Editorial

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Journal of Clinical and Molecular Pathology

2021
Vol.5 No.4:811

Viral Oncogenesis - Pathology

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Received date: December 03, 2021; **Accepted date:** December 17, 2021**; Published date:** December 24, 2021

Citation: Mohammed A (2021) Viral Oncogenesis - Pathology, J Clin Mol Pathol, Vol: 5 No: 4.

Description

Viral oncogenesis can be defined as the feature of the tumor viruses that induces benign or malignant proliferation of infected cells. Viruses are capable of reproducing by entering a living cell and manipulating the cell's machinery to create more viruses. During this viral replication process, certain virus's like DNA or RNA affects the host cell's genes in such a way that may cause it to become much cancerous. Gross early discoveries of viral causation of murine leukemias, the subsequent demonstration of retroviruses as causative factors, and the presence of similar viruses, particularly in lymphoproliferative disorders in cattle, cats, and various rodent species, justified efforts to find similar agents in human cancers for decades subsequently identified as the retroviruses.

Discussion

Most human oncogenic viruses have several traits in common, including the fact that they are DNA viruses with lengthy (co)evolutionary histories with their hosts and the ability to cause latent or chronic infections. They can reach large prevalence's while producing low case mortality, making them ideal candidates for virulence evolution theory. We utilize a mathematical modeling technique to study how the virus life cycle may generate selective forces favoring or acting against oncogenesis at the within-host or between-host level after analyzing the life histories of DNA oncoviruses. Tumor viruses are a diverse collection of viruses that cause malignancies in humans, benign tumors, and cancers in laboratory animals that have been infected experimentally. Despite the fact that they are members of separate and unrelated systematic entities, they share a number of characteristics. They generally encode growth-stimulating proteins, include DNA as genetic material, and survive in the host for a long time.

Viruses cause about 20% of human cancers, especially in low- and middle-income nations, to the point that viral infection is the leading cause of cancer in some parts of the world. Over the last two decades, our understanding of the connection between viral infection and oncogenesis has vastly increased. Viruses encode proteins that modify host cellular signalling pathways that control proliferation, differentiation, cell death, genomic integrity, and immune system recognition as obligatory intracellular parasites. Human cancers have been related to both DNA and RNA viruses. In AIDS patients, the intrathoracic symptoms of oncogenic viral infection are diverse. Understanding the epidemiologic, pathologic, and imaging aspects of these often-treatable disorders is critical to diagnosing and managing them.

However, given current developments in vaccine development and medicine development, it is reasonable to believe that we are getting closer to minimising the impact of these seven viruses on the global population. Although a thorough understanding of all of the complexities of interactions with the native host for all of the oncogenic viruses mentioned in this review is still lacking, it is known that the innate immune system may detect their existence via a network of sensors.

Conclusion

Oncogenic viruses have proved helpful in revealing important aspects of normal cellular function and disease. Recent developments indicate that they are still a useful tool for conducting and guiding basic research. Oncogenic viruses, for example, have revealed that once-separate cellular processes are now linked. Because both of these routes can trigger cell cycle arrest and promote host cell death during infection, it has been argued that there is overlap between the tumor suppressor and innate immune signaling pathways.