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Research Article

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

Xiying Wang[1], **Yuanxiao Li**[1] **and Xiaomei Wang**[2]

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

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

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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 based on FPK equation has not been considered.

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 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 the concentration of uninfected target cell $Z_1(t)$, infected cell $Z_2(t)$, and virus particles $Z_3(t)$.

$$\begin{aligned}
\dot{Z}_1(t) &= \lambda - \delta Z_1(t) - (1-\gamma)\beta Z_1(t) Z_3(t) \\
\dot{Z}_2(t) &= (1-\gamma)\beta Z_1(t) Z_3(t) - aZ_2(t) \\
\dot{Z}_3(t) &= (1-\eta) NaZ_2(t) - \mu Z_3(t) \\
&\quad - (1-\gamma)\beta Z_1(t) Z_3(t).
\end{aligned} \tag{1}$$

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 $Z_3(t) \in R^+$ and all parameters are in R^+. $(1-\gamma)$ $((0 < \gamma < 1))$ presents the reverse transcriptase inhibitor drug effect and $(1-\eta)$ $((0 < \eta < 1))$ is the protease inhibitor drug effect. The constant λ is the total rate of production of healthy cells per unit time, δ is the per capita death rate of healthy cells, β 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 μ presents the per capita death rate of infective virus particles.

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

$$J = \begin{bmatrix} -\delta - (1-\gamma)\beta Z_3(t) & 0 & -(1-\gamma)\beta Z_1(t) \\ (1-\gamma)\beta Z_3(t) & -a & (1-\gamma)\beta Z_1(t) \\ -(1-\gamma)\beta Z_3(t) & (1-\eta)Na & -\mu - (1-\gamma)\beta Z_1(t) \end{bmatrix}. \tag{2}$$

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

$$P_0 = (Z_1^*, Z_2^*, Z_3^*) \tag{3}$$

in which

$$\begin{aligned}
Z_1^* &= \frac{\mu}{\beta(1-\gamma)[N(1-\eta)-1]} \\
Z_2^* &= \frac{\beta\lambda(1-\gamma)N(1-\eta) - \beta\lambda(1-\gamma) - \delta\mu}{\alpha\beta(1-\gamma)[N(1-\eta)-1]} \\
Z_3^* &= \frac{\beta\lambda(1-\gamma)N(1-\eta) - \beta\lambda(1-\gamma) - \delta\mu}{\beta\mu(1-\gamma)}
\end{aligned} \tag{4}$$

Take parameters $\delta = 0.1day^{-1}$, $a = 0.5day^{-1}$, $\mu = 5day^{-1}$, $\gamma = 0.5$, $\eta = 0.5$ $\sigma_1 = 0.5$ and $\sigma_2 = 0.5$, $\beta = 1 \times 10^{-8} day^{-1}dm^3$, $\lambda = 10^7 day^{-1}dm^3$, $N = 100$ per cell. The characteristic values of Jacobian matrix for (1) which 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

implies that the system trajectories are ultimately confided 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.

$$\begin{aligned}
\dot{Z}_1(t) &= \lambda - \delta Z_1(t) - (1-\gamma)\beta Z_1(t) Z_3(t) \\
&\quad - \sigma_1 Z_1(t)\xi_1(t) \\
\dot{Z}_2(t) &= (1-\gamma)\beta Z_1(t) Z_3(t) - aZ_2(t) \\
&\quad - \sigma_1 Z_2(t)\xi_1(t) \\
\dot{Z}_3(t) &= (1-\eta) NaZ_2(t) - \mu Z_3(t) \\
&\quad - (1-\gamma)\beta Z_1(t) Z_3(t) - \sigma_2 Z_3(t)\xi_2(t).
\end{aligned} \tag{5}$$

Where $\xi_1(t)$ and $\xi_2(t)$ are dependent, Gaussian white noise and Gaussian colored noises with the intensity of noises σ_1 and σ_2, respectively, the following statistical properties:

$$\begin{aligned}
\langle \xi_1(t) \rangle &= \langle \xi_2(t) \rangle = 0, \\
\langle \xi_1(t)\xi_1(t') \rangle &= \frac{\sigma_1}{\tau_1} \exp\left[-\frac{|t-t'|}{\tau_1} \right], \\
\langle \xi_2(t)\xi_2(t') \rangle &= \frac{\sigma_2}{\tau_2} \exp\left[-\frac{|t-t'|}{\tau_2} \right], \\
\langle \xi_1(t)\xi_2(t') \rangle &= \langle \xi_2(t')\xi_1(t) \rangle = 0,
\end{aligned} \tag{6}$$

in which τ_1 and τ_2 are the self-correlation time of the noises.

Let k_{ij} be $ijth$ order intensity coefficient; then

$$\begin{aligned}
k_{11} &= \frac{\sigma_1}{\tau_1}, \\
k_{22} &= \frac{\sigma_2}{\tau_2}, \\
k_{12} &= k_{21} = 0.
\end{aligned} \tag{7}$$

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:

$$\begin{aligned}
&Z(t) = (Z_1(t), Z_2(t), Z_3(t)), \\
&F(Z(t),t) = (F_1(Z(t),t), F_2(Z(t),t), F_3(Z(t),t)) \\
&W(t) = (\xi_1(t), \xi_1(t), \xi_2(t))^T \\
&G(Z(t),t) \\
&= \begin{bmatrix} G_1(Z(t),t) & 0 & 0 \\ 0 & G_2(Z(t),t) & 0 \\ 0 & 0 & G_3(Z(t),t) \end{bmatrix}
\end{aligned} \tag{8}$$

in which

$$\begin{aligned}
F_1(Z(t),t) &= \lambda - \delta Z_1(t) - (1-\gamma)\beta Z_1(t) Z_3(t) \\
F_2(Z(t),t) &= (1-\gamma)\beta Z_1(t) Z_3(t) - aZ_2(t) \\
F_3(Z(t),t) &= (1-\eta) NaZ_2(t) - \mu Z_3(t) \\
&\quad - (1-\gamma)\beta Z_1(t) Z_3(t), \\
G_1(Z(t),t) &= -\sigma_1 Z_1(t) \\
G_2(Z(t),t) &= -\sigma_1 Z_2(t) \\
G_3(Z(t),t) &= -\sigma_2 Z_3(t).
\end{aligned} \tag{9}$$

Hence we can write (5) as

$$\dot{Z}(t) = F(Z(t),t) + G(Z(t),t) W(t), \quad (t \in T;\ Z(t_0) = Z_0) \tag{10}$$

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

$$\frac{\partial [P(Z,t \mid Z_0,t_0)]}{\partial t} = L_Z [P(Z,t \mid Z_0,t_0)], \tag{11}$$

where $L_Z[u]$ is a partial differential operator, defined as

$$L_Z[u] = -\sum_{i=1}^{3} \frac{\partial [f_i(Z,t)\,u]}{\partial Z_i} + \frac{1}{2}\sum_{i=1}^{3}\sum_{j=1}^{3} \frac{\partial^2 [b_{ij}(Z,t)\,u]}{\partial Z_i \partial Z_j}. \quad (12)$$

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

$$\begin{aligned} f_i(Z,t) &= F_i(Z,t) \\ &\quad + \pi \sum_{j=1}^{3}\sum_{l=1}^{3}\sum_{s=1}^{3} k_{ls} G_{jl}(Z,t) \frac{\partial}{\partial Z_j} G_{is}(Z,t) \\ b_{ij}(Z,t) &= 2\pi \sum_{l=1}^{3}\sum_{s=1}^{3} k_{ls} G_{il}(Z,t)\, G_{js}(Z,t), \end{aligned} \quad (13)$$

where

$$\begin{aligned} f_1(Z,t) &= F_1(Z,t) - \pi\sigma_1 k_{11} G_{11}(Z,t) \\ f_2(Z,t) &= F_2(Z,t) - \pi\sigma_1 k_{22} G_{22}(Z,t) \\ f_3(Z,t) &= F_3(Z,t) - \pi\sigma_2 k_{33} G_{33}(Z,t), \\ b_{11}(Z,t) &= 2\pi k_{11} G_{11}(Z,t)\, G_{11}(Z,t) \\ b_{12}(Z,t) &= 2\pi k_{12} G_{11}(Z,t)\, G_{22}(Z,t) \\ b_{13}(Z,t) &= 2\pi k_{13} G_{11}(Z,t)\, G_{33}(Z,t), \\ b_{21}(Z,t) &= 2\pi k_{21} G_{22}(Z,t)\, G_{11}(Z,t) \\ b_{22}(Z,t) &= 2\pi k_{22} G_{22}(Z,t)\, G_{22}(Z,t) \\ b_{23}(Z,t) &= 2\pi k_{23} G_{22}(Z,t)\, G_{33}(Z,t), \\ b_{31}(Z,t) &= 2\pi k_{31} G_{22}(Z,t)\, G_{11}(Z,t) \\ b_{32}(Z,t) &= 2\pi k_{32} G_{33}(Z,t)\, G_{22}(Z,t) \\ b_{33}(Z,t) &= 2\pi k_{33} G_{33}(Z,t)\, G_{33}(Z,t). \end{aligned} \quad (14)$$

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

$$\begin{aligned} &\lim_{t \longrightarrow t_0} P(Z,t \mid Z_0,t_0) = \delta(Z - Z_0) \\ &P(Z,t \mid Z_0,t_0)_{Z_i \longrightarrow \pm\infty} = 0, \quad (i = 1,2,3) \\ &\int_{-\infty}^{+\infty} P(Z,t \mid Z_0,t_0)\, dZ = 1. \end{aligned} \quad (15)$$

For convenience, we define

$$P(Z,t) = \int_{\Omega_0} P(Z,t \mid Z_0,t_0)\, P(Z_0,t_0)\, dZ_0, \quad (16)$$

where Ω_0 is a defined domain, determined by initial vector Z_0.

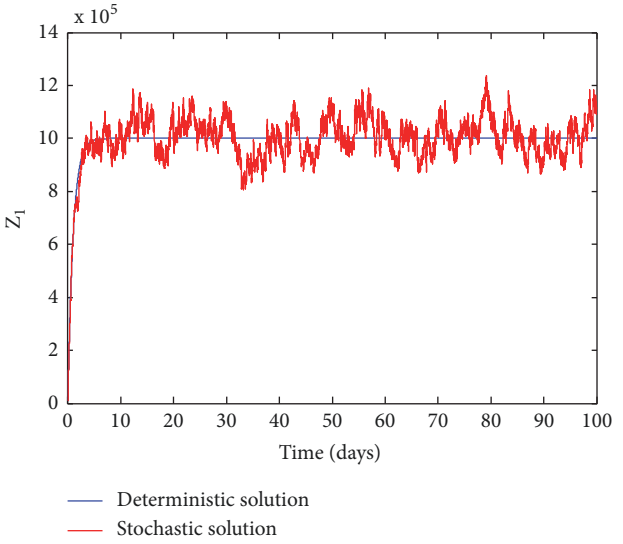

Figure 1: The approximate stationary solutions of Z_1 obtained for equations (1) and (5) with $\sigma_1 = 0.5$ and $\sigma_2 = 0.5$.

When $t \longrightarrow \infty$, $P(Z,t)$ is independent of time and satisfies reduced FPK equation.

$$\begin{aligned} &L_Z[P(Z,t)] = 0 \Longrightarrow \\ &-\sum_{i=1}^{3} \frac{\partial [f_i(Z,t)\, P(Z,t)]}{\partial Z_i} \\ &+ \frac{1}{2}\sum_{i=1}^{3}\sum_{j=1}^{3} \frac{\partial^2 [b_{ij}(Z,t)\, P(Z,t)]}{\partial Z_i \partial Z_j} = 0. \end{aligned} \quad (17)$$

By utilizing (17), the marginal probability density function of $Z_i (i = 1,2,3)$ can be determined as

$$P(Z_i,t) = \int_{-\infty}^{+\infty} P(Z,t)\, dZ_i, \quad (18)$$

where $Z_i = [Z_1, \ldots, Z_{i-1}, Z_{i+1}, \ldots, Z_n]$, $n = 3$.

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 $\delta = 0.1 day^{-1}$, $a = 0.5 day^{-1}$, $\mu = 5 day^{-1}$, $\gamma = 0.5$, $\eta = 0.5$ $\sigma_1 = 0.5$ and $\sigma_2 = 0.5$, $\beta = 1 \times 10^{-8} day^{-1} dm^3$, $\lambda = 10^6 day^{-1} dm^3$, $N = 100$ 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

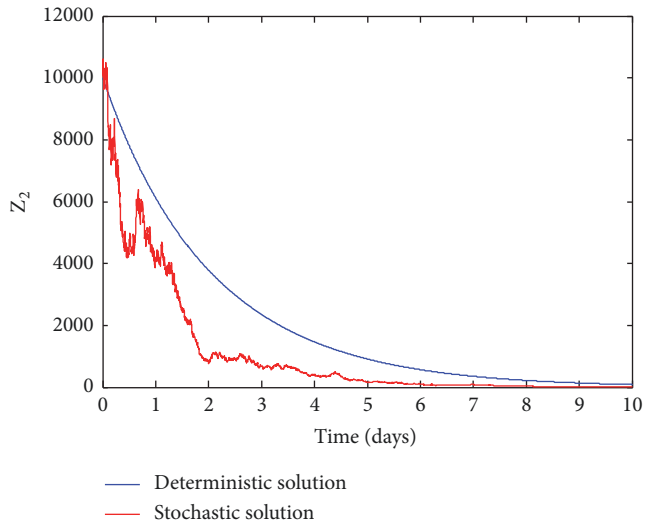

FIGURE 2: The approximate stationary solutions of Z_2 obtained for equations (1) and (5) with $\sigma_1 = 0.5$ and $\sigma_2 = 0.5$.

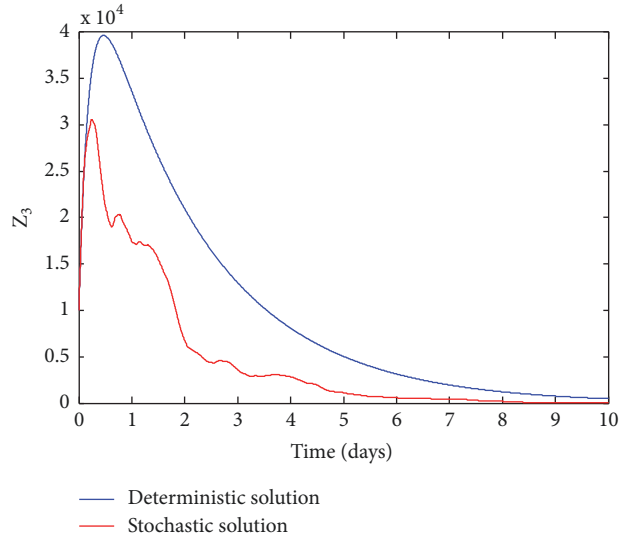

FIGURE 3: The approximate stationary solutions of Z_3 obtained for equations (1) and (5) with $\sigma_1 = 0.5$ and $\sigma_2 = 0.5$.

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 $\delta = 0.1day^{-1}, \beta = 1 \times 10^{-8}day^{-1}dm^3, \lambda = 10^6day^{-1}dm^3, N = 100$ per cell, $a = 0.5day^{-1}, \mu = 5day^{-1}, \gamma = 0.5, \eta = 0.5$ are fixed, the system factors are considered: one is the intensity of Gaussian white noise σ_1 and the other is the intensity of Gaussian colored noise σ_2.

Figures 4–6 show that when σ_1 and σ_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 0.95×10^6 and 1.05×10^6 and remains unchanged with σ_1 and σ_2 changed, which means that the noise-induced phase transitions do not occur. It is also clear that the decreases in stochastic noise intensity σ_1 can lead to higher peaks of the stationary probability density $P(Z_1)$. That is, litter intensity values σ_1 lead to larger probability that system will stay close to the equilibrium state. It can be seen from Figures 5-6 that

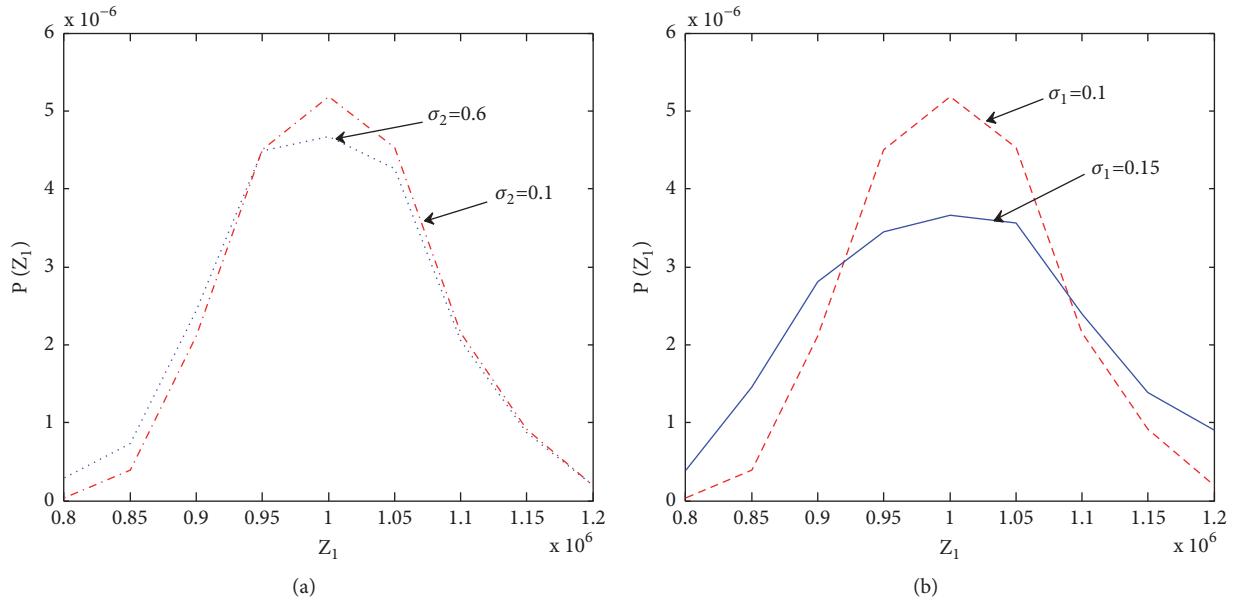

FIGURE 4: The stationary marginal probability density of uninfected target cells $Z_1(t)$. (a) $\sigma_1 = 0.1$ is fixed with $\sigma_2 = 0.1$ and $\sigma_2 = 0.6$. (b) $\sigma_2 = 0.1$ is fixed with $\sigma_1 = 0.1$ and $\sigma_1 = 0.15$.

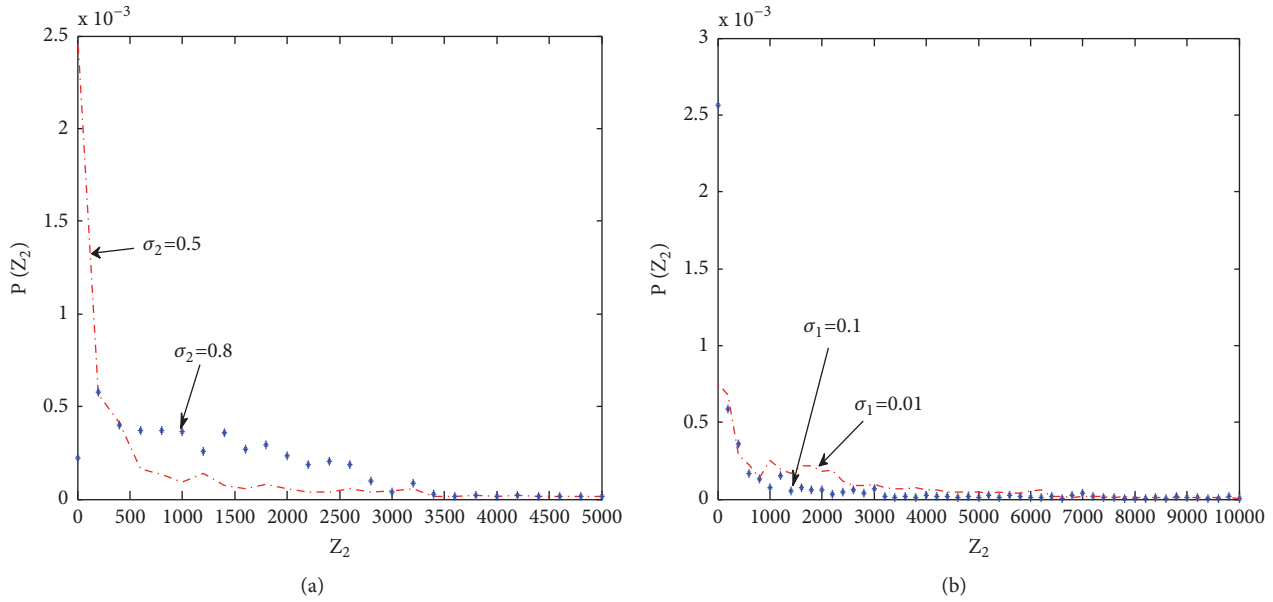

FIGURE 5: The stationary marginal probability density of infected target cells $Z_2(t)$. (a) $\sigma_1 = 0.08$ is fixed with $\sigma_2 = 0.5$ and $\sigma_2 = 0.8$. (b) $\sigma_2 = 0.5$ is fixed with $\sigma_1 = 0.1$ and $\sigma_1 = 0.01$.

the probabilities of infected cells and virus particles are decreasing rapidly and reach level no matter whether the parameters σ_1 and σ_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

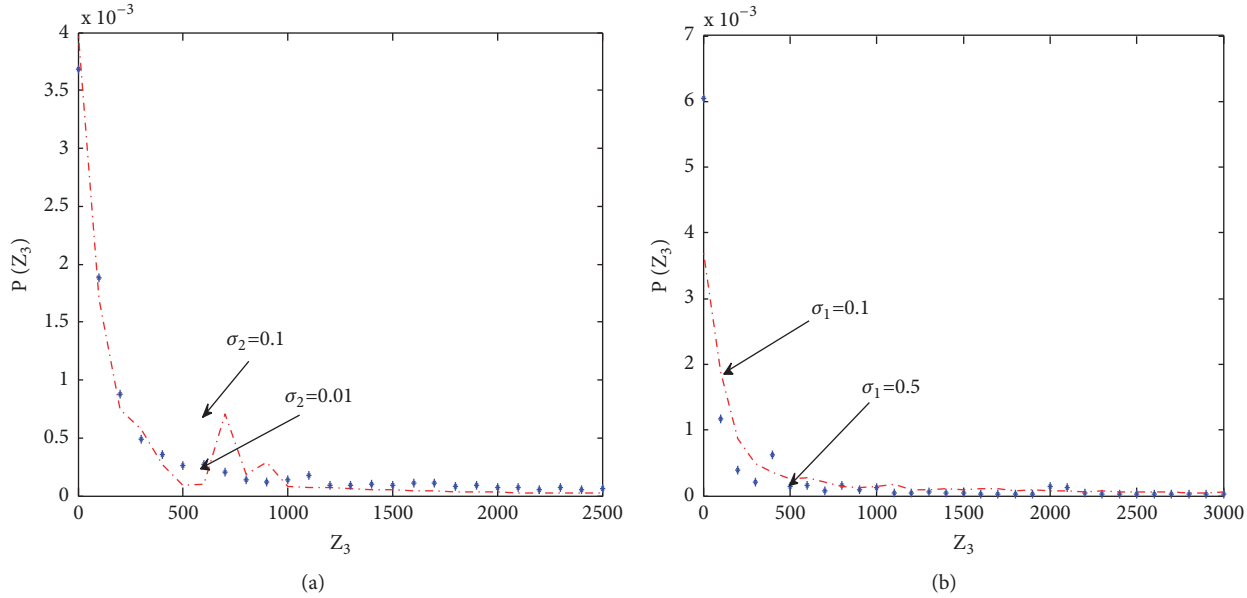

FIGURE 6: The stationary marginal probability density of virus particles $Z_3(t)$. (a) $\sigma_1 = 0.1$ is fixed with $\sigma_2 = 0.1$ and $\sigma_2 = 0.01$. (b) $\sigma_2 = 0.01$ is fixed with $\sigma_1 = 0.1$ and $\sigma_1 = 0.5$.

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.

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