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HIV/AIDS Model with Delay and the Effects of Stochasticity

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Abstract We present a deterministic HIV/AIDS model with delay. We then extend the model by adjoining terms capturing stochastic effects. The intensity of the fluctuations in the stochastic system is analytically evaluated using Fourier transform methods. We carry out simulations to assess differences in the dynamical behavior of the deterministic and stochastic models. Simulation results show that they are no significant differences in the behavior of the two models.

Keywords HIV/AIDS model · Incubation · Delay · Stochasticity

Mathematics Subject Classifications (2000) 92B34 · 92B60

1 Introduction

Mathematical models, mainly differential equation models, have been used to study the dynamics of diseases [1, 3, 4, 8, 14–16]. Many results have been compiled from

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these models. However, most models incorporate simplifying assumptions which may have an effect on model results when relaxed. In this paper, we assess the effects of variability introduced in populations by factors external to the populations (environmental stochasticity). We revisit the HIV/AIDS model studied in [10]. We add stochastic terms to incorporate variability introduced by a fluctuating environment. In a deterministic situation the difficulty to predict the future of a system accurately increases as we move outside the tightly controlled biochemical, physiological systems and the more complex dynamics of population ecosystems. One reason for this difficulty is that biological systems are subject to apparently random fluctuations. That is, either the state variables themselves or the parameters are perturbed at random times and by random events. Renshaw [12] pointed out that the most natural phenomena do not follow strictly deterministic laws but rather oscillate randomly about some averages so that the deterministic equilibrium is not an absolutely fixed state; instead it is a "fuzzy" value around which the biological system fluctuates. In fact, randomness or stochasticity plays a vital role in the structure and function of biological systems. There are two types of stochasticity: demographic stochasticity and environmental stochasticity [5, 11]. Since the ecological modelling is non-linear and multi-dimensional stochastic processes, the central obstacle to analyze the stochastic model is the lack of mathematical machinery available [9, 17]. A quantum leap in the mathematical sophistication of ecological modelling occurred when May [9] introduced stochastic differential equations to investigate limits to niche overlap in randomly fluctuating environment.

The model classifies the sexually active population into three classes that are: susceptibles, infectives and AIDS cases, with population numbers in each class denoted as functions of time by $S(t)$, $I(t)$ and $A(t)$ respectively. The total sexually interacting adult population is denoted by $N(t) = S(t) + I(t)$. Individuals enter the susceptible class at a constant rate b. The natural death rate is assumed to be proportional to the population number in each class, with rate constant μ. The model assumes a constant emigration rate m of individuals to other countries. There is an AIDS-related death constant ν in the AIDS class. The model assumes standard incidence of the form $\beta cSI/N$ where β is the probability of being infected from a sexual partner and c is the rate at which an individual acquires sexual partners per unit time. The model assumes a constant incubation period τ from the time of being infective to the development of AIDS symptoms. The probability that an individual remains in the incubation period at least t time units before developing AIDS is given by a step function with value 1 for $0 \le t \le \tau$ and value zero for $t > \tau$. The probability that an individual in the incubation period time t units has survived to develop AIDS and did not emigrate is $e^{-(\mu+m)\tau}$. The assumptions result in the following model equations for $t > \tau$,

$$
\begin{aligned}
S'(t) &= b - \beta c \frac{S(t)I(t)}{N(t)} - (\mu + m)S(t), \\
I(t) &= \int_{t-\tau}^{t} \beta c \frac{S(u)I(u)}{N(u)} e^{-(\mu+m)(t-u)} du, \\
A'(t) &= \beta c \frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} e^{-(\mu+m)\tau} - (\mu + \nu)A(t).
\end{aligned} \tag{1}
$$

The initial condition for model system (3) is given as,

$$S(\theta) = \varphi_1(\theta),\ I(\theta) = \varphi_2(\theta),\ A(\theta) = \varphi_3(\theta),\ \theta \in [-\tau, 0], \tag{2}$$

where $\varphi = (\varphi_1, \varphi_2, \varphi_3)^T \in C$ such that $\varphi_i(\theta) = \varphi_i(0) \geq 0$ $(\theta \in [-\tau, 0],\ i = 1, 2, 3)$, $\varphi_2(\theta) \geq 0$ $([-\tau, 0])$, and C denotes the Banach space $C([-\tau, 0], \mathbb{R}^3)$ of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^3$. By a biological meaning, we further assume that $\varphi_i(0) > 0$ for $i = 1, 2, 3$. Model system (1) as a delay differential equation becomes,

$$\begin{aligned}
S'(t) &= b - \beta c\frac{S(t)I(t)}{N(t)} - (\mu + m)S(t),\\
I'(t) &= \beta c\frac{S(t)I(t)}{N(t)} - \beta ck\frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\mu + m)I(t),\\
A'(t) &= \beta ck\frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\nu + \mu)A(t),
\end{aligned} \tag{3}$$

where $k = e^{-(\mu+m)\tau}$. We present the following mathematical results for model system (3) obtain in [10].

Theorem 1 *Let the initial data be $S(\theta) = S_0(\theta) \geq 0$, $I(\theta) = I_0(\theta) \geq 0$, $A(\theta) = A_0 \geq 0$ for all $\theta \in [-\tau, 0)$, with $S_0(0) > 0$, $I_0(0) > 0$, and $A_0(0) > 0$. Then solutions $S(t)$, $I(t)$ and $A(t)$ of system (3) are positive for all $t \geq 0$. For the model system (3), the region $\mathbb{R}$ is positively invariant and all solutions starting in $\mathbb{R}_{+0}$ or $\mathbb{R}_+$ approach, enter or stay in $\mathbb{R}$.*

Model system (3) has a disease-free equilibrium $\mathcal{E}_0 = \left(\frac{b}{m+\mu}, 0, 0\right)$. The spectral radius for model system (3) is given by $\mathcal{R}_0 = \frac{\beta c(1-k)}{(m+\mu)}$.

Theorem 2 *The disease-free equilibrium is locally and globally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Model system (3) has an endemic equilibrium, $\mathcal{E}_e = (S_e, I_e, A_e) = \left(\frac{b(1-k)}{(\mathcal{R}_0-k)(\mu+m)}, S_e(m+\mu)(\mathcal{R}_0 - 1), \frac{bk(\mathcal{R}_0-1)}{(\mathcal{R}_0-k)(\mu+\nu)}\right)$.

Theorem 3 *The endemic equilibrium is locally asymptotically stable if $\mathcal{R}_0 > 1$ and unstable otherwise.*

2 Stochastic Model

The random fluctuating environment always has the effect on the system parameters like death rate, constant input supply, recycling rate, and the conversion efficiency. We incorporate the random fluctuating environment as additive white noise fluctuation to the model system with discrete time delay. We study the intensities of the fluctuation of different populations. In this context we use additive white

noise to the system and calculate the population variance of the system about the non-trivial equilibrium point. The population variances characterize the stochastic stability of the system under consideration.

2.1 Stochastic Model and Variances of the Population

We now extend the delay model Eq. 3 to incorporate the effect of the randomly fluctuating environment on the system. The parameters of the model equations fluctuate about their average values due to random fluctuation of the environment [12]. We incorporate such randomness of the environment to the model Eq. 3 by additive white noises to the model system. Thus model system (3) becomes

$$\begin{aligned}
\frac{dS}{dt} &= b - \beta c\frac{S(t)I(t)}{N(t)} - (\mu + m)S(t) + \alpha_1\xi_1(t),\\
\frac{dI}{dt} &= \beta c\frac{S(t)I(t)}{N(t)} - \beta ck\frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\mu + m)I(t) + \alpha_2\xi_2(t),\\
\frac{dA}{dt} &= \beta ck\frac{S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\mu + \nu)A(t) + \alpha_3\xi_3(t),
\end{aligned} \tag{4}$$

where the random perturbations $\xi_j(t)$, $j = 1, 2, 3$ are assumed to be Gaussian white noise characterized by

$$< \xi_j(t) >= 0 \quad \text{and} \quad < \xi_j(t), \xi_k(t') >= \delta_{jk}\delta(t - t')$$

where $<>$ denotes ensemble average, δ_{jk} is Kronecker delta and $\delta(t - t')$ is the Dirac delta function. Gaussian white noise is a delta-correlated random process and it is very irregular. True white noise does not occur in nature. However, as can be seen by studying their spectra, thermal noise in electrical resistance, the force acting on a Brownian particle and climate fluctuations, disregarding the periodicities of astronomical origin etc. are white to a very good approximation. These examples support the usefulness of the white noise idealization in applications to natural systems. Furthermore, it can also be proved that the process (S, I, A), a solution of Eq. 4, is Markovian if and only if the external noises are white. These results explain the importance and appeal of the white noise idealization [2]. Here we have assumed the Stratonovich interpretation of stochastic differential equations (4), which conserves the ordinary rule of calculus and in this case the stochastic differential equations can be considered as an ensemble of ordinary differential equations [13]. In the deterministic population model (3), randomness has been added on the top of the deterministic dynamics. This is a commonly used approach and there is some limitations and weaknesses of the approach [6, 7]. Therefore, the method of analysis assumes that the system lies always in the vicinity of the equilibrium point $E^*(S^*, I^*, A^*)$, so that the linearized equations of the above system can be used. In the present case we want to evaluate the population variances (or mean-square fluctuations) which characterizes the stochastic stability of the system. We

will employ the method of Fourier transform [11]. We first linearize the above model system (4) around E_* with the perturbations $u_j(t)$, $j = 1, 2, 3$ as follows:

$$\begin{aligned}
\frac{du_1}{dt} &= \Big(-\beta c\frac{I^{*2}}{N^{*2}} - \mu - m\Big)u_1 - \beta c\frac{I^*}{N^*}u_2 + \alpha_1\xi_1(t),\\
\frac{du_2}{dt} &= \beta c\frac{I^{*2}}{N^{*2}}u_1(t) + \beta ck\frac{I^{*2}}{N^{*2}}u_1(t-\tau) + \Big(\beta c\frac{S^{*2}}{N^{*2}} - \mu - m\Big)u_2(t)\\
&\quad - \beta ck\frac{S^{*2}}{N^{*2}}u_2(t-\tau) + \alpha_2\xi_2(t),\\
\frac{du_3}{dt} &= -\beta ck\frac{I^{*2}}{N^{*2}}u_1(t-\tau) - \beta ck\frac{S^{*2}}{N^{*2}}u_2(t-\tau) + (\mu+\nu)u_3(t). \qquad (5)
\end{aligned}$$

One of the important assumption is that the dynamics of the system is analyzed at the vicinity of the equilibrium point E_*. Taking Fourier transform of the above Eq. 5, we obtain [11],

$$\tilde{\xi}(\omega) = L(\omega)\tilde{U}(\omega), \qquad (6)$$

where

$$\tilde{\xi}(\omega) = \begin{pmatrix} \alpha_1\tilde{\xi}_1(\omega)\\ \alpha_2\tilde{\xi}_2(\omega)\\ \alpha_3\tilde{\xi}_3(\omega)\end{pmatrix},\ L(\omega) = \begin{pmatrix} i\omega+\gamma+\delta & \eta & 0\\ -\gamma + k\gamma e^{-i\omega\tau} & i\omega+\delta-\theta+\theta e^{-i\omega\tau} & 0\\ -k\gamma e^{-i\omega\tau} & -k\theta e^{-i\omega\tau} & i\omega+\rho\end{pmatrix},$$
$$\tilde{x}(\omega) = \begin{pmatrix}\tilde{u}_1(\omega)\\ \tilde{u}_2(\omega)\\ \tilde{u}_3(\omega)\end{pmatrix}$$

and γ, δ, η, θ and ρ are as follows: $\gamma = \beta c\frac{I^{*2}}{N^{*2}}$, $\delta = \mu + m$, $\eta = \beta c\frac{I^*}{N^*}$, $\theta = \beta c\frac{S^{*2}}{N^{*2}}$ and $\rho = \mu + \nu$. We assume here that the matrix $L(\omega)$ is a non-singular matrix, then L^{-1} exists, and from Eq. 6, we obtain

$$\tilde{U}(\omega) = L^{-1}(\omega)\tilde{\xi}(\omega) = M(\omega), \tilde{\xi}(\omega) \qquad (7)$$

where $M(\omega) = \big(\frac{1}{DetL}L_{jk}\big)_{3x3}$, L_{jk} are the co-factors of the elements l_{jk} in the $DetL$, $j, k = 1, 2, 3$.

We now describe some preliminary results of the random population function. For a random function $f(t)$ with zero mean, the fluctuation intensity (variance) of $f(t)$ within the frequency interval $[\omega, \omega + d\omega]$ is given by $S_f(\omega)d\omega$ where $S_f(\omega)$ is the spectral density defined by [11],

$$S_f(\omega) = \lim_{T\to\infty}\frac{|f|^2}{T}.$$

The inverse transform of $S_f(\omega)$ is the autocovarince function

$$C_f(\tau') = \frac{1}{2\pi}\int_{-\infty}^{\infty} S_f(\omega)e^{i\omega\tau'}d\omega, \quad \tau' = t - t'.$$

The corresponding variance or fluctuation of $f(t)$ is given by

$$\sigma_f^2 = C_f(0) = \frac{1}{2\pi}\int_{-\infty}^{\infty} S_f d\omega. \qquad (8)$$

From Eq. 7, we have the mean value of the populations

$$\tilde{u}_j = \sum_{k=1}^{3} m_{jk}\tilde{\xi}_k(\omega), \tag{9}$$

where m_{jk}, $j, k = 1, 2, 3$, are the elements of the matrix $M(\omega)$. The spectral densities of u_j ($j = 1, 2, 3$), are given as:

$$S_{u_j} = \sum_{k=1}^{3} \alpha_k \left| m_{jk}(\omega) \right|^2, \quad (j = 1, 2, 3). \tag{10}$$

The variances or fluctuations of u_j, ($j = 1, 2, 3$), are given by [11]

$$\sigma_{u_j}^2 = \frac{1}{2\pi}\int_{-\infty}^{\infty} S_{u_j} d\omega = \frac{1}{2\pi}\sum_{k=1}^{3}\int_{-\infty}^{\infty} \alpha_k |m_{jk}(\omega)|^2 d\omega = \frac{1}{2\pi}\sum_{k=1}^{3}\int_{-\infty}^{\infty} \alpha_k \frac{|L_{jk}(\omega)|^2}{|DetL|^2} d\omega. \tag{11}$$

Since the matrix $L(\omega)$ as well as, the matrix $M(\omega)$ are complex, therefore, $DetL(\omega) = Re(DetL(\omega)) + iIm(DetL(\omega))$ and $L_{jk}(\omega) = Re(L_{jk}(\omega)) + iIm(L_{jk}(\omega))$ and $Re(DetL(\omega)) = \rho[(\gamma+\delta)(\delta-\theta+k\theta\cos\omega\tau) - \omega(\omega - k\theta\sin\omega\tau) + \gamma\eta(1-k\cos\omega\tau)] - \omega[\omega(\delta - \theta + k\theta\cos\omega\tau) + (\gamma+\delta)(\omega - k\theta\sin\omega\tau) + k\gamma\eta\sin\omega\tau]$, $Im(DetL(\omega)) = \rho[\omega(\delta - \theta + k\theta\cos\omega\tau) + (\gamma+\delta)(\omega - k\theta\sin\omega\tau) + k\gamma\eta\sin\omega\tau] + \omega[(\gamma+\delta)(\delta-\theta+k\theta\cos\omega\tau) - \omega(\omega - k\theta\sin\omega\tau) + \gamma\eta(1-k\cos\omega\tau)]$. Thus the variances are as follows ($j = 1, 2, 3$)

$$\sigma_{u_j}^2 = \frac{1}{2\pi}\sum_{k=1}^{3}\int_{-\infty}^{\infty} \alpha_k \frac{(ReL_{jk}(\omega))^2 + (ImL_{jk}(\omega))^2}{(ReDetL)^2 + (ImDetL)^2} d\omega. \tag{12}$$

Thus the variance for $S(t)$ (about its mean value given by Eq. 9) is given by

$$\sigma_{u_1}^2 = \frac{1}{2\pi}\sum_{k=1}^{3}\int_{-\infty}^{\infty} \alpha_k \frac{(ReL_{1k}(\omega))^2 + (ImL_{1k}(\omega))^2}{(ReDetL)^2 + (ImDetL)^2} d\omega, \tag{13}$$

where

$$\begin{aligned}
ReL_{11} &= \rho(\delta - \theta + k\theta\cos\omega\tau) - \omega(\omega - k\theta\sin\omega\tau),\\
ImL_{11} &= \rho(\omega - k\theta\sin\omega\tau) + \omega(\delta - \theta + k\theta\cos\omega\tau),\\
ReL_{12} &= \rho(\gamma - k\gamma\cos\omega\tau) - k\omega\gamma\sin\omega\tau,\\
ImL_{12} &= \gamma(\omega - k\cos\omega\tau + k\rho\sin\omega\tau),\\
ReL_{13} &= k\gamma[\delta\cos\omega\tau + \omega\sin\omega\tau] \text{ and}\\
ImL_{13} &= k\gamma[-\delta\sin\omega\tau + \omega\cos\omega\tau].
\end{aligned}$$

Similarly, the variance for $I(t)$ (about its mean value given by Eq. 9) is given by

$$\sigma_{u_2}^2 = \frac{1}{2\pi}\sum_{k=1}^{3}\int_{-\infty}^{\infty} \alpha_k \frac{(ReL_{2k}(\omega))^2 + (ImL_{2k}(\omega))^2}{(ReDetL)^2 + (ImDetL)^2} d\omega, \tag{14}$$

where

$$
\begin{aligned}
ReL_{21} &= -\eta\rho,\\
ImL_{21} &= \omega\eta,\\
ReL_{22} &= -\omega^2 + (\gamma+\delta)\rho,\\
ImL_{22} &= (\gamma+\delta+\rho)\omega,\\
ReL_{23} &= k[\theta(\gamma+\delta) - \gamma\eta]\cos\omega\tau + k\omega\theta\sin\omega\tau\\
ImL_{23} &= -k[\theta(\gamma+\delta) - \gamma\eta]\sin\omega\tau + k\omega\theta\cos\omega\tau
\end{aligned}
$$

The variance for the population $A(t)$ about its mean value is given by

$$
\sigma^2_{u_3} = \frac{1}{2\pi}\int_{-\infty}^{\infty} \alpha_3 \frac{(ReL_{33}(\omega))^2 + (ImL_{33}(\omega))^2}{(ReDetL)^2 + (ImDetL)^2} d\omega, \tag{15}
$$

where

$$
\begin{aligned}
ReL_{33} &= (\gamma+\delta)(\delta - \theta + k\theta\cos\omega\tau) - \omega(\omega - k\theta\sin\omega\tau) + \gamma\eta(1 - k\cos\omega\tau)\\
ImL_{33} &= (\delta - \theta + k\theta\cos\omega\tau)\omega + (\gamma+\delta)(\omega - k\theta\sin\omega\tau) + k\gamma\eta\sin\omega\tau.
\end{aligned}
$$

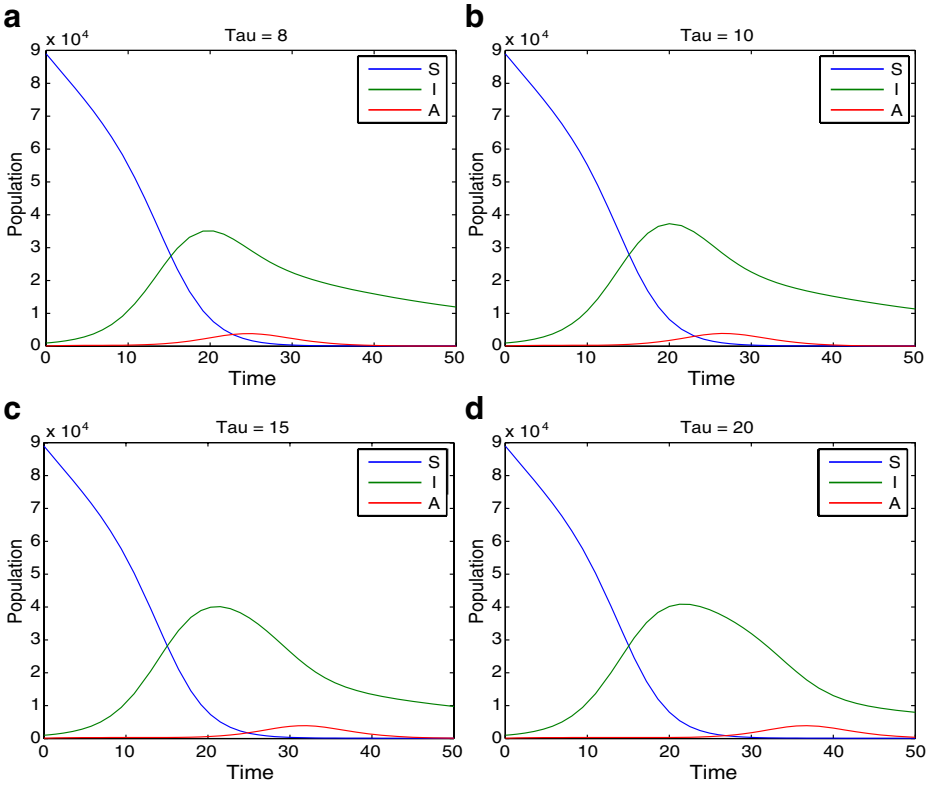

Fig. 1 Simulation of model system (3) showing plots of the susceptibles (S), infectives (I) and AIDS cases (A) for $\tau = 8, 10, 15, 20$ in **a**, **b**, **c**, and **d** respectively with $S(0) = 89{,}000$, $I(0) = 1{,}000$, $A(0) = 100$ on $[-\tau, 0]$ and other parameters values as in Table 1

The above three expressions represent variances or mean-square fluctuations of the populations $S(t)$, $I(t)$ and $A(t)$. One can find the parametric space for which the model system has stable equilibrium where the variances are relatively small. Large values of the variances of the population size give the parametric spaces for which the equilibrium state is unstable in which case the population extinct. This phenomena can be verified both analytically and numerically from the above three expressions.

3 Numerical Simulations

For model system (3), we carry out numerical simulations for the susceptibles (S), infectives (I) and AIDS cases (A) for $\tau = 8, 10, 15, 20$ and the simulation results are depicted in Fig. 1a–d respectively. We also performed the numerical simulations for the susceptibles (S), infectives (I) and AIDS cases (A) for $\tau = 8, 10, 15, 20$ for model system (4) and the simulation results are shown in Fig. 2a–d respectively. Both the numerical simulation results for the deterministic model system (3) and stochastic delay model system (4) illustrate that variation in delay values does not affect the dynamical behaviour of the system. In Fig. 3a–d we explore the strength

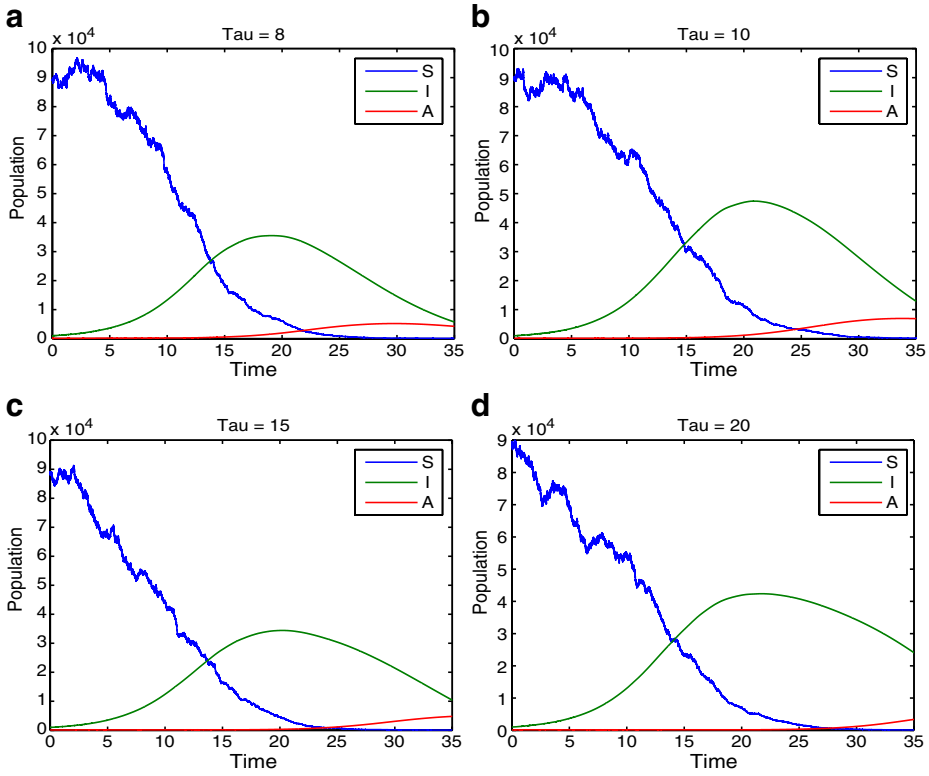

Fig. 2 Simulation of model system (4) showing plots of the susceptibles (S), infectives (I) and AIDS cases (A) for $\tau = 8, 10, 15, 20$ in **a**, **b**, **c**, and **d** respectively with $\alpha_1 = 5, \alpha_2 = 0.02, \alpha_3 = 0.02$, $S(0) = 89{,}000$, $I(0) = 1{,}000$, and $A(0) = 100$ on $[-\tau, 0]$ and other parameters values as in Table 1

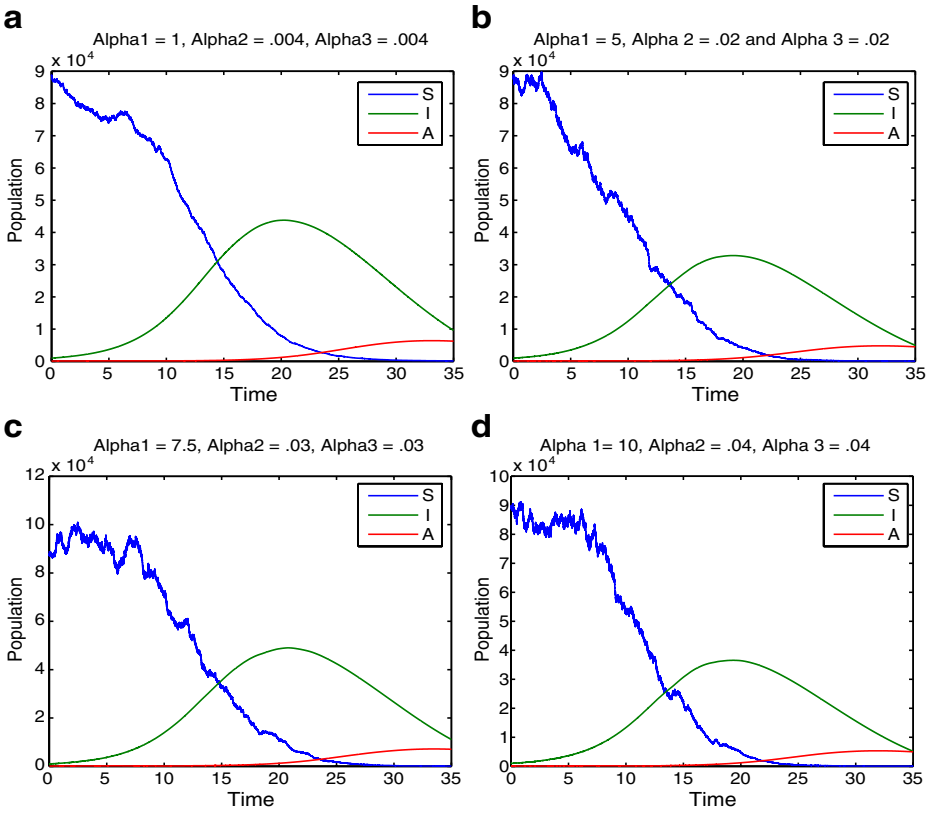

Fig. 3 Simulation of model system (4) showing plots of the susceptibles (S), infectives (I) and AIDS cases (A) for **a** $\alpha_1 = 1$, $\alpha_2 = 0.004$ and $\alpha_3 = 0.004$, **b** $\alpha_1 = 5$, $\alpha_2 = 0.02$ and $\alpha_3 = 0.02$, **c** $\alpha_1 = 7.5$, $\alpha_2 = 0.03$ and $\alpha_3 = 0.03$, **d** $\alpha_1 = 10$, $\alpha_2 = 0.04$ and $\alpha_3 = 0.04$, with $S(0) = 89{,}000$, $I(0) = 1{,}000$, $A(0) = 100$ on $[-\tau, 0]$ and other parameters values as in Table 1

of white noise on the dynamics of the the model and we observe that the change in the strength of the white noise does not affect the dynamical behaviour of the system instead of initial oscillation to the system.

Table 1 Data for the HIV/AIDS model

Parameter	Symbol	Value	Source
Recruitment rate	b	29 yr^{-1}	Estimate
Natural death rate	μ	0.02 yr^{-1}	[10]
AIDS-related death rate	ν	0.333 yr^{-1}	[10]
Emigration rate	m	0.01 yr^{-1}	[10]
Incubation period	τ	8 yr	[10]
Rate of acquiring new sexual partners	c	3 partners/year	[10]
Probability of transmission	β	0.011–0.95	[10]

4 Summary and Concluding Remarks

We have presented a deterministic HIV/AIDS model with a discrete time delay and extended the model to study the effects of random environments. Fourier transform methods were used to evaluate the intensities of the fluctuation of the population stochastic system. Numerical simulations of the deterministic and stochastic delayed model were carried out to assess the differences in the dynamical behaviour in the models. The results show that changes in delay and strength of the white noise does not affect the dynamical behavior of the system. Does this mean that: Introducing susceptible, infected and diseased individuals to a community will not change the dynamics of HIV/AIDS disease as long as the immigrating population behaves (captured by rates of infection) in the same manner as the rest of the community and also with similar demographic characteristics? Demographic stochasticity refers to the variability in population growth rates arising from random differences among individuals in survival and reproduction within a season. This variability will occur even if all individuals have the same expected ability to survive and reproduce and if the expected rates of survival and reproduction do not change from one generation to the next. Even though it will occur in all populations, it is important only in populations that are fairly small.

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