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Perspective

Characteristics of Cancer Associated with Tumour Suppressor Genes

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DESCRIPTION

The realisation that genetic alterations are responsible for the aetiology of cancers in both adults and children represents a significant advancement in cancer research over the past few decades. Every cell has these alterations, which can be inherited, although they are more frequently acquired somatically and are only present in tumour cells. Moreover, these changes have an impact on genes belonging to the following 3 categories: proto-oncogenes, tumour suppressor genes, and DNA repair genes. This article first briefly reviews tumour suppressor genes before concentrating on the function of proto-oncogenes in paediatric cancer.

Tumor suppressor genes

Tumor suppressor gene inactivation, which results in the loss of the normally negative control over cell proliferation, aids in the malignant transformation of a variety of cell types. The idea that two strikes, or mutations, are necessary for the onset of retinoblastoma was first put forth by Knudson. The cloning of the Retinoblastoma Tumour Suppressor Gene (RB1) and functional analyses of the retinoblastoma protein, Rb, later confirmed his hypothesis. Whereas the second mutation in retinoblastoma instances is always somatic, the first mutation of RB1 can be either constitutional or somatic. The first mutation in the inherited form of retinoblastoma, which has an early onset and a high prevalence of bilateral illness, is present in the germline. With nonhereditary retinoblastoma, however, both mutations are somatic.

Several of the proteins that tumour suppressor genes encode have an effect at particular stages of the cell cycle, like the Rb protein. For instance, the TP53 gene on chromosome 17 produces a nuclear protein of 53 kD that serves as a cell cycle checkpoint. P53 is a transcription factor that inhibits cell division at the G1 phase of the cell cycle to allow for DNA repair. Its expression is boosted by DNA damage. Moreover, cells with damaged DNA can undergo apoptosis when the TP53 gene is activated.

Patients with Li-Fraumeni syndrome, who often inherit a mutant TP53 gene from an afflicted parent, are found to have a germline mutation of one TP53 allele. Individuals with Li-Fraumeni syndrome are more likely than the general population to acquire sarcomas, breast cancer, brain tumours, adrenocortical cell carcinoma, and acute leukaemia by the age of 30.

Cyclin-Dependent Kinase (CDK) inhibitors are a significant family of tumour suppressor genes involved in cell cycle regulation and the development of human malignancies. These proteins, which include p15INK4B, p16INK4A, p18INK4C, p19INK4D, p19ARF, p21CIP1, p27KIP1, and p57KIP2, negatively control the cell cycle by preventing CDK phosphorylation of the Rb protein. INK4A is one of the most frequently altered genes in human malignancies, despite the fact that carcinogenic roles for the INK4B, INK4C, INK4D, CIP1, KIP1, and KIP2 genes appear to be limited. The cyclin D: CDK4/6 complexes, which are essential for controlling the cell cycle by phosphorylating Rb protein, are inhibited by the cell-cycle inhibitor p16INK4A protein.

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