The role of oncogenes and tumor suppressor genes in cancer progression.

Michael Patel*

Department of Internal Medicine, University Medical Center, The Netherlands

Introduction

Cancer is a multifaceted disease characterized by uncontrolled cell growth and proliferation. At the heart of this process lie genetic alterations in two critical groups of genes: oncogenes and tumor suppressor genes. These genes play opposing yet interconnected roles in maintaining cellular homeostasis, and mutations in either can disrupt the delicate balance between cell growth and death, leading to malignancy. Understanding their functions is essential for advancing cancer diagnosis, treatment, and prevention strategies [1].

Oncogenes are mutated or overexpressed versions of normal cellular genes known as proto-oncogenes. Proto-oncogenes play a role in normal cell growth, differentiation, and apoptosis. When these genes undergo mutations—whether through point mutations, gene amplification, or chromosomal rearrangements—they become oncogenes, driving uncontrolled cell proliferation and survival. Common examples include RAS, MYC, and HER2/ERBB2 [2].

In contrast to oncogenes, tumor suppressor genes function as cellular brakes, preventing excessive cell growth and promoting apoptosis when necessary. These genes act as a defense mechanism against cancer development. Key tumor suppressor genes include TP53 (p53), RB1 (Retinoblastoma protein), and BRCA1/BRCA2 [3].

Unlike oncogenes, which require a gain-of-function mutation, tumor suppressor genes typically require a loss-of-function mutation in both alleles (known as the "two-hit hypothesis"). The p53 protein, often referred to as the *"guardian of the genome,"* plays a central role in preventing cancer. It regulates cell cycle arrest, DNA repair, and apoptosis in response to cellular stress. Mutations in the TP53 gene are among the most common alterations observed in human cancers, allowing cells to evade apoptosis and accumulate additional mutations [4].

The RB1 gene regulates the cell cycle by controlling the G1/S checkpoint. Loss of RB1 function leads to unchecked cell cycle progression and uncontrolled proliferation. This mutation is famously associated with retinoblastoma and has been implicated in various other cancers, including osteosarcoma and small-cell lung cancer [5].

Cancer progression often involves the cooperation between oncogenes and tumor suppressor genes. For instance, oncogenic activation of *MYC* can drive proliferation, but its full oncogenic potential is often unleashed only when paired with a loss of tumor suppressor genes like *p53*. This interplay highlights the complexity of cancer biology and the need for multi-targeted therapeutic strategies [6].

Modern cancer therapies increasingly focus on targeting oncogenes and restoring tumor suppressor function. Drugs like trastuzumab (targeting HER2) and imatinib (targeting BCR-ABL) have revolutionized treatment outcomes. Additionally, gene therapy approaches aim to restore normal tumor suppressor gene function, such as reactivating p53 pathways [7].

While oncogenes can be effectively targeted with inhibitors, restoring tumor suppressor gene function remains challenging. Tumor suppressor genes are often inactivated through loss-of-function mutations, making them difficult therapeutic targets. Researchers are exploring novel strategies, including gene therapy, CRISPR-Cas9 gene editing, and synthetic lethality approaches [8].

Advances in genomic sequencing technologies allow for the identification of specific oncogene mutations and tumor suppressor gene alterations in individual patients. This knowledge enables personalized medicine approaches, where therapies are tailored to target a patient's unique genetic profile, improving treatment efficacy and minimizing side effects [9].

Ongoing research aims to unravel the intricate networks between oncogenes, tumor suppressor genes, and other cellular pathways. Innovative technologies like single-cell RNA sequencing and artificial intelligence in genomics are helping decode cancer's genetic complexity [10].

Conclusion

The interplay between oncogenes and tumor suppressor genes forms the foundation of cancer biology. Oncogenes drive uncontrolled proliferation, while tumor suppressor genes act as cellular safeguards. Understanding these mechanisms has paved the way for significant advancements in cancer therapeutics, diagnostics, and personalized medicine. However, many challenges remain, and continued research is vital to fully harness the therapeutic potential of these genetic insights in the fight against cancer.

References

1. Lee EY, Muller WJ. Oncogenes and tumor suppressor genes. Cold Spring Harb Perspect Biol. 2010;2(10):a003236.

***Correspondence to:** Michael Patel**,** Department of Internal Medicine, University Medical Center, The Netherlands, USA. E-mail: m.patel@erasmusmc.nl

***Received:** 1-Jan-2024, Manuscript No. aarrgs-25-157963; **Editor assigned:** 4-Jan-2024, PreQC No. aarrgs-25-157963 (PQ); **Reviewed:** 17-Jan-2024, QC No. aarrgs-25-157963; **Revised:** 24-Jan-2024, Manuscript No. aarrgs-25-157963 (R); **Published:** 31-Jan-2024, DOI: 10.35841/aarrgs-7.1.244*

***Citation:** Patel M. The role of oncogenes and tumor suppressor genes in cancer progression. J Res Rep Genet. 2025;7(1):244.*

2. Kontomanolis EN, Koutras A, Syllaios A, et al. Role of oncogenes and tumor-suppressor genes in carcinogenesis: A review. Anticancer Res. 2020;40(11):6009-15.
3. Yuspa SH, Długosz AA, Cheng CK, et al. Role of oncogenes and tumor suppressor genes in multistage carcinogenesis. J Invest Dermatol. 1994;103.
4. Osborne C, Wilson P, Tripathy D. Oncogenes and tumor suppressor genes in breast cancer: Potential diagnostic and therapeutic applications. Oncologist. 2004;9(4):361-77.
5. Osada H, Takahashi T. Genetic alterations of multiple tumor suppressors and oncogenes in the carcinogenesis and progression of lung cancer. Oncogene. 2002;21(48):7421-34.
6. Klein G. Oncogenes and tumor suppressor genes. Acta Oncologica. 1988;27(4):427-37.
7. Gariglio P. Oncogenes and tumor suppressor genes. Molecular Oncology Principles and Recent Advances. 2012;64:82.
8. Grandér D. How do mutated oncogenes and tumor suppressor genes cause cancer?. Med Oncol. 1998;15(1):20-6.
9. Aunoble BÉ, Sanches R, Didier E, et al. Major oncogenes and tumor suppressor genes involved in epithelial ovarian cancer. Int J Oncol. 2000;16(3):567-643.
10. Zhang B, Pan X, Cobb GP, et al. microRNAs as oncogenes and tumor suppressors. Dev Biol. 2007;302(1):1-2.

Citation: *Patel M. The role of oncogenes and tumor suppressor genes in cancer progression. J Res Rep Genet. 2025;7(1):244.*