone modifications, and non-coding RNA-mediated mechanisms.

DNA methylation is a modification to the DNA molecule that involves the addition of a methyl group to the cytosine nucleotide in a CpG dinucleotide context. DNA methylation is associated with gene silencing and is frequently altered in cancer. For example, hyper methylation of the promoter region of the tumor suppressor gene *CDKN2A* is a common event in many cancers and is associated with decreased expression of this gene.

Histone modifications, on the other hand, involve alterations to the proteins around which DNA is wrapped, called histones. These

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Received: 03 January, 2024, Manuscript No. jmgm-23-97259; **Editor assigned:** 05 January, 2024, PreQC No. P-97259; **Reviewed:** 17 January, 2024, QC No. Q-97259; **Revised:** 22 January, 2024, Manuscript No. R-97259; **Published:** 29 January, 2024, DOI: 10.37421/1747-0862.2024.18.649

modifications can alter chromatin structure and gene expression. For example, acetylation of histones is associated with gene activation, while methylation of histones can be associated with gene repression. Non-coding RNAs, including microRNAs and long non-coding RNAs, can also contribute to epigenetic alterations in cancer. These RNAs can regulate gene expression by binding to messenger RNA transcripts and inhibiting their translation into proteins or by regulating chromatin structure.

Precision medicine and genetic and epigenetic drivers of cancer

The identification of genetic and epigenetic drivers of cancer has led to the development of precision medicine approaches that aim to personalize cancer treatment based on the specific molecular alterations driving each individual patient's tumor. For example, tumors with mutations in the *EGFR* gene are sensitive to treatment with *EGFR* inhibitors, such as erlotinib and gefitinib. Similarly, tumors with mutations in the *BRAF* gene are sensitive to treatment with BRAF inhibitors, such as vemurafenib and dabrafenib.

Epigenetic alterations in cancer can also be targeted with precision medicine approaches. For example, inhibitors of DNA methyltransferases, such as azacitidine and decitabine, are approved for the treatment of some hematologic malignancies. Similarly, inhibitors of histone deacetylases, such as vorinostat and romidepsin, have been approved for the treatment of cutaneous T-cell lymphoma.

Conclusion

Cancer is a complex and heterogeneous disease that results from genetic and epigenetic alterations in the genome. The identification of genetic and epigenetic drivers of cancer has led to the development of precision medicine approaches that aim to personalize cancer treatment based on the specific molecular alterations driving each individual patient's tumor. Understanding the genetic and epigenetic drivers of cancer is critical for developing effective cancer therapies and improving patient outcomes.

How to cite this article: Deborah, Lyon. "Genetic and Epigenetic Drivers of Cancer Progression: Implications for Precision Medicine." *J Mol Genet Med* 18 (2024): 649.