AIRO JOURNALS
Free / Unpaid
Peer Reviewed
Multidisciplinary
National

ISSN:2321-3914
Volume 3 Issue 2
August 2023
Impact Factor: 10.1
Subject : Computer Science

AN EXAMINATION OF THE EFFECTS OF HIV ON PERMANENT SYSTEMS

BHABANI SANKAR RATHA
Research Scholar
Dr Pratap Singh Patwal
GUIDE NAME

DECLARATION: I AS AN AUTHOR OF THIS PAPER /ARTICLE, HERE BY DECLARE THAT THE PAPER SUBMITTED BY ME FOR PUBLICATION IN THE JOURNAL IS COMPLETELY MY OWN GENUINE PAPER.IFANY ISSUE REGARDING COPYRIGHT/PATENT/ OTHER REAL AUTHOR ARISES, THE PUBLISHER WILL NOT BELEGALLY RESPONSIBLE. IF ANY OF SUCH MATTERS OCCUR PUBLISHER MAY REMOVE MY CONTENT FROM THE JOURNAL WEBSITE. FOR THE REASON OF CONTENT AMENDMENT/OR ANY TECHNICAL ISSUE WITH NO VISIBILITY ON WEBSITE/UPDATES, I HAVE RESUBMITTED THIS PAPER FOR THE PUBLICATION.FOR ANY PUBLICATION MATTERS OR ANY INFORMATION INTENTIONALLY HIDDEN BY ME OR OTHERWISE, ISHALL BELEGALLY RESPONSIBLE. (COMPLETE DECLARATION OF THE AUTHOR ATTHE LAST PAGE OF THIS PAPER/ARTICLE

Abstract

Despite its common association with sequence analysis, database searching, and sequence comparison, bioinformatics is actually far more nuanced and useful than that. Bio-informaticians, often known as computational biologists, are responsible for a wide range of activities, including data collection, management, and presentation. Statistics, physics, computing, and engineering are just some of the many quantitative areas of study that are incorporated. Predictive methods developed with the aid of genome sequences will allow scientists to model the function and phenotype of an organism, which is the ultimate goal of analytical bio-informaticians.

INTRODUCTION

The only place a virus may reproduce successfully is inside the nucleus of a living cell. A wide variety of creatures, from mammals and plants to bacteria and archaea, are susceptible to virus infection. HIV is a virus that attacks human cells and weakens the immune system, making people more susceptible to illness. The fact that it needs a host in order to multiply is its defining characteristic. HIV is a main human cause of AIDS; hence the term. The human immunodeficiency virus (HIV) is a disease that develops later in life and causes a wide range of symptoms and indicators due to the body's inability to function properly. AIDS is a syndrome rather than a single disease because it affects so many different parts of the body and can produce so many different consequences and symptoms. Acquired immunodeficiency syndrome (AIDS) is a condition in humans characterised by a gradual breakdown of the immune system and the development of potentially fatal opportunistic infections. Human immunodeficiency virus (HIV) is a lent virus (a member of the retrovirus family) that causes AIDS.

The transmission of HIV takes place through bodily fluids such as blood, sperm, vaginal fluid, pre-ejaculate, and breast milk. HIV is prevalent in these body fluids as both free

ISSN:2321-3914
Volume 3 Issue 2
August 2023
Impact Factor: 10.1
Subject: Computer Science

virus particles and virus within infected immune cells. Pregnancy, birth, breast milk, and unsafe intercourse are the four most common ways that the virus is spread from mother to child (perinatal transmission).

Transmission of HIV through blood transfusions or infected blood products has been virtually eliminated in developed countries as a result of screening procedures.

The number of new HIV infections is increasing at an alarming rate, and the World Health Organization has declared a global pandemic (WHO). Roughly 34 million people worldwide have HIV as of 2010. About 16.8 million of them are female, while another 3.4 million are children under the age of 15. As a result, around 1.8 million people lost their lives in 2010, down from 3.1 million in 2001.

Classification of HIV: HIV-1 and HIV-2 are the two subtypes that have been identified. HIV-1 was the first virus found, and it was given the names LAV and HTLV-III. The majority of HIV infections worldwide are due to this strain because it is more virulent and contagious [54]. The Sooty Mangabey is the original host for HIV, while the Common Chimpanzee is the source for HIV-1 (Smm). HIV-2 has a lower infectivity than HIV-1, therefore fewer people will become infected from each exposure. HIV-2 is mostly found in West Africa because of its low transmissibility.

Stages of HIV infection: The incubation period, the acute infection, the latency stage, and AIDS are the four basic stages of HIV infection.

- Asymptomatic or clinically silent infection during the first incubation period (stage I) is defined as a CD4+ T cell count (also called CD4 count) of greater than 500 cells per microliter (uL). Generalized swelling of lymph nodes is possible and the process typically takes two to four weeks.

- Minor mucocutaneous signs, recurring upper respiratory tract infections, fever, lymphadenopathy, pharyngitis, rash, myalgia, malaise, and mouth and oesophageal ulcers are all indications of stage II acute infection. There is an average half-life of 28 days with a CD4 count of less than 500/uL.

- The third stage, known as latency, can last anywhere from two weeks to twenty years or more and is characterised by the presence of more advanced symptoms such as unexplained chronic diarrhoea lasting more than a month, severe bacterial infections such as tuberculosis of the lung, and a CD4 count of less than 350/uL.

- AIDS, the fourth and ultimate stage of HIV infection It illustrates the signs and symptoms of several opportunistic diseases. Kaposi's sarcoma, toxoplasmosis of the brain, and candidiasis of the oesophagus, trachea, bronchi, or lungs are only some of the severe symptoms that might occur. The viral load multiplies by the millions if the CD4 count drops below 200/uL [WHO case 2007].

HIV genome and structure: A comparison of HIV's structure to that of other retroviruses is presented in figure 1.4. It's around 60 times

ISSN:2321-3914
Volume 3 Issue 2
August 2023
Impact Factor: 10.1
Subject : Computer Science

smaller than a red blood cell, but it's still very big for a virus [36]; its diameter is about 120 nm. It has a conical capsid made up of 2,000 copies of the viral protein and contains two copies of positive single-stranded RNA that code for the virus's nine genes. Nucleocapsid proteins and enzymes like reverse transcriptase, proteases, ribonucleases, and integrase are coupled to the single-stranded RNA, which is then packaged into a virion. The virion particle's integrity is maintained by a matrix of viral protein that surrounds the capsid.

HIV enters macrophages and CD4+ T cells through fusion of the viral envelope with the cell membrane and the release of the HIV capsid, as shown in figure 1.4. When the trimeric envelope complex (gp160 spike) binds to CD4 and a chemokine receptor (often CCR5 or CXCR4, but others are known to interact), the virus gains access to the cell. gp120 attaches to integrin 47, activating LFA-1, the key integrin involved in establishing virological synapses, which allows for effective cell-to-cell dissemination of HIV-1. Both the CD4 binding domain and the chemokine receptor binding domain are present on the gp160 spike. First, gp120's CD4 binding domains connect to CD4 with great affinity. Once gp120 is coupled to the CD4 protein, a structural shift occurs in the envelope complex that reveals the chemokine binding domains of gp120, allowing them to engage with the specific chemokine receptor.

Transcription and replication: Upon entry of the viral capsid into the cell, the single-stranded (+) RNA genome is unbound from the viral proteins and copied into a complementary DNA (cDNA) molecule by an enzyme called reverse transcriptase. Mutations introduced during reverse transcription are likely to increase the virus's fitness, making it more difficult to treat or even impervious to the body's defences. During the production of cDNA, the reverse transcriptase destroys the viral RNA with its ribonuclease activity, and it synthesises sense DNA from anti-sense cDNA with its DNA-dependent DNA polymerase activity. The cDNA joins with its complementary strand to produce a double strand of viral DNA, which is subsequently taken up by the cell and taken to the nucleus. Another viral enzyme, integrase, is responsible for incorporating viral DNA into the genome of the host cell. Once the viral DNA has been integrated, the infection may enter a latent state and remain dormant. The most essential of these transcription factors, NF-B (NF kappa B), is up regulated in T-cell activation and is required for viral production. In other words, the cells that are actively working to combat the HIV infection are the ones at most risk of being destroyed by the virus.

In the process of viral replication, the integrated DNA provirus is translated into messenger RNA (mRNA) and subsequently spliced into smaller pieces. These fragments leave the nucleus and are translated into Tat (which promotes the creation of new viruses) and Rev in the cytoplasm. The newly synthesised Rev protein binds to viral mRNAs as it accumulates in the nucleus, releasing unspliced RNAs from the nucleus until they are processed.

Construction and release: Assembly of new HIV-1 virions, the last stage of the virus' life cycle, takes place at the host cell's plasma membrane. Translocated from the endoplasmic reticulum to the Golgi complex, the Env polyprotein is proteolytically broken to generate gp41 and gp120, the two HIV envelope glycoproteins. These then make their way to the host cell's plasma membrane,

ISSN:2321-3914
Volume 3 Issue 2
August 2023
Impact Factor: 10.1
Subject: Computer Science

where gp41 attaches gp120 to the infected cell's membrane. During the process of bud formation, the HIV genomic RNA and the polyproteins Gag and Gag-Pol bind with the inner surface of the plasma membrane. Either the developing bud or the immature virion that has detached from the host cell undergoes maturation. HIV proteases are responsible for cleaving the polyproteins during maturation into their component proteins and enzymes. Finally, a complete HIV virion is assembled from its many structural parts. Protease inhibitors can block this cleavage process. When the virus has completed its development, it can infect a new cell.

HIV MOLECULAR SEQUENCE CLASSIFICATION PROBLEMS AND HARDSHIPS

Several significant difficulties and opportunities exist in the broad field of biomedical informatics as biomedical research and healthcare continue to advance in the genomic /post genomic age. The current deluge of raw data, aggregate information, and evolving knowledge resulting from the study of the genome and its manifestation presents significant difficulties for the field of bioinformatics. Understanding the origins of this deluge of information and, more significantly, the vision of how this information might be used is helpful for understanding the potential for both biological research and bioinformatics in a broader perspective. The human genome project sped up the transition from linear to exponential growth in data production.

Bacterial, fungal, viral, and other pathogens' gene, microarray, nucleotide, and peptide sequence data create information and knowledge that can be traced to a more in-depth understanding of human health and disease. Opportunities to I better understand the structure of genetic networks and protein pathways, and (ii) determine the role that these structures play in determining cellular and organism phenotypes, coexist with these difficulties. (ii) create genomically informed tools for disease risk assessment, treatment response prediction, early diagnosis, and disease classification. As a result, improved research into how to address these problems and obstacles will emerge as a result of the interplay of computing techniques with the biological and biomedical areas. Nucleotides and amino acids, which make up biological data, are very variable.

CONCLUSION

SVM model predictions are highly accurate across all datasets, lending credence to the idea that using biological variables acquired from HIV could considerably boost performance. Since the HIV replication stages are located in the mitochondria, plasma membrane, cytoplasm, and extracellular space. The ability to forecast where proteins will be found within cells can aid in illness diagnosis. In this study, we only focus on proteins that only require a single localization site. SVM models' accuracy can be further enhanced by using more prediction sites. Understanding protein function relies on knowing where in the cell it is located, making this a crucial stage in the genome annotation process. Due to their proximity to the extracellular space or the cell surface, drug molecules have easy access to secreted proteins and plasma membrane proteins, respectively.

The HIV soluble proteins and HIV membrane proteins have been attempted to be categorised using physical and chemical

ISSN:2321-3914
Volume 3 Issue 2
August 2023
Impact Factor: 10.1
Subject: Computer Science

criteria. Repeatedly, the WEKA classifier correctly divides HIV membrane proteins into primary and secondary transmembrane regions. An accurate characterization of HIV membrane proteins relies heavily on their classification into distinct classes based on their amino acid makeup. Some functional characteristics of membrane proteins may be gleaned from a categorization based on the percentage of secondary transmembrane helices. Highly effective antiretroviral therapy (HAART) for HIV infection may make use of these characteristics. HIV is the virus responsible for AIDS, a fatal opportunistic infection. Future HIV drug development is aided by these novel approaches to studying membrane proteins and transmembrane helices. The best way to model the biological data is by categorization, and machine learning approaches are quick and cheap. The model will become even more accurate as more soluble and membrane protein data is added in the not-too-distant future.

REFERENCES

1. Dong Xu et al, Hydrogen bonds and salt bridges across protein–protein interfaces, *Protein Engineering*, vol.10, No.9 pages 999–1012, (1997).

2. Douek DC, Roederer M, Koup RA, "Emerging concepts in the immunopathogenesis of AIDS", *Annu. Rev. Med*., vol.60, pages 471–84, (2009).

3. Emanuelsson O, Nielsen H, von Heijne G," **ChloroP, a neural network-based method for predicting chloroplast transit peptides and their cleavage sites".** *Protein Sci*, vol.**8,** 978-984, (1999).

4. **Emily CY Su**, **Hua-Sheng Chiu**, **Allan Lo**, **Jenn-Kang Hwan**, **Ting-Yi Sung**and **Wen-Lian Hsu**, 'Protein subcellular localization prediction based on compartment-specific features and structure conservation", *BMC Bioinformatics*, vol. 8, no. 330, (2007).

5. Haedicke, J.; Brown, C.; Naghavi, H. "The brain-specific factor FEZ1 is a determinant of neuronal susceptibility to HIV-1 infection". Proceedings of the National Academy of Sciences, vol.106, no. 33, pages 14040–14045, (2009).

6. Han J. and Kamber M., Data mining :concepts and Techniques, (2010).

7. Hanbing Rao, Guobing Yang, Ningxin Tan, Ping Li, Zerong Li, Xiangyuan Li, "Prediction of HIV-1 Protease Inhibitors Using Machine Learning Approaches", *QSAR & Combinatorial Science*, vol. 28, Issue 11-12, pages 1346–1357, (2009).

8. Kitahata MM, Gange SJ, Abraham AG, et al., "Effect of early versus deferred antiretroviral therapy for HIV on survival". *N. Engl. J. Med*. Vol.360, no. 18, pages 1815–26. (2009),

9. Los Alamos National Laboratory Established 1943. "Fighting the world's most dangerous disease, Los Alamos Lab". Lanl.gov. (2010).

10. Mitaku S. Hirokawa T, Boon-Chieng S, "Classification of Membrane Proteins by Types of Transmembrane Helices using SOSUI system", *Bioinformatics*

ISSN:2321-3914
Volume 3 Issue 2
August 2023
Impact Factor: 10.1
Subject : Computer Science

Applications Note, 14, pages 132-140, (2004).

Author's Declaration

I as an author of the above research paper/article, hereby, declare that the content of this paper is prepared by me and if any person having copyright issue or patentor anything otherwise related to the content, I shall always be legally responsible for any issue. For the reason of invisibility of my research paper on the website/amendments/updates, I have resubmitted my paper for publication on the same date. If any data or information given by me is not correct, I shall always be legally responsible. With my whole responsibility legally and formally I have intimated the publisher (Publisher) that my paper has been checked by my guide (if any) or expert to make it sure that paper is technically right and there is no unaccepted plagiarism and hentriacontane is genuinely mine. If any issue arises related to Plagiarism /Guide Name /Educational Qualification /Designation /Address of my university/college/institution/Structure or Formatting/ Resubmission / Submission /Copyright / Patent/Submission for any higher degree or Job/Primary Data/Secondary Data Issues. I will be solely/entirely responsible for any legal issues. I have been informed that the most of the data from the website is invisible or shuffled or vanished from the data base due to some technical fault or hacking and therefore the process of resubmission is there for the scholars/students who finds trouble in getting their paper on the website. At the time of resubmission of my paper I take all the legal and formal responsibilities, If I hide or do not submit the copy of my original documents (Aadhar/Driving License/Any Identity Proof and Photo) in spite of demand from the publisher then my paper may be rejected or removed from the website anytime and may not be consider for verification. I accept the fact that as the content of this paper and the resubmission legal responsibilities and reasons are only mine then the Publisher (Airo International Journal/Airo National Research Journal) is never responsible. I also declare that if publisher finds any complication or error or anything hidden or implemented otherwise, my paper maybe removed from the website or the watermark of remark/actuality maybe mentioned on my paper. Even if anything is found illegal publisher may also take legal action against me

BHABANI SANKAR RATHA
Dr Pratap Singh Patwal