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Commentary

Role of Oncogenes in Cancer Genetics and Statistical Analysis of Genomic Data

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DESCRIPTION

Chromosomal changes are a feature of cancer; it has been assumed that the disease is caused primarily by changes in the genome of the affected cells. The idea that cancer is caused by genetic changes is almost intuitive, and advances in molecular biology and genomics have given us many tools to understand and possibly combat cancer. Because science has always been a continuum, genetic alterations in cancer must be understood in the context of cellular organisation, differentiation, tissue organisation, host response and susceptibility angiogenesis, and so on. The characteristics that are thought to distinguish cancer cells are also found in normal cells. Cancer cells, on the other hand, are distinguished by dysregulation and inappropriate expression of these characteristics. Oncogenes, tumour suppressor genes, and genes that maintain the integrity of the genome are the three major types of genes that are altered in cancer. It is important to remember that cancer is a multi-step process that requires several genetic alterations for a full-blown cancer phenotype.

Oncogenes

When critical functions are altered or malfunctioning, most normal cells will undergo a programmed form of rapid cell death (apoptosis). Oncogenes that are activated can cause cells that are supposed to die to survive and proliferate instead. Most oncogenes began as proto-oncogenes, which were normal genes involved in cell growth and proliferation or apoptosis inhibition. Normal genes that promote cellular growth that are up-regulated due to mutation (gain-of-function mutation) predispose the cell to cancer and are thus referred to as "oncogenes." Multiple oncogenes, as well as mutated apoptotic or tumour suppressor genes, usually work together to cause cancer. Hundreds of oncogenes have been identified in human cancer since the 1970s. Many cancer drugs target oncogene-encoded proteins. The Philadelphia chromosomal defect is a reciprocal translocation, in which parts of two chromosomes swap places. As a result of juxtaposing the ABL1 gene on chromosome to a portion of the BCR (Breakpoint Cluster Region) gene on chromosome, a fusion gene is formed. This is a reciprocal translocation that results in an elongated and truncated chromosome. This chromosomal translocation is known as according to the International System for human Cytogenetic Nomenclature (ISCN). ABL1 is an abbreviation for Abelson, the name of a leukaemia virus that carries a similar protein. Breakpoint Cluster Region (BCR) is a gene that encodes a protein that acts as a guanine nucleotide exchange factor for Rho GTPase proteins.

The statistical analysis of genomic data is made possible by bioinformatics technologies. Oncogenes functional properties have yet to be determined. Potential functions include tumour formation transformational capabilities and specific roles at each stage of cancer development. Following the discovery of somatic cancer mutations in a cohort of cancer samples, bioinformatic computational analyses can be performed to identify likely functional and likely driver mutations. There are three common approaches for identifying mutations: mapping mutations, assessing the effect of mutation on the function of a protein or a regulatory element, and detecting signs of positive selection across a cohort of tumours. The approaches are not necessarily sequential, but there are important precedence relationships between elements from the various approaches.

Operomics seeks to integrate genomics, transcriptomics, and proteomics in order to better understand the molecular mechanisms underlying cancer development whereas cross-species comparisons are used in comparative oncogenomics to identify oncogenes. This research involves studying cancer genomes, transcriptomes, and proteomes in model organisms such as mice, identifying potential oncogenes, and comparing them to human cancer samples to see if homologues of these oncogenes play a role in the development of human cancers. The genetic changes seen in mouse models are similar to those seen in human cancers. These models are created using techniques such as retroviral insertion mutagenesis or cancer cell graft transplantation.

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