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Vital Significance of Proto-Oncogenes and Tumor Suppressor Genes in Studying Cancer Genetics

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Opinion Article

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DESCRIPTION

The ability of cancer cells to proliferate, divide, and infiltrate without the typical restraint mechanisms at work sets them apart from their normal counterparts. Rather perversely, cancer cells resemble embryonic cells which are undifferentiated (not committed) and exhibit similar properties. Cancer cells frequently exhibit cytoskeletal disruption, albeit its exact aetiology is yet unknown. Cancer cells also exhibit many abnormalities of the various cell surface molecules and the signalling pathways. The surface receptors can be abnormal or even be increased in quantity and have the potential to switch on abnormal and uncontrolled growth.

When a certain somatic mutation (alteration) occurs in the DNA of one of the cancer genome's functional genes, a cell turns cancerous. And though we currently understand some about the origins of cancer, its progressive nature or why, in many cases, somatic mutations arise in the first place, we do recognize the ability of cancer to evolve through a multi-step process with sequential clonal and subclonal selection, following Darwinian iconic evolutionary principles. This is possibly best illustrated by a branching tree, where some mutations result in clonal heterogeneity when they are present in all cancer cells investigated (truncal), while other mutations are only present in specific subclones (branches). Scientists often use the term 'cancer plasticity' to define some of the characteristics associated with this clonal heterogeneity, which is a crucial factor contributing to the lethality of cancers.

Historically, the gene with the cancer-specific mutation, often in small discrete sequences of DNA, is termed an oncogene. În cellular biology parlance, the original cellular gene (which becomes an oncogene on developing mutations) is sometimes referred to as a proto-oncogene. Therefore, when this oncogene is activated, it will produce an aberrant protein (referred to as an oncoprotein), which modifies the cell's capacity to divide, typically by boosting its proliferation. Oncogenes were first discovered as the transforming (from normal to cancerous) elements (v- oncs) of acutely transforming retroviruses. These viruses have not been linked to cancer in humans, although they do cause cancer in animals. Oncogenes tend to be named after the mammalian species in which virus-induced tumours were first reported. The evidence that oncogenes are involved in human cancer was strengthened by the determination of the oncogene products. For example, the oncogene ERB-B (a term derived from the erythroblastic leukaemia viral oncogene) was found to have homology to Epidermal Growth Factor Receptor (EGFR), which plays an important role in breast cancer; another oncogene MYC is implicated in growth control and plays a causative role in several subtypes of lymphomas and a number of other cancers, including head and neck cancers. We also possess another class of genes called tumour suppressor genes, such as the Tumour Protein p53 gene (TP53), in which the genetic function is inactivated; since the protein p53 is essential for regulating cell division, apoptosis and preventing tumour development, it is sometimes referred to as 'guardian of the genome'. These two types of gene thereby orchestrate the lifespan of the mutated cell. Just as proto-oncogenes can become oncogenes when they mutate, cancer-specific alterations acquired by the tumour suppressor genes can also lead to cancerous growth.

Though genomic diversity within individual cancers was first described by Julian Huxley, it was only after the wider application of NGS that the identification of the genomic landscapes of most human cancers and the bewildering clonal heterogeneity became apparent. For example, we now know how many genes are mutated in individual cancers, understand mutation timing and some of the other key factors contributing to intra- and inter-tumour clonal heterogeneity, which fosters cancer evolution. We also know the sequence of all coding genes for over 22,000 cancers, and more than 3 million somatic mutations have been discovered. We recognize that cancers evolve over a significantly variable time frame and the clonal architecture, genotype and phenotype can alter over this time period, to allow expansion, migration and invasion. Each tumour is made up of cells that have anything from a few to thousands of somatic mutations. The majority of mutations are "passengers," but a small number (now undefinable) are "drivers," conferring a selective growth advantage to the cell in which they arise and thus contributing to the development of cancer.

'Point mutations', or changes to a single base pair, are included in the category of mutations; a minor genomic change within a gene known as an indels, or insertions and deletions, in which nucleotides are either inserted or deleted; rearrangements, or a genomic break and fusion that results in the loss of a regulatory region without a fusion partner or an aberrant juxtaposition of two genes; A genomic region called an amplification, which is present

in multiple copies in cancer cells, and a genomic region called a deletion, which covers a gene that has been removed from the genome.