



Viral Infections and Cancer: Analyzing the Link between Oncoviruses and Tumor Development

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DESCRIPTION

Cancer is a complex disease caused by a variety of factors, including genetic mutations, environmental influences and lifestyle choices. In recent decades, however it has become clear that viruses can also play a significant role in the development of certain types of cancer. These viruses, known as oncoviruses, are capable of directly or indirectly causing cancer by altering normal cellular processes. Research into the relationship between viral infections and cancer has uncovered important insights into how these viruses contribute to tumor development, offering new avenues for prevention, diagnosis and treatment. Oncoviruses are viruses that can cause cancer by infecting normal cells and disrupting their regulatory mechanisms. This disruption can lead to uncontrolled cell division, ultimately resulting in tumor formation. Oncoviruses can integrate their genetic material into the host's DNA or controls the cell's machinery to promote the production of viral proteins that interfere with normal cell functions. While not all viral infections lead to cancer, specific viruses have been identified as major contributors to certain cancer types.

Human Papillomavirus (HPV), Hepatitis B Virus (HBV), Hepatitis C Virus (HCV), Epstein-Barr Virus (EBV), Human T-Lymphotropic Virus Type 1 (HTLV-1), Kaposi's Sarcoma-Associated Herpesvirus (KSHV), each of these viruses has distinct mechanisms through which they promote carcinogenesis (the development of cancer), yet they share common pathways that lead to uncontrolled cell growth. Oncoviruses promote cancer through several mechanisms, including the integration of viral Deoxyribose Nucleic acid (DNA) into the host genome, the production of viral proteins that alter cellular regulation, and the induction of chronic inflammation. These processes can disrupt the normal balance between cell growth and cell death, leading to the accumulation of mutations and the development of cancerous cells. Some oncoviruses like HPV and HBV integrate their DNA into the host's genome. Once integrated, viral genes

can interfere with the normal regulation of cell division and apoptosis (programmed cell death).

Even without integrating their DNA into the host genome, some viruses promote cancer through the production of viral proteins that stimulate chronic inflammation. Chronic infection and inflammation create a microenvironment conducive to cancer development by generating Reactive Oxygen Species (ROS) that damage DNA and promoting the recruitment of immune cells that can release growth factors, promoting cellular proliferation. HCV, for instance, is associated with liver cancer due to the chronic inflammation it induces in the liver, leading to cirrhosis and eventual Hepatocellular Carcinoma (HCC). Oncoviruses enter the immune system by downregulating the expression of molecules that would alert the immune system to the presence of infected or abnormal cells. By suppressing immune responses, these viruses allow infected cells to survive and proliferate, increasing the likelihood of cancer development.

HPV is responsible for nearly all cases of cervical cancer, making it one of the most prominent examples of an oncovirus. High-risk strains of HPV, particularly HPV-16 and HPV-18, are responsible for the majority of HPV-related cancers, including not only cervical cancer but also anal, penile and oropharyngeal cancers. Chronic infection with HBV or HCV is a major cause of liver cancer (hepatocellular carcinoma). These viruses cause persistent liver inflammation, which over time leads to cirrhosis and increases the risk of cancerous cell growth in the liver. EBV is linked to several types of cancer, including nasopharyngeal carcinoma and certain lymphomas, such as Burkitt's lymphoma. EBV infection in epithelial cells of the nasopharynx can lead to tumor formation, particularly in individuals with specific genetic backgrounds and environmental factors. Ongoing research into viral carcinogenesis continues to offer new insights into cancer prevention, early detection and targeted treatments, providing hope for reducing the global burden of cancer caused by viral infections.

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