Research Article

The Stochastic Stability of Internal HIV Models with Gaussian White Noise and Gaussian Colored Noise

Xiying Wang¹,¹ Yuanxiao Li,¹ and Xiaomei Wang²

¹College of Science, Henan University of Technology, Zhengzhou 450001, China
²School of Mathematics Science, University of Electronic Science and Technology, Chengdu 610054, China

Correspondence should be addressed to Xiying Wang; wangxiying668@163.com

Received 20 January 2019; Accepted 5 March 2019; Published 19 March 2019

Academic Editor: Vicenç Méndez

Copyright © 2019 Xiying Wang et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

In this paper, the stochastic stability of internal HIV models driven by Gaussian white noise and Gaussian colored noise is analyzed. First, the stability of deterministic models is investigated. By analyzing the characteristic values of endemic equilibrium, we could obtain that internal HIV models reach a steady state under the influence of RTI and PI drugs. Then we discuss the stochastic stability of internal HIV models driven by Gaussian white noise and Gaussian colored noise, based on probability density functions. The functional methods are carried out to derive the approximate Fokker-Planck equation of stochastic internal HIV systems and further obtain the marginal probability density functions. Finally, numerical results show that the noise intensities have a great influence on uninfected cell, infected cell, and virus particles, for predicting the stability of stochastic dynamic systems subjected to Gaussian white noise and Gaussian colored noise.

1. Introduction

Governments and scientists all over the world have been concerning about the epidemic of HIV, with its high speed of spread around the world. As we all know that HIV has caused millions of deaths, and there are some millions of people living with HIV alone [1]. In June 2001, at a special session of the General Assembly on AIDS, world leaders made a commitment to ensure that resources for the global response to HIV/AIDS are substantial, sustained, and geared towards achieving results. As yet, there is no cure for HIV/AIDS. Together with the people of all sections of society, the medical world is working at utmost strain go study on the pathogenesis and properties of epidemic diseases [2, 3].

Mathematical models play a very important role in describing the Immunological response to infection with HIV, and making predictions about their behavior. Early models of HIV infection [4–6] were studied analytically and numerically by defining ordinary differential equations which are deterministic models. There are many authors to investigate how to control and predict HIV virus, based on deterministic HIV models [7–10].

In recent years, some authors [11–13] have added stochastic terms to incorporate variability introduced by a fluctuating environment or others. And Renshaw 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 [14]. In fact, stochasticity plays a vital role in the structure and function of biological systems. Nowadays, stochastic internal HIV systems have been concerned with the study of Gaussian white noise [15–17]. However, there are few studies on internal HIV systems subjected to combined Gaussian white noise and Gaussian colored noise. What is more, Gaussian distributions are not appropriate in some practical cases. Many experimental evidences, particularly in biological virus systems, indicate that most of the noises are not only Gaussian white noise, and there may be Gaussian colored noise or Non-Gaussian noise or others [18]. In this paper, we mainly discuss internal HIV systems subjected to Gaussian white noise and Gaussian colored noise.

The discussions of stochastic systems play a key role especially for those analyses on the basis of characteristics of
Lyapunov exponents, stationary densities, and characteristic function equations [19, 20]. Up to now, the authors [15–17] have proposed many theories and methods to study stochastic HIV systems, and the most important one is about stationary densities which have become an important way to examine basic statistical properties of stochastic systems, such as the stability, chaos, and bifurcation of stochastic systems. FPK method is an effect means to obtain stationary densities of stochastic dynamic systems and is often used in prediction of response process. For instance, Cetto, et al. [21] showed that a closed formula for the effective diffusion coefficient might be used to derive Fokker-Planck equations of the different approximate expressions. Ditlevsen, et al. [22] raised doubt about the validity of the spectral Fokker-Planck equation in its standard formulation and solved the equation with respect to stationary solutions in the particular case where the noise was Cauchy noise and the drift function was polynomial. Until now, stochastic stability of stochastic HIV models excited by Gaussian white noise and Gaussian colored noise, and discussed the fact that when noises are both Gaussian noises, these two cases agree very well [23].

In this paper, the characteristic values of epidemic equilibrium are calculated to consider the stability of HIV deterministic systems. And we mainly derive the approximate Fokker-Planck equation of internal HIV stochastic dynamic systems and get the general expression of their stationary densities. Considering variety of stochastic noise intensities, we analyze the changes of uninfected cell, infected cell, and virus particles, make predictions about the stability of stochastic dynamic systems subjected to Gaussian white noise and Gaussian colored noise, and discuss the fact that when noises are both Gaussian noises, these two cases agree very well [23].

The paper is organized as follows: in Section 2, we briefly review some basic facts about the stability HIV deterministic models which have involved in the concentration of uninfected target cell, infected cell, and virus particles, with the help of the characteristic values of epidemic equilibrium; Section 3 is devoted to derive the approximate Fokker-Planck equation of the HIV system with Gaussian white noise and Gaussian colored noise and further obtain the general expression of their stationary densities; in Section 4, numerical simulations results for the different stochastic noise intensities are carried out to predict about the behavior of the uninfected target cell, infected cell, and virus particles; in Section 5, we will present the conclusions and future directions to close this paper.

2. The Stability Analysis of HIV Deterministic Models

In the early stage of HIV infection, after reverse transcriptase inhibitor (RTI) and protease inhibitor (PI) drugs are given, virus particles are classified as either infections, not influenced, or as non-infection. On the basis of standard internal viral dynamics models [15, 16, 24–26], we will consider the following three-dimensional deterministic models which have involved in the concentration of uninfected target cell $Z_1(t)$, infected cell $Z_2(t)$, and virus particles $Z_3(t)$.

\[
\begin{align*}
\dot{Z}_1(t) &= \lambda - \delta Z_1(t) - (1 - \gamma) \beta Z_1(t) Z_3(t) \\
\dot{Z}_2(t) &= (1 - \gamma) \beta Z_1(t) Z_3(t) - aZ_2(t) \\
\dot{Z}_3(t) &= (1 - \eta) NaZ_2(t) - \mu Z_3(t) - (1 - \gamma) \beta Z_1(t) Z_3(t). \\
\end{align*}
\]

The initial conditions are $Z_1(0) = Z_{10}$, $Z_2(0) = Z_{20}$, $Z_3(0) = Z_{30}$. Here $Z_1(t)$, $Z_2(t)$ and $Z_3(t) \in \mathbb{R}^+$ and all parameters are in $\mathbb{R}^+$. $\lambda$ ($0 < \gamma < 1$) presents the reverse transcriptase inhibitor drug effect and $(1 - \eta)$ ($0 < \eta < 1$) is the protease inhibitor drug effect. The constant $\lambda$ is the total rate of production of healthy cells per unit time, $\delta$ is the per capita death rate of healthy cells, $\beta$ is the transmission coefficient between uninfected cells and infective virus particles, $a$ is the per capital death rate of infected cells, $N$ is the average number of infective virus particles produced by an infected cell in the absence of HAART during its entire infectious lifetime, and $\mu$ presents the per capita death rate of infective virus particles.

The Jacobian matrix for model system (1) is given as

\[
J = \begin{bmatrix}
-\delta - (1 - \gamma) \beta Z_1(t) & 0 & -(1 - \gamma) \beta Z_3(t) \\
(1 - \gamma) \beta Z_3(t) & -a & (1 - \gamma) \beta Z_1(t) \\
-(1 - \gamma) \beta Z_1(t) & (1 - \eta) Na - \mu & -(1 - \gamma) \beta Z_1(t) \\
\end{bmatrix}.
\]

The deterministic modes have been analyzed by Tuckwell et al. [25]. They show that if $R_0 = (1 - \gamma) \beta N \frac{(1 - \eta)}{\delta \mu + \beta \lambda (1 - \gamma)} \leq 1$, then the disease free equilibrium is the unique equilibrium. And if $R_0 = (1 - \gamma) \beta N \frac{(1 - \eta)}{\delta \mu + \beta \lambda (1 - \gamma)} > 1$, as well as the disease free equilibrium, then there is a unique equilibrium $P^0$ given by

\[
P_0 = (Z_1^*, Z_2^*, Z_3^*)
\]

in which

\[
\begin{align*}
Z_1^* &= \frac{\mu}{\beta (1 - \gamma)} \left\{ N (1 - \eta) - 1 \right\} \\
Z_2^* &= \frac{\beta \lambda (1 - \gamma) N (1 - \eta) + \beta \lambda (1 - \gamma) - \delta \mu}{\alpha \beta (1 - \gamma) [N (1 - \eta) - 1]} \\
Z_3^* &= \frac{\beta \lambda (1 - \gamma) N (1 - \eta) + \beta \lambda (1 - \gamma) - \delta \mu}{\beta \mu (1 - \gamma)} \\
\end{align*}
\]

Take parameters $\delta = 0.1 \text{day}^{-1}$, $a = 0.5 \text{day}^{-1}$, $\mu = 5 \text{day}^{-1}$, $\beta = 5 \text{day}^{-1}$, $\gamma = 0.5$, $\eta = 0.5$, $\sigma_1 = 0.5$ and $\sigma_2 = 0.5$, $\beta = 1 \times 10^8 \text{day}^{-1} \text{dm}^3$, $\lambda = 10^7 \text{day}^{-1} \text{dm}^3$, $N = 100$ per cell. The characteristic values of Jacobian matrix for (1) is in equilibrium $P^0$ are $\lambda_1 = -0.3589 + 0.4210i$, $\lambda_2 = -0.3589 - 0.4210i$, $\lambda_3 = -5.6355$. From that, we can see that one of the characteristic values is real number and less than zero, and others are conjugate complex whose real parts are less than zero. Therefore, based on Lyapunov stability’s law, internal viral dynamics models are asymptotically stable, which
Discrete Dynamics in Nature and Society

implies that the system trajectories are ultimately confined to a fixed point. In other words, the HIV deterministic systems reach a steady state under the influence of RTI and PI drugs.

When there is randomness in parameters such as the disease death rate, it is a standard technique to introduce environmental noise into the parameters in this way [14]. Stochastic effects are considered by Gaussian white noise, which is only ideal noise and may not exist in the real word. Therefore, both Gaussian white noise and Gaussian colored noise are investigated to reach on upon uninfected and infected CD4 cells, even CD4 cells and virus particles.

3. Stationary Probability Densities of Internal HIV Models with Gaussian White Noise and Gaussian Colored Noise

The Fokker-Planck equations have played an important role in the investigation of unusual statistic properties of dynamic systems, such as biology systems. In this paper, their statistic characteristics are predicted by deriving the approximate expressions of stationary probability densities for uninfected target cell, infected cell, and virus particles.

The models with Gaussian white noise and Gaussian colored noise in the paper seek to describe the dynamics of HIV-1 rival load during primary infection.

\[
\dot{Z}_1(t) = \lambda - \delta Z_1(t) - (1 - \gamma) \beta Z_1(t) Z_3(t) - \sigma_1 Z_1(t) \xi_1(t) \\
\dot{Z}_2(t) = (1 - \gamma) \beta Z_1(t) Z_3(t) - a Z_2(t) - \sigma_1 Z_2(t) \xi_1(t) \\
\dot{Z}_3(t) = (1 - \eta) Na Z_2(t) - \mu Z_3(t) - (1 - \gamma) \beta Z_1(t) Z_3(t) - \sigma_2 Z_3(t) \xi_2(t)
\]

in which \(\xi_1(t)\) and \(\xi_2(t)\) are dependent, Gaussian white noise and Gaussian colored noises with the intensity of noises \(\sigma_1\) and \(\sigma_2\), respectively, the following statistical properties:

\[
\langle \xi_1(t) \rangle = \langle \xi_2(t) \rangle = 0,
\]

\[
\langle \xi_1(t) \xi_1(t') \rangle = \frac{\sigma_1}{\tau_1} \exp \left[ -\frac{|t - t'|}{\tau_1} \right],
\]

\[
\langle \xi_2(t) \xi_2(t') \rangle = \frac{\sigma_2}{\tau_2} \exp \left[ -\frac{|t - t'|}{\tau_2} \right],
\]

\[
\langle \xi_1(t) \xi_2(t') \rangle = \langle \xi_2(t') \xi_1(t) \rangle = 0,
\]

in which \(\tau_1\) and \(\tau_2\) are the self-correlation time of the noises.

Let \(k_{ij}\) be \(i,j\)th order intensity coefficient; then

\[
k_{11} = \frac{\sigma_1}{\tau_1}, \quad k_{22} = \frac{\sigma_2}{\tau_2}, \quad k_{12} = k_{21} = 0.
\]

In order to derive the approximate Fokker-Planck equation of the HIV driven by Gaussian white noise and Gaussian colored noise, some signs are defined:

\[
Z(t) = (Z_1(t), Z_2(t), Z_3(t)), \\
F(Z(t), t) = (F_1(Z(t), t), F_2(Z(t), t), F_3(Z(t), t)) \\
W(t) = (\xi_1(t), \xi_1(t), \xi_2(t))^T \\
G(Z(t), t)
\]

in which

\[
F_1(Z(t), t) = \lambda - \delta Z_1(t) - (1 - \gamma) \beta Z_1(t) Z_3(t) \\
F_2(Z(t), t) = (1 - \gamma) \beta Z_1(t) Z_3(t) - a Z_2(t) \\
F_3(Z(t), t) = (1 - \eta) Na Z_2(t) - \mu Z_3(t) - (1 - \gamma) \beta Z_1(t) Z_3(t) \\
G_1(Z(t), t) = -\sigma_1 Z_1(t) \\
G_2(Z(t), t) = -\sigma_1 Z_2(t) \\
G_3(Z(t), t) = -\sigma_2 Z_3(t).
\]

Hence we can write (5) as

\[
\dot{Z}(t) = F(Z(t), t) + G(Z(t), t) W(t),
\]

\((t \in T; Z(t_0) = Z_0)\)

Based on above definitions, time-dependent joint probability density function of \(Z(t)\) satisfies the FPK equation.

\[
\frac{\partial}{\partial t} [P(Z,t | Z_0,t_0)] = L_Z [P(Z,t | Z_0,t_0)],
\]
where $L_Z[u]$ is a partial differential operator, defined as

$$L_Z[u] = -\sum_{i=1}^3 \frac{\partial}{\partial Z_i} \left[ f_i(Z, t) u \right] + \frac{1}{2} \sum_{i=1}^3 \sum_{j=1}^3 \frac{\partial^2}{\partial Z_i \partial Z_j} \left[ b_{ij}(Z, t) u \right].$$

(12)

Consider Wong-Zakai’s modification terms, drift coefficient and diffusion coefficient are provided as

$$f_i(Z, t) = F_i(Z, t)$$

$$b_{ij}(Z, t) = 2\pi \sum_{l=1}^3 \sum_{s=1}^3 k_{ls} G_{jl}(Z, t) G_{is}(Z, t),$$

where

$$f_1(Z, t) = F_1(Z, t) - \pi \sigma_1 k_{11} G_{11}(Z, t)$$
$$f_2(Z, t) = F_2(Z, t) - \pi \sigma_2 k_{22} G_{22}(Z, t)$$
$$f_3(Z, t) = F_3(Z, t) - \pi \sigma_1 k_{12} G_{12}(Z, t),$$

$$b_{11}(Z, t) = 2\pi k_{11} G_{11}(Z, t) G_{11}(Z, t)$$
$$b_{12}(Z, t) = 2\pi k_{12} G_{12}(Z, t) G_{22}(Z, t)$$
$$b_{13}(Z, t) = 2\pi k_{13} G_{13}(Z, t) G_{33}(Z, t),$$

$$b_{21}(Z, t) = 2\pi k_{21} G_{12}(Z, t) G_{11}(Z, t)$$
$$b_{22}(Z, t) = 2\pi k_{22} G_{22}(Z, t) G_{22}(Z, t)$$
$$b_{23}(Z, t) = 2\pi k_{23} G_{23}(Z, t) G_{33}(Z, t),$$

$$b_{31}(Z, t) = 2\pi k_{31} G_{13}(Z, t) G_{33}(Z, t)$$
$$b_{32}(Z, t) = 2\pi k_{32} G_{23}(Z, t) G_{22}(Z, t)$$
$$b_{33}(Z, t) = 2\pi k_{33} G_{33}(Z, t) G_{33}(Z, t).$$

(13)

Furthermore, initial condition, boundary condition, and normalization condition of $P(Z, t | Z_0, t_0)$ can be expressed as follows, respectively:

$$\lim_{t \to t_0} P(Z, t | Z_0, t_0) = \delta(Z - Z_0)$$

$$P(Z, t | Z_0, t_0)_{Z_i \to \pm \infty} = 0, \quad (i = 1, 2, 3)$$

$$\int_{-\infty}^{+\infty} P(Z, t | Z_0, t_0) dZ = 1.$$

(15)

For convenience, we define

$$P(Z, t) = \int_{\Omega_0} P(Z, t | Z_0, t_0) P(Z_0, t_0) dZ_0,$$

(16)

where $\Omega_0$ is a defined domain, determined by initial vector $Z_0$.

### 4. Numerical Simulations

In order to illustrate some of the effects of Gaussian white noise and Gaussian colored noise, we numerically solve the deterministic system of differential equations and stochastic system of stochastic differential equations. The parameter values for Figures 1–3 are $\delta = 0.1 \text{day}^{-1}$, $a = 0.5 \text{day}^{-1}$, $\mu = 5 \text{day}^{-1}$, $\gamma = 0.5$, $\sigma_1 = 0.5$ and $\sigma_2 = 0.5$, $\beta = 1 \times 10^{-8} \text{day}^{-1} \text{m}^3$, $\lambda = 10^6 \text{day}^{-1} \text{m}^3$, $N = 100$ per cell. When $\xi_1(t)$ and $\xi_2(t)$ are both Gaussian white noises, the results are consistent with findings in [23].

Figures 1–3 demonstrate the influence of Gaussian white noise and Gaussian colored noise onto the approximate stationary solutions. It is seen from Figure 1 that when the time increases, the number of uninfected target cells $Z_1(t)$ for noiseless conditions increases rapidly and reaches a set point.
level. However, for uninfected target cells \( Z_1(t) \) with noise, the number firstly increases rapidly and then changes in an appropriate range. And compared with the numerical results, we can find that the range is bigger and noise influenced is more intense. Figures 2-3 show that noises have great effect on infected cell \( Z_2(t) \) and virus particles \( Z_3(t) \), respectively. Infected cells and virus particles with noises in number are fewer than ones without noise, which means that Gaussian white noise and Gaussian colored noise are helpful to improve human’s immune system.

When the parameters \( \delta = 0.1 \text{day}^{-1} \), \( \beta = 1 \times 10^{-8} \text{day}^{-1} \text{dm}^3 \), \( \lambda = 10^6 \text{day}^{-1} \text{dm}^3 \), \( N = 100 \) per cell, \( a = 0.5 \text{day}^{-1} \), \( \mu = 5 \text{day}^{-1} \), \( \gamma = 0.5 \), \( \eta = 0.5 \) are fixed, the system factors are considered: one is the intensity of Gaussian white noise \( \sigma_1 \) and the other is the intensity of Gaussian colored noise \( \sigma_2 \).

Figures 4–6 show that when \( \sigma_1 \) and \( \sigma_2 \) take different values, respectively, noises have a great influence on the stationary marginal probability density functions. It is shown from Figure 5 that the peak of the stationary marginal probability density \( P(Z_1) \) appears between \( 0.95 \times 10^6 \) and \( 1.05 \times 10^6 \) and remains unchanged with \( \sigma_1 \) and \( \sigma_2 \) changed, which means that the noise-induced phase transitions do not occur. It is also clear that the decreases in stochastic noise intensity \( \sigma_1 \) can lead to higher peaks of the stationary probability density \( P(Z_1) \). That is, litter intensity values \( \sigma_1 \) lead to larger probability that system will stay close to the equilibrium state. It can be seen from Figures 5-6 that
the probabilities of infected cells and virus particles are decreasing rapidly and reach level no matter whether the parameters $\sigma_1$ and $\sigma_2$ are changed, which means that the infected cells and virus particles are approximate steady state.

5. Conclusions

The internal HIV models subjected to Gaussian white noise and Gaussian colored noise are mainly studied in this article. It is found that the intensities of noises influence greatly not only uninfected target cells, infected cell, and virus particles, but also their stationary marginal probability density functions. And compared with the internal HIV models with Gaussian white noises, systems driven by Gaussian white noise and Gaussian colored noise are more stable and more conform to reality. In our future work, we will need deep-going study on how to control infected cells and virus particles in short time to HIV
systems driven by Gaussian white noise and Gaussian colored noise.

Data Availability
The data used to support the findings of this study are included within the article.

Conflicts of Interest
The authors declare that there are no conflicts of interest regarding the publication of this paper.

Acknowledgments
This work was supported by the National Natural Science Foundation of China (Grant no. 11847081), the Key Scientific Research Projects of Henan Province (Grant nos. 17A110039 and 19A110007), the Fundamental Research Funds for the Henan Provincial Colleges and Universities in Henan University of Technology (Grant no. 2017QNJH18), and High-Level Personal Foundation of Henan University of Technology (no. 2017BS009).

References
[1] S. K. Saxena, S. Tiwari, and M. P. N. Nair, “A global perspective on HIV/AIDS,” Science, vol. 337, no. 6096, p. 798, 2012.
[2] R. A. Littlewood and P. A. Vanable, “A global perspective on complementary and alternative medicine use among people living with HIV/AIDS in the era of antiretroviral treatment,” Current HIV/AIDS Reports, vol. 8, no. 4, pp. 257–268, 2011.
[3] A. Hillmann, M. Crane, and H. J. Ruskin, “HIV models for treatment interruption: adaptation and comparison,” Physica A: Statistical Mechanics and its Applications, vol. 483, pp. 44–56, 2017.
[4] A. S. Perelson and P. W. Nelson, “Mathematical analysis of HIV-1 dynamics in vivo,” SIAM Review, vol. 41, no. 1, pp. 3–44, 1999.
[5] P. W. Nelson and A. S. Perelson, “Mathematical analysis of delay differential equation models of HIV-1 infection,” Mathematical Biosciences, vol. 179, no. 1, pp. 73–94, 2002.
[6] N. M. Dixit and A. S. Perelson, “HIV dynamics with multiple infections of target cells,” Proceedings of the National Academy of Sciences of the United States of America, vol. 102, no. 23, pp. 8198–8203, 2005.
[7] B. G. Williams, S. Gupta, M. Wollmers, and R. Granich, “Progress and prospects for the control of HIV and tuberculosis in South Africa: a dynamical modelling study,” The Lancet Public Health, vol. 2, no. 5, pp. e223–e230, 2017.
[8] X. Liu, H. Wang, Z. Hu, and W. Ma, “Global stability of an HIV pathogenesis model with cure rate,” Nonlinear Analysis: Real World Applications, vol. 12, no. 6, pp. 2947–2961, 2011.
[9] X. Wang, W. Xu I, Y. Cui, and X. I. Wang, “Mathematical analysis of hiv models with switching nonlinear incidence functions and pulse control,” Abstract and Applied Analysis, vol. 2014, Article ID 853960, 8 pages, 2014.
[10] R. F. Stengel, “Mutation and control of the human immunodeficiency virus,” Mathematical Biosciences, vol. 213, no. 2, pp. 93–102, 2008.
[11] D. Jasmina, J. S. Cristiana, and F. M. T. Delfim, “A stochastic SICA epidemic model for HIV transmission,” Applied Mathematics Letters, vol. 84, pp. 168–175, 2018.
[12] A. J. McKane and T. J. Newman, “Stochastic models in population biology and their deterministic analogs,” Physical Review E: Statistical, Nonlinear, and Soft Matter Physics, vol. 70, no. 4, p. 041902, 2004.
[13] X. Wang, X. Liu, W. Xu, and K. Zhang, “Stochastic dynamics of HIV models with switching parameters and pulse control,” Journal of The Franklin Institute, vol. 352, no. 7, pp. 2765–2782, 2015.
[14] G. P. Samanta, “A stochastic two species competition model: nonequilibrium fluctuation and stability,” *International Journal of Stochastic Analysis*, vol. 2011, Article ID 489386, 7 pages, 2011.

[15] Z. Huang, Q. Yang, and J. Cao, “Complex dynamics in a stochastic internal HIV model,” *Chaos, Solitons & Fractals*, vol. 44, no. 11, pp. 954–963, 2011.

[16] H. C. Tuckwell and E. Le Corfec, “A stochastic model for early HIV-1 population dynamics,” *Journal of Theoretical Biology*, vol. 195, no. 4, pp. 451–463, 1998.

[17] N. Dalal, D. Greenhalgh, and X. R. Mao, “A stochastic model of AIDS and condom use,” *Journal of Mathematical Analysis and Applications*, vol. 325, no. 1, pp. 36–53, 2007.

[18] H. G. Solari and M. A. Natiello, “Stochastic population dynamics: the poisson approximation,” *Physical Review E: Statistical, Nonlinear, and Soft Matter Physics*, vol. 67, no. 3, p. 031918, 2003.

[19] Y. Xu, X. Wang, H. Zhang, and W. Xu, “Stochastic stability for nonlinear systems driven by Lévy noise,” *Nonlinear Dynamics*, vol. 68, no. 1-2, pp. 7–15, 2012.

[20] D. Schertzer, M. Larchevêque, J. Duan, V. V. Yanovsky, and S. Lovejoy, “Fractional Fokker-Planck equation for nonlinear stochastic differential equations driven by non-Gaussian Lévy stable noises,” *Journal of Mathematical Physics*, vol. 42, no. 1, pp. 200–212, 2001.

[21] A. M. Cetto, L. De La Peña, and R. M. Velasco, “Approximate Fokker-Planck equation with colored Gaussian noise,” *Physical Review A: Atomic, Molecular and Optical Physics*, vol. 39, no. 5, pp. 2747–2748, 1989.

[22] O. Ditlevsen, “Invalidity of the spectral Fokker-Planck equation for Cauchy noise driven Langevin equation,” *Probabilistic Engineering Mechanics*, vol. 19, no. 4, pp. 385–392, 2004.

[23] N. Dalal, D. Greenhalgh, and X. Mao, “A stochastic model for internal HIV dynamics,” *Journal of Mathematical Analysis and Applications*, vol. 341, no. 2, pp. 1084–1101, 2008.

[24] D. Li and W. Ma, “Asymptotic properties of a HIV-1 infection model with time delay,” *Journal of Mathematical Analysis and Applications*, vol. 335, no. 1, pp. 685–691, 2007.

[25] F. Neri, J. Toivanen, L. Cascella, and Y. Ong, “An adaptive multimeme algorithm for designing HIV multidrug therapies,” *IEEE Transactions on Computational Biology and Bioinformatics*, 2009.

[26] M. S. Ciup, B. L. Bivort, D. M. Bortz et al., “Estimating kinetic parameters from HIV primary infection data through the eyes of three different mathematical models,” *Mathematical Biosciences*, vol. 200, no. 1, pp. 1–27, 2006.
