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**INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES**Available online at: <http://www.iajps.com>**Review Article****CANCER A GENETIC DISORDER - AN OVERVIEW**

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Abstract:

Cancer is primarily caused by mutations in growth, growth-inhibiting factor genes and pathways that inhibit the normal sequence of events associated with apoptosis. Normally cell cycle is controlled by signal transduction. Generally growth factors stimulate cell division and growth-inhibiting factors inhibit cell division. Healthy cells divide only when growth factor and growth-inhibiting factor balance favors cell division. Cancer cells divide without constraint. This paper reviews about the genes evidencing the possibility of cancer causing mechanism.

Key words: Mutation, Cell cycle, Signal transduction, Cell division and Genes.

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INTRODUCTION

Carcinogenic agents include chemicals in the environment, such as aniline and benzene, which are associated with the development of bladder cancer and leukemia, respectively. Environmental factors, such as excessive sun exposure, also may result in cancer. Viruses, including the human papilloma virus and hepatitis B, may be associated with the development of cancer. Some of the chemotherapy agents cause secondary cancers after therapy has been completed. Numerous factors may contribute to the development of cancer [1].

Tumours may arise from epithelial, connective (i.e., muscle, bone, and cartilage), lymphoid, and nerve tissue. A *lipoma* is a benign growth that resembles fat tissue. Precancerous cells have cellular changes that are abnormal but not yet malignant and may be described as *hyperplastic* or *dysplastic*. Hyperplasia occurs when a stimulus is introduced and reverses when the stimulus is removed. Dysplasia is an abnormal change in the size, shape, or organization of cells or tissues. Carcinomas arise from epithelial cells, whereas sarcomas arise from muscle or connective tissue. Adenocarcinomas arise from glandular tissue [1].

The four primary treatment modalities of cancer are surgery, radiation, chemotherapy, and biologic therapy. Surgery is useful to gain tissue for diagnosis of cancer and for treatment, especially those cancers with limited disease. Radiation plays a key role not only in the treatment and possible cure of cancer but also in palliative therapy. Together, surgery and radiation therapy may provide local control of symptoms of the disease. However, when cancer is widespread, surgery may play little or no role, whereas radiation therapy localized to specific areas may palliate symptoms. Drugs like alkylating agents, antimetabolites and hormones are used in cancer chemotherapy [1].

CANCER GENES

Cancer arises through a series of somatic alterations in DNA that result in unrestrained cellular proliferation. Most of these alterations involve actual sequence changes in DNA (i.e., mutations). They may originate as a consequence of random replication errors, exposure to carcinogens (e.g., radiation), or faulty DNA repair processes. While most cancers arise sporadically, familial clustering of cancers

occurs in certain families that carry a germline mutation in a cancer gene [2].

TYPES OF CANCER GENES

Retrovirus

Peyton Rous in the early 1900s revealed that a chicken sarcoma could be transmitted from animal to animal in cell-free extracts, suggesting that cancer could be induced by an agent acting positively to promote tumor formation. The agent responsible for the transmission of the cancer was a retrovirus (Rous sarcoma virus, RSV) and the oncogene responsible was identified 75 years later as *v-src* [2,3].

Point Mutation

Point mutation is a common mechanism of oncogene activation. For example, mutations in one of the *RAS* genes (*HRAS*, *KRAS*, or *NRAS*) are present in up to 85% of pancreatic cancers and 45% of colon cancers but are less common in other cancer types, although they can occur at significant frequencies in leukemia, lung, and thyroid cancers [2,3].

DNA Sequence Amplification

The second mechanism for activation of oncogenes is DNA sequence amplification, leading to over expression of the gene product. This increase in DNA copy number may cause cytologically recognizable chromosome alterations referred to as *homogeneous staining regions* (HSRs) integrated within chromosomes or *double minutes* (dmins) if extra chromosomal. The recognition of DNA amplification is accomplished through various cytogenetic techniques such as comparative genomic hybridization (CGH) or fluorescence in situ hybridization (FISH), which allow the visualization of chromosomal aberrations using fluorescent dyes. The first indication of the existence of tumor-suppressor genes came from experiments showing that fusion of mouse cancer cells with normal mouse fibroblasts led to a nonmalignant phenotype in the fused cells [4].

Retinoblastoma

Retinoblastoma (OMIM-180200) shows sporadic and hereditary forms and fits the pattern of a two-hit model, most common eye tumor of children, occurs from birth to 4 years of age and early treatment with gamma radiation is 90% effective. Two-hit mutation model for retinoblastoma (OMIM-180200), Sporadic retinoblastoma, which occur 60% of retinoblastoma

cases and develops in children with no family history, affects one eye, and hereditary retinoblastoma common in 40% of retinoblastoma cases. Multiple tumors involving both eyes [5].

Tumor Suppressor Genes

First discovered in 1960s by Henry Harris. Harris fused tumor cells with normal cells and discovered some of the hybrid cells were normal. Harris hypothesized that the normal cells produced gene products that suppressed uncontrolled cell proliferation. Some cancers show deletions of specific sites (tumor repressor genes) that normally inhibit cell growth and division. e.g., breast cancer, colon cancer, lung cancer [6,7].

CONCLUSION

In our review it is evidenced that cancer is majorly caused by genetically associated reasons rather than any other causes. Hence further research in cancer should be carried out in this area to develop good molecule to cure cancer permanently without significant side effects.

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