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# The effect of stochastic migration on an HIV/AIDS transmission model.

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**Abstract**

In [3] we developed a mathematical model of the transmission dynamics of HIV/AIDS in Nigeria. In this paper, we consider the effect of stochastic migrating into the susceptible class. A system of stochastic ordinary differential equations (SODEs) was then formulated. This was analyzed. Also the Fokker-Planck equation $\frac{\partial P}{\partial t} = -\sum_{i=1}^{n} \frac{\partial}{\partial x_i} A_i P + \frac{1}{2} \sum_{i=1}^{n} \sum_{j=1}^{n} \frac{\partial^2}{\partial x_i \partial x_j} B_{ij} P$ is 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.

**Keywords:** Stochastic migration, HIV/AIDS, Susceptible, Fokker-Planck equation, SODE

## 1.0 Introduction

A lot of stochastic models have been developed for HIV/AIDS, some of which include the works by Tuckwell and Le Corfec, Gallop, R, Kesinger, J., and Sleeman, C.K.

Tuckwell, H.C. and Lee Corfec, E. [10] 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 virons 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, R. [2] presented a stochastic model with sufficient parameters describing the behavior of the epidemic. By embedding non-linear difference equations in the stochastic process in decrease 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 nonlinear difference 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. Provided were examples of this methodology applied 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 were also investigated.

Kesinger,J.[4] 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, C.K [6] 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 processe. 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, P and Watmough , J. [1] 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, J.P. [5] 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 works in this area can be found in [7,8, 9].

In this paper, we investigate the effect of stochastic migration on the model developed in [3] as presented in (1.1) below.

$$\left.\begin{aligned}
\frac{dS_{ij}}{dt} &= \Lambda - S_{ij}\left(\mu + \lambda_{ij} + \xi_2\right)\\
\frac{dI_{1ij}}{dt} &= S_{ij}\lambda_{ij} - I_{1ij}\left(\mu + \gamma_{1ij}\right)\\
\frac{dI_{2ij}}{dt} &= I_{1ij}\gamma_{1ij} - I_{2ij}\left(\mu + \gamma_{2ij}\right)\\
\frac{dI_{3ij}}{dt} &= I_{2ij}\lambda_{2ij} - I_{3ij}\left(\mu + \gamma_{3ij}\right)\\
\frac{dA_{ij}}{dt} &= I_{3ij}\gamma_{3ij} - \delta A_{ij}\\
\frac{dR}{dt} &= \xi_1 N + \xi_2 S_{ij} + \xi_3 P - \left(\eta + \mu\right)R\\
\frac{dN}{dt} &= \beta' P - \left(\mu + \nu\right)N, \nu = \alpha_2 + \xi_1
\end{aligned}\right\} \qquad (1.1)$$

## 2.0 The stochastic migration

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

$$\Lambda = \mu S^0 \tag{2.1}$$

which is the migration term. Hence in an infinitesimal time interval *dt*, the new migration into the susceptible class is

$$\mu S^0 \mathrm{dt} + \mu S^0 \Psi dW_t \tag{2.2}$$

where $S^0$ 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

$$dS_{ij}(t) = \left[\mu\left(S^0 - S_{ij}(t)\right) - \left(\lambda_{ij} + \xi_2\right) S_{ij}(t)\right]dt + \mu S^0 \Psi dW_t \tag{2.3}$$

For the disease-free equilibrium, $\lambda_{ij} = 0$

$$\Rightarrow \mathrm{dS_{ij}}(t) = \mu\left[S^0 - (1+\xi_2)\,\mathrm{S_{ij}}(t)\right]dt + \mu S^0 \Psi dW_t \tag{2.4}$$

Let $(1+\xi_2)S_{ij}(t) - S^0 = \mathrm{X}(t)$, hence (2.4) becomes

$$d\mathrm{X}(t) = (1+\xi_2)\left[-\mu \mathrm{X}(t) + \mu S^0 \psi dW_t\right]$$

Let $\mu(1+\xi_2) = \mu'$

$$\Rightarrow d\mathrm{X}(t) = -\mu' \mathrm{X}(t)dt + \mu' S^0 \psi dW_t = -\mu'\left[\mathrm{X}(t)dt - S^0 \psi dW_t\right] \tag{2.5}$$

Let $S^0 \psi = u_0$, then (2.5) becomes $d\mathrm{X}(t) = -\mu' \mathrm{X}(t)dt + \mu' u_o dW_t$ (2.6)

## 2.1 Characteristics of the process

From (2.6) three situations arise and we now discuss them.

**Case I:** $\mu' = 0$:

When $\mu' = 0$, X is a constant function of time. If $\xi_2 = 0$, it implies that $S_{ij}(t) - S^0$ is a constant. This means that the difference between the number of susceptibles 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 $\mu > 0$. This implies that after a long time ($t \to \infty$), the number of susceptibles will be equal to the carrying capacity.

**Case III:** $\mu' \neq 0, u_0 \neq 0$:

This is the most interesting scenario. In this case, (2.6) is the well- know ***Langevin's equation,*** whose solution is the equally well-known ***Ornstein-Uhlebeck's process.*** The explicit solution of (2.6) is

$$X(t) = X_0 e^{-\mu' t} + \mu' u_0 \int_0^t e^{-\mu'(t-s)} dW_t, \qquad t \in I\Re \tag{2.7}$$

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

Given (2.7), three basic things can be observed about the process *X*.

(i) Since $W_t$ is the Wiener process, *X* is Guassian if and only if X(0) is Guassian. This means that the difference between the number of susceptibles and its carrying capacity is Guassian iff it was Guassian at the beginning (at $t = 0$)

(ii) If $E[X(0)^2] < \infty$, (that is, if the mathematical expectation of $X(0)^2$ is finite), then the expectation (mean), variance and covariance functions of X are given by

$$E\left[X(t)\right] = X_0 e^{-\mu' t}$$

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

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

where $t \wedge s = \min(t,s)$.

(iii) If X(0) is Guassian, with mean zero and variance $\frac{\mu' u_0^2}{2}$, then $X$ is a stationary Guassian process with mean zero and covariance $\rho(s,t) - \frac{\mu' u_0^2}{2} e^{-\mu'|t-s|}$.

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

## 2.2 The Fokker-Planck Equation

The Fokker-Planck 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 stochastic ordinary differential equations (SODEs) written in vector form as

$$dX = A(X,t)dt + B(X,t)dW_t \qquad (a)$$

where $A$ 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-Planck equation.

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

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

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

the Dirac's delta function.

Now applying Fokker-Planck equation on (2.6) we have

$$\frac{\partial}{\partial t} = \frac{\partial}{\partial t}(\mu x P) + \frac{(\mu S^0 + \mu u_0)^2}{2} \frac{\partial^2 P}{\partial x^2} \qquad (2.8)$$

with 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 (2.8) in $x$ and let $\phi(s)$ be the Fourier transform,

$$\phi(s,t) = \int P((x,t) e^{-ist} dx$$

Now (2.8) is transformed to $\frac{\partial \phi}{\partial x} - i \frac{\partial^2 \phi}{\partial t \partial s} + \frac{(\mu' S^0 + \mu' u_0)^2}{2} s^2 \phi = 0$ with the initial conditions $\phi = (s,0) = e^{-s} - x_0$.

Solving this we have two ordinary differential equations for the characteristic curves

$$\frac{dt}{dz} = 1,\ t(0) = 0,\ \frac{ds}{dz} = \mu s,\ s(0) = \zeta$$

Solving these by separation of variables gives, $t$ = z, s = $\zeta e^{\mu z}$ which is z= t and $\zeta = se^{-\mu t}$. This yields the ODE

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

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

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

Solving this by separation of variables gives

$$\varphi = \exp\left\{\left[\frac{-(\mu' S^0 \varphi)^2}{4\mu'} \zeta^2 (1 - e^{-2\mu' z}) - i\zeta x_0\right]\right\}$$

$$\varphi(s,t) = \exp\left\{\left[\frac{-(\mu' S^0 \varphi)^2}{4\mu'} s^2 e^{-\mu' t} (1 - e^{-2\mu' z}) - isx_0\right] e^{-\mu' t}\right\}$$

This is the characteristic function of a Guassian, take the inverse Fourier transformation to get

$$P(X,t)=\frac{\mu'}{\pi(\mu' S^0\psi)^2}\exp\left(\frac{-\mu'(x-x_0e^{-\mu' t})^2}{(\mu' S^0\psi)^2}\right)$$

Hence, $X$ is Gaussian with $E[X(t)]=X(0)e^{-\mu' t}=x_0e^{-\mu' t}$, $Var[X(t)]=\frac{(\mu' S^0\psi)^2}{2\mu'}(1-e^{-2\mu' t})$. So, $E[S(t)]=S^0+S(0)e^{-\mu' t}$

$Var[S(t)]=\frac{(\mu' S^0\psi)^2}{2\mu'}(1-e^{-2\mu' t})$. As t $\to \infty$, the stationary solution gives $E(S) = S^0$

$$Var\,(S) = \frac{(\mu' S^0\psi)^2}{2\mu'}$$

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.

## 3.0 Summary and Conclusion

The effect of stochastic migration into the susceptible class was considered.
It was observed that:

i) The difference between the number of susceptibles in time t and the carrying capacity of the susceptible class will be constant when people die solely as a result of AIDS, ($\mu$ =0). At this point none of the susceptibles joins the resistant group ($\xi_2$ =0).

ii) After a long time of the start of the AIDS epidemics (t $\to \infty$), the number of susceptibles will be equal to the carrying capacity of the susceptible class.

iii) The difference between the number of susceptibles and its carrying capacity is Guassian if and if it was Guassian at the beginning (at t = 0).

iv) The mean and variance of the number of susceptibles as t $\to \infty$, are respectively $E(S)=S^0, Var(S)=\frac{(\mu' S^0\psi)^2}{2\mu'}$

where E(S) is the expected value of S and Var(S) is the variance of S.

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


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