Research Journal of Oncology

Open access Short Communication

Tumor Suppressors: Guardians against Cancer

Charlotte Martin*

Department of Tumor Biology, University of Copenhagen, Denmark

INTRODUCTION

Tumor suppressor genes are vital components in the defense against cancer, acting as cellular "brakes" to control cell division, repair DNA damage, and prevent abnormal growth. When these genes function correctly, they inhibit the formation of tumors and maintain normal cellular function. However, when tumor suppressor genes are damaged or mutated, cells can grow uncontrollably, potentially leading to cancer. Understanding the mechanisms and roles of tumor suppressor genes is essential for advancing cancer prevention, diagnosis, and treatment. Tumor suppressor genes are a category of genes that help prevent cancer by producing proteins that regulate cell growth and division. Under normal conditions, these proteins maintain a balance between cell proliferation and cell death, keeping cellular growth under control. When tumor suppressor genes are mutated, their regulatory function is lost, enabling cells to divide unchecked. Unlike oncogenes, tumor suppressor genes need to be inactivated or lost to contribute to cancer development. The two-hit hypothesis, proposed by geneticist Alfred Knudson, suggests that both alleles of a tumor suppressor gene must be mutated or inactivated for cancer to develop. This theory has been instrumental in understanding how mutations in tumor suppressors lead to cancer. Several tumor suppressor genes are well-studied and play a significant role in preventing cancer. Among the most notable are TP53, RB1, and BRCA1/BRCA2.

DESCRIPTION

Often referred to as the "guardian of the genome", TP53 is one of the most commonly mutated genes in cancer. It encodes the p53 protein, which plays a crucial role in cell cycle regulation, DNA repair, and apoptosis (programmed cell death). When DNA damage occurs, p53 can halt cell division to allow for DNA repair or initiate cell death if the damage is irreparable. Mutations in TP53 lead to loss of these functions, allowing damaged cells to survive and proliferate, which can result in tumor formation. The Retinoblastoma protein encoded by the RB1 gene regulates the cell cycle, particularly at the G1/S checkpoint. By inhibiting cell cycle progression, Rb prevents cells from replicating before they are ready. Mutations in RB1 can lead to uncontrolled cell division, contributing to cancers such as retinoblastoma, a rare form of eye cancer that affects young children. BRCA1 and BRCA2, these genes are involved in DNA repair, particularly in the repair of double-strand breaks. When BRCA1 or BRCA2 is mutated, DNA repair processes are compromised, leading to genomic instability and an increased risk of breast, ovarian, and other cancers. Inherited mutations in these genes significantly raise an individual's risk of developing certain cancers, which is why genetic testing for BRCA mutations is recommended for individuals with a family history of these cancers. Tumor suppressor genes employ several mechanisms to maintain cellular health and prevent cancer. Cell Cycle control many tumor suppressor proteins act as cell cycle checkpoints, ensuring that cells do not progress through the cell cycle until they are ready. By halting cell division, they prevent the propagation of damaged DNA, which can lead to mutations and cancer. Tumor suppressors also play an essential role in repairing DNA damage. Proteins produced by genes like BRCA1 and BRCA2 help to correct errors that occur during DNA replication. Without functional DNA repair mechanisms, cells accumulate mutations that may lead to cancer. Induction of Apoptosis, when cells experience irreparable damage, tumor suppressor genes like TP53 can trigger apoptosis. This prevents the survival and replication of cells with damaged DNA, reducing the risk of cancer. Understanding tumor suppressor genes has significant implications for cancer therapy. In some cases, restoring the function of these genes can help to halt or slow tumor growth. For instance, researchers are exploring ways to reactivate p53 in cancers where it has been inactivated, potentially stopping tumor progression. Additionally, the loss of tumor suppressor function can make cancer cells more dependent on alternative survival pathways, which can be targeted with drugs. This approach, known as synthetic lethality, has been particularly successful in cancers with BRCA1 or BRCA2 mutations, where

Received:	02-September-2024	**Manuscript No:**	IPRJO-24-22008
Editor assigned:	04-September-2024	**PreQC No:**	IPRJO-24-22008 (PQ)
Reviewed:	18-September-2024	**QC No:**	IPRJO-24-22008
Revised:	23-September-2024	**Manuscript No:**	IPRJO-24-22008 (R)
Published:	30-September-2024	**DOI:**	10.36648/iprjo-8.3.21

Corresponding author Charlotte Martin, Department of Tumor Biology, University of Copenhagen, Denmark, Email: cmartin@copenhagenuni.dk

Citation Martin C (2024) Tumor Suppressors: Guardians against Cancer. Res J Onco. 8:21.

inhibitors of the PARP enzyme are used to target cancer cells selectively. While the role of tumor suppressor genes is well understood, there are challenges in directly targeting them for cancer therapy. Since tumor suppressors are often inactivated rather than overactive, conventional drug design is difficult. Instead, researchers focus on compensating for the loss of tumor suppressor function or exploiting the vulnerabilities created by this loss. Future research in gene editing, synthetic lethality, and targeted therapies holds promise for improving cancer treatment by addressing the inactivation of tumor suppressor genes [1-4].

CONCLUSION

Tumor suppressor genes are vital in protecting cells from cancerous transformation. By controlling cell division, repairing DNA damage, and inducing apoptosis, they serve as essential safeguards for cellular integrity. However, when these genes are inactivated, the risk of cancer significantly increases. Continued research into the mechanisms and therapeutic targeting of tumor suppressor genes holds promise for better cancer prevention and more effective treatments, underscoring the critical role of these “guardians” in our ongoing battle against cancer.

ACKNOWLEDGEMENT

None.

CONFLICT OF INTEREST

The author’s declared that they have no conflict of interest.

REFERENCES

1. Guo XE, Ngo Bryan, Modrek AS, Lee WH (2014) Targeting tumor suppressor networks for cancer therapeutics. Curr Drug Targets. 15(1):2-16.
2. Liu Y, Hu X, Han C, Wang L (2015) Targeting tumor suppressor genes for cancer therapy. Bioessays. 37(12):1277-1286.
3. Fontela CM, Mandinova A, Aaronson SA, Lee SW (2016) Emerging roles of p53 and other tumour-suppressor genes in immune regulation. Nat Rev Immunol. 16(12):741-750.
4. Heritz JA, Backe SJ, Mollapur M (2024) Molecular chaperones: Guardians of tumor suppressor stability and function. Oncotarget. 15:679-696.