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Cancer-Causing Viruses: A Comprehensive Review of Oncogenic Mechanisms and Clinical Implications

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الفيروسات المسببة للسرطان: مراجعة شاملة للآليات المسببة للسرطان والآثار السريرية

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ABSTRACT

Background

Viruses are recognized as significant contributors to cancer development, accounting for approximately 15–20% of human malignancies worldwide.

Aim

This review aims to provide a comprehensive overview of the major cancer-causing viruses and to elucidate their oncogenic mechanisms and clinical implications.

Key point

The review focuses on *Human Papillomavirus (HPV)*, *Hepatitis B and C viruses (HBV,*and *HCV)*, *Epstein–Barr virus (EBV)*, *Human T-cell Leukemia Virus type 1 (HTLV-1)*, and *Kaposi's Sarcoma-associated Herpesvirus (KSHV)*. Each virus employs distinct molecular strategies to manipulate host cellular pathways, promoting uncontrolled cell proliferation, evasion of apoptosis, and genomic instability hallmarks of cancer. The review explores viral integration, expression of viral oncogenes, immune evasion, and chronic inflammation as key mechanisms in virus-induced carcinogenesis. Additionally, the clinical implications of these findings are discussed, including the development of diagnostic biomarkers, and targeted antiviral therapies.

Conclusions

Understanding the interplay between viral infection and tumor biology offers valuable insights into cancer prevention, early detection, and novel therapeutic approaches.

Keywords: Cancer, Viruses, *HBV, HCV, EBV, KSHV, HTLV-1*

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INTRODUCTION

Cancer is one of the most dreaded diseases of today's world. It represents a global public health disaster of colossal dimensions affecting individuals, and their families, communities as well as health care system worldwide [1]. During the last few decades, cancer incidence rates of several malignancies have increased remarkably globally, thus scientists are focusing more on understanding the biological and molecular perspectives of cancer development and progression to find better preventions as well as treatment strategies [2]. Carcinogenesis is a complex and multi-step process that results from the interaction of various genetic and environmental factors such as genetic mutations, deregulation of cell-cycle control, chronic inflammation, exposure to carcinogens and viral agents, which are progressively being recognized as strong promoters for tumour development [3]. Current estimates suggest 16-22% of human cancers are due to direct viral infection which underscores the essential nature of investigating the involvement of viruses in cancer initiation [4].

Viruses capable of causing cancer are called carcinogenic viruses and they form virus groups that interfere with the regulation systems of host cells through either integrating their own genes or expressing specific proteins which control cellular division as well as apoptosis [5, 6]. Some of these viruses are able to incorporate their own nucleic acids into the genome of host cells, which can result in up-regulation or down-regulation of oncogenes and tumor suppressor genes[7].

Among the most prominent viruses known to cause cancer are human papillomavirus (HPV), which is closely linked to cervical cancer and other reproductive cancers, and Epstein-Barr virus (EBV), which was associated with several species of lymphoma and nasopharyngeal cancer. Hepatitis B and C viruses (HBV and HCV) also play a key role in the development of liver cancer, while Kaposi's sarcoma-associated herpesvirus (HHV-8) is associated with this type of cancer, particularly among immuno-compromised individuals, especially patients afflicted with human immunodeficiency virus (HIV) **[8].** These viruses vary in their degree of association with cancer, ranging from a weak association to a direct association exceeding 90%, as is the case with some HPV-associated cervical cancers[9].

Oncovirus research has changed the way in which we consider basic cellular pathways that are dysregulated during cancer [10]. These studies have identified multiple nodal regulatory hubs in the cells such as those mediated by p53, Rb and PI3K/Akt signaling pathways that control the pro-survival/prodeath balance within the cell. The study of viral interference with these pathways has also provided an opportunity to develop targeted diagnostic and therapeutic tools that target these mechanisms, leading to the emergence of novel therapeutic strategies, including vaccines against oncoviruses such as HPV and HBV, and which have successfully decreased the occurrence of some cancers[9, 11].

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2 CLASSIFICATION OF ONCOGENIC VIRUSES

Human tumor viruses are categorized into various virus families, especially the RNA virus families Retroviridae and Flaviviridae [12]. The DNA viral families comprise Hepadnaviridae, Herpesviridae, and Papillomaviridae. Viruses significantly associated with human malignancies include: (i) HTLV-1 (adult T-cell leukemia (ATL))[13]. Human papillomavirus (HPV) is linked to cervical carcinoma, cutaneous malignancies in persons with epidermodysplasia verruciformis (EV), head and neck cancers, and multiple anogenital neoplasms[14]. HHV-8 is linked to Kaposi's sarcoma, primary effusion lymphoma, and Castleman's disease[9, 15]. (iv) Epstein-Barr Virus (EBV) is correlated with Burkitt's Lymphoma (BL)[16], nasopharyngeal carcinoma (NPC), post-transplant lymphomas, and Hodgkin's disease[17]; (v) Hepatitis B Virus (HBV) and Hepatitis C Virus (HCV) are associated with hepatocellular carcinoma (HCC)[18, 19]. Here are some viruses that have been linked to human cancers: (i) simian vacuolating virus 40 (SV40), which is associated with brain, bone, and mesothelioma cancers[20]; (ii) BK virus (BKV), which is linked to prostate cancers[21]; (iii) JC virus (JCV), which is linked to brain cancers[22]; and (iv) human endogenous retroviruses (HERVs), which are linked to germ cell tumors, breast, and ovarian cancers. Melanoma is one type of cancer[23]; (v) HMTV is associated with breast cancer [24]; while TTV is associated with cancer of the gastrointestinal tract, lungs and breast cancer, myeloma, as well as additional malignancies [25] . HMTV is associated with breast cancer; while TTV is associated with cancer of the gastrointestinal tract, lungs and breast cancer, myeloma, as well as additional malignancies [24]. Overview of viruses with established or potential associations with human cancer. Research on RNA and DNA tumor viruses has led to the discovery of tumor suppressor genes and oncogenes, significantly enhancing our comprehension of the etiology of carcinogenesis, whether triggered by viruses or other factors.

2.1 DNA viruses (such as HPV, EBV, HBV, HHV-8)

One common mechanism by which oncogenic DNA viruses induce cancer is by integrating their DNA in the genome for the host cell. This integration may inactivate tumor suppressor genes or stimulate oncogenes, contributing to cell cycle disruption and cancer transformation.

2.2 RNA viruses (such as HTLV-1, HCV)

RNA tumor viruses, also known as retroviruses, are among the most important viruses associated with tumor formation and the stimulating of cancer cell growth in living organisms. Their importance lies in their ability to alter the genetic materials in the host cell and cause cellular changes that lead to tumors [26]. Medically, these viruses pose a significant challenge in diagnosing and treating cancer, but they are also used today as vital tools in research. They have helped identify tumor suppressor genes and oncogenes and contributed to the development of gene delivery systems in gene therapy research[27].

2.2 .1 HTLV-1

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HTLV-1 was a complex delta-type retrovirus that causes tropical spastic paralysis/HTLV-1-associated myelopathy (TSP/HAM) and adult T-cell leukemia (ATL)[28]. The HTLV-1 virus was native to the Caribbean, Africa, South America, and Japan. Only a small fraction (2-6%) of the approximately 20 million people infected with HTLV-1 will develop ATL. It appears that HTLV-1 infection is insufficient to induce T-cell transformation, given the long clinical delay and relatively low accumulated lifetime danger of ATL. Specific biological events are ambiguous; nonetheless, multiple factors, including the virus, host cells, and immunological elements, contribute to the development of ATL [28] (Figure 1). The main transactivator of HTLV1 is the multifunctional viral helper protein Tax, according to multiple studies [29]. Among the many cellular transcriptional signaling networks that Tax affects is the nuclear factor kappa B (NF-κB) pathway, which controls the expression of viral genes through viral long terminal repeats (LTRs) [30].

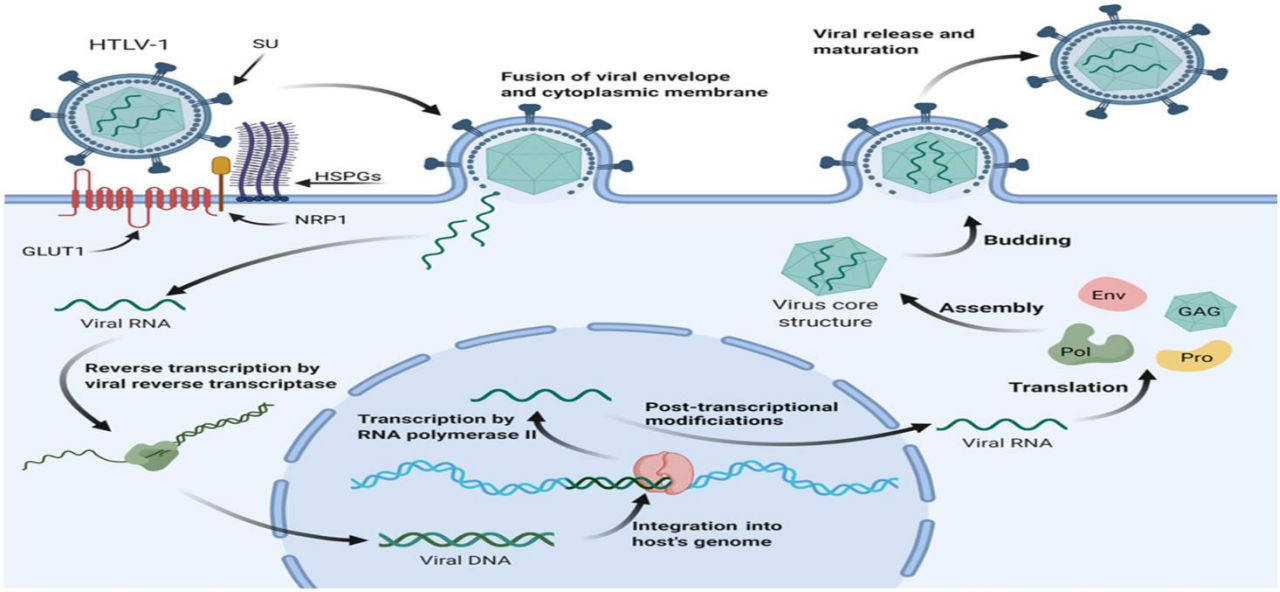

Fig. 1. Mechanisms of cell infection and multiplication by HTLV-1. The surface subunit (SU) of HTLV-1 glycoproteins engages with heparan sulphate proteoglycans (HSPGs) present in the cytoplasmic membrane of the target cell. A complex is then established between the viral envelope, heparan sulphate proteoglycans (HSPGs), neuropilin-1 (NRP1), and glucose transporter-1 (GLUT1). The envelope merges with the cytoplasmic membrane, releasing the viral RNA into the cytoplasm, where it undergoes reverse transcription and is transported to the nucleus as viral DNA for integration into the host genome. The provirus is subsequently transcribed by the cell's RNA polymerase II, and following post-transcriptional changes, the mature viral mRNA is delivered to the cytoplasm. The translation of viral mRNA and alternatively spliced mRNAs produces the proteins essential for viral assembly within the host cell, including envelope glycoprotein (Env), polymerase (Pol), protease (Pro), and structural proteins (GAG). These proteins, along with two copies of viral RNA, translocate to the budding site and are expelled from the cell's surface to subsequently develop into infectious viral particles through protease-dependent action[31].

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Among the viral components, the tax protein is pivotal in the pathogenesis of HTLV-1. Tax is a transactivator protein that modulates the transcription of both viral and host cellular genes[32] . It activates transcription through interaction with the viral long terminal repeats (LTRs) and initiates signaling cascades that modulate standard cell cycle regulation, apoptosis and escape from the immune system [33]. One of the most well-characterized pathways affected by Tax is the nuclear factor kappa B (NF-κB) signaling pathway[34]. Tax constitutively activates both the canonical and non-canonical NF-κB pathways, leading to the persistent expression of survival and proliferation genes. This chronic NF-κB activation results in enhanced cell survival, resistance to apoptosis, and immune dysregulation, all of which contribute to the transformation of CD4+ T-cells and the development of ATL[35]. Furthermore, Tax also interacts with other key cellular pathways, CREB/ATF pathways, enhancing viral gene transcription, p53 tumor suppressor inhibition, reducing apoptotic response, Cell cycle dysregulation via modulation of cyclins and CDK inhibitors, and DNA repair interference, contributing to genetic instability in infected cells[32].

In contrast to other established DNA tumor viruses that typically necessitate the persistent expression of viral oncoproteins to maintain transformation [36, 37], tax transcripts are found in merely 40% of adult T-cell leukemias (ATLs) [38] , suggesting the Tax could have been crucial for beginning transformation, although not for maintaining the altered phenotype. The principal target of the host's cytotoxic T lymphocyte (CTL) responses are Tax; thus, the inhibition of Tax expression allows infected host cells to evade immune detection and promotes the preferential selection of these cells during the progression of ATL [38]. Cell of ATL may diminish Tax expression via several mechanisms, including the deletion of the viral promoter responsible for tax transcription, the 5′-LTR [38], mutations within the tax gene , and epigenetic modifications affecting the 5′-LTR [39].

2.2.2 Hepatitis C virus (HCV)

Hepatitis C virus represents one of the most prominent viruses that infects the liver, triggering persistent inflammation, which in turn can cause major consequences including cirrhosis and hepatocellular cancer [40]. Figure 2 shows the mechanism HCV induced hepatocellular carcinoma, this virus, which is a member of the Flaviviridae family and the Hepacivirus genus, it is the only positive-sense RNA virus known to cause cancer in humans, with a genome approximately 9.6 kilobases long, encoding structural and nonstructural proteins that play essential roles in its viral cycle and pathogenesis [41]. The viral genome includes an open reading frame (ORF) that is translated into a long polypeptide that is cleaved into 10 proteins by viral and cellular proteins. The structural proteins include Core, E1, and E2, while the nonstructural proteins include NS2, NS3, NS4A, NS4B, NS5A, and NS5B. These proteins play important roles in viral replication, immune evasion, and modulation of cellular responses, making them vital targets for drug therapy[42]. HCV is primarily transmitted through contact with contaminated blood. Common modes of transmission include transfusion of unscreened blood or blood products, sharing of

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needles among drug users, needlestick injuries in healthcare facilities, vertical transmission from mother to fetus, and sexual contact[43]. An estimated 58 million people are infected with the virus globally, with approximately 1.5 million new infections recorded annually[44]. Strategies to combat HCV rely on improving early detection, expanding testing, and providing treatment to all groups, especially high-risk groups. The World Health Organization has set an ambitious goal of eliminating hepatitis C as a public health threat by 2030, by reducing new infections by 90% and deaths by 65% [45].

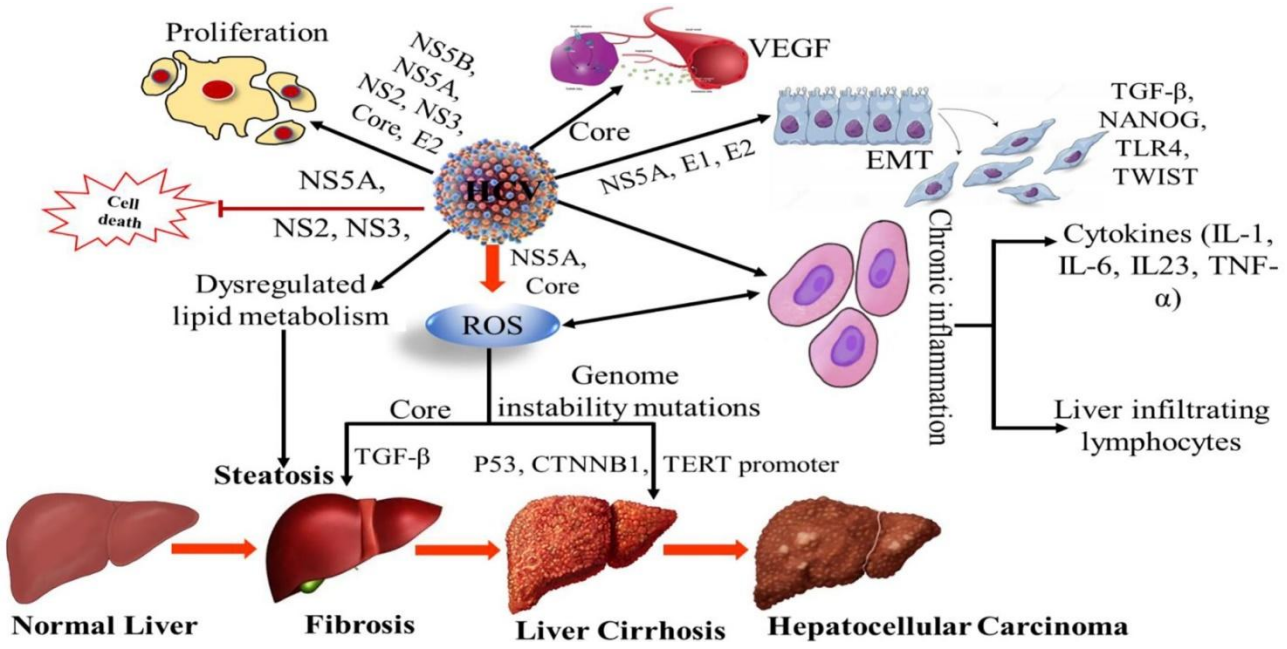

Figure 2. Mechanism of HCV induced hepatocellular carcinoma[46].

3. DNA TUMOR VIRUSES

DNA tumor viruses are a group of viruses that contain deoxyribonucleic acid (DNA) as their genetic material and are characterized by their ability to transform host cells into tumors[47]. These viruses introduce their genetic material into the cell and interfere with the molecular mechanisms the regulate the cell cycle, leading to loss of control of cell division and the development of cancerous features[47]. These infections can lead to the formation of benign or malignant tumors, and some types are considered established carcinogens in humansAlthough some DNA carcinoma viruses, like human papillomavirus (HPV), epidermidis gonorrhea (EBV), hepatocellular carcinoma (KSHV), and cytomegalovirus (CMV), can only induce cancers in specific animal models, others, like human adenovirus (HAV), can change cultured cells and create tumors in various hosts[48]. Genome tumor viruse oncogenes a considered essential for viral replication and are viral in origin, as opposed to cellular, as are oncogenes expressed by animal

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retroviruses[49]. The oncogenic process initiated by DNA tumor viruses involves the expression of viral oncogenes, which are crucial for viral replication and cellular transformation. Unlike retroviral oncogenes, which are derived from host cellular proto-oncogenes, the oncogenes of DNA tumor viruses are of purely viral origin [50].

Research on small DNA carcinoma viruses, such as adenoviruses, polyomaviruses, and papillomaviruses, has significantly enhanced the comprehension of the fundamental molecular mechanisms underlying virally-induced cellular transformation[51]. It was pRB, a 105 kDa protein linked to Ad E1A in adenovirus-transforming cells, initially identified as a target for tiny DNA tumor viruses oncoprotein. Subsequently, SV40 Tag and HPV16 E7 were also found to target pRB. Ad E1A, SV40 TAg, and HPV E7 can inactivate growth-regulating activities by interacting with pRB and destroying or altering cellular complexes that typically contain pRB [52]. When cells expressing E1A, SV40 TAg, or HPV E7 display elevated levels of free E2F, the cell cycle-dependent regulation for E2F-responsive genes becomes impaired[53]. Due to the key role of E2F-responsive genes in cell cycle progression, the interaction between pRB and smaller DNA tumor viral oncoproteins begins S-phase, a crucial stage of these viruses' life cycles. The primary pRB binding sites of SV40 TAg, HPV E7, and Ad E1A are comparable, the functional results of interactions amongst viral oncoproteins are distinct. Although both the hypo- and hyper-phosphorylated forms of pRB can be bound to by the Ad E1A protein, which in turn deactivates the protein [54], the SV40 TAg protein interacts with the G1-specific, growth-suppressing hypophosphorylated form, and the HPV16 E7 protein binds preferentially to the hypophosphorylated form, leading to its proteasome-mediated degradation[55]. It was previously discovered that p53 linked to SV40 TAg in cells transformed by SV40, but recent studies have demonstrated that it also links to high-risk HPV E6 and Ad E1B, making it the second main cellular tumor suppressor targeted with oncoproteins from short DNA tumor viruses. After being classified as an oncogene because of a point mutation and cloning from a cancer cell line, the wild type variation of the p53 protein was found to have tumor suppressor capabilities [56]. As a result of their interactions, SV40 TAg, HPV E6, and Ad E1B inhibit p53's tumor suppressor activities . p53 activates transcription by binding to particular sequences in DNA. It combines signal transduction pathways that detect cellular stress [57], however it is not needed for regular cell proliferation, despite its "guardian of the genome" title. The structural dissimilarity amongst SV40 TAg, HPV E6, and Ad E1B and SV40 TAg, HPV E7, and Ad E1A is highlighted by their distinct interactions with p53. P53 levels are higher in cells expressing SV40 TAg or Ad E1B relative to normal cells because its metabolic half-life is increased. However, when compared to normal cells, those harboring high-risk HPV E6 have lower p53 levels[58] .

3.1 Human papillomavirus (HPV)

Human papillomavirus (HPV) is among the most prevalent viruses that infect the skin and mucous membranes of humans. This virus belongs to the Papillomaviridae family and is characterized by a circular, double-stranded, non-enveloped DNA structure, which gives it high durability in environmental conditions[59]. There are more than 200 known subtypes of this virus,

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some of which are classified as low-risk (such as types 6 and 11) and others as high-risk (such as 5 and 8) due to their association with serious cancers, such as cervical cancer[60]. Although infection of HPV is common, progression to cancer is relatively rare and often requires additional factors such as a weakened immune system, smoking, or long-term use of birth control pills. Certain genetic conditions, such as Fanconi anemia, also increase the risk of HPV-related cancer[61]. Figure 3**,** illustrates a set of key molecular markers that contribute to the development of cervical cancer, all associated with human papillomavirus (HPV) infection. The process begins with the virus invading target tissues (cellular tropism), where the virus has a selective ability to infect cervical epithelial cells, particularly in the transition zone[62]. This infection interacts with a genetic predisposition to cancer, where genetic mutations or other genetic factors play a role in enhancing the negative response to infection. Cancer development is also influenced by the amount of virus in the body (viral load), with a high viral load being associated with an increased risk of cancerous transformation [63]. The physical state of the virus whether it is in a circular state or integrated into the host cell's genome also influences the mechanism by which regulatory genes in the cell are inactivated[64]. One of the most notable changes is the loss of function of the E2 protein, which regulates the expression of the oncogenes E6 and E7, leading to uncontrolled elevation of these genes' expression. Viral proteins E6 and E7 inhibit important cell cycle-regulating proteins such as p53 and Rb, disrupting cell division control and pushing the cell toward malignant transformation. Furthermore, abnormal regulation of host cell protein functions occurs, contributing to cellular homeostasis. Epigenetic mechanisms play an additional role, contributing to the inhibition or activation of genes associated with survival or cell death without altering the DNA sequence. The figure also shows the cell's transition to the mesenchymal phenotype, an important step in the ability of cancer cells to invade and spread[65].

Also HPV linked to a rare genetic skin disorder known as epidermodysplasia verrucosum (EV). This lesions may progress to skin cancer, especially in regions of the body exposed to sunlight. Skin cancer in epidermodysplasia verruciformis (EV) patients was the inaugural type of HPV-associated malignancy linked to HPV infection[66] ; subsequent research has indicated that HPVs 5 and 8, along with analogous cutaneous HPVs, may contribute to the pathogenesis of non-melanoma skin cancers (NMSC), particularly in immunocompromised individuals [67]. Cutaneous HPV infections are prevalent in people of all ages, and several viruses, including the ostensibly high-risk HPV5, may be a component of the normal skin flora and can be detected in extracted hair follicles[68].

HPV is predominantly spread via direct sexual contact, encompassing skin-to-skin interaction. Transmission might happen via contaminated surfaces or from a pregnant woman with an infection to her offspring during parturition. Not all carriers of the virus cause symptoms, which contributes to its silent spread. It is estimated that most sexually active adults will be infected with at least one type of HPV during their lifetime, without knowing it[69].

Researchers are still trying to pin down the exact molecular functions of cutaneous HPVs and how they interact with other potential NMSC-causing co-factors [70]. Studies have shown that cutaneous HPV E6 [71]. proteins can cause Bak, a pro-apoptotic Bcl-2 family member, to degrade.

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It is believed that cells carrying cutaneous HPV may exhibit a reduced propensity for apoptosis following UV-induced DNA damage, as Bak is crucial in regulating this response to UV exposure [72]. This might allow HPV-infected cells with major genetic abnormalities to survive and multiply, paving the way for transformation. In vitro cell transformation has been shown to occur in some cutaneous HPVs [73]. In addition, transgenic mice that have the cutaneous human papillomavirus early region genes develop skin hyperplasia and tumors. It has been suggested that cutaneous HPVs may help initiate carcinogenesis but aren't necessary for upholding the altered appearance, or that they promote alteration by means other than cell autonomously mechanisms, given that HPV genomes are typically detected in a small percentage of malignant cells [74]. A crucial element in tumorigenesis is the incorporation of the viral genome into the host cell's genome, leading to the persistent and irregular expression of the viral proteins E6 and E7, which inhibit tumor suppressor genes such as p53 and Rb. This persistent expression keeps cells in an abnormal state and may later lead to malignant transformation, especially with additional mutations in the cellular DNA[75].

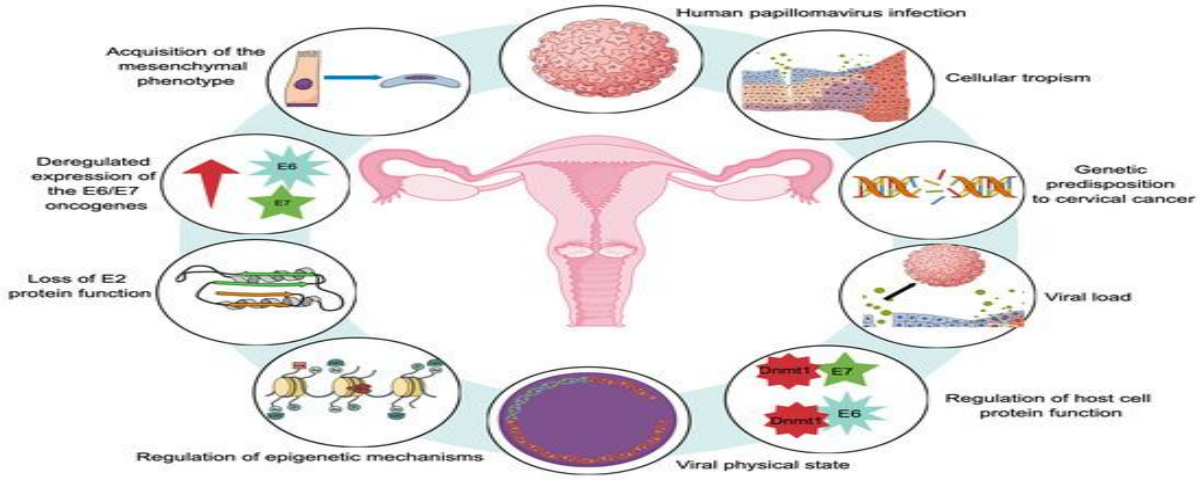

Figure 3. Proposed molecular hallmarks for cervical carcinogenesis caused by HPV[76]

3.2 Hepatitis B virus (HBV)

Hepatitis B virus (HBV) is an enveloped, double-stranded DNA virus belonging to the Hepadnaviridae family[77]. It is one of the most important hepatitis viruses affecting humans and can cause acute or chronic hepatitis, and in some cases, may lead to cirrhosis or hepatocellular carcinoma (HCC). HBV is transmitted through blood or body fluids, including sexual contact, blood transfusions, contaminated needles, or from mother to fetus[78].

A considerable number of cases of HCC can be traced back to HBV. Between (16 and 45) % A subset of infected persons develops chronic active hepatitis (CAH), potentially resulting in failure of the liver, cirrhosis, or HCC [79]. Tobacco smoke, aflatoxin B, and ethanol are environmental

carcinogens that accelerate this process. Cirrhosis can lead to HCC in cases of chronic alcoholic hepatitis (CAH), which is characterized by fibrosis, inflammation, and hepatic necrosis. This is because hepatocytes regenerate faster after chronic necrosis, which speeds up the process of mutation accumulation and, ultimately [80]. Cirrhotic patients have a higher risk of hepatocellular carcinoma (HCC) than non-cirrhotic patients [81].

Both CAH and cirrhosis seem to have a role in liver carcinogenesis; however, evidence exists, including the link between blood HBV DNA levels and HCC risk, suggesting that HBV has an active role in carcinogenesis. Consequently, the etiology of HBV-associated HCC seems to stem from a confluence of chronic inflammation and carcinogenic mechanisms encoded by HBV [82]. Reverse transcription is the key to HBV replication, just like it is for retroviruses. However, unlike retroviruses, HBV replication does not depend on viral genome integration into the host chromosome, although this does help the viral genome endure [83]. Since the HBV genome is commonly integrated into the host chromosome of patients with both chronic active hepatitis and hepatocellular carcinoma, the integration event happens before tumor growth. Changes in the viral and surrounding cellular sequences can occur throughout the ever-changing process of HBV integration in response to chronic inflammation and increased hepatocyte proliferation [84]. Genomic instability and proto-oncogene activation may result from HBV integration The integration event might result in chromosomal deletions and transpositions of virus genomic sequencing across chromosomes. Unfortunately, not all cases of HBV-associated HCC involve integration of the HBV genome; in fact, about 20% of patients do not show any indications of integration at all [85].

Genomic alterations, along with the marked transcriptional dysregulation seen in hepatocellular carcinoma (HCC), are closely associated with risk factors. Hepatitis B virus (HBV)-associated HCCs are clearly defined by proliferative subclasses (G1-G3 transcriptional subgroups), particularly in HCCs that exhibit stem cell features (G1 transcriptional subgroup)[86]. HBV-associated tumors occur through two main mechanisms: direct and indirect. In the indirect effect, which accounts for approximately 60–90% of cases, chronic HBV infection induces persistent inflammation in the liver tissue, which over time leads to cirrhosis[87]. Fibrosis is a precancerous state that creates a favorable cellular environment for cancer development as a result of chronic histological and structural changes[88]. In the direct effect, which accounts for 10–40% of cases, the virus integrates its genetic material (HBV DNA) into the genome of hepatocytes or retains it as closed circular DNA (cccDNA) within the nucleus, leading to chromosomal instability. This is likely due to the ability of HBV to induce genomic instability through viral integrases and HBx protein activity, along with the observed frequency of mutations in TP53 and IRF2, two genes essential for chromosome stability (**Figure 4**)[89].

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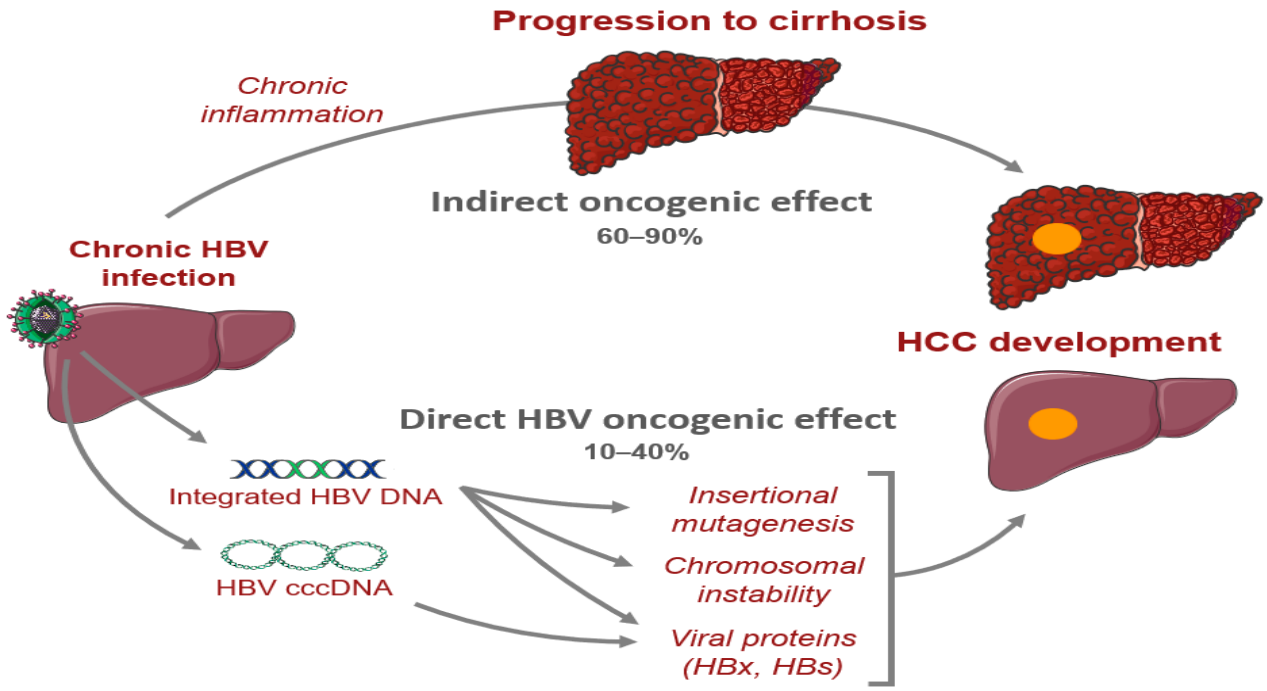

Figure 4. Oncogenic mechanisms related to hepatitis B virus[89].

3.3 Epstein–Barr virus (EBV)

Epstein–Barr virus (EBV) is one of the most well-known viruses in the Herpesviridae family, first discovered in 1964. EBV is classified as a double-stranded DNA virus and was among the first viruses associated with stimulating tumor growth in humans, earning it a classification as a carcinogen by the International Agency for Research on Cancer (IARC)[90]. EBV is commonly spread through saliva, initially infecting epithelial cells of the mouth and pharynx, and then moving on to infect B cells of the immune system. In most cases, the infection remains latent for life without significant clinical symptoms. However, under certain conditions, such as immunocompromised individuals or genetic factors, the virus can contribute to the malignant transformation of infected cells[91]. EBV is associated with a number of malignancies in humans, most notably nasopharyngeal carcinoma, which is prevalent in certain regions such as Southeast Asia and North Africa[92]. The virus is also responsible for the development of African Burkitt lymphoma, particularly when infection is accompanied by mutations in the MYC gene, and contributes to the development of B-cell lymphomas associated with immunodeficiency conditions such as those that occur after organ transplantation or HIV infection. Furthermore, the virus is associated with EBV-associated gastric cancer and some other cancers such as salivary gland cancers and breast cancers, as suggested in some studies[93]. At the molecular level, EBV plays a key role in carcinogenesis through the production of several proteins during the latent phase, such as EBNA1, EBNA2, LMP1, and LMP2[94]. These proteins contribute to stimulating vital cellular pathways responsible for cell survival and cell division, such as activating the NF-κB signaling

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pathway and the PI3K/AKT pathway, disrupting normal cell cycle control and contributing to malignant transformation. For example, the LMP1 protein is one of the most important viral factors associated with carcinogenesis, as it mimics cell growth signals and increases cell resistance to apoptosis[94, 95].

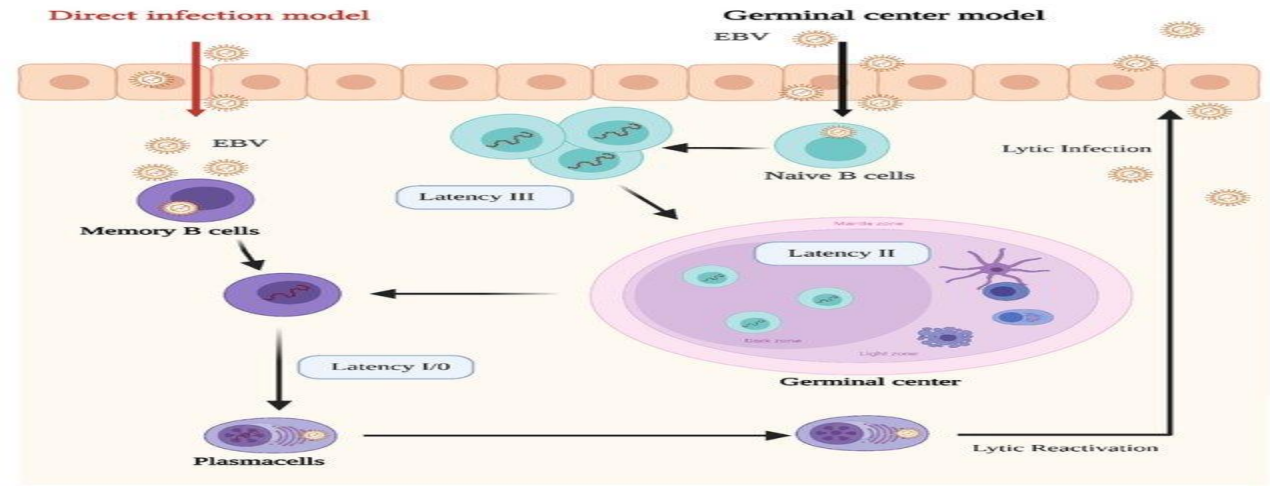

Figure 5. Schematic depiction of the life cycle and latency phases of the Epstein–Barr Virus (EBV). In the germinal center model, Epstein-Barr virus infects submucosal naive B cells and initiates the latency III program. At this stage, the proliferation and growth of the infected B cell population is facilitated by the expression of all latent genes of EBV. The infected cells subsequently enter the germinal center, where they proliferate and undergo maturation. The Latency II program is now established. Certain infected B cells exit the germinal center as memory B cells. At this stage, EBV infection remains latent (latency I/0); but, if infected memory cells develop into plasma cells, lytic reactivation may be initiated. In the direct infection model, memory B cells are infected directly[96]

Diagnosis of EBV infection often relies on measuring virus-specific antibody levels or using modern techniques such as polymerase chain reaction (PCR) to detect the presence of viral DNA in clinical samples. These tests have become key tools for determining the relationship between EBV and various types of cancer. Furthermore, New research is improving our knowledge of how the virus interacts with the immune system. This could lead to future vaccines or immunotherapies that target specific components of EBV, representing a promising advance in the field of prevention and targeted treatment of EBV-associated tumors[97].

3.4 Kaposi's sarcoma-associated herpesvirus (KSHV)

The γ-herpesvirus subfamily includes the double-stranded human rhadinovirus known as HHV8 (or KSHV). It has been recently discovered and, similar to other γ-herpesviruses, it maintains a state of lifelong latency in B cells[98]. There is a correlation between KSHV and all types of KS, primary effusion lymphomas (PELs), and multicentric Castleman's disease (MCD).

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It has been demonstrated beyond a reasonable doubt that KSHV has the potential to cause cancer, particularly in immunocompromised individuals: The KSHV virus is responsible for the transformation of endothelial cells , and epidemiological studies have linked it to human cancers [99]. KSHV is primarily transmitted through bodily fluids such as saliva and blood. It enters cells and undergoes two main phases: latent and productive. In the latent phase, the virus expresses a limited set of genes that enable it to remain in the cell for long periods without being easily detected by the immune system. In the productive phase, the virus expresses a broader set of genes that promote the spread of infection to new cells[100].

KSHV is closely associated with the development of three major malignant diseases: Kaposi's sarcoma, primary effusion lymphoma (PEL), and virus-associated multiple Castleman disease. Kaposi's sarcoma is the most common form, often presenting as multiple skin lesions characterized by purple or dark brown coloration[101] . In advanced cases, it can spread to involve internal organs. Primary effusion lymphoma (PEL) is a rare and highly aggressive type that typically affects serous cavities such as the pleura or peritoneum, while multiple Castleman disease is characterized by an increase in the size and number of lymph nodes, leading to severe systemic symptoms[102].

4. OTHER TYPES OF VIRUS

4.1 Polyomaviruses

Polyomaviruses are small, non-enveloped viruses with circular double-stranded DNA genomes, typically around 5,000 base pairs in size[103]. These viruses belong to the family polyomaviridae family of viruses has been found in many different kinds of hosts, including both animals and humans. Human polyomaviruses, such as JC virus (JCV), BK virus (BKV), Merkel cell polyomavirus (MCPyV), and others, have been extensively studied due to their association with opportunistic infections and cancer development under immunosuppressive conditions[104]. The biological behavior of polyomaviruses is characterized by a latent infection in their host tissues, with reactivation occurring primarily when the immune system is compromised. In healthy individuals, infections are usually asymptomatic. However, in immunocompromised patients such as transplant recipients or those with HIV/AIDS polyomaviruses can cause serious diseases, including progressive multifocal leukoencephalopathy (PML) caused by JCV and nephropathy linked to BKV[105]. Similar to SV40, it has been hypothesized that BKV and JCV is implicated among humans malignancies; nevertheless, there is no conclusive evidence that either virus directly induces human cancer or serves as a co-factor in its development. Given the prevalence of both BKV and JCV in the human population, the investigation into whether or not there is a connection between the two viruses and the development of cancer in humans is a challenging endeavor. Reports reveal alterations in JCV within several humans brain cancers; however, these investigations have not established any causal link [106]. This is particularly concerning with regard to JCV. In addition, there is evidence that suggests that BKV may play a component in the development of cancer in humans. BKV is able to infect epithelial cells in the urinary system for

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an extended period of time, and multiple investigations have established a connection between BK virus infections and prostate cancer [107].

Most recently, studies have demonstrated the carcinogenic potential of specific polyomaviruses, notably Merkel cell polyomavirus (MCPyV), that was associated with Merkel cell carcinoma (MCC), a rare yet aggressive kind for cancer of the skin. MCPyV integrates into the host genome and expresses viral oncoproteins (such as Large T antigen) that interfere with cell cycle regulation, thereby promoting tumorigenesis [108]. Molecularly, the oncogenic mechanisms involve the inactivation of tumor suppressor pathways, notably the p53 and retinoblastoma (pRb**)** pathways, which are critical for maintaining normal cell proliferation and apoptosis. The persistent expression of viral T antigens is essential for both the initiation and maintenance of the malignant phenotype in infected cells[109].

Given the increasing recognition of the role of polyomaviruses in human cancers, there is a growing interest in developing targeted diagnostic tools and potential therapeutic strategies. For instance, serological assays and molecular detection of MCPyV DNA in tumors are becoming valuable diagnostic adjuncts in MCC management [110]. Despite these advances, many aspects of polyomavirus biology and their complete role in carcinogenesis remain incompletely understood, warranting further research in order to clarify how they engage with the host's immunological system and cellular apparatus[111].

4.2 Adenoviruses

Adenoviruses are medium-sized, non-enveloped viruses with a linear double-stranded DNA genome, belonging to the family Adenoviridae. They are known to infect a wide range of vertebrate hosts, including humans, and are capable of causing a variety of clinical syndromes such as respiratory tract infections, conjunctivitis, gastroenteritis, and, in rare cases, systemic infections[112]. In humans, more than 50 serotypes have been identified, which are classified into seven species (A to G) based on genetic, biochemical, and immunological properties.

While most adenovirus infections are self-limiting in immunocompetent individuals, they can cause life-threatening diseases in immunocompromised patients, particularly in bone marrow transplant recipients and individuals with primary immunodeficiencies[113] . Adenoviruses have a strong tropism for epithelial cells, and they can establish persistent or latent infections, particularly in lymphoid tissues such as the adenoids and tonsils. Preliminary studies indicated that certain Ad serotypes may not be associated with cancer cases in humans, even if these viruses display extremely high levels of transformation in cultures of cells and models of animals [114]. On the other hand, this matter has been revisited in recent times, and a study has reported the discovery of adenovirus DNA in juvenile brain tumors [115]. Nevertheless, there is still a lack of clarity regarding the putative causative relationship that exists between the infection of Ad and the formation of malignant brain tumors[116]. In the past, adenoviruses were thought to have the ability to convert and cause cancer due to E1A and E1B oncoproteins. Long periods of time have

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shown this to be true. However, recent studies have shown that E4 ORFs could potentially play a role in cellular transformation, perhaps through a "hit-and-run" mechanism [117]. This is the case with certain serotypes, at least. As a result, it is probable that the potential contribution of adenovirus infections to certain elements of the formation of human tumors needs to be given critical consideration once again.

4.3 Torque Teno Virus (TTV)

Originally identified in a patient with non-A-E hepatitis, TTV is a member of the Circoviridae family and a circular, single-stranded DNA virus [118]. Numerous TTV types and subtypes with substantial genetic variation have been discovered since its first identification. The effects of TTV, which begin in early childhood and continue into adulthood, are cumulative. The association among TT viruses and pathogenicity, namely liver issues, remains unproven, despite multiple attempts [119]. An elevated TTV load may have predictive value in HCV-related liver disease, according to one study; however, it is still unclear whether or not a high TTV load speeds up the progression of HCV-associated disease. Additional research pointed to an abnormally high frequency of TTV-related DNA sequences in other cancers of human, such as myeloma, intestinal cancer, lungs cancer, and breast cancer [120]. This study does not establish a causal connection between TTV infection and carcinogenesis due to the absence of normal control tissues.

4.4 Xenotropic murine leukemia virus-related virus (XMRV)

The hypothesis that genetic deficiencies in RNase L may facilitate infection by an oncogenic virus and potentially lead to the onset of prostate cancer (PC) has been prompted by the discovery that mutations in the structural gene for RNase L [121] are associated with the familial prostate cancer susceptibility locus Hpc1. RNase L is an effector in the interferon-induced innate viral response [122]. Codons from tumors in prostate cancer patients with a mutation in RNase L were used in the PCR cloning process that led to the discovery of Xenotropic murine leukemia virus-related virus (XMRV), a newly found gamma retrovirus. While the cancer cells in prostate cancer did not have the XMRV protein, it was found in the stroma and hematopoietic cells [123]. Consequently, the causal association between XMRV and prostate cancer remains ambiguous. Nonetheless, XMRV may potentially have a role in carcinogenesis via an indirect method.

5. MECHANISMS OF VIRAL CARCINOGENESIS

- **Integration into host genome**

A critical step in the process of viral carcinogenesis is the integration of the viral genome into the DNA of the host cell, particularly in viruses such as human papillomavirus (HPV) and hepatitis B virus (HBV)[124]. When this integration occurs, it has the potential to activate oncogenes or deactivate tumor suppressor genes, causing genomic instability and changes in gene expression. For example, in the case of HPV, integration inactivates the E2 gene, allowing for the persistent expression of the oncoproteins E6 and E7, which interfere with the functions of tumor suppressor proteins such as p53 and Rb. In the case of HBV, integration can activate oncogenes or inactivate tumor suppressor genes, contributing to the development of liver cancer[125] .

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- **Viral oncogenes (e.g. E6/E7 in HPV)**

Some viruses produce oncogenes that express proteins that interfere with cell cycle regulation. In HPV, the proteins E6 and E7 are considered key drivers of carcinogenesis. E6 promotes the breakdown of the p53 protein, whereas E7 disrupts the function of the Rb protein, leading to loss of cell cycle control and the accumulation of genetic mutations. These proteins are potential therapeutic targets, as inhibiting them can halt the growth of cancer cells[126].

- **Immune evasion mechanisms**

Viruses have developed many mechanisms to circumvent the immune system, allowing them to survive and replicate within the host. These mechanisms include reducing the presentation of antigens on the surface of infected cells, inhibiting interferon signaling, and modifying host gene expression[127]. For example, Epstein-Barr virus (EBV) produces proteins that suppress T cell responses, allowing it to remain latent within lymphocytes. This immune evasion contributes to the development of virus-associated tumors[128].

- **Chronic inflammation and indirect mechanisms**

Chronic inflammation is one of the primary indirect mechanisms contributing to the development of cancer resulting from viral infection. Although many oncogenic viruses do not contain oncogenes in the traditional sense, their chronic presence within cells and their persistent stimulation of the immune system can trigger a series of biological and environmental changes that create an environment conducive to cellular transformation[129].

During persistent viral infections, such as hepatitis B virus (HBV) or hepatitis C virus (HCV) infections, the chronic immune response triggers the sustained release of inflammatory mediators such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interferons. These mediators initiate cellular signaling pathways, including JAK/STAT and NF-κB, resulting in enhanced proliferation of cells, diminished apoptotic and elevated intracellular oxidative stress levels[130].

Chronic oxidative stress contributes to DNA damage and the disruption of DNA repair systems, increasing the likelihood of genetic mutations and chromosomal changes that predispose to cancer. In addition, prolonged immune responses can lead to structural changes in tissue, such as fibrosis and chronic scarring, which hinder normal tissue regeneration and stimulate the uncontrolled proliferation of damaged cells[131].

In the case of the liver, for example, chronic infection with HBV or HCV leads to chronic active hepatitis, which eventually progresses to cirrhosis and, eventually, hepatocellular carcinoma. Here, carcinogenesis is linked not only to the virus itself but also to environmental changes caused by recurrent inflammation and persistent tissue damage[132].

This indirect mechanism is an important focus in understanding viral carcinogenesis, especially in cases where direct viral integration or clear expression of viral oncogenes does not occur. Therefore, monitoring chronic inflammation and inhibiting its pathways is a new approach in preventing infection-related cancer, alongside antiviral therapy[7].

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Limitations and Future Research Suggestions.

Despite the exhaustive summary of major oncogenic viruses and their molecular and clinical relevance, his review is limited by reliance on previously published studies, and the significant heterogeneity between available experimental and clinical results. Future studies could be better designed large-scale clinical and molecular studies that can reveal more about virus-specific oncogenic pathways, host–virus interactions and population-based disparities. Second, with the development of high throughput technologies and precision medicine, it is imaginable that effective biomarkers and targeted antiviral or immunotherapeutic strategy for virus-related cancer would be facilitated.

CONCLUSION

Oncogenic viruses contribute to the initiation and progression of multiple types of human cancers. This review shows how oncolytic viruses actively participate in both direct and indirect ways, like chronic inflammation and evasion from immune response, to cell transformation and tumour progression. There is an overwhelming body of evidence that several viruses like HPV, EBV, HBV, HCV and KSHV play a significant role in the etiology of many cancers which highlights the intricate relationship between infection and oncogenesis. An increasing knowledge about these viruses, including their role inactivation of tumor-suppressive genes, viral integration into the host genome and interference in immune responses offers profound insights to cancer biology. In other words, the management of virus-induced cancers will require a holistic programme of molecular studies - clinical interventions - public health that address these cancers through optimal prevention, diagnosis and treatment.

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There are non-conflicts of interest.

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الخلاصة

المقدمة

تُعرف الفيروسات بأنها عوامل رئيسية تُسهم في تطور السرطان، حيث تُمثل ما يقارب 15–20% من الأورام الخبيثة البشرية حول العالم.

الهدف

تُقدم هذه المراجعة نظرة عامة شاملة على أبرز الفيروسات المُسرطنة، ، وتوضيح آلياتها المسببة للأورام وآثارها السريرية.

النقاط الرئيسية

تركز المراجعة على فيروس الورم الحليمي البشري (HPV)، وفيروسات التهاب الكبد B (HBV) وC (HCV)، وفيروس إبشتاين بار (EBV)، وفيروس ابيضاض الدم التائي البشري من النوع 1 (HTLV–1)، وفيروس الهربس المُرتبط بورم كابوزي (KSHV). يستخدم كل فيروس استراتيجيات جزيئية مُميزة للتلاعب بمسارات الخلايا المُضيفة، مُعززًا تكاثر الخلايا غير المُتحكم فيه، ومُتجنبًا موت الخلايا المُبرمج، وعدم الاستقرار الجيني – وهي السمات المُميزة للسرطان. تستكشف المراجعة التكامل الفيروسي، والتعبير عن الجينات المُسرطنة الفيروسية، ومُتجنبًا المناعة، والالتهاب المُزمن كآليات رئيسية في التسرطن المُحفز بالفيروس. بالإضافة إلى ذلك، تُناقش الآثار السريرية لهذه النتائج، بما في ذلك تطوير المؤشرات الحيوية التشخيصية، والعلاجات المُضادة للفيروسات المُستهدفة.

الاستنتاج

يُتيح فهم التفاعل بين العدوى الفيروسية وبيولوجيا الأورام رؤى قيّمة في مجال الوقاية من السرطان، والكشف المبكر عنه، والأساليب العلاجية الحديثة.

الكلمات المفتاحية: السرطان، الفيروسات، فيروس التهاب الكبد ب، فيروس التهاب الكبد ج، فيروس إبشتاين–بار، فيروس الهربس البسيط (KSHV)، فيروس سرطان الدم الليمفاوي الحاد (HTLV–1)