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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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Table of Contents Recent Advances In Virus Diagnosis

1. Understanding the eBook Recent Advances In Virus Diagnosis
 - The Rise of Digital Reading Recent Advances In Virus Diagnosis
 - Advantages of eBooks Over Traditional Books
2. Identifying Recent Advances In Virus Diagnosis
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Recent Advances In Virus Diagnosis
 - User-Friendly Interface
4. Exploring eBook Recommendations from Recent Advances In Virus Diagnosis
 - Personalized Recommendations
 - Recent Advances In Virus Diagnosis User Reviews and Ratings
 - Recent Advances In Virus Diagnosis and Bestseller Lists
5. Accessing Recent Advances In Virus Diagnosis Free and Paid eBooks

- Recent Advances In Virus Diagnosis Public Domain eBooks
- Recent Advances In Virus Diagnosis eBook Subscription Services
- Recent Advances In Virus Diagnosis Budget-Friendly Options
- 6. Navigating Recent Advances In Virus Diagnosis eBook Formats
 - ePub, PDF, MOBI, and More
 - Recent Advances In Virus Diagnosis Compatibility with Devices
 - Recent Advances In Virus Diagnosis Enhanced eBook Features
- 7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Recent Advances In Virus Diagnosis
 - Highlighting and Note-Taking Recent Advances In Virus Diagnosis
 - Interactive Elements Recent Advances In Virus Diagnosis
- 8. Staying Engaged with Recent Advances In Virus Diagnosis
 - Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Recent Advances In Virus Diagnosis
- 9. Balancing eBooks and Physical Books Recent Advances In Virus Diagnosis
 - Benefits of a Digital Library
 - Creating a Diverse Reading Collection Recent Advances In Virus Diagnosis
- 10. Overcoming Reading Challenges
 - Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
- 11. Cultivating a Reading Routine Recent Advances In Virus Diagnosis
 - Setting Reading Goals Recent Advances In Virus Diagnosis
 - Carving Out Dedicated Reading Time
- 12. Sourcing Reliable Information of Recent Advances In Virus Diagnosis
 - Fact-Checking eBook Content of Recent Advances In Virus Diagnosis
 - Distinguishing Credible Sources
- 13. Promoting Lifelong Learning
 - Utilizing eBooks for Skill Development

- Exploring Educational eBooks

14. Embracing eBook Trends

- Integration of Multimedia Elements
- Interactive and Gamified eBooks

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