

# Effect Of Stochastic Migration On an Hiv/Aids Transmission Model

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## Abstract:

We developed a mathematical model of HIV/AIDs on varying population in [1]. In this paper we considered the effect of stochastic migrating into the susceptible class of the developed model. A system of stochastic ordinary differential equations (SODEs) was then formulated and this was analyzed. Also the Fokker-Planck equation,

$$\frac{\partial P}{\partial t} = - \sum_{i=1}^n \frac{\partial}{\partial x_i} A_i P + \frac{1}{2} \sum_{i=1}^n \sum_{j=1}^n \frac{\partial^2}{\partial x_i \partial x_j} B_{ij} P,$$

was used to transform the system into a system of deterministic partial differential equation. This latter equation was analyzed and it was shown that the stochastic migration has no significant effect on the model.

**Conclusion:** From the analysis of the effect of stochastic migration into the susceptible class of the HIV/AIDS model in [1], several important results emerge. First, when the natural mortality rate is zero—meaning individuals die only from AIDS—the difference between the number of susceptible individuals at time  $t$  and the carrying capacity of the susceptible class remains constant. Second, in the long-term behavior of the system, the susceptible population approaches the carrying capacity, indicating convergence regardless of initial fluctuations. Third, the difference between the susceptible population and its carrying capacity follows a Gaussian distribution if and only if it was Gaussian at the initial time, demonstrating preservation of distributional structure under the model dynamics. Finally, the mean and variance of the susceptible population, expressed as  $E(S)$  and  $\text{Var}(S)$ , respectively, confirm that stochastic migration introduces no significant deviation from the deterministic behavior of the system. Collectively, these findings show that stochastic migration into the susceptible class has no meaningful impact on the overall dynamics of the HIV/AIDS model.

**Key Word:** HIV/AIDS transmission model; Stochastic migration; Stability analysis; Epidemiological Modeling; Planck Equation, Perturbation.

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## 1.0 Introduction

A lot of stochastic models have been developed for HIV/AIDS, some of which include the works by Tuckwell *et al.*, (1998). They developed a simple stochastic mathematical model and investigated for early HIV -1 population dynamics. The model, which was a multi-dimensional diffusion process, included activated uninfected CD4 (+) T-cells, latently and actively infected CD4 (+) T -cells, and free virions occurring in plasma. Stochastic effects were assumed to arise in the process of infection of CD4 (+) T -cells and transitions might occur from uninfected to latently or actively infected cells by chance mechanisms. Using the best currently available parameter values, the intrinsic variability in response to a given initial infection was examined by solving the stochastic system numerically. They estimated the statistical distribution of the time of occurrence and the magnitude of the early peak in viral concentration. The maximum of the viral load had a value in the experimental range and its time of occurrence had a 95% confidence interval from 19.4 to 25.1 days. On the stochastic nature of the growth, they explored the effects of Perturbations in the parameter values in order to assess the additional stochastic effects of between patient variability. They found that changes in the initial number of virions or dose size, the rate at which latently infected CD4 (+) T -cells were converted to the actively infected form and the fraction of latent cells had only minor effects on the size, speed and variability of the response. In contrast, decreased speed and magnitude but

greater variability in response were obtained when the death rate of uninfected CD4 (+) T - cells and the initial number of uninfected activated CD4 (+) T-cells were decreased.

They also determined the distribution of the time to reach a given virion density. From this distribution the probability of detection of the virus as a function of time could be estimated. The numerical results obtained were in the range of experimental values and were discussed in relation to recently proposed detection and testing procedures.

Gallop (2000) presented a stochastic model with sufficient parameters describing the behavior of the epidemic. By embedding non-linear difference equations in the stochastic process in decreasing time, a more thorough understanding of the epidemic was achieved. To visually enhance the investigation of the epidemics behavior, comparison of trajectories of the deterministic model and those computed from the samples Monte Carlo realizations were made. To derive threshold conditions, non-linear differential equations were derived from the non linear equations. Threshold conditions were determined by investigation of the stability of the Jacobean Matrix for the embedded system of nonlinear differential equations. Threshold conditions for the model were formulated and the sensitivities of these conditions were analyzed under slight deviations of the parameter space. Examples of this methodology were provided for application to the HIV/AIDS epidemics in the heterosexual community. Comparison of the behavior of the two modeling structures, stochastic and deterministic, with respect to the threshold conditions was also investigated.

Kesinger (2000), in another paper compared some discrete time deterministic and stochastic models. His presentation was divided into two parts. In the first part, deterministic and stochastic discrete-time SIS (Susceptible-infected-susceptible) and SIR (susceptible-infected-removed) models were analyzed and compared. The stochastic models were markov chains. Models with constant population size and general force of infection were analyzed. Then a more general SIS model with variable population size was analyzed. In the second part of the presentation discrete time models for the solution of infectious diseases in plant pathosystems were analyzed. Deterministic and stochastic models based on the gene-for-gene hypothesis were developed. The evolution of plant maintenance and pathogen virulence was studied.

Sleeman (2000), implemented models accommodating partnerships and heterogeneity with respect to behavioral risk classes and used them to study the evolution of epidemics in various populations. For sexually transmitted diseases with multiple stages like HIV/AIDS, the selection of sexual partners according to disease stage was considered. Computer intensive experimentation was the goal, with a more complete use of latent risk functions and competing risks governing transitions to the infected state than in earlier models. The mathematical structure was used to make connections between the stochastic processes. And a system of non-linear differential equations embedded in the process, thus enabling a search for threshold conditions for the stochastic process. Van den Driessche and Watmough(2002)presented a precise definition of the basic reproduction number,  $R_0$ , for a general compartmental disease transmission model based on a system of ordinary differential equations (ODEs). It was shown that if  $R_0 < 1$ , the disease free equilibrium is locally asymptotically stable where as it is unstable if  $R_0 > 1$ .

Thus  $R_0$  is a threshold parameter for the model. An analysis of the local center manifold yielded a simple criterion for the existence and stability of super - and sub-threshold endemic equilibrium for  $R_0$  near one. This criterion, together with the definition of  $R_0$  was illustrated by treatment, multi-group, staged progression, multi-strain and vector- host models and can be applied to more complex models. The results are significant for disease control

Medlock, (1999) presented a simple deterministic susceptible - infective - removed (SIR) model of HIV transmission in a high-risk population as a system of ordinary differential equations and was analyzed using techniques from dynamical systems. He later added a stochastic migration term to the deterministic model to model variation in the influx of individuals to the susceptible class. This model was then presented as a system of stochastic ordinary differential equations. Fokker Planck equation was used to transform this system to a simple deterministic partial differential equation which was then analyzed. It was then shown that the system is insensitive to small fluctuations in migration. Other useful works relevant to this paper and referred to in this work are [1, 5, 11 and 12].

**2.0 Main Analysis**

**2.1 The Effect of Stochastic Migration to the Susceptible Class on HIV/AIDS Transmission**

Recall that our model in (1) is

$$\left. \begin{aligned}
 \frac{dS}{dt} &= \lambda N - \frac{h_1(\beta_1 - a_1)IS}{N} - \frac{h_3(\beta_3 - a_2)I_C S}{N} - (\mu + \xi + \theta)S \\
 \frac{dI}{dt} &= \frac{h_1(\beta_1 - a_1)IS}{N} + \frac{h_3(\beta_3 - a_2)I_C S}{N} - (\gamma + \mu)I + (1 - k_1)k_2 I \\
 \frac{dI_C}{dt} &= f_1\gamma + \xi S I_C - (\Psi + \mu)I_C \\
 \frac{dI_T}{dt} &= f_2\gamma + \theta S I_T + g\Psi I_C + \omega A - (\alpha + \mu)I_T + (1 - a_3)A \\
 \frac{dA}{dt} &= (1 - f_1 - f_2)\gamma + (1 - g)\Psi I_C + \alpha I_T - (\omega - \mu_A + \mu)A - a_3 A
 \end{aligned} \right\} (2.1)$$

From (2.1), the equation for the susceptible class is

$$\frac{dS}{dt} = \lambda N - \frac{h_1(\beta_1 - a_1)IS}{N} - \frac{h_3(\beta_3 - a_2)I_C S}{N} - (\mu + \xi + \theta)S$$

Note that  $N = S + I + I_C + I_T + A$  and in a pure susceptible class (where there is no infection),

$$I = I_C = I_T = A = 0$$

$$\Rightarrow N = S, \text{ hence, } \frac{dS}{dt} = \lambda N - \eta S = \lambda S^0 - \eta S = \Lambda - \eta S \text{ where } \eta = (\mu + \xi + \theta)$$

$$\therefore \frac{dS}{dt} = \Lambda - \eta S, \text{ where } \eta = (\mu + \xi + \theta) \tag{2.2}$$

**2.2 The Stochastic Migration to the Susceptible Class**

In our model, we assumed that the compartments (population) are closed to migration. That is, when migration into the susceptible class has a stochastic fluctuation. For simplicity, let

$$\Lambda = \lambda S^0 \tag{2.3}$$

Is the migration term hence, in an infinitesimal time interval  $dt$ , the new migration into the susceptible class is

$$\lambda S^0 dt + \lambda S^0 \Psi dW_t \tag{2.4}$$

where  $\eta = (\mu + \xi + \theta) S^0$  is the carrying capacity,  $W_t$  is the Wiener process and  $\Psi > 0$  is the noise coefficient, which determines the effect of the stochastic term. Hence, our model becomes

$$dS(t) = [\lambda(S^0 - S(t)) + (\lambda - \eta)S(t)]dt + \lambda S^0 \Psi dW_t \tag{2.5}$$

For the disease-free equilibrium,  $\lambda = 0$

$$\Rightarrow dS(t) = \lambda[S^0 - (1 + \eta)S(t)]dt + \mu S^0 \Psi dW_t \quad (2.6)$$

Let  $(1 + \eta)S(t) - S^0 = X(t)$

Hence, (2.6) becomes,

$$dX(t) = (1 + \eta)[-X(t) + \lambda S^0 \Psi]dt + \lambda S^0 \Psi dW_t.$$

Let  $\lambda(1 + \eta) = \lambda'$ .

$$\Rightarrow dX(t) = \lambda'X(t)dt + \lambda'S^0\Psi dW_t = -\lambda'[X(t)dt - S^0\Psi dW_t]. \quad (2.7)$$

Let  $S^0\Psi = u_0$ , then (2.7) becomes

$$dX(t) = -\lambda'X(t)dt + \lambda'u_0dW_t. \quad (2.8)$$

### 2.3 Characteristics of the Process

From (1.8) three situations arise and we now discuss them as cases below

Case I:  $\lambda' = 0$

when  $\lambda' = 0$ , X is a constant function of time. If  $\eta = 0$ , it implies that  $S(t) - S^0$  is a constant. This means that the difference between the number of susceptible in time t and the carrying capacity of the susceptible class will be constant when the natural mortality rate is zero (that is, when people die only as a result of AIDS).

Case II:  $u_0 = 0$

When  $u_0 = 0$ , X decays exponentially with time if  $\lambda' > 0$ . This implies that after a long time ( $t \rightarrow \infty$ ), the number of susceptible will be equal to the carrying capacity.

Case III:  $\lambda' \neq 0, u_0 \neq 0$

This is the most interesting scenario. In this case, (2.8) is the well-known Langevin's equation, whose solution is the equally well-known Ornstein-Uhlenbeck's process. The explicit solution of (2.8) is

$$X(t) = X_0 e^{-\lambda't} + \lambda'u_0 \int_0^t e^{-\lambda'(t-s)} dW_s, t \in \mathfrak{R} \quad (2.9)$$

Where  $X_0 = X(0)$  is the value of X at  $t = 0$ .

Given (2.9), three basic observations can be made about the process X.

- (i) Since  $W_t$  is the Wiener process, X is Gaussian if and only if  $X(0)$  is Gaussian. This means that the difference between the number of susceptible and its carrying capacity is Gaussian if and only if it was Gaussian at the beginning (at  $t = 0$ ).
- (ii) If  $E[(X(0))^2] < \infty$ , (that is, if the mathematical expectation of  $X^2(0)$  is finite), then the expectation (mean), variance and covariance functions of X are given by
 
$$E[X(t)] = X_0 e^{-\lambda't}$$

$$V(t) = Var(X(t)) = \frac{\lambda'u_0^2}{2} + \left( V(0) - \frac{\lambda'u_0^2}{2} \right) e^{-2\lambda't} \quad (2.10)$$

$$\rho(s,t) \equiv Cov(X(s), X(t)) = \left[ V(0) - \frac{\lambda'u_0^2}{2} (e^{2\lambda'(t \wedge s)} - 1) \right] e^{-\lambda'(t+s)} \quad (2.11)$$

Where  $t \wedge s = \min(t, s)$

(iii) If  $X(0)$  is Gaussian, with mean zero and covariance  $\rho(s,t) = -\frac{\lambda'u_0^2}{2} e^{-\lambda'|t-s|}$  (2.12)

These results (the characteristics of the process  $X$ ) could be alternatively obtained by using the Fokker-Plank equation as shown in the next subsection.

### 3.0 The Fokker-Plank Equation

The Fokker-Plank equation is a transformation from a system of stochastic ordinary differential equations (S. O. D. Es) in variable  $X$  into a system of deterministic partial differential equations (PDEs) for the distribution for the variable  $X$ . Given the system of  $n$  (S.O.D.Es) written in vector form as:

$$dX = A(X,t)dt + B(X,t)dW_t \quad (3.1)$$

$A$  is the  $n$ -vector of the deterministic terms of the system;  $B$  is the  $n$ -vector of the stochastic terms of the sequence and  $dW_t$  is an  $n$ -dimension Wiener process. The corresponding partial differential equation is the Fokker-Plank equation.

$$\frac{\partial P}{\partial t} = \sum_{i=1}^n \frac{\partial}{\partial x_i} A_i P + \frac{1}{2} \sum_{i=1}^n \sum_{j=1}^n \frac{\partial^2 B_{ij} P}{\partial x_i \partial x_j} \quad (3.2)$$

where  $x_i$  and  $A_i$  are the  $i^{th}$  components of  $X$  and  $A$  respectively and

$$P(x,t / x_0,0) = P[x(t) = x / x(0) = x_0] = \delta(x - x_0), \quad (3.3)$$

with  $\delta$  being the Dirac's delta function.

Now applying Fokker-Plank equation, we have

$$\frac{\partial}{\partial t} = \frac{\partial}{\partial t} (\lambda'xP) + \frac{(\lambda'S^0 + \lambda'u_0)^2}{2} \frac{\partial^2 P}{\partial x^2} \quad (3.4)$$

the initial condition  $P(x,0 / x_0,0) = \delta(x - x_0)$  where  $\delta$  is the Dirac's delta function.

Take the Fourier transform of (1.16) in  $x$  and let  $\phi(s)$  be the Fourier transform,

$$\left[ \phi(s,t) = \int P(x,t) e^{ist} dx \right]$$

Now (3.4) is transformed to

$$\frac{\partial \phi}{\partial t} - i \frac{\partial^2 \phi}{\partial t \partial x} + \frac{(\lambda'S^0 + \lambda'u_0)^2}{2} s^2 \phi = 0 \quad (3.5)$$

the initial condition  $\phi(s,0) = e^{-s} - x_0$ .

Solving this (3.5) we have two ordinary differential equations for the characteristic curves:

$$\left. \begin{aligned} \frac{dt}{dz} &= 1, t(0) = 0 \\ \frac{ds}{dz} &= \lambda' s, s(0) = \zeta \end{aligned} \right\} \quad (3.6)$$

Solving 3.6 by separation of variables gives,

$$\begin{aligned} t &= z \\ s &= \zeta e^{\lambda' z} \end{aligned}$$

where  $z = t$  and  $\zeta = s e^{-\lambda' t}$ .

This yields the ordinary differential equation (ODE)

$$\frac{d\varphi}{dz} + \frac{(\lambda' S^0 + \lambda' \Psi)^2}{2} s^2 \varphi = 0, \varphi(0) = e^{-\zeta x_0}$$

Where  $\varphi$  is known as the characteristic function of P

Substituting  $t = z$  and  $s = \zeta e^{\lambda' z}$

$$\Rightarrow \frac{d\varphi}{dt} + \frac{(\lambda' S^0 \varphi)^2}{2} \zeta - 2\lambda' t \varphi = 0, \varphi(0) = e^{-\zeta x_0}$$

Solving by separation of variables gives,

$$\begin{aligned} \varphi &= \exp\left\{\left[\frac{-(\lambda' S^0 \varphi)^2}{2} \zeta^2 (1 - e^{-2\lambda' z}) - i\zeta x_0\right]\right\} \\ \varphi(s, t) &= \exp\left\{\left[\frac{-(\lambda' S^0 \varphi)^2}{4\lambda'} s^2 e^{-\lambda' t} (1 - e^{-2\lambda' z}) - i s x_0\right] e^{-\lambda' t}\right\} \end{aligned}$$

This is the characteristic function of a Gaussian. Take the inverse Fourier transformation of equation to get

$$P(X, t) = \frac{\lambda'}{\pi(\lambda' S^0 \Psi)^2} \exp\left(\frac{-\lambda'(x - x_0 e^{-\lambda' t})^2}{(\lambda' S^0 \Psi)^2}\right)$$

Hence, X is Gaussian with

$$E[X(t)] = X(0)e^{-\lambda' t} = x_0 e^{-\lambda' t}$$

$$Var[X(t)] = \frac{(\lambda' S^0 \Psi)^2}{2\lambda'} (1 - e^{-2\lambda' t})$$

Taking the first two moments of  $S(t)$  we have

$$\left. \begin{aligned} E[S(t)] &= S^0 + S(0)e^{-\lambda t} \\ \text{Var}[S(t)] &= \frac{(\lambda' S^0 \Psi)^2}{2\lambda'} (1 - e^{-2\lambda t}) \end{aligned} \right\} \quad (3.7)$$

As  $t \rightarrow \infty$  in (3.7) the stationary solution gives,

$$E(S) = S^0$$

$$\text{Var}[S(t)] = \frac{(\lambda' S^0 \Psi)^2}{2\lambda'} = \frac{\lambda'}{2} (S^0 \Psi)^2$$

Since  $E(S)$  does not contain  $\Psi$  (the noise coefficient which determines the effect of the stochastic term), we conclude that the model (system) is insensitive to small fluctuations in migration. The effect of stochastic migration into the susceptible class was considered and it was observed that:

- i. The difference between the number of susceptible in time  $t$  and the carrying capacity of the susceptible class will be constant when people die solely as a result of AIDS. At this point none of the susceptible join the resistant group ( $\eta = 0$ )
- ii. After a long time of the start of AIDS epidemic ( $t \rightarrow \infty$ ), the number of susceptible will be equal to the carrying capacity of the susceptible class.
- iii. The difference between the number of susceptible and its carrying capacity is Gaussian if and only if it was Gaussian at the beginning
- iv. (at  $t = 0$ ).
- v. The mean and Variance of the number of Susceptible as  $t \rightarrow \infty$ , are respectively

$$E(S) = S^0 \text{ and } \text{Var}[X(t)] = \frac{(\lambda' S^0 \Psi)^2}{2\lambda'} = \frac{\lambda'}{2} (S^0 \Psi)^2$$

Where  $E(S)$  is the expected value of  $S$  and  $\text{Var}[S(t)]$ .

We therefore conclude that stochastic migration into the susceptible class has no significant effect on the model.

#### 4.0 Conclusion

From analysis of the effect of stochastic migration to the susceptible class of the HIV/AIDS Model of [1], we make four conclusions thus:

- i. The difference between the number of susceptible in time  $t$  and the carrying capacity of the susceptible class will be constant when the natural mortality rate is zero (that is), when people die only as a result of AIDS.
- ii. After a long time from the start of HIV/AIDS disease, the number of susceptible will be equal to the carrying capacity.
- iii. The difference between the number of susceptible and its carrying capacity is Gaussian if and only if it was Gaussian at the beginning
- iv. The mean and Variance of the number of Susceptible as  $t \rightarrow \infty$ , are respectively

$$E(S) = S^0 \text{ and } \text{Var}[X(t)] = \frac{(\lambda' S^0 \Psi)^2}{2\lambda'} = \frac{\lambda'}{2} (S^0 \Psi)^2 \text{ where } E(S) \text{ is the expected value of } S \text{ and}$$

$\text{Var}[S(t)]$ . We therefore conclude that stochastic migration into the susceptible class has no significant effect on the model.

### References

- [1]. Achugamonu, P.C., Nwobi, F.N. & Inyama, S.C. (2016). Mathematical Model of HIV/AIDS on varying population. African Journal of Mathematics and Computer Science Research.
- [2]. Chukwu, M. and John, S. (2014)The spread of HIV in Africa. Nature.**351**, (581-589).
- [3]. Gallop, R. (2000) Determination of threshold condition for non-linear stochastic Partnership Models for Hetro-sexually Transmitted Disease with Stages. Conference on Stochastic Models in Epidemiology, Purdue.
- [4]. Kesinger, J. (2000) Comparison of some Discrete-Time Deterministic and Stochastic Epidemic Models. Conference on Stochastic Models in Epidemiology, Purdue.
- [5]. Medlock, J. P. (1999)The Effect of Stochastic Migration on an SIR Model for the Transmission of HIV. Georgia Institute of Technology.
- [6]. Obasi, C. (2013) Mathematical model of control measures in the spread of
- [7]. Cholera.M.Sc Thesis Department of Mathematics UNN. unpublished paper.
- [8]. Sleeman, C. K. (2000) Computer Implementation of Stochastic Partnership Models for HIV/AIDS. Conference of Stochastic Models in Epidemiology, Prudue.
- [9]. Tan, W. (2000a) Stochastic Models of HIV Parthenogenesis under HAART and Development of Drug Resistance. Conference on Stochastic Models in Epidemiology, Purdue.
- [10]. Tan, W. (2000b) Stochastic Modeling of AIDS Epidemiology and HIV Pathogenesis. Scientific, Singapore.
- [11]. Tan, W. (2002) Stochastic Models with Application to Genetics, Cancers, AIDS and other Biomedical Systems Series on Concrete and Applicable Mathematics. **4**, World Scientific, New Jersey, 241-246, 337-341.
- [12]. Tuckwell, A. C., and Le Corfec, E. (1998) A Stochastic Model for Early HIV – 1 Population Dynamics, Journal of Theoretical Biology.**195**, 451-463.
- [13]. UNAIDS 2015 Technical Report on HIV/AIDS
- [14]. UNAIDS 2021 Technical Report on HIV/AIDS
- [15]. Vanden Driessche, P., and Watmough .J. (2002) Reproduction Numbers and Sub-Threshold epidemic Equilibrium for compartmental models of diseases transmission. Mathematical Biosciences,**180**, 29-48.