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Editorial: Recent advances in diagnosis, prognosis, and therapy of oncogenic virus- driven tumors

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Editorial on the Research Topic

Recent advances in diagnosis, prognosis, and therapy of oncogenic
virus-driven tumors

In recent years, the understanding of the molecular mechanisms underlying virus-induced tumorigenesis has grown exponentially, leading to breakthroughs in diagnosis, prognosis, and therapy. The development of cancer malignancy is multifaceted, involving a complex interplay of various factors rather than relying solely on one aspect. This intricate process encompasses genetic mutations, epigenetic modifications, environmental factors, and lifestyle choices. While numerous factors play a role in cancer, it is noteworthy that certain viruses, known as oncogenic viruses, can drive the cancer onset and progression in humans (1, 2). Remarkably, oncogenic viruses are implicated in a notable proportion of human cancers, accounting for >20% of cases across different cancer types (3, 4).

Oncogenic viruses are commonly classified as either DNA or RNA viruses. Notable viral agents, including high-risk types of human papillomavirus (HPV) (5), human polyomaviruses (HPyV) (6), Epstein-Barr virus (EBV), hepatitis B virus (HBV), hepatitis C virus (HCV), human immunodeficiency virus type 1 (HIV-1), human T cell lymphotropic virus type 1 (HTLV-1), and human herpesvirus type 8 (HHV-8 or Kaposi's sarcoma herpesvirus, KSHV), have been designated as human carcinogens (Group 1) by the International Agency for Research on Cancer (IARC) (<https://monographs.iarc.who.int/list-of-classifications/>), while additional viruses, such as Merkel cell polyomavirus (MCPyV), are currently considered as probably carcinogenic in humans (Group 2A). Furthermore, several other viruses have been postulated, to varying degrees, to potentially contribute to oncogenesis. Of note, patients affected by immune system impairments, such as those immune-suppressed/immune-compromised, are more prone to develop oncogenic virus-driven tumors because unable to counteract in full, with their immune system, the oncogenic potential of DNA and RNA tumor viruses.

Viruses are recognized for their ability to replicate by hijacking host mechanisms to serve their own purposes. Oncogenic viruses possess the unique ability to induce tumorigenesis, facilitating the spread of malignancy from infected cells to adjacent healthy cells (1, 3, 4). The

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