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Tumor Suppressor Genes: The Guardians against Cancer

Isaac Kapel*

Department of Biochemistry, Sejong University, South Korea

INTRODUCTION

Tumor suppressor genes play a crucial role in maintaining cellular integrity by regulating cell growth, division, and death. When functioning correctly, these genes act as safeguards against cancer by preventing uncontrolled cell proliferation and ensuring that damaged cells are eliminated. However, mutations or loss of function in tumor suppressor genes can lead to the development and progression of various cancers. This article delves into the functions of tumor suppressor genes, their role in cancer, and emerging therapeutic strategies aimed at targeting these critical regulators. Tumor suppressor genes are involved in several key cellular processes: Tumor suppressor genes help regulate the cell cycle, ensuring that cells only divide when appropriate. For example, the p53 protein, encoded by the TP53 gene, acts as a checkpoint regulator by assessing DNA damage and inducing cell cycle arrest or apoptosis if damage is detected.

DESCRIPTION

Tumor suppressor genes are essential for maintaining genomic stability by facilitating DNA repair mechanisms. The BRCA1 and BRCA2 genes are well-known for their roles in repairing double-strand breaks in DNA. Mutations in these genes can lead to an increased risk of breast and ovarian cancers. Tumor suppressors also regulate programmed cell death. The p53 protein, for instance, can initiate apoptosis in response to severe DNA damage, preventing the proliferation of potentially cancerous cells. Tumor suppressors like E-cadherin help maintain proper cell-cell adhesion, which is crucial for preventing tumor metastasis. Loss of E-cadherin function can lead to increased cell mobility and invasion, contributing to cancer spread. Several tumor suppressor genes have been extensively studied for their roles in cancer development: The TP53 gene, which encodes the p53 protein, is one of the most well-known tumor suppressors. p53 functions as a "guardian of the genome" by detecting cellular stress and damage, and initiating repair processes or cell death. Mutations in TP53 are found in a wide variety of cancers, including lung, breast, and colorectal cancers. These mutations often result in a loss of p53 function, allowing cells with genetic damage to continue dividing. The BRCA1 and BRCA2 genes are critical for DNA repair through homologous recombination. Mutations in these genes significantly increase the risk of breast and ovarian cancers. Individuals with BRCA1 or BRCA2 mutations may have up to a 70% lifetime risk of developing breast cancer. These genes are also involved in maintaining genomic stability and preventing tumor progression. The RB1 gene encodes the retinoblastoma protein, which regulates the cell cycle by inhibiting progression from the G1 phase to the S phase. Loss of Rb function, often due to mutations in RB1, leads to uncontrolled cell cycle progression and is a key event in retinoblastoma, a rare childhood eye cancer, as well as in other cancers such as small cell lung cancer. The PTEN gene encodes a lipid phosphatase that negatively regulates the PI3K/Akt signaling pathway, which is involved in cell survival and growth.

CONCLUSION

Loss of PTEN function can lead to uncontrolled cell proliferation and is implicated in various cancers, including prostate, endometrial, and brain cancers. PTEN mutations can also contribute to tumor progression by enhancing resistance to apoptosis. In conclusion, tumor suppressor genes are critical regulators of cellular growth and stability, and their dysfunction plays a pivotal role in cancer development. Advances in our understanding of these genes and their associated pathways hold promise for developing targeted therapies and improving cancer treatment outcomes. As research progresses, tumor suppressor genes will continue to be central to efforts in combating cancer and enhancing patient care.

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Corresponding author Isaac Kapel, Department of Biochemistry, Sejong University, South Korea, E-mail: kapel@is.re.kr

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