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Perspective

Conceptual Advances of Non-Transforming Retroviruses Activate Cellular Proto-Oncogenes

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DESCRIPTION

The revolution in cancer biology that has taken place during the past 20 years has utilized viruses as important tools. It is difficult to imagine that today's knowledge of the molecular causes of cancer would exist without the contributions of viral carcinogenesis. Viruses have emerged as the key to uncovering the secrets of cell growth control, despite being initially perceived as strange organisms that caused cancer in animals but were of little value to people. They have offered a conceptual framework that is relevant to all neoplasia, including cancer caused by viruses, and have uncovered the functional underpinnings of the genetic basis of cancer.

Over the past 20 years, tumour viruses have played two important roles in the study of cancer: first, as instruments for identifying and dissecting cell signaling and growth control pathways, and second, as recently recognized organisms responsible for human neoplasia. The DNA tumour viruses have been the main players in both fields, while the RNA tumour viruses have played a significant role in the first field of endeavor. This article will interpret significant advances and conceptual shifts brought about by viral carcinogenesis over the previous 20 years. The intriguing intricacies of unique virus-cancer systems cannot be presented due to space limitations, however recent publications can be consulted for in-depth explanations of the properties of various viruses, characteristics of various tumour virus systems and the ways that viral transforming genes work. Other articles in this issue discuss the biochemical and molecular specifics of cellular functions that tumour viruses may influence. Reverse transcription is a peculiar trait of the life cycle shared by retroviruses. Soon after infection, a virion-associated enzyme called reverse transcriptase converts the viral RNA genome into a double-stranded DNA copy that is then integrated into the cell's chromosomal DNA with the aid of the viral integrase enzyme. The cellular genome contains a wide variety of potential locations for proviral integration. With the exception of the fact that transcription is often regulated by sequences in the viral long arm, the integrated copy, known as the provirus, is similar to a cellular gene.

As proviruses are nearly never deleted from the genome, retroviral infection of a cell is irreversible. The basic virology investigations conducted in the 1960s and the Rous Sarcoma Virus (RSV), the transmissible cause of a chicken sarcoma that Rous discovered in 1911, gave the means for the identification of oncogenes. Genetic research demonstrating that replication and transformation reflected distinct viral gene activities and that the transforming gene (*src*) was not necessary for viral proliferation was made possible by the creation of a quantitative *in vitro* focus assay for RSV. A probe specific for the *src* gene was created after the ground-breaking discovery of reverse transcriptase, and most unexpectedly, it was discovered that it could hybridize to typical cell DNA. The fact that a tumour virus had a gene connected to a cellular gene has significant ramifications. In the 1950s and 1960s, a large number of animal retroviruses with oncogenic potential were discovered, mostly in mice and chickens. It was later shown that every oncogene carried by those transforming retroviruses originated in the cell. Protein kinases, growth factor receptors, growth factors, G proteins, transcription factors, and adaptor proteins are examples of the proto-oncogene classes of progenitor cellular genes that are involved in mutagenic signaling and growth control. Unexpectedly, an oncogene from a chicken sarcoma virus was discovered to be associated with a cellular transcription factor. In transforming retroviruses, more than 30 transduced oncogenes have been found, the most likely oncogene transduction pathway. Chimeric virus-cell transcripts may result from the integration of a provirus upstream of a proto-oncogene, and recombination during the subsequent cycle of replication may result in the integration of the cellular gene into the viral genome.

The inserted cellular sequences are typically duplicates of spliced transcripts without introns. This capture process typically results in the loss of viral genes, resulting in viruses that are incapable of reproducing themselves and rely on replication-competent helper viruses to deliver the viral activities required for

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reproduction. No appreciable amount of spontaneously occurring cancers is induced by acutely transforming retroviruses harboring oncogenes. Oncogene theft is an uncommon occurrence, and given the faulty phenotypic of these viruses, it is unlikely that they would persist for very long in nature. The tumour virologists' laboratory research provided assurance for the survival of the known transforming retroviruses.