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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 HIV¹, human papillomavirus (HPV)², hepatitis B and C³, Epstein-Barr virus (EBV)⁴, and Kaposi's sarcoma-associated herpesvirus (KSHV)⁵, can integrate their genetic material into the host genome, resulting in mutations. HPV plays a significant role in the development of cervical cancer⁶, while hepatitis B and C viruses are major causes of liver cancer⁷. Epstein-Barr virus (EBV) is associated with several types of lymphoma⁸. 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 vessels⁹.

The mechanisms of genotoxicity encompass a range of processes that result in direct or indirect damage to the DNA molecule¹⁰. 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 agents¹¹.

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 infections¹². 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 replication¹³.

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 replication¹⁴. 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^{15,16}.

Retroviruses such as HIV have been observed to employ specific strategies for interacting with the DDR, which significantly impacts their replication processes¹⁷. 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 infections¹⁸.

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^{19,20}.

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

¹Department of Synthesis and Technology of Drugs, Medical University of Białystok, Białystok, Poland. ²Department of Biotechnology, Medical University of Białystok, Białystok, Poland. ³Chromatin Dynamics and Metabolism in Cancer, CNRS UMR9018 Institut Gustave Roussy, Université Paris Saclay, 39, rue Camille-Desmoulins, 94805 Villejuif, France. ⁴Department of Pharmacy, Faculty of Medicine and Life Sciences, University of Latvia, Jēgavas Street 1, LV1004 Riga, Latvia. e-mail: yegor.vassetzky@cnrs.fr; nikolajs.sjakste@lu.lv

Viral Consequences

Daniel V. Lim



Viral Consequences:

Viral Consequences John N. Fujii,2000-07-04 Jason Majors a freelance writer is caught up in a viral pandemic caused by Ivan Wiesnovsky a biotechnician who leaves vials of viruses in cities as he takes a round the world trip Once involved Jason finds himself searching for the origins causes and consequences of the resulting viral pandemic **Neoplastic Transformation, Mechanisms and Consequences** Hilary Koprowski,1977 **Anthropogenic Impacts on the Microbial Ecology and Function of Aquatic Environments** Maurizio Labbate,Justin R. Seymour,Federico Lauro,Mark V. Brown,2016-09-06 Aquatic ecosystems are currently experiencing unprecedented levels of impact from human activities including over exploitation of resources habitat destruction pollution and the influence of climate change The impacts of these activities on the microbial ecology of aquatic environments are only now beginning to be defined One of the many implications of environmental degradation and climate change is the geographical expansion of disease causing microbes such as those from the Vibrio genus Elevating sea surface temperatures correlate with increasing Vibrio numbers and disease in marine animals e g corals and humans Contamination of aquatic environments with heavy metals and other pollutants affects microbial ecology with downstream effects on biogeochemical cycles and nutrient turnover Also of importance is the pollution of aquatic environments with antibiotics resistance genes and the mobile genetic elements that house resistance genes from human and animal waste Such contaminated environments act as a source of resistance genes long after an antibiotic has ceased being used in the community Environments contaminated with mobile genetic elements that are adapted to human commensals and pathogens function to capture new resistance genes for potential reintroduction back into clinical environments This research topic encompasses these diverse topics and describes the affect s of human activity on the microbial ecology and function in aquatic environments and describes methods of restoration and for modelling disturbances *Antiviral Agents and Human Viral Diseases* George J. Galasso,Richard J. Whitley,Thomas C. Merigan,1997 *The Effects of Radiation and Radioisotopes on the Life Processes* Charles M. Pierce,U.S. Atomic Energy Commission,1963 *Diagnosis, Characterization, and Impact of Bovine Viral Diarrhea Virus (BVDV) Infection in Dairy Cattle Under Field Management Conditions* Claudia A. Muñoz-Zanai,2002 *The Effects of Radiation and Radioisotopes on the Life Processes: Index* ,1963 *Impact of Co-receptor Tropism on Human Immunodeficiency Virus Type 1 (HIV-1) Viral Output of Primary Cells* Ann-Marie Michelle Cruz,2006 HIV 1 most often utilizes CD4 and one of two co receptors CCR5 and CXCR4 in order to enter host cells Interestingly many patients in early and in late disease maintain high circulating levels of CCR5 tropic R5 viruses even though the target population of CCR5 cells appears to be small Such viral loads may be sustained because relative to CXCR4 tropic X4 infection RS infection of permissive CID4 T cells results in the production of significantly more infectious viral particles per cell To investigate this possibility the average amount of virus produced per infected cell by R5 and X4 HIV 1 in isolated CID4 T cell populations PBMC was determined as well as the average amount of

virus released per infected target CD4 T cell following isogenic R5 and X4 HIV 1 infections of dispersed human tonsil tissue Prior stimulation using CD3 and CD28 antibodies was performed in the case of PBMC Virus production was measured by flow cytometry and by p24 ELISA to quantitate both cell associated and cell released viral capsid protein and infectious virus was quantitated via TCID50 assay We provide evidence that R5 HIV 1 compensates for a small target cell population by producing to 100W fold more infectious virus per CCR5 target cell than X4 HIV 1 This phenomenon was observed in both of the infection models that were used evidence that this mechanism of R5 HIV 1 predominance is not restricted to lymphoid tissue but relevant to blood lymphocyte populations as well This phenomenon may contribute to the predominance of R5 HIV 1 in early in vivo infection

Ecological Impact of GMO Dissemination in Agro-ecosystems Tamás Lelley, Ervin Balázs, Mark Tepfer, 2003 Viral Safety and Evaluation of Viral Clearance from Biopharmaceutical Products Fred Brown, Anthony S. Lubiniecki, 1996 Assurance that biopharmaceuticals do not contain unwanted infectious agents is a vital safety consideration in the use of these products This can only be ensured by careful screening of the cells and other materials used in their production and subsequent testing of the final product This publication reports the proceedings of an international conference which focused on the methods of how to avoid contamination of cell cultures with virus during the production process of biologicals with the aim of providing harmonized guidelines to ensure product safety Scientists from industry and regulatory agencies present updates on the current state of knowledge about assays for viral contamination and procedures for inactivation and removal Risks associated with the processing of biopharmaceuticals are identified and data on the actual experiences of testing and industrial laboratories are presented and discussed in the context of the clinical setting Drawing together a wealth of new data this book will be essential reading for all scientists concerned with the development production and quality control of biopharmaceutical products

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