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Oncogenes: Mechanisms, Types, and Role in Cancer Progression

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INTRODUCTION

Oncogenes play a fundamental role in the development and progression of cancer. Originally derived from proto-oncogenes, these mutated genes promote uncontrolled cell proliferation, survival, and resistance to apoptosis, contributing to tumorigenesis. Cancer results from the accumulation of genetic alterations that disrupt normal cellular regulation, leading to uncontrolled growth and malignancy. Oncogenes are a class of genes that, when mutated or dysregulated, drive the cancerous transformation of normal cells. Derived from normal cellular proto-oncogenes, which control essential processes like cell growth, differentiation, and survival, oncogenes can be activated through mutation, gene amplification, or chromosomal rearrangements. This review highlights the mechanisms of oncogene activation, major types of oncogenes, and their impact on cancer progression. Understanding oncogenes not only sheds light on cancer biology but also provides targets for novel therapeutic interventions, marking oncogenes as essential in precision medicine approaches.

DESCRIPTION

This article discusses the primary mechanisms underlying oncogene activation, categorizes key oncogenes, and examines their roles in cancer development. Proto-oncogenes become oncogenes through three primary mechanisms. Single nucleotide changes can result in a gain-of-function mutation in a proto-oncogene. The RAS gene family is a classic example, where point mutations lead to constitutive activation of RAS proteins, driving uncontrolled cell proliferation and survival signals. An increase in the number of copies of a proto-oncogene can lead to overexpression. For example, MYC amplification, often seen in breast and lung cancers, results in excessive transcriptional activity that fuels cell division and inhibits differentiation. Rearrangements that relocate proto-oncogenes to different genetic loci can result in oncogene activation. A well-known example is the Philadelphia chromosome, where a translocation between chromosomes 9 and 22 creates the BCR-ABL fusion gene in Chronic Myeloid Leukemia (CML). The BCR-ABL fusion protein has abnormal tyrosine kinase activity, promoting unchecked cell division. Oncogenes can be categorized based on their roles in cellular signaling and processes. Oncogenic growth factor receptors are often constitutively active, bypassing normal regulatory mechanisms. The ERBB2 (HER2) receptor, overexpressed in a subset of breast cancers, promotes aggressive cell growth and resistance to apoptosis. Proteins like the RAS family (KRAS, NRAS, and HRAS) and RAF (e.g., BRAF) are integral to signaling cascades that regulate cell proliferation. Mutations in these proteins lead to continuous activation of the MAPK and PI3K pathways, contributing to many cancers, including melanoma and colon cancer. MYC is a key transcription factor that controls the expression of numerous genes involved in cell growth and division. When dysregulated, MYC contributes to tumorigenesis by promoting rapid cell cycling and metabolic changes that favor cancer cell survival. Oncogenes like cyclin D1 drive progression through the cell cycle. Overexpression of cyclin D1, common in breast cancers, bypasses the G1/S checkpoint, allowing cells to divide without proper regulation. Proteins that prevent apoptosis, such as BCL-2, can act as oncogenes. BCL-2 overexpression, particularly in certain types of lymphomas, helps cancer cells evade apoptosis, promoting survival and resistance to chemotherapy. Oncogenes drive multiple hallmarks of cancer, including sustained proliferative signaling, evasion of growth suppressors, and resistance to apoptosis. By altering normal cell signaling pathways, oncogenes enable cells to bypass key regulatory checkpoints, allowing for unchecked cell division.

CONCLUSION

Oncogenes are critical drivers of cancer and represent valuable targets for therapeutic intervention. The elucidation of oncogene mechanisms and pathways has not only deepened our understanding of cancer biology but has also paved the way for precision medicine approaches. Future research focused on overcoming drug resistance and understanding oncogene interactions within the tumor microenvironment will be essential for advancing cancer treatment.

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