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Discovery of Viral Oncogenes

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Abstract

In a wide range of animals and people, retroviruses are cancer-causing. The study of retroviruses has shed light on the mechanisms underlying human oncogenesis, including the identification of viral and cellular proto-oncogenes. The processes through which non-acute retroviruses-retroviruses without oncogenes-cause cancer are the focus of this review. The recurring idea is that these cancers develop as a result of viral DNA integration activating cellular proto-oncogenes by insertional activation. Review of early studies on proto-oncogene insertional activation in cancers brought on by viruses. Searches for common insertion sites (CISs) in virus-induced malignancies have helped researchers studying non-acute retroviruses find new proto-oncogenes. Retroviral infection of genetically susceptible mice (retroviral tagging) has been used to discover cellular proto-oncogenes active in particular oncogenic pathways and has helped to shed light on the cooperation between various proto-oncogenes in the development of tumours. The availability of the mouse genome sequence, high throughput DNA sequencing, and the PCR cloning of viral integration sites have all sped up the pace of proto-oncogene identification. Insertional activation has been shown to be a substantial danger in gene therapy studies using retroviral vectors to treat genetic abnormalities. Studies on non-acute retroviral oncogenesis shed light on the mechanics of oncogenesis as well as potential hazards.

Keywords: Proto-oncogene • Oncogenesis • Retroviral vector

Introduction

Research on oncogenic viruses has significantly advanced cancer molecular biology. Significant accomplishments include the finding of tumour suppressor genes, the identification of signal transduction pathways, the discovery of cellular protooncogenes and viral oncogenes. The history of cancer virology began roughly one hundred years ago with the identification of the RNA-containing retroviruses that cause avian sarcoma and acute leukaemia. The study of animal cancer viruses accelerated with the identification of DNA viruses that could transform cells in culture and the development of quantitative tests for transformation by DNA and RNA-containing tumour viruses in the late 1960s and early 1970s. Research on retroviruses was accelerated in 1970 by the identification of reverse transcriptase in these pathogens. In fact, RNA and DNA tumour viruses were at the forefront of cancer molecular biology before molecular cloning [1,2].

It was possible to physically extract virus particles and create specialised hybridization probes for viral DNA and RNA at a time when biological genes could not be examined in the same way. The understanding of RNA and DNA tumour viruses, many of which are animal viruses, is crucial for understanding human cancer. To start, many of the theories of viral oncogenesis uncovered in these studies have immediate application to human cancers, even those not brought on by viruses. In addition, a significant portion of human malignancies-roughly 15%-contain a viral component. Hepatitis B virus, Epstein-Barr virus, Kaposi's sarcoma herpesvirus, Merkel cell polyomavirus, human papillomavirus, and high-risk strains of HPV are all human DNA tumour viruses (HBV) [3]. Two human RNA viruses that cause cancer are the retrovirus Human T-cell Leukemia Virus Type I (HTLV-I) and the flavivirus Hepatitis C Virus (HCV). Additionally, a retrovirus (HIV-1 and -2) is what causes AIDS, and the immunodeficiency that characterises AIDS strongly contributes to the development of malignancies.

Most of these cancers have a viral component to them (KSHV and Kaposi's sarcoma, which was first discovered in AIDS patients, are two examples). The majority of the contributions are centred on retroviruses because they have been researched for more than fifty years. Acute transforming viruses are distinguishable from non-acute retroviruses, which do not carry viral oncogenes and generate tumours more slowly. Acute transforming viruses carry viral oncogenes and rapidly induce cancer. The typical acute transforming retrovirus is the v-src oncogene-carrying Rous sarcoma virus (RSV) [4].

Description

Non-coding RNAs called microRNAs (miRNAs) control a variety of cellular functions by directly binding to mRNA to influence translation efficiency and mRNA abundance and altering protein levels. Recent research has shown that miRNAs can influence RNA virus proliferation and pathogenicity either directly by binding to the RNA virus genome or indirectly by changing the host transcriptome as a result of the virus. Here, we go through what is currently known about how RNA viruses and cellular miRNAs interact. We also go over how miRNA expression can directly influence viral pathogenesis in cells and tissues. Understanding how cellular miRNAs function during viral infection may help researchers discover new ways to prevent the proliferation of RNA viruses or control the targeting of viral vectors at the cell level [5].

Conclusion

V-src was produced from the cellular proto-oncogene c-src. The other acute transforming retroviruses also carry captured copies of cellular proto-oncogenes. Viral oncogenes created from cellular proto-oncogenes boost cell growth or division constitutively by using the same methods as these oncogenes do. Acute transforming retroviruses and their oncogenes were once the subject of in-depth research, but the attention has now shifted to the typical functions and regulation of cellular protooncogenes. As a result of the viral long terminal repeats (LTRs) interacting with the proto-oncogenes, non-acute retroviruses typically induce tumours by transcriptionally activating cellular proto-oncogenes (LTR activation of protooncogenes).

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Conflict of Interest

None.

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