Commentary
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Cancer Genetics

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Description

Cancer has been assumed to be a disease caused mostly by changes in the genome of the affected cells since Boveri discovered that chromosomal changes are a characteristic of the disease. The idea that cancer is caused by genetic changes is virtually intuitive nowadays, and developments in molecular biology and genomics have provided us with a plethora of tools to better understand and possibly treat cancer. The genetic mutations in cancer must be understood in the context of cellular organisation, differentiation, tissue organisation, host response, vulnerability angiogenesis, and so on, because science has always lived in a continuum.

Normal cells share many of the characteristics that distinguish cancer cells. Cell division, migration, and even invasion are among them (as exemplified by the trophoblast cells). Dysregulation and incorrect expression of these characteristics, on the other hand, distinguish cancer cells. Oncogenes, tumour suppressor genes, and genes that maintain genome integrity are the three principal types of genes that are altered in cancer. It's important to remember that cancer is a multi-step process, and a full-blown cancer phenotype requires numerous genetic changes.

Oncogenes

These are cellular genes that result in a malignant phenotype when they are mutated and/or incorrectly expressed in a way that increases their activity. Oncogenes like src, ras, and myc are well-known examples. The src gene, for example, codes for a tyrosine kinase, while the ras gene codes for a G protein and the myc gene codes for a nuclear protein involved in DNA replication. Acutely transforming retroviruses were the first to contain oncogenes (Rous Sarcoma Virus). These viruses cause a neoplastic phenotype when they infect immortalised but untransformed cells in vitro. It was later shown that these viral oncogenes were not naturally occurring viral genes, but rather were taken from the cellular genome and altered or overexpressed to cause cellular transformation. The requirement for oncogene cooperativity is consistent with the multi-step theory of carcinogenesis obtained from classical investigations, because a single mutant oncogene cannot convert initial cells. Oncogene cooperation frequently necessitates collaboration between oncogenes from distinct groups, such as nuclear oncogenes (e.g. myc with cytoplasmic e.g. ras).

Tumour suppresor genes (TSG)

These genes work in the cell to regulate cell division, similar to the brakes on an automobile. Loss of genetic materials is also a significant event in the development of neoplasia, as cytogenetic methods can indicate. The loss of genetic matter has been further defined using molecular methods. One allele of a TSG is usually lost, while the other is inactivated by point mutation. The Retinoblastoma gene was the first to illustrate TSG principles (RB). TSGs commonly affected include the p53 gene (affected in about half of all human cancers), the Wilms tumour gene, the p16 gene, and others.

Genes controlling genomic integrity

These genes are also known as caregiver genes. Inactivation of these genes causes genomic instability, which greatly raises the likelihood of oncogene and TSG mutations. The hMSH2 and hMLH1 genes, which are typically altered in human malignancies, have been intensively researched. Homologues of such genes can be traced back to yeasts, suggesting the underlying commonality of these biological processes, as with most oncogenes and TSGs. Microsatellite repeat sequences expand and contract at an unusually fast rate due to DNA mismatch repair errors. Hereditary Non Polyposis Colon Cancer (HNPCC) is an example of inherited faults in such genes, where examination of microsatellite repeats in leucocyte DNA is used to diagnose affected siblings in a family [1-5].

Conflict of Interest

None

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