MEDWIN PUBLISHERS
Committed to Create Value for Researchers

Virology & Immunology Journal
ISSN: 2577-4379

The Complicated Relationship between Cancer Disease and Viral Infection: Facts and Understanding

Hassan BAR[1]* and Mohammed AH[2]

[1]Department of Pharmacy, Al Rafidain University College, Iraq

[2]School of Pharmacy, Monash University Malaysia, Malaysia

***Corresponding author:** Bassam Abdul Rasool Hassan, Department of Pharmacy, Al Rafidain University College, Iraq, Email: bassamsunny@yahoo.com

Mini Review

Volume 7 Issue 2

Received Date: June 07, 2023

Published Date: June 23, 2023

DOI: 10.23880/vij-16000313

Abstract

Environmental, lifestyle, host-related, infectious, and genetic factors have all been connected to the oncogenic process. Knowing what causes cancer and what variables increase one's risk is crucial for targeting screening and prevention efforts towards high-risk populations. Infectious agents have been identified as a significant preventable risk factor for cancer among various other risk factors. Among this cohort, viruses are the predominant etiological agents implicated in the pathogenesis of neoplastic disorders in humans.

Keywords: Cancer; Virus; Oncogenesis

Introduction

The oncogenic process has been linked to various factors including environmental, lifestyle, host-related, infectious, and hereditary factors. Comprehending the aetiology and risk factors of cancer serves as a valuable mechanism for identifying populations at elevated risk and enhancing screening protocols and preventative measures [1]. Infectious agents have been identified as a significant preventable risk factor for cancer among various other risk factors. Among this cohort, viruses are the predominant etiological agents implicated in the pathogenesis of neoplastic disorders in humans. Over the last few decades, in cancer biology, viruses have played a significant impact., making significant contributions to our understanding of the pathways involved in cell signaling and growth control that ultimately leads to the development of cancer [1]. Kaposi's sarcoma-associated herpesvirus (KSHV; also known as human herpesvirus 8), human immunodeficiency virus (HIV), human T-cell lymphotropic virus type 1 (HTLV-1), human papilloma virus (HPV), and Merkel cell polyomavirus (MCPyV) are all recognised as human oncoviruses. This finding is predicated on data gathered from several studies conducted over the last half-century in the fields of experimental science, clinical medicine, and epidemiology [2]. Infection with the bacterium *Helicobacter pylori* is responsible for an additional 5% of malignancies worldwide, most commonly stomach cancer. There is a huge range in the estimated number of incident cases connected with different viruses around the world, from 640,000 for HPV to 3,000 for HTLV. There are significant implications for turning the understanding of virus-induced malignancies into public health treatments, as approximately 85% of the burden of virus-induced cancers is borne by individuals in developing countries of the world. Moreover, some viruses are more likely to cause cancer in one sex than the other. Nearly 90% of HPV-related malignancies affect women, but 2/3 of HBV, HCV, and EBV cancers affect men [1,2].

Mechanisms of Oncogenesis

The oncogenic pathways of numerous tumour viruses entail persistent expression of particular viral genes that modulate activities related to proliferation, evasion of apoptosis, and/or immune response via interacting with gene targets within cells. Oncoproteins, such as E6 and E7 of HPVs, LMP1 of EBV, Tax of HTLV-1, and T antigen of MCPyV, are among the known examples [2,3].

Liver cancer progression may be directly influenced by HCV and HBV proteins, it is more likely that these viruses induce cancer indirectly through the persistence of infection, which leads to chronic inflammation and tissue damage [2,3]. It is possible that KSHV exerts its effects mainly through the modification of intricate cytokine/chemokine networks. MicroRNAs that are encoded by viruses, particularly those found in KSHV and EBV, are directly involved in the development of cancer [1-3]. In certain viruses, such as MCPyV and HPV, the process of malignant progression typically entails genetic alteration of the host genome through mutation and/or viral integration, thereby impeding its ability to reproduce [1-3].

The acquisition of HIV infection poses a significant risk for the development of various types of cancers, particularly those that are linked with other viral infections. HIV infection is thought to influence oncogenesis in a roundabout way, as it hinders the regular immune functions of the host that would otherwise regulate or eradicate oncovirus infections and/or monitor the emergence of tumours through immunosurveillance [1-4]. Regarding, the EBV is a widely distributed gamma herpesvirus that infects a large portion of the global population without causing any noticeable symptoms. In instances of compromised immune function, the Epstein-Barr virus has the potential to initiate malignancies in humans that originate from both epithelial and lymphoid tissues. The oncogenic capacity of EBV is evidenced by the transformation of dormant B cells into lymphoblastoid cell lines (LCLs) through in vitro infection. The utilisation of cell lines and primary infection by using virus particles with modified genomes, in conjunction with recent technological advancements, has facilitated the comprehension of the fundamental mechanisms involved in EBV-induced B-cell lymphomagenesis [1,5].

The incidence of cancer caused by HPV infection accounts for roughly 5% of all cancer cases and is linked to 30% of all cancers that are pathogen-related. Cervical cancer ranks third in prevalence among women globally, with approximately 70% of cases attributed to high-risk human papillomaviruses (HR HPVs) of genotypes 16 and 18. The primary mode of transmission for HPV infection is sexual contact, although there are also potential methods in which viruses can spread laterally and vertically. Following HPV infection of basal keratinocytes or ectoendocervical transition zone cells, the viral DNA remains present in the episomal form. Typically, the immune system is responsible for the elimination of infected cells. In some instances, the process of elimination may prove ineffective, leading to the persistence of HPV infection. The process of HPV replication within dividing epithelial cells is concomitant with a rise in the expression levels of the E6 and E7 oncoproteins [1,6-8]. The oncoproteins in question are accountable for inducing genomic instability, interfering with the cell cycle, promoting cell proliferation, enabling cellular immortality, and ultimately leading to the malignant transformation of cells that have been infected with HPV. Furthermore, the oncoproteins E6 and E7 have the capability to trigger immunosuppression, thereby hindering the immune system's ability to identify HPV-infected and transformed cells. E6 and E7 expression are both upregulated after HPV becomes integrated into the host cell's DNA, which in turn contributes to the development of HPV-associated malignancies. The administration of prophylactic HPV vaccines has the potential to prevent more than 80% of anogenital cancers that are associated with HPV [1,6-8].

Hepatocellular carcinoma (HCC) is a prevalent form of cancer on a global scale. The prevailing belief is that a significant proportion, namely 80%, of hepatocellular carcinomas are associated with persistent infections caused by the hepatitis B (HBV) or hepatitis C (HCV) viruses. Persistent hepatitis B virus (HBV) and hepatitis C virus (HCV) infections have the potential to modify the functioning of hepatocytes in comparable manners and could potentially employ analogous pathways to impact the progression of HCC. Considerable advancements have been made in comprehending the molecular biology of Hepatitis B Virus (HBV) and Hepatitis C Virus (HCV) and recognising the cellular signal transduction pathways that are modified by the infections caused by HBV and HCV [1,9]. The exact molecular pathways connecting HBV and HCV infections with the emergence of HCC remain incompletely comprehended. Nonetheless, there exists substantial evidence indicating that the inflammatory reactions to viral infections, the consequential damage and regeneration of hepatocytes, and the functions of HBV- or HCV-encoded proteins all play a role in the transformation of hepatocytes [1,9].

Endothelial cell infection or hematopoietic progenitor cell infection by KSHV induces alterations in their morphology, glucose metabolism, growth rate, lifespan, and gene expression, ultimately leading to the development of Kaposi's sarcoma. The oncogenic properties of KSHV are evidenced by the significant upregulation of various pro-angiogenic molecules upon infection of endothelial cells. These molecules include members of the VEGF-VEGFR

and angiopoietin families, as well as cyclooxygenase 2 (COX2) and angiogenin. Nonetheless, it is worth noting that within the majority of experimental setups, the endothelial cell infection with KSHV in vitro results in alterations in morphology and an elongated lifespan, thereby conferring a heightened capacity for survival in the face of apoptotic stimuli [1,10]. However, it is important to acknowledge that such infection does not necessarily lead to complete neoplastic transformation. Furthermore, despite the presence of oncogenic genes in KSHV that have the potential to induce all malignant phenotypes related to KS, the incidence of KS resulting from KSHV infection in the general population is low. This highlights the presence of cofactors, such as HIV or drug-induced immunosuppression, which are necessary for the virus to initiate tumorigenesis [1,10].

Conclusion

The identification and delineation of oncoviruses have been a prominent area of investigation in the field of biomedical research for the past few decades. The aforementioned has yielded significant revelations pertaining to fundamental cellular biology and the processes underlying the development of cancer.

References

1. Carrillo-Infante C, Abbadessa G, Bagella L, Giordano A (2007) Viral infections as a cause of cancer. International journal of oncology 30(6): 1521-1528.

2. Schiller JT, Lowy DR (2021) An Introduction to Virus Infections and Human Cancer. Recent Results Cancer Research 217: 1-11.

3. Li X, An L, Jin Y, Gui J, Lin W, et al. (2023) Editorial: Viral infection, tumor development and host immune response. Front Cell Infect Microbiol 13: 1177032.

4. Olivares-Hernández A, Figuero-Pérez L, Miramontes-González JP, López-Gutiérrez Á, González-Sarmiento R, et al. (2021) Immune system disorders, cancer and viral infections: A new treatment opportunity for the immune checkpoint inhibitors. Life 11(12): 1400.

5. Saha A, Robertson ES (2019) Mechanisms of B-cell oncogenesis induced by Epstein-Barr virus. J Virol 93(13): e00238-e00319.

6. Münger K, Baldwin A, Edwards KM, Hayakawa H, Nguyen CL, et al. (2004) Mechanisms of human papillomavirus-induced oncogenesis. Journal of virology 78(21): 11451-11460.

7. Araldi RP, Sant'Ana TA, Módolo DG, de Melo TC, Spadacci-Morena DD, et al. (2018) The human papillomavirus (HPV)-related cancer biology: An overview. Biomed Pharmacother 106: 1537-1556.

8. Vonsky M, Shabaeva M, Runov A, Lebedeva N, Chowdhury S, et al. (2019) Carcinogenesis associated with human papillomavirus infection. mechanisms and potential for immunotherapy. Biochemistry (Moscow) 84(7): 782-799.

9. Bouchard MJ, Navas-Martin S (2011) Hepatitis B and C virus hepatocarcinogenesis: lessons learned and future challenges. Cancer Lett 305(2): 123-143.

10. Mesri EA, Cesarman E, Boshoff C (2010) Kaposi's sarcoma herpesvirus/ Human herpesvirus-8 (KSHV/ HHV8), and the oncogenesis of Kaposi's sarcoma. Nat Rev Cancer 10(10): 707-719.