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Role of Stochastfic Models in HIV Transmission

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Abstract

Stochastic models are developed to estimate the likely time which is otherwise known as expected time to seroconversion has been a matter of research interest. These stochastic models provide the basis for developing the suitable methods using it could become possible to estimate the likely time at which the seroconversion taken place. In this paper we discuss about the various stochastic models which predict the transmission of HIV.

Key words: Seroconversion, Antigenitic Diversity, Acquired Imuino Virus, Incubation period

The papers previewed here may be classified as those pertaining to the following areas in the study of HIV infection and AIDS.

AMS classification: 26A33, 49K15

1 Introduction

Human Immuno Deficiency Virus (HIV) causes the viral infection that leads to Acquired Immune Deficiency Syndrome (AIDS). It interruptions dejected the body's immune system, leaving the victim exposed to a variety of life-aggressive oppurunistic infections, neurological disorders, or unusual malignancies (Centre for Disease Control 1987). The emergence of HIV infection and the resulting AIDS has become a matter of great concern not only to the public but also to governments in various countries around the world. The consequences of HIV infection include suffering and a shortened lifespan for those infected. This would help a lot to take all precautionary measures in advance to reduce the spread of the virus as follows.

1. To contain the progression of the infection.
2. To find out the possible drugs and methods of intervention so that the process of serconversion can be delayed.
3. To find out the factors which contribute to process of seroconversion.

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It is proposed to progress stochastic to forecast models the life time and likely number of persons who would become seropositive cases which depends upon the way of life a precautionary measures which they take. Even though it would very difficult to get a relevant data from the individual concerned, the hospital records may be used for the purpose of data collection. The main factors which are socio economic, physiological individual behavior can be taken and their contribution and significance in the process of seroconversion can be identified.
The papers previewed here may be classified as those pertaining to the following areas in the study of HIV infection and AIDS.

1. Models relating to the transmission and dynamics of HIV spread,
2. Models relating to the distributions of random vari- ables (Latency period, Incubation period and Seroconversion time) and
3. Models relating to infectivity, projection and other aspects.

2 Models Relating to the Transmission and Dynamics of HIV Spread

[3] Anderson (1988) has discussed on the epidemiology of HIV infection taking into consideration the variable cul- tivation period, infectious period and the heterogeneousness in sexual activity. The author considers that a homosexual community of size N can be divided into X =number of susceptible, Y = number of infected and A = AIDS patients ($N = X + Y + A$) The simplest set of equations that are used for the transmission of HIV in a closed communal (i.e., no influx of susceptible from outside and no deaths other than is given by those who die due to AIDS)

$$\frac{dX}{dt} = -\lambda X$$
$$\frac{dY}{dt} = -\lambda X - \nu Y$$
$$\frac{dA}{dt} = -\nu Y - \alpha A$$
$$\frac{dN}{dt} = -\alpha X$$

where the parameter $\nu = \frac{1}{D}$ describes the rate of exit the infected class Y to connection the AIDS class A, where D is the average duration of stay in Y. Here λ is the per capital force of infection defined as

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$$\lambda = \frac{\beta CN}{N}$$

where C is the average number of sexual associates per of tirne and β is the probability of infecting a susceptible partner. The expression for R_0 which is the average number of secondary cases of infection is obtained as $R_0 = \beta CD$ with the postulation that the infected personalities are infectious during the duration of their stay in class Y . The expression for doubling time t_d , is given by

$$t_d = t\left[\frac{ln(2)}{R_0 - 1}\right]$$

The author has also discussed various extensions of this model taking into consideration variable incubation, infection periods and also the variations in the rate of sexual partners.

Understand the use of mathematical models the AIDS epidernic has been presented by [11] Hymann and Stantley (1988). The authors have indicated how the various factors, qualitative and quantitative, should be incorporated into a mathematical model that is used for depicting the blowout of AIDS epidernic. The concepts like population risk structure the sexual activities and their impact on risk, the drug use etc., have been discussed in detail in this paper. In addition to this the authors have given some simplified mathematical models for blowout of the epidemic. [9] Isham (1988) has given a examination of the mathematical modeling of the transmission subtleties of HIV infection and AIDS. The paper deals with a simple epidemic model of deterministic type is discussed initially. A fixed people of size n is detached into two groups namely a group of $X(t)$ the susceptible and $Y(t)$ the infectives, where $X(t) + Y(t) = n$ and $X(t)Y(t)$ are big enough so that they can be viewed as continuous variables. Assuming the population thus mixes homogeneously at every small time interval $(t, t+\delta)$ The number of contacts between a susceptible and an infectious is proportional to $X(t)$ and $Y(t)$ (and δ) and that a fixed proportion of these contacts result in the susceptible being infected. It has been shown that the number of new cases of infection in time intervals is $\alpha X(t), Y(t), \delta$ forsome α which is a constant of proportionality. $X(t)$ satisfies the differential equation $\frac{dX(t)}{dt} = \frac{\alpha X(t)}{Y(t)} = \alpha X(t)[n - X(t)]$ and the solution of this equation is given by $dy(t)ldt$

is the rate at which the new contagions occur and it can be obtained from the solution of the differential equations given above. Assuming $X(0) \approx n$, for t

$$Y(t) = Y(0)e^{n\alpha t}$$

The expression for doubling time is obtained as

$$t^d = (n\alpha)^{-1} ln2$$

The author has also discussed a simple model for the spread of AIDS in terms of stochastic behavior and obtained a deterministic guesstimate to the stochastic process. In doing so, a number of assumptions are made. They are enumerated as follows:

1. The blowout of HIV infection is within a closed male homosexual community,
2. The population of a fixed size n at time t is divided into $X(t)$ inclined and $Y(t)$ infectives,
3. The latent period of infection is considered to be negligible,
$$\frac{dX(t)}{dt} = \frac{[-\beta KX(t)Y(t)]}{[n]}$$
4. Each susceptible acquires new sexual partners at k. A deterministic calculation of the stochastic process is given by the differential equation
$$\frac{dX(t)}{dt} \frac{[\beta KX(t)Y(t)]}{[n]}$$

satisfied $yX(t)$. Assurning that at time 0, the number of infectives is small the expression for $Y(t)$ is

$$Y(t) \approx Y(0)e^{-\beta Kt}$$

The express for doubling tirne is

$$t_d \approx (\beta K)^{-1} ln2$$

Further variations of this model have also been discussed. The heterogeneity of sexual activity and its influence are also considered in this paper. The incubation

distribution is also discussed.

[21]Tan and Hsu (1989) have discussed a stochastic model for AIDS blowout in a homosexual population. In doing so, the authors have used Kolmogorov's forward equation. The authors also state that many biological issues such as cultivation time and communal factors affecting AIDS spreader subjected to extensive random variation. In developing the stochastic models, the authors assume a population consisting of four types of persons, namely S (Susceptible person), L (Latent person), I (Infective person), A (AIDS case) $S(t)$, $L(t)$,$I(t)$ and $A(t)$ are taken to be the number of persons in the four categories at time t. A set of assumptions are also provided by the authors before developing the model. These assumptions include

(i) Homogeneous mixing of persons among the category S and I,
(ii) $S(t)$ is assumed to be large for all $t \geq t_0$ so that S(t)is expected to be a deterministic function of t.

Under some expectations regarding AIDS blowout$(S \to L)$, latency $(L \to I)$ and incubation $(I \to A)$, theprobability generating function (pgf) of $L(t)$, $I(t)$ and $A(t)$ at time t_0 is given by

$$q(t_0,t) = Q(xy,z,t_0,t)$$

Using the p.g.f, the first order differential equation called Kolomogorov's forward equation for the p.g.f has beene- obtained. The expected value of $L(t)$,$I(t)$ and $A(t)$ are also obtained.

The possessions of changing the contact rate betweenthe susceptible person and the infective person is alsoinvestigated in this paper.

[7] Billard and Zhao (1991) have discussed a 3 stagestochastic epidemic model with applications to AIDS. Inthe three stage model discussed, the assumption is that attime t the population size is N, of which $X(t)$ are infectedindividuals, and $Y(t)$ the over-all number of AIDS casesso that $S(t)$, the number susceptible individuals in the population,

$$S(t) = N - X(t) - Y(t)$$

It is also assumed that AIDS epidemic is a time continuousMarkov process. The infinitesimal transition probabilitiesin the time interval $(t,t+h)$ are

$$Pr[X(t+h) = x+1, Y(t+h) = y]/[X(t) = x, Y(t) = y] = \lambda(x,y,t)h + o(h)$$

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$$Pr[X(t+h)=x-1,Y(t+h)=(y+1)]/[X(t)=x,Y(t)=y]=\mu(x,y,t)h+o(h)$$

Pr [wo or further changes in $(t,t+h)$]=$o(h)$

$$\text{Where } o(h) \text{ is well-defined by } lim_{h\to 0}\frac{o(h)}{h}=0$$

Let us represent

$$P_{x,y}(t)=Pr\frac{[X(t)=x,Y(t)=y]}{[X(0)=x_0,Y(O)=y_0]}$$

Then the forward differential difference equations can be inscribed as

$$\begin{aligned}\frac{d}{dt}P_{x'y}(t)=&-[\lambda(x,y,t)+\mu(x,y,t)]P_{x'y}\\&+\lambda(x-1,y,t)+\mu(x,y,t)P_{(x-1)'y(t)}\\&+\mu(x+1,y-1,t)P_{(x+1)'y-1}(t)for(x,y)\in B\end{aligned}$$

where B is the state space of the Markov process.

The result of the set of a differential difference equa-tions has been obtained in this paper. The expression forthe mean and variance of AIDS cases in a closed population and also in an open population have been obtained.
[12] M (1991) has discussed a stochastic model for thegrowth of AIDS epidemic in a heterosexual population.The author has listed out a number of classical modelswhich have not taken into account the concept of bisexualpopulation. The author has introduced a two sex modelfor the blowout of HIV in a heterosexual population. Theprogression of HIV disease in any individual is labeledusing 6 states e_0,e_1,e_2,e_3,e_4 and e_5 where e_0 is thesusceptible state, e_1 is the infected but not seropositivestate, e_2 is the seropositive but asymptomatic state, e_3 is the AIDS related complex state, e_4 state of full blownAIDS and e_5 is the death due to AIDS. The conditionalprobabilities governing the transitions between the statesare assumed to be constant over time. Couples in partnership taken into account in a heterosexual population;and the state of the couples is denoted j,k which meansthat the female $j,k=0,1,2,3,4$ which shows that both the partners are alive. Based on these assumptions andalso taking into account the risk of infection due to theuse of drug intravenously, a matrix of probabilities B isdefined. In this paper using random functions, the courseof the epidemic has been obtained. The results have beenobtained by using computer simulation.

[5] Arca et al. (1992) have authored a paper on thecommunication between intravenous drug users and heterosexual population. A general mathematical model related to the transition dynamics of HIV infected is givenin this paper. A compartmental structure $(X(t))$, Infected $Y(t)$ and Removed $Z(t)$ and with two possible transitions from X to Y and Y to Z, is considered. The change perunit in the number X of susceptible is given by

$$\frac{d(X)}{dt} = -\lambda(X,Y,t)X(t)$$

Here, $\lambda(X,Y,t)$ is the power of infection at $'t'$ given by

$$\lambda(X,Y,t) = c, \frac{(Y(t))}{(X(t)+Y(t))+\beta(t)}$$

where, c is the rate at which people establish contacts, β is the probability of getting infected in a single contact.Similarly the equations for the infected and removed are

$$\frac{d(Y)}{dt} = \lambda(X,Y,t)X(t) - \nu Y(t)$$

$$\frac{d(Y)}{dt} = \nu Y(t)$$

where, $\nu = [E(D)]^{-1}$ which gives the constant rate atwhich the infected developed AIDS, and D is the lengthof the incubation period.

[17] Perelson et al. (1993) have discussed the dynamics of HIV infection of $CD4+T$ cells. A model for the communication of HIV with $CD4+T$ cells is considered taking 4 different populations namely uninfected T cells,latently infected T cells, actively infected T cells and free of virus. 2 steady states such as uninfected state with no virus existing and an epidemically infected state in which virus infected T cells present are considered in this model.The model mainly of HIV uses the fact that the depletion of $CD4+T$ cells as a consequence of HIV infected.

[14] Naresh et al. (2006) have considered a non-linear mathematical model for the HIV widespread, which blowout through both horizontal and vertical communication in a population of different sizes. Vertical communication can be achieved by transplacental transmission of the pathogen. In recent years, some vertical transmission studies have been directed to designate the impact of various epidemiological and demographicfactors.

[15] Nirav Dalal (2006) observes in his study that many mathematical models have been established to designate viral dynamics of HIV-I, typically consuming a system of ordinary differential equations.
[23] Verotta and Schaedeli (2002) used nonlinear models to depict the viral dynamics of HIVI, which may involve various factors connected with resurgence. They discussed a nonlinear model of HIVI dynamics that includes revelation, handling adherence, and the emergence of HIVI resistance.
[16] Nirav Dalal et al. (2008) have analyzed the stochastic model representing the dynamics of the HIV interval virus. They have shown that their model does not have negative solutions since it is important in any population dynamics model. They explained the stability part of the model and showed that the number of infected cells and virus articles almost certainly exponentially asymptotically tended to zero. Their work showed that stochastic differential equations offer additional option for modeling virus dynamics.
[19] Samira Khalili et al. (2008), discussed the sim- ulation results in their study and examined the effect of the latency of the start of treatment on the infection dynamics. They observed that starting treatment imme- diately resulted in a significant decrease in the Infection Probability Percentage (IPP). They concluded that IPP can be determined using the stochastic model;
[26] Yong Sheng Ding et al. (2008) conclude in their paper that the disease transmission rate should fall below its critical level in order to reduce the percentage of the population infected with HIV in the total population.
[1] Adriana Weinberg et al. (2009) have discussed and found that resistance to antiretroviral drugs is present in HIV-infected pregnant women. The 14% pervasiveness of confrontation to zidovudine and lamivudine in antiretroviral-skilled women suggests that substitute nucleoside analogue reverse transcriptase inhibitors (NRTIs) are desirable in this patient population. [16] Shan mei et al. (2010) introduced the idea of a complex agent network in their work to model the HIV epidemic by joining multi-agent systems in which the agents depict people who might have sexual interactions.
[18] Ron Brookmeyer (2010) discussed the present methods and methods for measuring the HIV/AIDS epidemic, as well as their strengths and weaknesses. The author recaps the main sources of error and glitches with these and other methods and confers ways to improve their reliability.
[2] Akpa and Oyejola (2010 have discussed a review of modeling the communication

dynamics of HIV/AIDS waves, and this learning gives an overview of some of the models planned by different authors to describe the epidemiology and the epidemiological significances of the HIV/AIDS epidemic and how around of these could be improved to accommodate them adapt to situations in other countries. Four different types of basic modeling approaches can be used to develop mathematical models for HIV/AIDS and also for some other infectious sicknesses. Four different categories of basic modeling methods can be used to progress mathematical models for HIV/AIDS and also for some further infectious viruses.

The four types are the deterministic model, the stochastic model, the statistical model and the Karman filter model ([24] Wan Yuan (2000)). [6] Beena Thomas et al. (2011) have discussed the unique sociocultural issues faced by men who have sex with men (MSM) in India and how they relate to HIV risk, which could maximize the benefits of future prevention efforts. This article attempts to identify the specific challenges of providing of an effective HIV prevention program for this diverse and socially excluded at-risk group.

[25] A stochastic HIV infection model with latent infec- tion and antiviral therapy by Yan wang, Luju lin and Tang ting zhao (2018— is the work dealing with the formulation of a stochastic model with cell-to-cell infection. [22] Ting ting zhao Julia contributed a paper in 2018 looking at the stochastic HIV infection model that includes both latent infections and combinations during therapies] Competi- tive numerical analysis for stochastic HIV/AIDS epidemic model in a two sex population by Mohamed Rafiq et al (2019) This paper is an attempt to explain the responsible numerical analysis of the stochastic model in a bisexual population considering antiviral therapy.

3 Conclusion

As the models discussed above with various contribution make us to know about applications of stochasic models in HIV transmission and in the area of reduction of virus.This help us to develop new models in the transmission area. Also to give suggestions to eradicate the spread of HIV.

References

[1] Adirana Weinberg, Jeri Foster-Harwood, Eliza- beth J, Mcforland, Jennifer Pappas, Jill K. Davies, Kay Kenzie, Emily Barr A, Suzanne M. Paul, Carol Sabenblatt, Elizabeth Soda, Anna Varquez, Miran J. Lewin. Resistent to

antiretroviral in HIV infected pregnant women, Journal of Clinical Verology, Vol. 45, 39-42, 2009.

[2] Akba OM, Oyejola BA, Modelling the transmis- sion dynamics of HIV/AIDS epidemics : an introduction and a review. J Inect Dev. ctries, Vol. 4, No. 10, 597-608, 2010.

[3] Ali Raza, Muhamed Rafiq, Dumitru Baleanu, Muhammad Shoaib Arif, Muhammad Naveed, Kaleem Ashraf. Competitive numerical analysis for stochastic H—V/A—DS epidemic model in a two-sex population. Vol. 13, No. 6, 305-315, 2019.

[4] Anderson RM, The epidemiology of HIV infection: Variable incubation plus infectious periods and heterogenity in sexual activity, J.R. Statist. Soc. A, Vol. 151, No. 1, 66-93, 1988.

[5] Arca, Perucci CA, Spadea T, The epidemic dynam- ics of HIV-I in Italy modeling the interaction between intravaneous drug users and homosexual population, Statistics in Medicine, Vol. 11, 1657-1684, 1992.

[6] Beena Thomas, Mathew J. Mimiga, Senthil kumar, Soumiya swaminathan. Reason for a concentrated epidemic strategies for prevention, Vol. 134, No. 6, 920-929, 2011.

[7] Billiard L , Zhao, Three stage stochastic model for application of AIDS, Math. Bio. Sci.,Vol. 107, No. 2, 431-449, 1991.

[8] Isham V, Mathematical modeling of the transmission dynamics of HIV infection and AIDS : A review, J. R. Statist. Soc. A, Vol. 151, Part 1, 5-30, 1988.

[9] Julia Brauer, Leonard Held, Bruno Ledcr Gerber. Predictive cross-valuation for the choice of linear mixed effects models with application to data from the Swiss HIV cohort study. Biometrics, Vol. 68, 53-61, 2012.

[10] Jun Liu, Yan wang, Luju Liu, Tingting Zhao. A Stochastic model with latent infection and Antiretroviral therapy. Discrete dynamics in nature and society, 2018.

[11] Hymann, Stanley. Mathematical models to understand the AIDS Epidemic. Math. Bio. Sci., Vol. 90,415-473, 1988.

[12] Mode MJ, Stochastic model for the development of an AIDS epidemic. Vol. 107, No. 2, 491-520, 1990.

[13] Mohammed Rafiq, Dhamitru Balenu, Mohammed Shoaib, Mohammed Naveed, Kaleem Ashraf. IET Systems, Bioogy, Vol. 13, No. 6, 2019.

[14] Naresh R, Tripathi A, Omer S, Modeling the spread of AIDS epidemic with vertical transmission. Applied Mathematics and Computation, Vol. 178, 262-272,2006.

[15] Nirav Dalal. Application of Stochastic and ordinary differential equations to HIV dynamics. Unpublished Ph.D.,Thesis, University of Strathylyde. 2006.

[16] Nirav Dalal, David green singh, Xurong Mao. Stochastic model for iternal HIV dynamics. Journal of Mathematical Analysis and applications, Vol. 341, No. 2, 10S4-1101, 2008.

[17] Pelereson AS, Denise EK, RobBoer D, Dynamics of HIV infection of CD4+T cells. Mathematical BioSciences, Vol. 114, 81-125, 1993.

[18] Ron Brookmeyer. Measuring the HIV / AIDS epi- demic : Approaches and challenges. Epidemiol Rev.,Vol. 32, 26-37, 2010.

[19] Samira Khalili, Antonios Armaou, Sensitivity analysis of HIV infection response to treatment via stochastic modeling. Chemical Engineering Science, Vol. 63, 1330-1341, 2008.

[20] Shanmei PMA, Slooot, Rick Quax, Yifan Zhu, We- ing Wang. Complex agent networks explaining the HIV epidemic among homosexual men in Amsterdam. Mathematics and Computer Simulation, Vol. 80, 1018-1030, 2010

[21] Tan WY, Hsu H, Some stochastic models of AIDS spread. Statistics in Medicine, Vol. 8, 121-143, 1989.

[22] Tingting Zhao, Luju Lin, Yan Wan. Discrete Dynamics in Nature and Society, Vol. 2018, Article ID 5175383, 14 pages, 2018.

[23] Verrotta D, Schadeli F, Non linear dynamics models characterizing long term virological data from AIDS clinical trials. Math. Bioscience,Vol. 176, 163-183, 2002.

[24] Wan Yuan T, Stochastic modeling of AIDS epidemiology and HIV pathogenisis. World Scientific Publising Co. Pvt. Ltd., Singapore, Vol. 431, 200

[25] Yan Wang, Ting Ting zhao, Jun Liu. Viral dynamics of an HIV stochastic model with cell-to-cell infection. CTL immune response and distributed delays. Math. Biosci. Eng. 7126-7154, 2019.

[26] Yong Sheng Ding, Min Xu, Liang Jian Hu, Asymp- totic behaviour and stability of a stochastic model for AIDS transmission. Applied Mathematics and Computation, Vol. 204, 99-108, 2008.