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THE IMPACT OF QUASI-SPIECIES OF HIV ON THE ESTIMATION TIME TO GET AIDS

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Abstract

It is a proven fact that the antigenic diversity induced by the HIV plays a major role towards the progression of the infection to the AIDS condition. Because of the fact that no successful medicine is available for curing HIV/AIDS, the only possibility for keeping the patient alive is to prolong the period of getting AIDS condition since from infection. The amount of antigenic diversity contributed by not only the virus entered through every infective contact, but also their quasi-species. As such is the case, the antigenic diversity induced by the quasi-species of the virus entered through successive infective contacts may form an order statistic. This article considers this fact and based on which a stochastic model has been developed to estimate the time to get AIDS condition since from infection.

1. Introduction

The HIV virus, which suppress immune system and hence induces the susceptibility to infections and diseases, is the etiological agent of AIDS. HIV presents numerous antigenic variants, complicating the immune system's ability to recognize and respond to the antigens, which significantly increase the viral load and fastens the onset of AIDS conditions. It is reasonable to consider that the HIV-infected individual can only endure the virus with a specific amount of antigenic diversity, referred to as the Antigenic Diversity Threshold (ADT) (Nowak and May, 1991). Each infective contact may contribute to the antigenic diversity of HIV and added to that the replication of several quasi-species further enhances the levels of this diversity. An infected individual's life expectancy may be diminished by means getting AIDS condition sooner, if the total accumulated antigenic diversity exceeds the Antigenic Diversity Threshold (ADT).

Many authors have investigated various aspects of antigenic diversity in their research endeavors. After a prolonged and variable incubation period, an HIV infection severely damages the immune system, leading to the development of Acquired Immunodeficiency Syndrome (AIDS), as observed by Martin A. Nowak and Robert M. May (1991). The notion that AIDS occurs when the diversity of HIV antigenic variants in an infected individual exceeds a critical threshold, beyond which the immune system cannot maintain itself, is based on the

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changing nature of the interaction between immune response and viral antigenic variation. Stilianakis et al. (1994) created a model to predict the non-monotonic risk of AIDS transition that is partially characterized by the Antigenic Diversity threshold. Nowak et al. (1997) refined Stilianakis' model to highlight the fact that the threshold of antigenic diversity explains the prolonged and variable delays observed between HIV infection and the development of AIDS and to clarify the relationship that exists between each of them. Estimating how long is expected to exceed the Antigenic Diversity Threshold (ADT) is essential, as it indicates the probability of an infected individual manifesting AIDS symptoms. In a stochastic model created by Jaisankar R. and Sabeurnisha A. (2017) to estimate the period of time required to cause AIDS development based on the antigenic diversity produced by each sexual contact with an infected individual with the assumption that the Antigenic Diversity Threshold (ADT) is a random variable. Veena A and Kannan R (2023) developed a model that based on shock models and the cumulative damage process and the model estimates the expected time for seroconversion when the accumulated antigenic diversity crosses a threshold level.

The present study develops a stochastic model for predicting the time to get AIDS condition, considering the antigenic diversity induced by both variants of HIV acquired through infectious contacts and those related to their corresponding viral quasi-species. It is to be noted that the antigenic diversity contributed by the virus's quasi-species generated through the HIV entered through each of the successive infective contact may constitute an order statistic. This article examines this phenomenon and, based on it, a stochastic model has been constructed to estimate the time from HIV infection to the development of AI DS.

2. Methodology

2.1 Assumptions of the Model

- HIV spread through sexual contacts with infected individuals only.
- A person has several contacts at random, which may include some infectious individuals as well.
- When an individual is having sexual contacts with HIV infected partners, a random number of HIV is getting transmitted at each contact.
- The quasi-species of viruses, developed through the replication of HIV entered during each infectious contact, also contribute to the antigenic diversity.
- The amount of virus spread through each contact is assumed to be a random variable follows exponential distribution.
- Each HIV virus develops its own viral quasi-species after a certain duration, which is also assumed to follow the exponential distribution.
- The antigenic threshold level, represented as Z, is considered to be an exponential random variable.

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- Over a period of time, say (0, t), an individual has *n* contacts at random, of which *k* are infected contacts.

2.2 Notations

- U_i - A random variable that represents Antigenic Diversity caused or induced by the i^{th} contact with a HIV-infected person which follows an exponential distribution, say, $g(.)$ with parameter λ.
- V_i – A random variable representing the Antigenic diversity induced by the quasi-species of U_i after some time period which is assumed to follow exponential distribution, say, $h(.)$ with parameter $\boldsymbol{\gamma}_i$.
- $I_k(.)$ represents the probability distribution of the total antigenic diversity induced by the *k* infective sexual contacts.
- $J_k(.)$represents the probability distribution of the total antigenic diversity induced by the quasi-species of *k* infective sexual contacts.
- *H(.)* – The probability distribution of the antigenic diversity induced through the i^{th}contact and its corresponding quasi-species respectively.
- $\widetilde{U}$ - Total antigenic diversity induced by *k* contacts
- $\tilde{V}$ - Total antigenic diversity induced by the quasi-species of U_i
- *Z* – A random variable representing the stochastic Antigenic Diversity Threshold (ADT), which is assumed to follow an exponential distribution with parameter μ.
- *T* – Stochastic time at which Antigenic diversity threshold *Z* is passed.

2.3 THE PROPOSED MODEL

Suppose that over a period of time duration, say, *t*, a person is having *n* number of sexual contacts with randomly selected partners. Assume that out of *n* contacts, *k* are infective and each of which may contribute to the antigenic diversity. Added to this the quasi-species produced by the replication of HIV virus entered may also contribute to the antigenic diversity. In this context, since the contacts are successive, the later part may be explained through an order statistic in which every term is assumed to follow exponential distribution with different parameters. In this model the sum of these terms is taken up which follow a hypo-exponential distribution. Now, the probability that the antigenic diversity generated from *k* sexual contacts with infected persons and their associated viral quasi-species over a specified duration, remains below the threshold level is expressed by,

$$P\left[\left(\widetilde{U}+\tilde{V}\right)<Z\right]=\int_0^\infty H(z)\mu e^{-\mu z}dz$$

$$=\int_0^\infty \left(I_k(.)*J_k(.)\right)\mu e^{-\mu z}dz$$

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$$= \left(\frac{\lambda}{\lambda+\mu}\right)^k \prod_{i=1}^{k} \frac{\gamma_i}{\gamma_i+\mu}$$

The probability that there are *n* random contacts during the time interval (0, t) is explained by a Poisson process with parameter, say, α, and out of *n, k* being the number of infected contacts is explained by a Binomial distribution with parameter, say, *p*.

Hence, the probability that Antigenic diversity developed through *n* contacts that does not cross the antigenic diversity threshold level becomes,

$$P(T > t) \quad = \sum_{n=0}^{\infty} \frac{e^{-\alpha t}(\alpha t)^n}{n!} * \sum_{k=0}^{n} \binom{n}{k} q^{n-k}(1-q)^k * \left(\frac{\lambda}{\lambda+\mu}\right)^k \prod_{i=1}^{k} \frac{\gamma_i}{\gamma_i+\mu}$$

And the density function of the same is given by,

$$f(t) = \sum_{n=0}^{\infty} \frac{e^{-\alpha t}(\alpha t)^n}{n!}\left(\alpha - \frac{n}{t}\right) * \sum_{k=0}^{n} \binom{n}{k} q^{n-k}(1-q)^k * \left(\frac{\lambda}{\lambda+\mu}\right)^k \prod_{i=1}^{k} \frac{\gamma_i}{\gamma_i+\mu}$$

From this, the mean time to cross the Antigenic diversity threshold level and its variance can be derived as,

$$E(T) = \frac{1}{\alpha} \sum_{n=0}^{\infty} \sum_{k=0}^{n} \binom{n}{k} q^{n-k}(p)^k * \left(\frac{\lambda}{\lambda+\mu}\right)^k \prod_{i=1}^{k} \frac{\gamma_i}{\gamma_i+\mu}$$

$$V(T) = \frac{2}{\alpha^2} \sum_{n=0}^{\infty} (n+1) \sum_{k=0}^{n} \binom{n}{k} q^{n-k}(p)^k \left(\frac{\lambda}{\lambda+\mu}\right)^k \prod_{i=1}^{k} \frac{\gamma_i}{\gamma_i+\mu} - \left(\frac{1}{\alpha} \sum_{k=0}^{n} \binom{n}{k} q^{n-k}(p)^k * \left(\frac{\lambda}{\lambda+\mu}\right)^k \prod_{i=1}^{k} \frac{\gamma_i}{\gamma_i+\mu}\right)^2$$

3 Results and Discussion

The behaviour of the constructed model is evaluated using a simulation studies, and the sample mean and variance of the time to exceeds the antigenic diversity threshold are computed using random numbers simulated from the corresponding distribution for various parameter combinations. The following table and graphs show the observed values of *E(T)* and *V(T)*.

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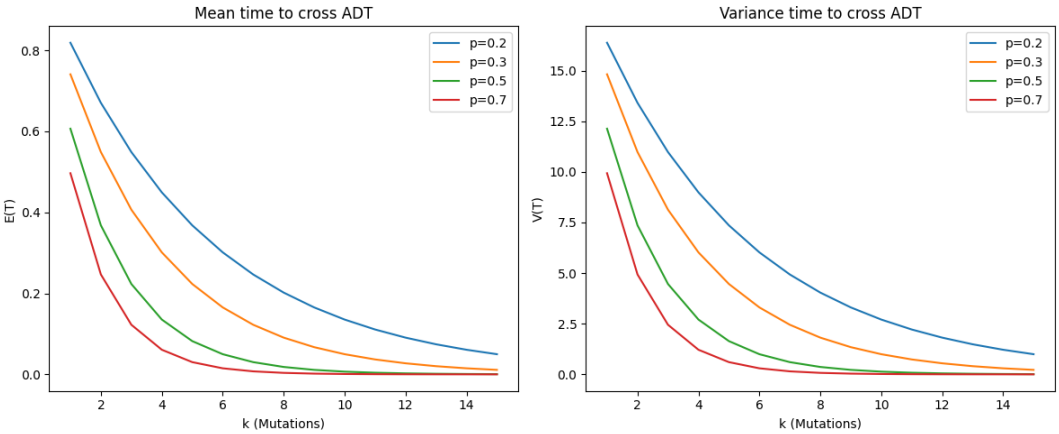

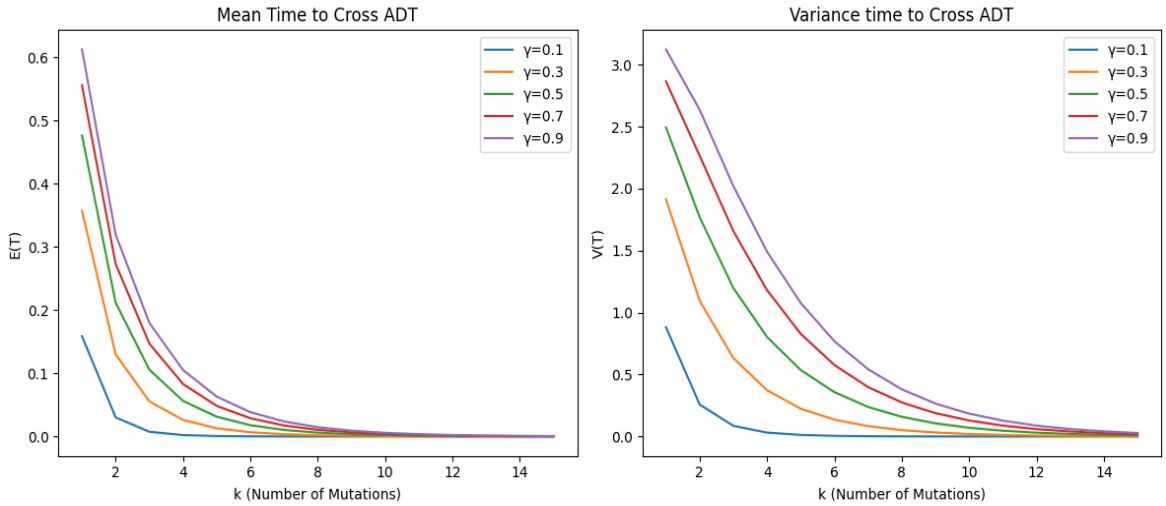

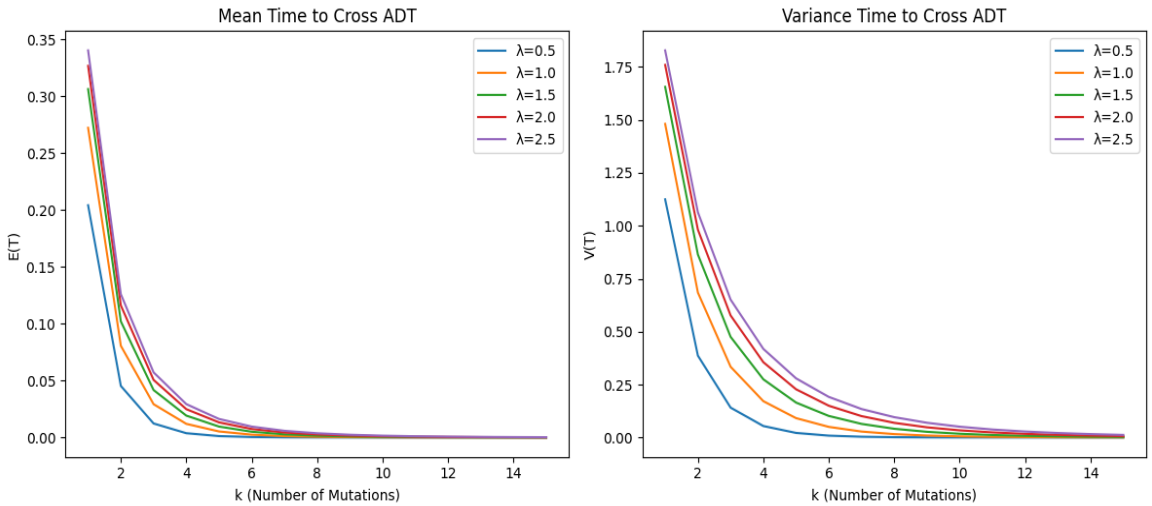

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The graphs based on the numerical study demonstrates the impact of key parameters on the expected time *E(T)* and variance *V(T)* for time to exceed the antigenic diversity threshold (ADT). As the number of infective contacts *k* increases, both *E(T)* and *V(T)* decrease, indicating that higher contact rates accelerates the progression towards the AIDS condition. It can also be interpreted as elevated mutation rates lead to declines in *E(T)* and *V(T)* as the increased mutagenesis promotes faster antigenic diversification. Also, it is seen that if the host population in which the infected individual lives is more vulnerable for infection he may have more number of infective contacts and hence will cross the antigenic diversity threshold soon. Further, it can be noted that if the damage caused to the immune system through every contact has significant role in crossing the Antigenic diversity threshold. Contacts with highly infectious individuals make a rapid increae increase Antigenic diversity levels and as a consequence the infected individual attains the AIDS condition soon. These results highlight the critical interaction between viral dynamics and evolutionary pressures in driving antigenic diversity. That is, even if an infected individual avoid further contacts with infected, reaching AIDS condition depends on the antigenic diversity induced by the replication of virus already entered through infective contacts.

4 Conclusions

Overall, there are two aspects, namely, the number of contacts with infected individuals and the antigenic diversity induced through the quasi species of the virus entered regarding the time it takes to reach AIDS condition in an infected individual which is attributable to antigenic diversity were taken. If an infected person is having sexual contacts with more infected people, threshold of antigenic diversity is crossed earlier so also when the contacts are with higher levels of antigenic diversity. Conversely, if an infected person avoids further contacts with infected individuals the time taken for crossing the antigenic diversity level may get expanded and hence he will attain the AIDS condition at a later time. Knowing and understanding these factors enables better forecasting of AIDS condition in an infected individual.

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