

Genomic Analysis of Human Retroviruses

Williams M Down*

Department of Antiviruses, University of Sydney, Sydney, Australia

ABOUT THE STUDY

Retroviruses have an extremely intricate and well regulated life cycle, which has been the subject of much research for decades. When a virus enters a cell, its genome is reverse-transcribed, integrated into the host genome, and all structural and regulatory proteins are then translated. A new virion formed from the proteins and the viral DNA buds off the host cell and develops into a fresh infectious virion. The virus cannot create contagious viral offspring if any one of these processes is flawed. It is now feasible to learn more about this family of viruses, including specifics on how they govern and coordinate the many stages of the virus life cycle, due to recent developments in structural and molecular approaches.

Through giving a general overview of structural and biochemical investigations to comprehend these processes, the molecular analysis of the life cycle's assembly and maturation stages is presented [1]. The variations in these procedures among distinct retrovirus groups, the retrovirus genome is selectively recruited and packed into released particles together with two non-covalently linked versions at the site of virus particle formation. The nuclear export of the genome's unspliced RNA, the movement of the genome to the viral assembly sites, and a particular interaction with Gag, the primary structural protein of the virus, are all necessary for retroviral RNA packaging [2]. An update on recent developments in our understanding of the mechanism of RNA packaging for retroviruses, such as HIV-1, HIV-2, and HTLV-1, that are known to cause disease in humans, as well as developments in our knowledge of the specifics of nuclear export of genomic RNA, genome translocation to virus assembly sites, and genomic RNA dimerization.

In the human genome, Transposable Elements (TEs) make up around 45% of the total. During hominoid radiation, TEs have multiplied at random and incorporated into functional genes. They resemble somewhat enlarged left-handed Z-DNAs and right-handed B-DNA double helices. With an average ratio of 8%, human chromosomes are broadly dispersed with HERV families. A 5'-Long Terminal Repeat (LTR)-gag-pol-env-3'-LTR structure is

present in them [3,4]. The U3 enhancer and promoter region, transcribed R region, and U5 region are all present in LTRs.

LTRs can operate as regulatory elements and affect how the host's genes are expressed. By combining Z-hunt and Deep Z data for human functional genes, we explain the alternative promoters derived from LTR elements that overlap Z-DNA in this review. We also provide proof that Z-DNA-containing LTR segments in GSDML have regulatory function. Z-DNA and LTR elements' combined regulatory activity enables us to comprehend how genes behave in connection to numerous human disorders [5,6]. For the treatment of HIV, a specific class of antiviral drugs known as Antiretroviral Therapy (ART) is available. It is estimated that up to 20 million people globally are infected with HTLVs, however only a tiny proportion of those affected go on to acquire ATL or HAM/TSP [3].

Human AIDS is a condition caused by the retrovirus known as the Human Immunodeficiency Virus (HIV). SIV, a retrovirus that affects chimpanzees and gorillas, is related to HIV in a close way.

Infectious RNA- or DNA-containing viruses known as exogenous retroviruses spread from one creature to another [2]. Viruses are divided into two categories according to the Baltimore categorization system, which classifies them together according to how they synthesize messenger RNA: Group VI includes viruses with single-stranded RNA and a DNA intermediary in their life cycle, whereas Group VII includes viruses with double-stranded DNA and an RNA intermediate in their life cycle.

Animal malignancies and slow-growing tumours are brought on by retroviruses, which are also linked to slow-moving animal illnesses such horse infectious anaemia. A malignancy known as adult T-cell leukaemia is brought on by a retrovirus called Human T-cell Lymphotropic Virus type 1 (HTLV-1) (ATL). Additionally, it can result in HTLV-1-Associated Myelopathy/Tropical Spastic Paraparesis (HAM/TSP), a neurological disorder. Although it has not been proven that HTLV-2, a similarly related virus, causes human illness, it is connected with very modest neurological problems.

Correspondence to: Williams M Down, Department of Antiviruses, University of Sydney, Sydney, Australia, E-mail: williamdown@mdo.my.au

Received: 18-Jul-2022, Manuscript No. JAA-22-18892; **Editor Assigned:** 22-Jul-2022, Pre QC No. JAA-22-18892 (PQ); **Reviewed:** 12-Aug-2022, QC No. JAA-22-18892; **Revised:** 18-Aug-2022, Manuscript No. JAA-22-18892; **Published:** 25-Aug-2022. DOI: 10.35248/1948-5964.22.14.239.

Citation: Down WM (2022) Genomic Analysis of Human Retroviruses. J Antivir Antiretrovir. 14: 239.

Copyright: © 2022 Down WM. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

CONCLUSION

Retroviruses stand apart from other infectious agents in both the way they interact with the host cell and organism and the effects of this connection, which can sometimes extend beyond the life of the infected host and affect the host's descendants. No other higher eukaryotic infectious agent consistently integrates its genetic material into the host genome, frequently acquires host genes into its genome, can infect the host's germ line, and has contributed to as many different facets of contemporary biology. This brief part serves as a link between the perspective offered in the section that came before it, which describes the interactions between a single virus and a single cell, and the numerous intricate details of how retroviruses interact with the entire host.

REFERENCES

1. Padilla LA, Leung BS, Carson LF. Evidence of an association between human papillomavirus and impaired chemotherapy-induced apoptosis in cervical cancer cells. *Gynecol Oncol.* 2002;85: 59-66.
2. de Freitas L, Soares CP, Fontana CR. Synergistic effect of photodynamic therapy and cisplatin: a novel approach for cervical cancer. *J Photochem Photobiol B.* 2014;140: 365-373.
3. Strom TJ, Trotti AM, Kish J, Russell JS, Rao NG. Comparison of every 3 week cisplatin or weekly cetuximab with concurrent radiotherapy for locally advanced head and neck cancer. *Oral Oncol.* 2015;51: 704- 708.
4. Chen J, Solomides C, Parekh H, Simpkins F, Simpkins H. Cisplatin resistance in human cervical, ovarian and lung cancer cells. *Cancer Chemother Pharmacol.* 2015;75: 1217-1227.
5. Cao WQ, Zhai XQ, Ma JW, Fu XQ, Zhao BS. Natural borneol sensitizes human glioma cells to cisplatin-induced apoptosis by triggering ROS-mediated oxidative damage and regulation of MAPKs and PI3K/AKT pathway. *Pharm Biol.* 2020;58: 72-79.
6. Manohar S, Leung N. Cisplatin nephrotoxicity: A review of the literature. *J Nephrol.* 2018;31: 15-25.