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Journal of Cancer Science and Research

Commentary

Role of Decoding Oncogenic Gene Dysregulation in Cancer Development

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ABOUT THE STUDY

Cancer, a group of diseases characterized by uncontrolled cell growth and proliferation, remains to be one the biggest worldwide medical issues. Cancer arises due to the dysregulation of genes that control vital cellular processes. These genes, known as oncogenes, play vital role in initiating and sustaining the transformation of normal cells into cancer cells.

Causes and effects of cancer

Oncogenes are normal genes that play an important role in the regulation of cell growth, differentiation, and survival. When functioning effectively, they help maintain tissue homeostasis. However, under certain conditions, such as genetic mutations or other external influences, these genes can become dysregulated and transform into oncogenes. These oncogenes then drive cancer development by promoting uncontrolled cell division and blocking normal cell death.

Mechanisms of oncogenic gene dysregulation

Mutation: Mutations in oncogenes can result from various factors, such as exposure to carcinogens (e.g., UV radiation, tobacco smoke), replication errors, or inherited genetic mutations. These mutations can lead to the over stimulation of oncogenes, stimulating the development of cancer.

Gene amplification: In some cases, oncogenes can grow to a higher level i.e., there are multiple copies of the gene present. This can result in an excess of protein products that promote cell growth and division.

Chromosomal translocation: Chromosomal translocations involve the exchange of genetic material between different chromosomes, leading to the fusion of genes. This fusion can produce new oncogenes, which are highly active and promote cancer development.

Epigenetic alterations: Epigenetic modifications, such as DNA methylation and histone acetylation, can also dysregulate oncogenes. These modifications can either suppress the expression of tumor suppressor genes or activate oncogenes, promoting cancer.

Common oncogenic pathways

RAS pathway: Mutations in the RAS family of genes are common in many cancers, particularly pancreatic and colorectal cancers. RAS proteins are involved in transmitting signals that regulate cell growth and division.

PI3K/AKT/mTOR pathway: Dysregulation of this pathway is usually leads to cancer. It controls cell survival, proliferation and is connected to various cancers, including breast and prostate cancer.

EGFR Pathway: The Epidermal Growth Factor Receptor (EGFR) pathway is involved in regulating cell growth and division. Variations in this pathway are common in lung, colorectal, and glioblastoma cancers.

MYC Pathway: MYC is a transcription factor that controls the expression of genes involved in cell growth and division. Dysregulation of MYC is observed in many types of cancer.

Consequences of oncogenic gene dysregulation

The dysregulation of oncogenic genes have significant effects on the cellular level and plays a vital role in the development and progression of cancer:

Resistance to cell death: Oncogenes can promote resistance to apoptosis, the programmed cell death process. This allows cancer cells to persist and accumulate.

Invasion and metastasis: Oncogenic gene dysregulation can facilitate the entry of cancer cells into surrounding tissues and promote metastasis, leading to cancer's spread to other parts of the body.

Angiogenesis: Oncogenes can stimulate the formation of new blood vessels (angiogenesis) around the tumor, ensuring a steady supply of nutrients and oxygen.

Immune evasion: Some oncogenic pathways contribute to the evasion of the immune system, enabling cancer cells to avoid detection and destruction.

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Received: 29-Aug-2023, Manuscript No. JCSR-23-27755; **Editor assigned:** 31-Aug-2023; PreQc No. JCSR-23-27755 (PQ); **Reviewed:** 14-Sep-2023, Qc No. JCSR-23-27755; **Revised:** 21-Sep-2023, Manuscript No. JCSR-23-27755 (R); **Published:** 28-Sep-2023, DOI: 10.35248/2576-1447.23.8.554.

Citation: Ayohal S (2023) Role of Decoding Oncogenic Gene Dysregulation in Cancer Development. J Can Sci Res. 8:554.

Oncogenic gene dysregulation and cancer research

Oncogenic gene dysregulation is of most important in cancer research. Researchers are dedicated to analyzing the complex molecular mechanisms underlying oncogenic transformation to identify potential targets for therapeutic interventions. Here are some important areas of research:

Targeted therapies: The identification of specific oncogenes and associated pathways has led to the development of targeted therapies that aim to block or inhibit these oncogenic signals. For example, tyrosine kinase inhibitors have been successful in treating certain types of cancer with specific mutations.

Personalized medicine: Advances in genomics have led to personalized cancer treatment. By analyzing a patient's genetic makeup, researchers are able to modify therapy plans to target the specific cancer-causing oncogenes.

Epigenetic therapies: Researchers are exploring ways to reverse epigenetic alterations that dysregulate oncogenes. Drugs targeting epigenetic modifications offer the potential to reprogram cancer cells into a less volatile condition.

Immunotherapy: Immunotherapy, which use the immune system's capability to target cancer cells, has become a revolutionary in cancer treatment. By reactivating the immune response against cancer cells, immunotherapies can be effective even in cases of oncogenic gene dysregulation.

Novel biomarkers: Research in oncogenic gene dysregulation has led to the discovery of new biomarkers that help in early cancer diagnosis and prognosis. These biomarkers provide valuable information for clinicians in making informed decisions about patient care.

CONCLUSION

Oncogenic gene dysregulation is an important factor influencing the cancer development. It plays a central role in the uncontrolled cell growth, survival, and metastasis that characterize cancer. As our understanding of oncogenic gene dysregulation continues to evolve, also treatment options and strategies. Research efforts in this field use the commitment of more effective cancer therapies, increased survival rates, and improved outcomes for patients. The ongoing dedication of scientists and medical professionals take steps to decode oncogenic gene dysregulation which is important in the fight against this complex and challenging disease.