The role of oncogenes in cancer progression: Mechanisms and therapeutic targets.

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Introduction

Cancer is a complex disease driven by genetic and molecular alterations that disrupt normal cellular functions. Among these alterations, oncogenes play a critical role in cancer progression by promoting uncontrolled cell growth, survival, and metastasis. Understanding oncogene function, their mechanisms of activation, and potential therapeutic targets is essential for developing effective cancer treatments [1].

Oncogenes are mutated or abnormally expressed genes that drive tumor formation and progression. They originate from normal cellular genes called proto-oncogenes, which regulate cell growth, differentiation, and survival. When proto-oncogenes undergo mutations, gene amplifications, or chromosomal translocations, they become oncogenes, leading to unregulated cellular proliferation and tumorigenesis [2].

There are several mechanisms by which proto-oncogenes transform into oncogenes. Genetic mutations, often caused by environmental carcinogens, radiation, or inherited genetic defects, can lead to the constant activation of oncogenes. Gene amplification results in excessive production of oncogene-encoded proteins, further driving uncontrolled cell division. Additionally, chromosomal translocations can create fusion proteins with novel oncogenic properties, as seen in chronic myeloid leukemia (CML) with the BCR-ABL fusion gene [3].

Several well-characterized oncogenes contribute to cancer progression. The RAS oncogene family (KRAS, NRAS, and HRAS) regulates cell signaling pathways involved in proliferation and survival; mutations in these genes are common in lung, colorectal, and pancreatic cancers. MYC, another oncogene, controls cell cycle progression and metabolism, and its overexpression is associated with aggressive cancers. The HER2/ERBB2 oncogene, frequently amplified in breast cancer, promotes uncontrolled cell growth by activating multiple signaling pathways [4].

Oncogenes not only drive cancer cell proliferation but also influence the tumor microenvironment. By altering cellular signaling, they can promote angiogenesis, immune evasion, and metastasis. For example, oncogenic mutations in VEGF (vascular endothelial growth factor) stimulate new blood vessel formation, allowing tumors to access oxygen and nutrients. Additionally, oncogenes can modulate immune checkpoints, helping cancer cells evade immune detection [5].

The discovery of oncogenes has led to the development of targeted therapies designed to inhibit their function. Small molecule inhibitors and monoclonal antibodies have been developed to block oncogene-driven pathways. For example, tyrosine kinase inhibitors (TKIs) such as imatinib effectively inhibit BCR-ABL in CML, while EGFR inhibitors like erlotinib target mutated EGFR in lung cancer [6].

Despite advances in targeted therapies, several challenges remain. Many oncogene-driven cancers develop resistance to therapy through secondary mutations, alternative signaling pathways, or tumor heterogeneity. For example, mutations in KRAS make targeted therapy difficult, as effective inhibitors have only recently been developed. Additionally, not all oncogenes have direct druggable targets, requiring alternative therapeutic approaches [7].

To overcome resistance, researchers are developing next-generation inhibitors, combination therapies, and novel drug delivery systems. KRAS inhibitors such as sotorasib and adagrasib have recently shown promise in targeting KRAS G12C-mutated lung cancer. Combining oncogene-targeted therapies with immunotherapy, such as immune checkpoint inhibitors, is also being explored to enhance treatment efficacy [8].

Immunotherapy has emerged as a promising approach in cancer treatment. Tumors with specific oncogene alterations may be more susceptible to immune-based therapies. For instance, CAR-T cell therapy is being investigated to target oncogenic proteins expressed on the surface of cancer cells. Additionally, immune checkpoint inhibitors, such as PD-1/PD-L1 blockers, have shown efficacy in cancers with high oncogenic mutation burdens [9].

Advancements in genomic sequencing, CRISPR-based gene editing, and artificial intelligence are revolutionizing oncogene research. Precision oncology aims to tailor treatments based on the specific oncogenic mutations present in a patient's tumor, maximizing efficacy while minimizing side effects. Continued research on oncogene function and resistance mechanisms will be essential for developing next-generation cancer therapies [10].

Conclusion

Oncogenes play a fundamental role in cancer initiation and progression, making them critical targets for cancer therapy. While significant progress has been made in developing

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oncogene-targeted drugs, challenges such as drug resistance and tumor heterogeneity remain. Ongoing research and innovative therapeutic approaches offer hope for improving cancer treatment outcomes. A deeper understanding of oncogenes will pave the way for more effective, personalized cancer treatments in the future.

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