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Cancer and Genetic

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Cancer is an uncontrolled cell proliferation resulted of deterioration in mechanisms that regulate normal behaviors of a cell. Tumor, defined as also uncontrolled cell divisions, affects the function of tissue and organs by spreading to other parts of the body.

Given the facts that control of cell division in cancer is lost, and the division and growth of cell is under control of genes, a generalized statement can be made that genetic factors are effective in almost every type of cancer. In some cancers, an abnormal gene is considered to be a primary factor, while in others environmental factors are, and thus uncontrolled cell proliferation is considered as a secondary factor. Whatever factor is effective, it is now known that all types of cancer occur as a result of mutation or mutations in somatic cells and these mutations affect a series of gene expressions.

In this review, we focused on tumor suppressor genes and oncogenes that are effective in cancer formation. Additionally, it was given some information about viruses leading to cancer.

Tumor Suppressor Genes

Tumor suppressor geneis a gene that suppresses cell proliferation and tumor growth under normal conditions. Loss of normal function in both alleles and damaging or inactivation of these genes affect cell proliferation negatively, and then cause tumor formation. The presence of a normal allele suppresses tumor growth. Mutations of tumor suppressor genes are recessive at the cellular level. Unlike oncogenes, both alleles of tumor suppressor genes should lose function for tumor growth. For this case, some mutational events are required. Some of them are base changes and deletions. Others include nondisjunction in mitosis and chromosome loss caused by some mechanisms, and these events occur more frequently.

APC, BRCA1, BRCA2, p53, PTEN, PTCH, INK4, Rb, Smad2 and Smad4 are examples of known tumor suppressor genes. Of these, Rb gene is the first examined tumor suppressor gene and responsible for retinoblastoma which is rare eye tumor in childhood. Patients can survive and establish a family when it is treated early. Studies have shown that Rb gene also plays a role in adult tumors. In most tumors, Rb gene is either disappeared or inactivated.

P53 protein has very important roles in many cellular events such as DNA repair, control of cell cycle, genomic stability, regulation of chromosome separation, regulation of gene expression, aging, and programmed cell death. This protein composed of 393 amino acids, is encoded by a gene lining within a 20 kb in length with 11 exon and located on the short arm of the chromosome 17. Cell cycle arrest provides a

cell time to repair the damage in DNA before cell goes through to the replication process. Loss of P53 function causes the elimination of cell arrest and this results in increased mutation frequency and instability in the cell genome. Genomic instability is a common feature of most cancer cells.

The proteins encoded by BRCA1 and BRCA2 genes are also responsible for controlling the cell cycle checkpoints and repairing the double strand DNA breaks. In cancer development, the roles of mutations within tumor suppressors and oncogenes acting as stability genes are not due to a cell proliferation, but they are mainly due to an increased frequency of mutations resulted from inactivation of that genes.

Oncogenes

Characteristics of tumor cell cultures are different from that of normal cell cultures. Transformed cells grow in less constrained conditions and they often do not need to attach to a rigid surface, so they appear to be round in shape. Their serum requirements are decreased. They show a massive structure on the floor of cell culture dish. Some oncogenes observed in human tumors are the homologues of retrovirus oncogenes described previously, while others are described as new oncogenes. The first identified human oncogene is RasH oncogene. In human tumors,ras gene family has three members including RasH, RasK and RasN. Ras oncogenes play a role in most human tumors, for example in 50% of colon cancers and 25% of lung cancers. In addition to Ras, there are many known oncogenes such as abl, bcl-2, erbB, c-myc, PDGFR, P13K. Point mutation, insertion, translocation or gene amplification can activate protooncogenes.

Tumor Viruses

There are viruses that causes cancer directly in humans or experimental animals. They are called tumor viruses. Most common tumor viruses are Hepatitis B and C, Papilloma viruses, Epstein-Bar virus, HIV virus, Merkel cell polyoma virus, Herpes viruses and Retroviruses. Transforming activity of a tumor virus originates from the specific gene or genes present in viral genome. Some genes that are active in the early stages of viral lytic cycle are responsible for the oncogenic potential. When transformation occurs, genes integrated to the genome of transformed cell are abnormally expressed. These oncogenes produce proteins transformed. These oncogenes have no cellular counterparts and work by inhibiting cellular tumor suppressors.

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