

The Role of DNA Viruses in Human Cancer

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ABSTRACT: This review discusses the possible involvement of infections-associated cancers in humans, with virus infections contributing 15% to 20% of total cancer cases in humans. DNA virus encoded proteins interact with host cellular signaling pathways and control proliferation, cell death and genomic integrity viral oncoproteins are known to bind cellular Deubiquitinases (DUBs) such as cyclinomatosis tumor suppressor, ubiquitin-specific proteases 7, 11, 15 and 20, and A-20 to improve their intracellular stability and cellular signaling pathways and finally transformation. Human papillomaviruses (cervical carcinoma, oral cancer and laryngeal cancer); human polyomaviruses (mesotheliomas, brain tumors); Epstein-Barr virus (B-cell lymphoproliferative diseases and nasopharyngeal carcinoma); Kaposi's Sarcoma Herpesvirus (Kaposi's Sarcoma and primary effusion lymphomas); hepatitis B (hepatocellular carcinoma (HCC)) cause up to 20% of malignancies around the world.

KEYWORDS: DNA viruses, cancer, oncogenic viruses

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Introduction

Cancer is a devastating disease which affects people and their families as well as health care systems by 10.9 million new cases and 6.7 million deaths annually.¹ The cell-free transmission of oral dog warts was described by McFadyen and Hobday in 1907, and similar findings were published in 1898 by Ciuffo for human warts.^{2,3} The Epstein-Barr virus (EBV) discovery from Burkitt's lymphoma (BL) in 1964 and the hepatitis B discovery in human sera in 1970, resulted new studies in the roles of viruses in human cancer.⁴ Human papillomavirus (HPV) 16 and 18 DNAs were isolated from human cervical cancer specimens by nucleic acid hybridization.⁵ DNA tumor viruses in permissive cells, carried on replication leading to cell lysis and cell death and in nonpermissive cells mostly integrated into the cell chromosomes.^{6,7} DNA viruses and other cofactors such as immune suppression and the environment (smoking, drinking, and genetic susceptibility) have effects on the development of viral-associated malignancies.^{4,8-10}

Human Oncogenic DNA Viruses

Carcinogenic process contributed by oncogenic viruses with the association of 15% to 20% viral infection.¹¹ Oncogenic viruses have used different strategies for evading the host immune response to establish long-term persistent infections.¹²⁻¹⁴ Human tumor viruses belong to a number of DNA virus families such as Hepadnaviridae (HBV), Herpesviridae, Papillomaviridae (HPV), human polyomaviruses (HPyV),

human herpes virus 8 (HHV8), Epstein-Barr Virus (EBV) are involved in various cancers.¹⁵⁻¹⁷ DNA oncogenes of viruses are necessary for replication of the virus and also controlling mammalian cell growth. The retinoblastoma gene product (pRB) is the first cellular tumor suppressor, which interact with The E2F genes, which play roles in cell cycle progression, which targeted by DNA virus oncoproteins. The second suppressor tumor is p53. These modulations, which are further interpreted in later chapters, eventually cause S-phase induction. Tumor suppressor genes participate in cell growth and mutation in one allele causes great risk for malignancy development (Figure 1).¹⁸⁻²⁰

Oncogenesis

Oncogenic viruses comprise of both DNA and RNA viruses. Virus oncogenes are responsible for encoding the viral proteins that are necessary for the virus to replicate. Oncogenic viruses lead cell transformation and development of malignant tumors.²¹ Protooncogenes (c-onc genes) are the cellular genes such as protein kinases, growth factors, growth factor receptors, DNA binding proteins, and their activation with mutation leads to uncontrolled cell growth. Point mutations, deletions, or chromosomal translocations transform C-onc genes into V-onc²² (Table 1).

We will describe each of the DNA viruses that are involved in cancer development and progression as follows:

Human papillomavirus (HPV). The Papillomaviridae family of viruses includes the small, non-enveloped, double-stranded DNA viruses known as Papillomaviruses (PVs). HPV virus is divided into 2 groups: mucosal and cutaneous, with a range of

Correction (October 2023): Article updated to include Mehdi Fazlalipour in the author byline along with his affiliations 1 and 2. The "Author Contributions" section is amended.



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