Editorial
Volume 6:4, 2021

Journal of Molecular Histology and Medical Physiology

Open Access

Genes that Predispose People to Cancer

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Introduction

Cancer is a disease characterised by uncontrolled cell proliferation caused by genetic mutations and misregulation of genetic information expression. Cancer cells proliferate haphazardly and selfishly, ignoring the ordered developmental possibilities for multicellular creatures, causing harm to the host and ultimately to themselves. Point mutations, copy number variations, chromosomal rearrangements, epigenetic changes, or abnormal forms of key regulator proteins in the form of amyloids can all create problems with cell growth regulation. There is just a single main initiation event in a few cases, but in the majority of cases, a series of events leads to alterations in regulatory networks, which leads to tumour development and invasiveness. Defects in genetic information maintenance and expression predispose cells to cancer and impact the results of therapeutic treatments.

The novel parallel analysis of cancer-specific DNA sequence variations in genomes and gene expression, which affected not individual genes but molecular pathways divided into four functional groups: signalling, metabolic, cytoskeleton, and DNA repair, revealed that the genes in the latter group had the highest mutation enrichment and upregulation levels. Members of the signalling and cytoskeleton groups were enriched by genes with numerous SNVs and suffered the most significant downregulation of gene expression, suggesting that they may play a role as carcinogenesis initiators. In a TP53-rat model of angiosarcoma, dysregulation of gene expression indicated an equal number of upregulated and downregulated genes. Upregulated genes for DNA helicases, chromosomal maintenance complexes, recombination, and replication, like those in the previous study, belong to the DNA repair group. Another technique to discover key genes in cancer and find tumour cell vulnerabilities is to use the Functional Signature Ontology (FUSION) approach of genome-wide, loss-of-function screening. Researchers can find new treatment targets using a gene expression-based high-throughput screening technique.

Recent research on tumour genomes found that mutations in replicative DNA polymerase genes increased genome instability, resulting in a propensity to cancer. However, colon and endometrial cancer-associated mutations (both spontaneous and hereditary) mostly affect the transcription factor of polymerase, which participates in leading strand DNA synthesis, suggesting that this polymerase performs a unique function throughout replication and human development.

Defects in DNA mismatch repair (Lynch syndrome) have long been known to increase the risk of colon and other cancers. The syndrome can also be identified in the aetiology of sarcomas, according to the current research. Fanconi anaemia is linked to malfunctions in the machinery that deals with the abnormal replication of damaged DNA with inter-strand DNA crosslinks. The relevance of Fanconi anaemia genes in noncanonical pathways like mitochondrial homeostasis, inflammation, and virophagy is highlighted during a review. The disease's diverse symptoms might be aggravated by an impairment of the cytoprotective pathways, which could increase the basic abnormalities in DNA repair processes in such individuals.

Hereditary ovarian cancer and, more often, breast cancer develops early in life and is passed down through several generations. DNA methylation patterns serve as a model for cancer susceptibility epigenetic inheritance, with a focus on the co-methylated network. Mutations in the DNMT3a gene, which is responsible for de novo methylation, are thought to be linked to multiple paragangliomas and papillary thyroid cancer.

How to cite this article: Khan Javed. Genes that Predispose People to Cancer. *J Mol Hist Med Phys* 6 (2021) 19

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Received 06 July 2021; **Accepted** 09 July 2021; **Published** 17 July 2021