Journal of Cancer Research and Immuno-Oncology

OPEN ACCESS Freely available online

Commentary

Role of Oncogenes and Tumor Suppressor Genes in Cancer Progression

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DESCRIPTION

Cancer is not a random disease it's a biological consequence of genetic miscommunication. At the heart of this miscommunication are two fundamental players of oncogenes and tumor suppressor genes. Their roles in normal cellular function are critical, but when their balance is disrupted, cells can escape the body's regulatory checks and begin a path toward malignancy. Understanding how these genes contribute to cancer progression is not only key to decoding the disease but also to unlocking new therapies.

Oncogenes: Fueling the fire of uncontrolled growth

Oncogenes are the mutated, overactive versions of normal genes called proto-oncogenes. In healthy cells, proto-oncogenes regulate growth, division, and survival. When mutated or overexpressed, they transform into oncogenes genes that push the gas pedal of the cell cycle, often beyond the point of control. A classic example is the *RAS* gene family, which encodes proteins involved in signal transduction pathways that promote cell proliferation. Mutations in *RAS* particularly in *KRAS*, *NRAS*, and *HRAS* result in a constantly active protein that sends continuous growth signals, even in the absence of external stimuli. These mutations are found in a wide array of cancers, including pancreatic, colon, and lung cancers. Another well-known oncogene is *MYC*, a transcription factor that regulates genes involved in cell growth and metabolism. When *MYC* is amplified or mutated, it promotes rapid cellular proliferation and metabolic reprogramming hallmarks of aggressive tumors. Likewise, *HER2*, a receptor tyrosine kinase overexpressed in some breast cancers, drives growth signals that make tumors particularly fast-growing and invasive. The presence of oncogenes not only fuels tumor growth but also provides opportunities for targeted therapies. Drugs like trastuzumab (targeting *HER2*) and tyrosine kinase inhibitors (like imatinib for *BCR-ABL* in chronic myeloid leukemia) have revolutionized treatment by specifically targeting these overactive proteins. However, oncogenes are notoriously adaptable, and resistance to such therapies often develops through secondary mutations or pathway rewiring.

Tumor suppressor genes: Guardians of the cell cycle

In contrast to oncogenes, tumor suppressor genes act like the brakes on cellular growth. They regulate DNA repair, monitor cell cycle checkpoints, and initiate apoptosis (programmed cell death) when errors occur. When these genes are inactivated through mutations, deletions, or epigenetic silencing the cell loses crucial control mechanisms, enabling the emergence of cancer. Perhaps the most iconic tumor suppressor is *TP53*, often referred to as "the guardian of the genome." This gene encodes *p53*, a protein that responds to DNA damage by halting the cell cycle and initiating repair or apoptosis. Mutations in *TP53* are found in over 50% of human cancers. When *p53* is inactivated, cells with damaged DNA are allowed to survive and divide, accumulating more mutations that drive tumor progression.

Another crucial tumor suppressor is *RB1*, which regulates the G1/S transition in the cell cycle. Loss of RB1 function removes a key checkpoint, allowing unchecked entry into DNA synthesis and division. In retinoblastoma, a childhood eye cancer, biallelic inactivation of *RB1* is the primary driver of tumor formation. Similarly, the *BRCA1* and *BRCA2* genes are responsible for homologous recombination repair of DNA. Mutations in these genes impair DNA repair mechanisms, increasing susceptibility to breast, ovarian, and other cancers. Unlike oncogenes, which typically act in a dominant fashion (only one mutated allele can drive change), tumor suppressors often follow the "two-hit hypothesis" Both copies of a critically important for cellular tumor suppressor gene must be inactivated to lose function. This difference in genetic behavior adds complexity to therapeutic strategies targeting these genes.

Cancer is, in many ways, a disease of imbalance. Oncogenes push cells to divide, survive, and metastasize, while tumor suppressors work to keep those impulses in check. When both systems are dysregulated, the result is a cell that can divide uncontrollably, ignore death signals, invade tissues, and resist treatment. This balance is not simply disrupted by random chance; environmental factors such as UV radiation, smoking, and viral infections (e.g., HPV in cervical cancer) can induce mutations that tip the scale. Additionally, epigenetic changes like DNA methylation can silence tumor suppressor genes without altering the DNA sequence, adding another layer of complexity.

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Received: 14-Feb-2025, Manuscript No. JCRIO-25-38363; **Editor assigned:** 17-Feb-2025, PreQC No. JCRIO-25-38363 (PQ); **Reviewed:** 03-Mar-2025, QC No. JCRIO-25-38363; **Revised:** 10-Mar-2025, Manuscript No. JCRIO-25-38363 (R); **Published:** 17-Mar-2025, DOI: 10.35248/2684-1266.25.11.240

Citation: Menon S (2025) Role of Oncogenes and Tumor Suppressor Genes in Cancer Progression. J Cancer Res Immunooncol. 11.240.

Therapeutically, restoring this balance is a major goal. While we can inhibit some oncogenes with drugs, reactivating tumor suppressor genes has proven more challenging. Recent strategies include using small molecules to restore *p53* function, *CRISPR*-based gene editing, or targeting downstream effects of tumor suppressor loss. Immunotherapy also offers a promising avenue, as tumors with damaged p53 or high mutation loads often generate neoantigens that can be recognized by the immune system.

CONCLUSION

The battle between oncogenes and tumor suppressor genes is at the heart of cancer biology. It is a war between acceleration and restraint, between growth and control. By understanding the roles these genes play in cancer progression, we are not only deciphering the language of tumors but also identifying points of vulnerability. As research continues, a deeper appreciation of this genetic tug-of-war will inform more precise, more durable, and more personalized treatments. Cancer may be complex, but in its core genetic drivers, it reveals the keys to its own undoing.