Advances in Cancer Prevention
ISSN: 2472-0429

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Abstract

Oncogenes are the tumour causing genes and have important role in development of many cancers. In 1970, SRC oncogene was discovered in chicken retrovirus. As a result of some mutation in the otherwise normal proto-oncogenes, their deregulation occurs and uncontrolled proliferation of cells starts and leads to cancer.

Keywords: Diagnosis; Cancer; Prevention; Scintigraphy; Health outcomes; Imaging

Introduction

At genomic level, only single oncogenic allele is required to alter normal gene function because of its dominant property. The origin of oncogene can be cellular i.e., from inside the body or viral i.e. from some virus. Gene Duplication, addition, insertion, deletion or chromosomal translocation, chromosomal re-arrangement of certain proto-oncogenes alters their function and converts them into oncogenes. These mutations overexpress the protein to an uncontrolled level, which may lead to tumour. These mutations may occur due to external factors or internal factors or both like viral infection, radiation or chemicals, injury and disease. Among these mutations, viral infection is the rare cause of oncogene activation in animals but is of great importance for understanding oncogene function [1]. Viral infection Retrovi7(r)4(8p T1ETQq4)] T6-7(r)4(o)-1 T1ETB-6(f)13(e)4(c.Q2(eira07 Tm[( )] T1ETBT06 Tc[(or)] T1ETBT1 0 0 1r 0 1At)-14(io06F6 9 518)4(c.Q8)] T1[(4(io06Fca5

products of Rb and INK-4 genes. Retinoblastoma is the tumour of the eye. Two mutagenic events are required for the etinoblastoma development in sporadic cases whereas only one mutagenic event is needed in individuals with inherited form of the disease in which it displays autosomal dominant inheritance [6]. In normal cells, Cdk2 and cyclin D complexes regulate the entry through the constraint point, thereby phosphorylating and inactivating pRb. pRb also impedes the entry through the constraint point in the G1 phase of the cell cycle by repressing the transcription of many genes involved in cell cycle advancement. The INK4 tumour suppressor gene also regulates movement through the restriction point by encoding Cdk inhibitor p16. Inactivation of INK4 results in uncontrolled phosphorylation of Rb [7]. The p53 plays its role by regulating cell cycle and programmed cell death. The p53 can arrest the cell cycle upon DNA damage. It allows the DNA to repair or cause the programmed cell death. This is achieved by activating a number of genes involved in controlling and regulating the cell cycle. Mutation in p53 in tumorigenic cells results in uncontrolled cell proliferation and inefficient DNA repair. P53 mutations are estimated to be the most common in tumors of humans, approximately greater than that. Breast cancer and genes are linked to familial breast cancer. Breast Cancer-1 gene consists of 100 Kb DNA and 21 exons. It has a zinc-finger domain like that in the DNA binding proteins. Breast cancer-1 is a tumour suppressor gene. BRCA-2 is located on chromosome. Tumour suppressor genes can be studied at the levels of DNA, mRNA, and proteins in the normal and cancerous cells using various methods. Tests for the detection of heterozygosis can be helpful for identifying individuals predisposed to retinoblastoma and other malignancies. Higher frequency of p53 mutations also offers