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Carcinogenicity of Deoxycholate: A Secondary Bile Acid of Inherited Genes

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Editorial

Carcinogenesis, also called oncogenesis or tumorigenesis, is the formation of a cancer, whereby normal cells are transformed into cancer cells. The process is characterized by changes at the cellular, genetic, and epigenetic levels and abnormal cell division. Cell division is a physiological process that occurs in almost all tissues and under a variety of circumstances. Normally the balance between proliferation and programmed cell death, in the form of apoptosis, is maintained to ensure the integrity of tissues and organs. According to the prevailing accepted theory of carcinogenesis, the somatic mutation theory, mutations in DNA and epimutations that lead to cancer disrupt these orderly processes by disrupting the programming regulating the processes, upsetting the normal balance between proliferation and cell death. This results in uncontrolled cell division and the evolution of those cells by natural selection in the body. Only certain mutations lead to cancer whereas the majority of mutations do not.

Variants of inherited genes may predispose individuals to cancer. In addition, environmental factors such as carcinogens and radiation cause mutations that may contribute to the development of cancer. Finally random mistakes in normal DNA replication may result in cancer causing mutations. A series of several mutations to certain classes of genes is usually required before a normal cell will transform into a cancer cell.On average, for example, 15 "driver mutations" and 60 "passenger" mutations are found in colon cancers. Mutations in genes that regulate cell division, apoptosis (cell death), and DNA repair may result in uncontrolled cell proliferation and cancer.

Cancer is fundamentally a disease of regulation of tissue growth. In order for a normal cell to transform into a cancer cell, genes that regulate cell growth and differentiation must be altered.[6] Genetic and epigenetic changes can occur at many levels, from gain or loss of entire chromosomes, to a mutation affecting a single DNA nucleotide, or to silencing or activating a microRNA that controls expression of 100 to 500 genes. There are two broad categories of genes that are affected by these changes. Oncogenes may be normal genes that are expressed at inappropriately high levels, or altered genes that have novel properties. In either case, expression of these genes promotes the malignant phenotype of cancer cells. Tumor suppressor genes are genes that inhibit cell division, survival, or other properties of cancer cells. Tumor suppressor genes are often disabled by cancer-promoting genetic changes. Finally Oncovirinae, viruses that contain an oncogene, are categorized as oncogenic because they trigger the growth of timorous tissues in the host. This process is also referred to as viral transformation.

Genetic and epigenetic

There is a diverse classification scheme for the various genomic changes that may contribute to the generation of cancer cells. Many of these changes are mutations, or changes in the nucleotide sequence of genomic DNA. There are also many epigenetic changes that alter whether genes are expressed or not expressed. Aneuploidy, the presence of an abnormal number of chromosomes, is one genomic change that is not a mutation, and may involve either gain or loss of one or more chromosomes through errors in mitosis. Large-scale mutations involve either the deletion or duplication of a portion of a chromosome. Genomic amplification occurs when a cell gains many copies (often 20 or more) of a small chromosomal region, usually containing one or more oncogenes and adjacent genetic material. Translocation occurs when two separate chromosomal regions become abnormally fused, often at a characteristic location.

A well-known example of this is the Philadelphia chromosome, or translocation of chromosomes 9 and 22, which occurs in chronic myelogenous leukemia, and results in production of the BCR-abl fusion protein, an oncogenic tyrosine kinase. Small-scale mutations include point mutations, deletions, and insertions, which may occur in the promoter of a gene and affect its expression, or may occur in the gene's coding sequence and alter the function or stability of its protein product. Disruption of a single gene may also result from integration of genomic material from a DNA virus or retrovirus, and such an event may also result in the expression of viral oncogenes in the affected cell and its descendants.

DNA damage can also be caused by substances produced in the body. Macrophages and neutrophils in an inflamed colonic epithelium are the source of reactive oxygen species causing the DNA damage that initiates colonic tumorigenesis, and bile acids, at high levels in the colons of humans eating a high-fat diet, also cause DNA damage and contribute to colon cancer.

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