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A Report on Oncovirus

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Description

Infection caused 17.8% of human malignancies in 2002, according to the World Health Organization's International Agency for Research on Cancer, with one of seven viruses accounting for 11.9 percent. A study in 2020 discovered that 16 percent of 2,658 samples from 38 different forms of cancer were linked to a virus. These tumours could be readily prevented (for example, HPV vaccines), identified with simple blood tests, and treated with less-toxic antiviral drugs.

Tumor viruses infect their hosts and produce non-neoplastic diseases such as acute hepatitis or mononucleosis in the case of the hepatitis B virus and mononucleosis in the case of the Epstein–Barr virus. After infection, a small percentage of people (or animals) will acquire cancer. This has made determining whether or not a virus causes cancer more difficult. The well-known Koch's postulates, which were devised by Robert Koch in the 19th century to determine the likelihood of Bacillus anthracis causing anthrax disease, do not apply to viral diseases.

For starters, viruses can't be properly isolated in pure culture—even the most strict isolation procedures can't completely eliminate undetectable contaminating viruses with identical density characteristics—and they have to be grown on cells. Second, for most tumour viruses, asymptomatic virus infection and carriage is the norm, which contradicts Koch's third principle. The challenges in applying Koch's postulates to virus-induced malignancies have been described by Relman and Fredericks. Finally, because human viruses have a host constraint, experimentally transmitting a suspected cancer virus is unethical. Other criteria, such as A. B. Hill's, are more relevant to cancer virology but have limits in determining causality.

Tumor viruses come in a range of shapes and sizes, including: Cancer can be caused by viruses with a DNA genome, such as adenovirus, and viruses with an RNA genome, such as the Hepatitis C Virus (HCV). Retroviruses with both DNA and RNA genomes can also cause cancer (Human T-lymphotropic virus and hepatitis B virus, which normally replicates as a mixed double and single-stranded DNA virus but also has a retroviral replication component). Tumor viruses frequently do not cause cancer in their natural hosts, preferring instead to infect dead-end species. Adenoviruses, for example, do not cause cancer in humans, but instead cause colds, conjunctivitis, and other acute infections. When inoculated into specific rodent species, such as Syrian hamsters, they become tumorigenic. Some viruses, such as Epstein–Barr virus and Kaposi's sarcoma-associated herpesvirus, are tumorigenic when they enter a cell and remain as circular episomes or plasmids, replicating independently from the host cell DNA. Other viruses, such as polyomaviruses and papillomaviruses, are only carcinogenic when they integrate into the host cell genome as a result of a biological accident [1-5].

The human papillomavirus, hepatitis B and C viruses, the Epstein–Barr virus, the human T-lymphotropic virus, the Kaposi's Sarcoma-associated Herpesvirus (KSHV), and the Merkel cell polyomavirus are the primary viruses linked to human cancers. Viruses appear to be the second most important risk factor for cancer formation in humans, second only to tobacco use, according to experimental and epidemiological studies. Acutely transforming and slowly transforming tumours are the two types of virally generated cancers. A gene encoded for an overactive oncogene called viral-oncogene (v-onc) is carried by viral particles in acutely transforming viruses, and the infected cell is transformed as soon as v-onc is produced. In contrast, the virus genome is inserted near a proto-oncogene in the host genome in slowly changing viruses, especially because viral genome insertion is an essential feature of retroviruses. Overexpression of that proto-oncogene is caused by the viral promoter or other transcription regulatory elements, which leads to uncontrolled cellular growth. Slowly transforming viruses have a far longer tumour latency than rapidly transforming viruses, which already carry the viral oncogene, because viral genome insertion is not specific to proto-oncogenes and the chance of insertion near that proto-oncogene is minimal.

Conclusion

Vaccines to prevent cancer have been developed thanks to advancements in cancer research. The hepatitis B vaccination is the first vaccine to be approved for the prevention of cancer (hepatocellular carcinoma) caused by infection with the causative virus. Gardasil, a human papillomavirus vaccination, was licenced by the US Food and Drug Administration in 2006. The vaccine protects against four HPV varieties, which cause 70% of cervical malignancies and 90% of genital warts when combined. The Advisory Committee on Immunization Practices (ACIP) of the US Centers for Disease Control and Prevention (CDC) advised that females aged 11–12 receive the vaccine in March 2007, and stated that females as young as 9 and as old as 26 are also candidates for immunisation.

Conflict Of interest

None

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