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# Genotoxic consequences of viral infections

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Viral diseases continually threaten human health as evolving pathogens introduce new risks. These infections can lead to complications across organ systems, with impacts varying by virus type, infection severity, and individual immune response. This review examines the genotoxic stress caused by viral infections and its pathological consequences in humans.

Many aspects of viral infections are not directly related to the action of the virus in question. Complications associated with infections can be permanent or curable. One critical aspect of post-viral complications is the genotoxic effects of certain viruses. Genotoxicity refers to the ability of certain agents, including viruses, to damage the genetic material within host cells, potentially leading to cell dysfunction and cancer development. Genotoxic viruses, including HIV<sup>1</sup>, human papillomavirus (HPV)<sup>2</sup>, hepatitis B and C<sup>3</sup>, Epstein-Barr virus (EBV)<sup>4</sup>, and Kaposi's sarcoma-associated herpesvirus (KSHV)<sup>5</sup>, can integrate their genetic material into the host genome, resulting in mutations. HPV plays a significant role in the development of cervical cancer<sup>6</sup>, while hepatitis B and C viruses are major causes of liver cancer<sup>7</sup>. Epstein-Barr virus (EBV) is associated with several types of lymphoma<sup>8</sup>. Kaposi's sarcoma-associated herpesvirus (KSHV) is linked to Kaposi's sarcoma, a type of cancer that forms in the lining of blood and lymph vessels<sup>9</sup>.

The mechanisms of genotoxicity encompass a range of processes that result in direct or indirect damage to the DNA molecule<sup>10</sup>. Direct DNA damage can be defined as the formation of adducts, or direct chemical bonds to DNA; DNA strand breaks, which disrupt the integrity of the genome; and DNA alkylation, where alkyl groups added to DNA lead to abnormal replication. Indirect DNA damage may result from the production of reactive oxygen species (ROS), which oxidize the nitrogenous bases of DNA, or from the inhibition of DNA repair mechanisms, thereby increasing the likelihood of mutation. Errors in the replication of DNA may be caused by intercalation, whereby chemical compounds occupy the space between DNA base pairs, resulting in replication errors. Furthermore, chromosomal abnormalities, including chromosomal aberrations (deletions, duplications, translocations, inversions) and aneuploidy (alterations in chromosome number), are also induced by genotoxic agents<sup>11</sup>.

The role of DNA damage in host cells in the life cycle of viruses is complex, with effects varying depending on the virus type, replication mechanism and host response. A multitude of viruses have evolved intricate mechanisms to exploit or manipulate host DNA damage response (DDR),

frequently utilizing these pathways to enhance replication, evade immune defenses, or establish persistent infections<sup>12</sup>. While DNA damage can impede viral replication by disrupting essential cellular processes, it can also facilitate viral propagation by activating pathways that viruses exploit for their own replication<sup>13</sup>.

In the event of DNA damage, cells initiate a series of repair mechanisms collectively known as DDR (Fig. 1). However, this activation presents a potential challenge for DNA viruses, as it can result in cell cycle arrest or apoptosis, thereby limiting viral replication<sup>14</sup>. To counteract this, several DNA viruses have developed strategies to interfere with key DDR signaling proteins, such as ATM and ATR kinases, which are crucial for the detection and repair of DNA breaks<sup>15,16</sup>.

Retroviruses such as HIV have been observed to employ specific strategies for interacting with the DDR, which significantly impacts their replication processes<sup>17</sup>. During the course of an HIV infection, the integration of viral DNA into the host genome involves the participation of DNA repair enzymes, which can result in the triggering of a DNA damage response in the host cell. HIV effectively exploits DDR components, such as the non-homologous end-joining (NHEJ) pathway, to enhance the efficiency of its integration and establish a long-term presence within the host genome, creating latent reservoirs that contribute to persistent infections<sup>18</sup>.

Furthermore, DNA damage resulting from viral infections can prompt host cells to adopt a state that is conducive to viral replication or persistence. Persistent DNA damage may result in the induction of cellular senescence or the activation of chronic inflammation, thereby creating an environment that is conducive to the replication of certain viruses. For example, cytomegalovirus (CMV) exploits cellular senescence to establish latency, exploiting this altered cellular state to evade immune detection and enabling reactivation under conditions of immune suppression<sup>19,20</sup>.

In the present review, we evaluate how different viruses induce genotoxic stress, their interaction with the DNA repair machinery, and finally, how this may lead to potentially oncogenic consequences, e.g. mutations and/or chromosomal aberrations. We excluded directly oncogenic viruses

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# Viral Consequences

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