

Dynamics of an HIV/AIDS transmission model with protection awareness and fluctuations

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Abstract

We establish a stochastic HIV/AIDS model involving the susceptible with protection awareness within a total population. The mechanism of dynamic behaviors of the stochastic HIV/AIDS model with protection awareness are investigated in order to control the spread of HIV/AIDS. We firstly show that the stochastic model admits a global positive solution with any initial positive values. By constructing Lyapunov functions, the ergodic stationary distribution under the condition $R_0^s > 1$, and the extinction under the condition $R_0^e < 1$ for the stochastic model are further obtained respectively. Moreover, by using positive preserving truncated Euler-Maruyama method (PPTEM), the related numerical simulations are performed, which demonstrate the quantitative properties of persistence and extinction of the solution. Precisely, the increasing of protection efficiency of the susceptible with

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protection awareness reduces the scale of the infected individuals with AIDS; and the continuous antiretroviral therapy (ART) also benefits the control of the scale of the infected individuals with HIV/AIDS; the media reports with more instructions and details such as taking post-exposure prophylaxis (PEP) within 72 hours to help the individuals avoid the infection to meet the 90%-90%-90% plan of WHO.

Key words: HIV/AIDS infection, protection awareness, stationary distribution, extinction

1. Introduction

Infectious diseases caused one-quarter of the global deaths [1]. Various factors such as media campaigns, population migration and temperature changes influenced the spreading of infectious diseases. Since June 6 of 1981, the first global case of HIV (Human Immunodeficiency Virus) infection was announced, human beings have been fighting against HIV for over four decades [2]. As of the year 2022, over 37.7 million individuals have been infected with HIV [3]. As we have known today, the transmission of HIV took place through blood, semen, cervical (or, vaginal secretions) and breast milk as well. Especially, an infected individual was unaware of the protection and was lack of active treatment, HIV often broke down the immune system of the infected individual and eventually turned into the acquired immune deficiency syndrome (AIDS). Although there was no drug or vaccine for HIV, the antiretroviral therapy (ART) could prolong the life expectancy of an infected individual and make it approach that of the uninfected individuals. Meanwhile, the infected individuals with ART treatment do not retransmit

HIV to their sexual partners [3]. From 2000 to 2018, the number of new HIV infected individuals fell by 37%, HIV-related deaths fell by 45%, and ART treatment saved 13.6 million individuals. At the end of 2018, over 23.3 million individuals had received ART treatment [3]. Thus, UNAIDS puts forward the 90%-90%-90% plan (90% of all people living with HIV know their HIV status, 90% of all people with diagnosed HIV infection receive sustainable treatment, and 90% of all people receiving treatment have viral load suppression), and sets the great goal of eliminating the AIDS epidemic by 2030 in General Assembly of the United Nations [4].

Mathematical models of HIV/AIDS and their kinetic behavior analysis can well predict the development trend of HIV/AIDS. Many scholars have already studied HIV/AIDS models and their kinetic behaviors. For example, Silva and Torres [5] obtained the results on the global stability of the HIV/AIDS model by considering bilinear incidence rates. Ghosh et al. [6] studied the effect of medias and self-imposed psychological fears on disease dynamics by separating the susceptible into the unaware and the aware individuals. Later, Zhao et al. [7] modified the model established by Fatmawati et al. [8], and considered piecewise fractional differential equations and investigated the effect of protection awareness towards the HIV transmission. They neglected the random contacts of MSM population through the mobilities of MSM population among distinct sites, which were still the major mechanisms of spread of HIV/AIDS at present. While indeed the random contacts of MSM population often depended on the scale of MSM population. Therefore, in this paper, we assumed that the HIV/AIDS transmissions were inevitably affected by random contacts of MSM population. Mathematically,

we supposed that the HIV/AIDS model was fluctuated by the white noises in the real circumstances. In the recent contributions, the epidemic models with fluctuations were chosen to investigate their long-term dynamics. For instance, Mao et al. [9] found that the small fluctuations to the deterministic models effectively suppress the rapid increment of the population. Other recent epidemic models in [10–17] also governed the fluctuations to describe the diversities of their models. More precisely, [16, 17] found that small fluctuations produced the long-term persistence, and large fluctuations led to the extinction of infectious diseases in stochastic HIV/AIDS models. Liu [18] discovered that the higher order fluctuations made HIV/AIDS eradicated under sufficient conditions. Meanwhile, Wang [19] also figured out the extinction and persistence in the mean depended on the fluctuations of main parameters.

We formulate a stochastic HIV/AIDS model with protection awareness by considering the environmental noises into the model of Fatmawati et al. [8]. We next provide the expression of the basic reproduction number for the deterministic model. In Section 3, we prove theoretically that there exists a unique positive solution to the stochastic model, and the existence of a unique ergodic stationary distribution is investigated. Further, we give a new threshold R_0^e for the extinction of HIV/AIDS, and the corresponding numerical simulations are demonstrated to verify the theoretical results. Then we conclude that the detailed publicity has great impact on the control of AIDS compared with the extensive publicity. Moreover, the continuous antiretroviral therapy (ART) is helpful to control the number of the individuals with HIV/AIDS. Meanwhile, we figure out the main improvements compared

with other contributions, and give some suggestions to control the long-term dynamics of HIV/AIDS.

2. Establishment of the mathematical model

The number that an infected contacts with the susceptible per unit of time is called the contact number, which is usually related to the total population N and denoted as $U(N)$. Let the probability of infection per exposed individual be β_0 , and $\beta_0 U(N)$ is called the effective exposure number, which represents the infectivity of an infected individual per unit of time. The total population is usually separated by the susceptible individuals, the immune individuals, and the exposed individuals. So, the proportion of the susceptible individuals in the whole population is $\frac{S(t)}{N(t)}$, which is not being infected by the infected individuals. Then, the number of the susceptible being infected effectively at time t is

$$\beta_0 U(N) \frac{S(t)}{N(t)} I(t)$$

which is called the incidence rate. We assume in this paper that the contact rate between the susceptible and the infected is proportional to the total population, i.e., $U(N) = kN(t)$. Let $\beta = \beta_0 k$, then the incidence rate is rewritten as $\beta S(t)I(t)$, which is called the bilinear incidence rate. Many researchers have applied the bilinear incidence rates into their HIV/AIDS models [20–23] for further discussions.

Fatmawati et al. [8] developed an HIV/AIDS model within a total population, where the susceptible with the protection awareness was concerned. Precisely, they separated the total population into five different groups: the susceptible without protection awareness (short for S_u), the susceptible with

protection awareness (short for S_a), the infected without ART treatment (short for I), the infected with ART treatment (short for C) and the infected who eventually developed into AIDS (short for A) therein. Moreover, the total people size N was expressed as

$$N = S_a + S_u + I + C + A.$$

Nowadays, the fast and prompt public transportation brings the random contacts of MSM population more frequently, based on recent work by Fatmawati et al. [8], we establish an HIV/AIDS model under the bilinear incidence scenario consisting of five equations in this paper:

$$\begin{aligned} \dot{S}_u(t) &= \Lambda - \beta S_u I - (\alpha + \mu) S_u, \\ \dot{S}_a(t) &= \alpha S_u - (1 - \varepsilon) \beta S_a I - \mu S_a, \\ \dot{I}(t) &= \beta I (S_u + (1 - \varepsilon) S_a) + \eta C + \nu A - (\rho + \gamma + \mu) I, \\ \dot{C}(t) &= \rho I - (\eta + \mu) C, \\ \dot{A}(t) &= \gamma I - (v + \delta + \mu) A, \end{aligned} \quad (2.1)$$

with the positive initial values

$$\begin{aligned} S_u(0) = S_{u0} \geq 0, S_a(0) = S_{a0} \geq 0, I(0) = I_0 \geq 0, \\ C(0) = C_0 \geq 0, A(0) = A_0 \geq 0. \end{aligned}$$

where Λ is a constant recruitment rate within the total population, β is the transmission rate per capita for each new HIV infected individual, and ε is the protection efficiency of S_a , μ is the natural death rate of the total population, α is the conversion rate from S_u to S_a , the conversion rates η , ν ,

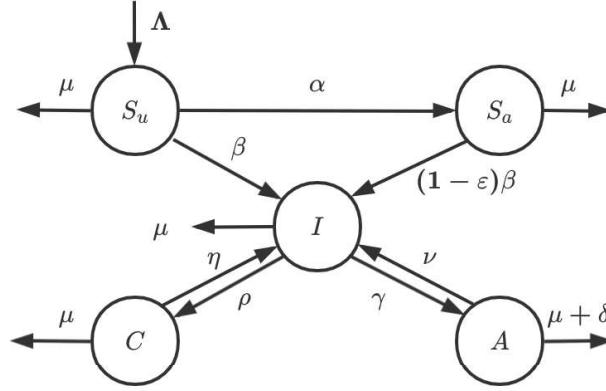


Figure 2.1 Transfer mechanism of model (2.1)

γ , and ρ respectively represent the conversion rates from C to I , A to I , I to A , and I to C ; and δ is the case-fatality rate of AIDS. Moreover, The mutual transfer mechanisms of each group in model (2.1) is demonstrated clearly in Figure 2.1. Let us add five equations of model (2.1) and then one gets that

$$\dot{N}(t) = \Lambda - \mu N - \delta A \leq \Lambda - \mu N.$$

By comparing theorems, the positive invariant set of model (2.1) is derived

$$\Omega = \left\{ (S_u, S_a, I, C, A) \in \mathbb{R}_+^5 : 0 \leq N \leq \frac{\Lambda}{\mu} \right\}.$$

We only consider the dynamical properties of model (2.1) in the set Ω . The basic reproduction number of model (2.1) can be obtained from the next generation matrix approach [24, 25] as follows:

$$R_0 = \frac{\beta [\mu + (1 - \varepsilon)\alpha] k_1 k_2 \Lambda}{\mu(\mu + \alpha) [\mu(k_2(k_1 + \gamma) + \rho k_1 + \gamma\delta) + \eta\gamma\delta]}, \quad (2.2)$$

with $k_1 = \mu + \delta + \nu$, $k_2 = \mu + \eta$. By the similar proof of Theorem 2 in Fatmawati et al. [8], we obtain that when $R_0 < 1$ model (2.1) has a unique boundary equilibrium point

$$P_0 = \left(\frac{\Lambda}{\mu + \alpha}, \frac{\alpha\Lambda}{\mu(\mu + \alpha)}, 0, 0, 0 \right),$$

which is locally asymptotically stable in the set Ω . We also provide the expression of an endemic equilibrium point

$$P^* = (S_u^*, S_a^*, I^*, C^*, A^*),$$

of model (2.1) when $R_0 > 1$, where

$$S_u^* = \frac{\Lambda}{\beta I^* + \alpha + \mu}, S_a^* = \frac{\alpha\Lambda}{((1 - \varepsilon)\beta I^* + \mu)(\beta I^* + \alpha + \mu)},$$

$$C^* = \frac{\rho I^*}{k_2}, A^* = \frac{\gamma I^*}{k_1}.$$

Substituting S_u^* , S_a^* , C^* , A^* into the third equation of model (2.1) and letting

the left side be zero, then one finds that

$$\begin{aligned}
 f(I^*) &= \beta I^* \left(\frac{\Lambda}{\beta I^* + \alpha + \mu} + \frac{(1 - \varepsilon)\alpha\Lambda}{((1 - \varepsilon)\beta I^* + \mu)(\beta I^* + \alpha + \mu)} \right) + \eta \frac{\rho I^*}{k_2} \\
 &\quad + v \frac{\gamma I^*}{k_1} - (\rho + \gamma + \mu) I^* \\
 &= I^* (\beta \Lambda k_1 k_2 ((1 - \varepsilon)\beta I^* + \mu) + \beta \Lambda k_1 k_2 (1 - \varepsilon)\alpha \\
 &\quad + (k_1 \eta \rho + k_2 v \gamma - k_1 k_2 (\rho + \gamma + \mu)) (\beta I^* + \alpha + \mu) ((1 - \varepsilon)\beta I^* + \mu)) \\
 &\quad (k_1 k_2 (\beta I^* + \alpha + \mu) ((1 - \varepsilon)\beta I^* + \mu))^{-1} \\
 &= I^* (A^* I^{*2} + B^* I^* + C^*) (k_1 k_2 (\beta I^* + \alpha + \mu) ((1 - \varepsilon)\beta I^* + \mu))^{-1},
 \end{aligned}$$

where

$$\begin{aligned}
 P_1 &= (1 - \varepsilon)\beta^2 (k_1 \eta \rho + k_2 v \gamma - k_1 k_2 (\rho + \gamma + \mu)) \\
 &= - (1 - \varepsilon)\beta^2 (\mu \nu \rho + \mu \delta \rho + \mu^2 \rho + \eta \delta \rho + \eta \mu \rho + \mu \delta \gamma + \mu^2 \gamma \\
 &\quad + \mu (\nu + \delta + \mu) (\eta + \mu)) < 0, \\
 P_2 &= \beta ((1 - \varepsilon)\alpha + (2 - \varepsilon)\mu) (k_1 \eta \rho + k_2 v \gamma - k_1 k_2 (\rho + \gamma + \mu)) \\
 &\quad + k_1 k_2 \Lambda \beta^2 (1 - \varepsilon), \\
 P_3 &= k_1 k_2 \Lambda \beta (\mu + (1 + \varepsilon)\alpha) + \mu (\mu + \alpha) (k_1 \eta \rho + k_2 v \gamma - k_1 k_2 (\rho + \gamma + \mu)) \\
 &= \mu (\mu + \alpha) (\mu [k_2 (k_1 + \gamma) + \rho k_1 + \gamma \delta] + \eta \gamma \delta) (R_0 - 1).
 \end{aligned}$$

Obviously $f(0) = 0$. When $R_0 > 1$, we have $P_1 < 0$ and $P_3 > 0$. According to the Descartes sign rule, $f(I^*)$ has a unique positive real root regardless of the sign of P_2 . Therefore, the endemic equilibrium point P^* exists.

By the approaches given in Evans et al. [31] and Tan et al. [32], together with the contributions described by stochastic differential equations in [26–30], we are motivated to introduce the independent environmental fluctua-

tions into model (2.1), in which the independent environmental fluctuations are multiplicative white noises, and they are proportional to S_u, S_a, I, C, A . More precisely, when $\Delta t \rightarrow 0$, we consider a Markov process

$$X(t) = (S_u(t), S_a(t), I(t), C(t), A(t))^T$$

with the following descriptions:

$$\begin{aligned}\mathbb{E}[S_u(t + \Delta t) - S_u(t)|X_t = x] &\approx [\Lambda - \beta S_u(t)I(t) - (\alpha + \mu)S_u(t)] \Delta t, \\ \mathbb{E}[S_a(t + \Delta t) - S_a(t)|X_t = x] &\approx [\alpha S_u(t) - (1 - \varepsilon)\beta S_a(t)I(t) - \mu S_a(t)] \Delta t, \\ \mathbb{E}[I(t + \Delta t) - I(t)|X_t = x] &\approx [\beta I(t) (S_u(t) + (1 - \varepsilon)S_a(t)) + \eta C(t) + v A(t) \\ &\quad - (\rho + \gamma + \mu)I(t)] \Delta t, \\ \mathbb{E}[C(t + \Delta t) - C(t)|X_t = x] &\approx [\rho I(t) - (\eta + \mu)C(t)] \Delta t, \\ \mathbb{E}[A(t + \Delta t) - A(t)|X_t = x] &\approx [\gamma I(t) - (v + \delta + \mu)A(t)] \Delta t,\end{aligned}$$

and

$$\begin{aligned}\text{Var}[S_u(t + \Delta t) - S_u(t)|X_t = x] &\approx \sigma_1^2 S_u^2(t) \Delta t, \\ \text{Var}[S_a(t + \Delta t) - S_a(t)|X_t = x] &\approx \sigma_2^2 S_a^2(t) \Delta t, \\ \text{Var}[I(t + \Delta t) - I(t)|X_t = x] &\approx \sigma_3^2 I^2(t) \Delta t, \\ \text{Var}[C(t + \Delta t) - C(t)|X_t = x] &\approx \sigma_4^2 C^2(t) \Delta t, \\ \text{Var}[A(t + \Delta t) - A(t)|X_t = x] &\approx \sigma_5^2 A^2(t) \Delta t,\end{aligned}$$

where $\sigma_i^2 > 0$ are the intensities of white noises for $i = 1, 2, 3, 4, 5$. We

therefore derive a stochastic epidemic model as follows:

$$\begin{aligned}
 dS_u(t) &= [\Lambda - \beta S_u(t)I(t) - (\alpha + \mu)S_u(t)] dt + \sigma_1 S_u(t)dB_1(t), \\
 dS_a(t) &= [\alpha S_u(t) - (1 - \varepsilon)\beta S_a(t)I(t) - \mu S_a(t)] dt + \sigma_2 S_a(t)dB_2(t), \\
 dI(t) &= [\beta I(t) (S_u(t) + (1 - \varepsilon)S_a(t)) + \eta C(t) + vA(t) \\
 &\quad - (\rho + \gamma + \mu)I(t)] dt + \sigma_3 I(t)dB_3(t), \\
 dC(t) &= [\rho I(t) - (\eta + \mu)C(t)]dt + \sigma_4 C(t)dB_4(t), \\
 dA(t) &= [\gamma I(t) - (v + \delta + \mu)A(t)]dt + \sigma_5 A(t)dB_5(t),
 \end{aligned} \tag{2.3}$$

where $B_i(t)$ are independent standard Brownian motions with the initial values $B_i(0) = 0$ ($i = 1, 2, 3, 4, 5$).

3. Existence and uniqueness of positive solution

Let $X(t)$ be a homogeneous Markov process in \mathbb{R}^d , which satisfies the stochastic differential equation

$$dX(t) = b(X(t))dt + \sum_{r=1}^k g_r(X(t)) dB_r(t), \quad k \leq d.$$

The diffusion matrix is defined as

$$A(X) = (a_{ij}(X))_{d \times d}, \quad a_{ij}(X) = \sum_{r=1}^k g_r^i(X)g_r^j(X).$$

Lemma 3.1 [33] Assume that there exists a bounded open region $G \subset \mathbb{R}^d$ with regular boundaries Γ , and it has the following properties:

(i) The minimum eigenvalue of the diffusion matrix $A(X)$ is non-zero in its domain G and one of its neighborhoods.

(ii) The average time τ for the path from z to set G is finite when $z \in \mathbb{R}^d \setminus G$, and $\sup_{z \in K} E^z \tau < \infty$ holds for each compact subset $K \in \mathbb{R}^d$.

Then, the Markov process $X(t)$ has a unique ergodic stationary distribution $\pi(\cdot)$. Let $f(X)$ be an integrable function of π , for all $X \in \mathbb{R}^d$, the following formula holds:

$$\mathbb{P} \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t f(X(s)) ds = \int_{\mathbb{R}^d} f(X) \pi(dX) \right\} = 1.$$

Remark 3.1 The proof of Lemma 3.1 can be found on pages 106-109 of Khasminskii [33]. If there exists a positive M such that

$$\sum_{i,j=1}^d a_{ij}(X) \xi_i \xi_j \geq M |\xi|^2, \xi \in \mathbb{R}^d,$$

then property (i) holds.

Now, we will give two useful results, Lemma 3.2 and Lemma 3.3, by using of Theorem 2.1 and Theorem 3.1 in [34]. In fact, we write down the conclusions without giving the details of the proofs.

Lemma 3.2 *Let $X(t)$ be a solution of model (2.3) initiated with $X_0 \in \mathbb{R}_+^5$, so*

$$\lim_{t \rightarrow \infty} \frac{1}{t} (S_u(t) + S_a(t) + I(t) + C(t) + A(t)) = 0,$$

and

$$\begin{aligned} \lim_{t \rightarrow \infty} \frac{S_u(t)}{t} = 0, \lim_{t \rightarrow \infty} \frac{S_a(t)}{t} = 0, \lim_{t \rightarrow \infty} \frac{I(t)}{t} = 0, \\ \lim_{t \rightarrow \infty} \frac{C(t)}{t} = 0, \lim_{t \rightarrow \infty} \frac{A(t)}{t} = 0 \quad a.s. \end{aligned}$$

Lemma 3.3 *Suppose that $\mu > (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2 \vee \sigma_4^2 \vee \sigma_5^2)/2$, let $X(t)$ be a*

solution of model (2.3) initiated with $X_0 \in \mathbb{R}_+^5$. Then

$$\begin{aligned} \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S_u(s) dB_1(s) &= 0, \quad \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S_a(s) dB_2(s) = 0, \\ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t I(s) dB_3(s) &= 0, \quad \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t C(s) dB_4(s) = 0, \\ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t A(s) dB_5(s) &= 0. \quad a.s.. \end{aligned}$$

Before we start to study the dynamical behaviors of the stochastic epidemic model (2.3), the existence of a global positive solution is of importance. Next, we show that there exists a unique global positive solution to model (2.3) for any given initial values.

Theorem 3.1 *Model (2.3) has a unique global positive solution $X(t) \in \mathbb{R}_+^5$ initiated with $X_0 \in \mathbb{R}_+^5$ for any $t \geq 0$.*

Proof It is obvious to check that the local Lipschitz condition is satisfied for model (2.3) initiated with $X_0 \in \mathbb{R}_+^5$, so there exists a unique local solution $X(t)$ for $t \in [0, \tau_e)$. To prove that $X(t)$ is global, our work is to verify $\tau_e = \infty$. Indeed, let $n_0 > 1$ be large enough satisfying each component of $X(t)$ lies in $[\frac{1}{n_0}, n_0]$. Define the stopping time

$$\begin{aligned} \tau_n = \inf \left\{ t \in [0, \tau_e) : \min \{S_u(t), S_a(t), I(t), C(t), A(t)\} \leq \frac{1}{n} \right. \\ \left. \text{or } \max \{S_u(t), S_a(t), I(t), C(t), A(t)\} \geq n \right\} \end{aligned}$$

for any integer $n > n_0$. Let $\inf \emptyset = \infty$. As $n \rightarrow \infty$, it is obvious that $\{\tau_n\}_{n \geq n_0}$ is monotonically increasing. We set $\tau_\infty = \lim_{n \rightarrow \infty} \tau_n$, then we get $\tau_\infty \leq \tau_e$ by the definition of stopping time. We claim that $\tau_\infty = \infty$. What

we claim is checked, which will end the proof. By contradiction, there exists a pair of positive constants $T > 0$ and $\varepsilon \in (0, 1)$ such that the probability that $\tau_\infty \leq T$ is larger than ε . We rewrite as $\mathbb{P}\{\tau_n \leq T\} \geq \varepsilon$ for $n \geq n_0$. Define a C^2 -function $U : \mathbb{R}_+^5 \rightarrow \mathbb{R}_+$ by

$$\begin{aligned} U(X(t)) &= \left(S_u - \theta - \theta \ln \frac{S_u}{\theta} \right) + (S_a - 1 - \ln S_a) + (I - 1 - \ln I) \\ &\quad + (C - 1 - \ln C) + (A - 1 - \ln A), \end{aligned}$$

where $\theta \in \mathbb{R}_+$. By the scalar Itô's formula, we get

$$\begin{aligned} dU(X(t)) &= \mathcal{L}U(X(t))dt + \sigma_1 (S_u - \theta) dB_1(t) + \sigma_2 (S_a - 1) dB_2(t) \\ &\quad + \sigma_3 (I - 1) dB_3(t) + \sigma_4 (C - 1) dB_4(t) + \sigma_5 (A - 1) dB_5(t), \end{aligned}$$

where $\mathcal{L}U(X(t)) : \mathbb{R}_+^5 \rightarrow \mathbb{R}_+$ is

$$\begin{aligned} \mathcal{L}U(X(t)) &= -\frac{\theta\Lambda}{S_u} + \theta\beta I + \theta(\alpha + \mu) + \frac{\theta}{2}\sigma_1^2 - \frac{\alpha S_u}{S_a} + (1 - \varepsilon)\beta I \\ &\quad + \mu + \frac{1}{2}\sigma_2^2 - \beta(S_u + (1 - \varepsilon)S_a) - \frac{\eta C}{I} - \frac{\nu A}{I} \\ &\quad + \rho + \gamma + \mu + \frac{1}{2}\sigma_3^2 - \frac{\rho I}{C} + \eta + \mu + \frac{1}{2}\sigma_4^2 - \frac{\gamma I}{A} \\ &\quad + \nu + \delta + \mu + \frac{1}{2}\sigma_5^2 + \Lambda \\ &\quad - \mu(S_u + S_a + I + C + A) - \delta A \\ &\leq (\theta + 1 - \varepsilon)\beta I - \mu I + \theta \left(\alpha + \mu + \frac{1}{2}\sigma_1^2 \right) + \Lambda + 4\mu \\ &\quad + \rho + \gamma + \eta + \nu + \delta + \frac{1}{2}(\sigma_2^2 + \sigma_3^2 + \sigma_4^2 + \sigma_5^2). \end{aligned}$$

We let $\theta = \frac{\mu}{\beta} - 1 + \varepsilon$, then

$$\begin{aligned} \mathcal{L}U(X(t)) &\leq \left(\frac{\mu}{\beta} - 1 + \varepsilon\right) \left(\alpha + \mu + \frac{1}{2}\sigma_1^2\right) + \Lambda + 4\mu + \rho + \gamma + \eta + \nu + \delta \\ &\quad + \frac{1}{2}(\sigma_2^2 + \sigma_3^2 + \sigma_4^2 + \sigma_5^2) := Q, \end{aligned}$$

which further gives

$$\begin{aligned} dU(X(t)) &\leq Qdt + \sigma_1(S_u - \theta)dB_1(t) + \sigma_2(S_a - 1)dB_2(t) \\ &\quad + \sigma_3(I - 1)dB_3(t) + \sigma_4(C - 1)dB_4(t) \\ &\quad + \sigma_5(A - 1)dB_5(t). \end{aligned} \tag{3.1}$$

Integrating (3.1) from 0 to $\tau_n \wedge T = \min\{\tau_n, T\}$, taking the expectation, we get

$$\mathbb{E}(U(X(\tau_n \wedge T))) \leq U(X_0) + Q\mathbb{E}(\tau_n \wedge T) \leq U(X_0) + QT. \tag{3.2}$$

When $n \geq n_0$, let $\Omega_n = \{\tau_n \leq T\}$, then the inequality $\mathbb{P}\{\tau_n \leq T\} \geq \varepsilon$ transforms into $\mathbb{P}\{\Omega_n\} \geq \varepsilon$. For each $\omega \in \Omega_n$, S_u takes value $n - \theta - \theta \ln \frac{n}{\theta}$ or $\frac{1}{n} - \theta + \theta \ln(\theta n)$ at time $\tau_n \wedge T$, so do S_a , I , C and A .

Obviously, inequality (3.2) can be transformed into

$$\begin{aligned} U(X_0) + QT &\geq \mathbb{E}[\mathbb{I}_{\Omega_n(\omega)}U(X(\tau_n \wedge T))] \\ &\geq \varepsilon \left[\left(n - \theta - \theta \ln \frac{n}{\theta}\right) \wedge \left(\frac{1}{n} - \theta + \theta \ln(\theta n)\right) \right], \end{aligned}$$

where $\mathbb{I}_{\Omega_n(\omega)}$ is the index function of $\Omega_n(\omega)$. Let $n \rightarrow \infty$, we get

$$\infty > U(X_0) + QT = \infty.$$

This is a contradiction. The proof is complete.

4. Existence of a unique ergodic stationary distribution

Sufficient conditions for the existence of stationary distribution and ergodicity of model (2.3) are given below, which also implies that HIV/AIDS is persistent in the mean. We denote the stochastic index

$$R_0^s = \frac{\beta [\mu + (1 - \varepsilon) \alpha] k_1 k_2 \Lambda}{k_5 k_6 (k_1 k_2 (k_4 + \frac{1}{2} \sigma_3^2) - (k_1 + \frac{1}{2} \sigma_5^2) \rho \eta - (k_2 + \frac{1}{2} \sigma_4^2) \gamma \nu)},$$

with

$$k_1 = \mu + \delta + \nu, \quad k_2 = \mu + \eta, \quad k_3 = \frac{\beta (\mu + (1 - \varepsilon) \alpha) \Lambda}{\mu (\mu + \alpha)},$$

$$k_4 = \rho + \gamma + \mu, \quad k_5 = \mu + \frac{1}{2} \sigma_2^2, \quad k_6 = \mu + \alpha + \frac{1}{2} \sigma_1^2.$$

When $\sigma_1 = \sigma_2 = \sigma_3 = \sigma_4 = \sigma_5 = 0$, R_0^s degenerates to R_0 in (2.2).

Theorem 4.1 *Model (2.3) has a unique stationary distribution, and it is ergodic when $R_0^s > 1$.*

Proof The diffusion matrix

$$A(X) = \text{diag} \{ \sigma_1^2 S_u^2, \sigma_2^2 S_a^2, \sigma_3^2 I^2, \sigma_4^2 C^2, \sigma_5^2 A^2 \}$$

of model (2.3) is positive definite, then condition (i) is clearly established.

Therefore, we only need to prove that condition (ii) holds. First, we create

a C^2 -function $G : \mathbb{R}_+^5 \rightarrow \mathbb{R}_+$ by

$$\begin{aligned} G(X(t)) &= M(-c_1 \ln S_u - c_2 \ln S_a + c_3 k_1 k_2 \beta \frac{1}{\alpha} S_a - c_3 k_1 k_2 \ln I \\ &\quad + c_3 \rho \eta \ln A + c_3 \gamma \nu \ln C) + (S_u + S_a + I + C + A)^{m+1} \\ &\quad - \ln S_u - \ln S_a - \ln C - \ln A \\ &:= MV_1 + V_2 + V_3 + V_4 + V_5 + V_6, \end{aligned}$$

where $c_i \in \mathbb{R}_+$ for $i = 1, 2, 3$; $M > 0$ is a sufficiently large positive number, m is a sufficiently small positive number, M and m satisfy

$$\begin{aligned} &-3M \left(\sqrt[3]{R_0^s} - 1 \right) + (M\beta^* + (2 - \varepsilon)\beta)\varepsilon_1 + B + \alpha + \mu \\ &+ \frac{1}{2}\sigma_1^2 + \mu + \frac{1}{2}\sigma_2^2 + k_2 + \frac{1}{2}\sigma_4^2 + k_1 + \frac{1}{2}\sigma_5^2 \leq -2, \end{aligned} \quad (4.1)$$

and

$$\mu - \frac{1}{2}m (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2 \vee \sigma_4^2 \vee \sigma_5^2) > 0, \quad (4.2)$$

here β^* and B are defined in (4.3)-(4.4) respectively. We obtain that

$$\lim_{n \rightarrow \infty} \inf_{(S_u, S_a, I, C, A) \in \mathbb{R}_+^5 \setminus U} G(S_u, S_a, I, C, A) = +\infty,$$

where

$$U = \left(\frac{1}{n}, n \right) \times \left(\frac{1}{n}, n \right) \times \left(\frac{1}{n}, n \right) \times \left(\frac{1}{n}, n \right) \times \left(\frac{1}{n}, n \right).$$

Since $G(S_u, S_a, I, C, A)$ is a continuous function, there must be a minimum

value \tilde{G} . Define a non-negative C^2 -function

$$V_1(S_u, S_a, I, C, A) = G(S_u, S_a, I, C, A) - \tilde{G},$$

then we apply the Itô's formula on V_1 :

$$\begin{aligned} \mathcal{L}V_1 &= -c_1 \left(\frac{\Lambda}{S_u} - \beta I - (\alpha + \mu) \right) + \frac{1}{2}c_1\sigma_1^2 - c_2 \left(\frac{\alpha S_u}{S_a} - (1 - \varepsilon)\beta I - \mu \right) \\ &\quad + \frac{1}{2}c_2\sigma_2^2 - c_3k_1k_2 \left(\beta(S_u + (1 - \varepsilon)S_a) + \frac{\eta C}{I} + \frac{\nu A}{I} - k_4 \right) \\ &\quad + \frac{1}{2}c_3k_1k_2\sigma_3^2 + c_3\rho\eta \left(\frac{\gamma I}{A} - k_1 \right) - \frac{1}{2}c_3\rho\eta\sigma_5^2 + c_3\gamma\nu \left(\frac{\rho I}{C} - k_2 \right) \\ &\quad - \frac{1}{2}c_3\gamma\nu\sigma_4^2 + c_3k_1k_2\beta\frac{1}{\alpha}(\alpha S_u - (1 - \varepsilon)\beta S_a I - \mu S_a) \\ &= - \left(c_1 \frac{\Lambda}{S_u} + c_2 \frac{\alpha S_u}{S_a} + c_3k_1k_2\beta \left(\frac{\mu}{\alpha} + 1 - \varepsilon \right) S_a \right) + c_1 \left(\alpha + \mu + \frac{1}{2}\sigma_1^2 \right) \\ &\quad + c_2 \left(\mu + \frac{1}{2}\sigma_2^2 \right) + c_3 \left[k_1k_2 \left(k_4 + \frac{1}{2}\sigma_3^2 \right) - \left(k_1 + \frac{1}{2}\sigma_5^2 \right) \rho\eta \right. \\ &\quad \left. - \left(k_2 + \frac{1}{2}\sigma_4^2 \right) \gamma\nu \right] + \left(c_1\beta + c_2(1 - \varepsilon)\beta + c_3\rho\gamma \left(\frac{\eta}{A} + \frac{\nu}{C} \right) \right) I \\ &\quad - c_3k_1k_2 \left(\frac{\eta C}{I} + \frac{\nu A}{I} + (1 - \varepsilon)\beta^2\frac{1}{\alpha}S_a I \right). \end{aligned}$$

Using $a + b + c \geq 3\sqrt[3]{abc}$ for positive a, b and c , we get

$$\begin{aligned} \mathcal{L}V_1 &\leq -3\sqrt[3]{c_1c_2c_3\beta[\mu + (1 - \varepsilon)\alpha]k_1k_2\Lambda} + c_1 \left(\alpha + \mu + \frac{1}{2}\sigma_1^2 \right) \\ &\quad + c_2 \left(\mu + \frac{1}{2}\sigma_2^2 \right) + c_3 \left(k_1k_2 \left(k_4 + \frac{1}{2}\sigma_3^2 \right) - \left(k_1 + \frac{1}{2}\sigma_5^2 \right) \rho\eta \right. \\ &\quad \left. - \left(k_2 + \frac{1}{2}\sigma_4^2 \right) \gamma\nu \right) + (c_1\beta + c_2(1 - \varepsilon)\beta + c_3\rho\gamma(\eta + \nu))I, \end{aligned}$$

we let

$$\begin{aligned}
 c_1 &= \frac{1}{\alpha + \mu + \frac{1}{2}\sigma_1^2}, & c_2 &= \frac{1}{\mu + \frac{1}{2}\sigma_2^2}, \\
 c_3 &= \frac{1}{k_1 k_2 (k_4 + \frac{1}{2}\sigma_3^2) - (k_1 + \frac{1}{2}\sigma_5^2) \rho \eta - (k_2 + \frac{1}{2}\sigma_4^2) \gamma \nu}, \\
 \beta^* &= c_1 \beta + c_2 (1 - \varepsilon) \beta + c_3 \rho \gamma (\eta + \nu), \\
 \hat{M} &= \mu - \frac{1}{2} m (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2 \vee \sigma_4^2 \vee \sigma_5^2),
 \end{aligned}$$

and then

$$\mathcal{L}V_1 \leq -3 \left(\sqrt[3]{R_0^s} - 1 \right) + \beta^* I. \quad (4.3)$$

Similarly,

$$\begin{aligned}
 \mathcal{L}V_2 &= (m+1) N^m (\Lambda - \mu N - \delta A) \\
 &\quad + \frac{1}{2} (m+1) m N^{m-1} (\sigma_1^2 S_u^2 + \sigma_2^2 S_a^2 + \sigma_3^2 I^2 + \sigma_4^2 C^2 + \sigma_5^2 A^2) \\
 &\leq (m+1) N^m (\Lambda - \mu N) \\
 &\quad + \frac{1}{2} (m+1) m N^{m+1} (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2 \vee \sigma_4^2 \vee \sigma_5^2) \\
 &\leq (m+1) \Lambda N^m - (m+1) \hat{M} N^{m+1} \\
 &\leq B - \frac{1}{2} (m+1) \hat{M} (S_u^{m+1} + S_a^{m+1} + I^{m+1} + C^{m+1} + A^{m+1}),
 \end{aligned} \quad (4.4)$$

where

$$B = \sup_{N \in (0, \infty)} (m+1) \left(\Lambda N^m - \frac{1}{2} \hat{M} N^{m+1} \right) < \infty.$$

We thus derive

$$\begin{aligned}\mathcal{L}V_3 &= -\frac{1}{S_u} [\Lambda - \beta S_u I - (\alpha + \mu) S_u] + \frac{1}{2S_u^2} (\sigma_1^2 S_u^2) \\ &= -\frac{\Lambda}{S_u} + \beta I + (\alpha + \mu) + \frac{1}{2} \sigma_1^2,\end{aligned}\quad (4.5)$$

$$\mathcal{L}V_4 = -\frac{\alpha S_u}{S_a} + (1 - \varepsilon) \beta I + \mu + \frac{1}{2} \sigma_2^2, \quad (4.6)$$

$$\mathcal{L}V_5 = -\frac{\rho I}{C} + k_2 + \frac{1}{2} \sigma_4^2, \quad (4.7)$$

$$\mathcal{L}V_6 = -\frac{\gamma I}{A} + k_1 + \frac{1}{2} \sigma_5^2. \quad (4.8)$$

From (4.3)-(4.8) we can get

$$\begin{aligned}\mathcal{L}V &\leq -3M \left(\sqrt[3]{R_0^s} - 1 \right) + (M\beta^* + (2 - \varepsilon) \beta) I + B - \frac{\Lambda}{S_u} + \alpha + \mu \\ &\quad - \frac{1}{2} (m + 1) \hat{M} (S_u^{m+1} + S_a^{m+1} + I^{m+1} + C^{m+1} + A^{m+1}) \\ &\quad + \frac{1}{2} \sigma_1^2 - \frac{\alpha S_u}{S_a} + \mu + \frac{1}{2} \sigma_2^2 - \frac{\rho I}{C} + k_2 + \frac{1}{2} \sigma_4^2 - \frac{\gamma I}{A} + k_1 + \frac{1}{2} \sigma_5^2.\end{aligned}\quad (4.9)$$

We define a bounded region

$$\begin{aligned}H &= \left\{ X(t) \in \mathbb{R}_+^5 : \varepsilon_1 \leq S_u(t) \leq \frac{1}{\varepsilon_1}, \varepsilon_1^2 \leq S_a(t) \leq \frac{1}{\varepsilon_1^2}, \varepsilon_1 \leq I(t) \leq \frac{1}{\varepsilon_1}, \right. \\ &\quad \left. \varepsilon_1^2 \leq C(t) \leq \frac{1}{\varepsilon_1^2}, \varepsilon_1^2 \leq A(t) \leq \frac{1}{\varepsilon_1^2} \right\},\end{aligned}$$

where $\varepsilon_1 > 0$ is sufficiently small and satisfies:

$$-\frac{\Lambda}{\varepsilon_1} + F \leq -1, \quad (4.10)$$

$$-\frac{\alpha}{\varepsilon_1} + F \leq -1, \quad (4.11)$$

$$-3M \left(\sqrt[3]{R_0^s} - 1 \right) + (M\beta^* + (2 - \varepsilon)\beta)\varepsilon_1 + F - e \leq -1, \quad (4.12)$$

$$-\frac{\rho}{\varepsilon_1} + F \leq -1, \quad (4.13)$$

$$-\frac{\gamma}{\varepsilon_1} + F \leq -1, \quad (4.14)$$

$$-\frac{1}{2}(m+1)\hat{M}\frac{1}{\varepsilon_1^{m+1}} + F \leq -1, \quad (4.15)$$

$$-\frac{1}{2}(m+1)\hat{M}\frac{1}{\varepsilon_1^{2m+2}} + F \leq -1, \quad (4.16)$$

$$-\frac{1}{4}(m+1)\hat{M}\frac{1}{\varepsilon_1^{m+1}} + F \leq -1, \quad (4.17)$$

combined with (4.1), we denote

$$F := B + e + \alpha + \mu + \frac{1}{2}\sigma_1^2 + \mu + \frac{1}{2}\sigma_2^2 + k_2 + \frac{1}{2}\sigma_4^2 + k_1 + \frac{1}{2}\sigma_5^2,$$

$$e = \sup_{I \in (0, \infty)} \left\{ -\frac{1}{4}(m+1)\hat{M}I^{m+1} + (M\beta^* + (2 - \varepsilon)\beta)I \right\} < \infty.$$

Obviously $\mathbb{R}_+^5 \setminus H = D_1 \cup D_2 \cup \dots \cup D_{10}$, where

$$\begin{aligned} D_1 &= \{X(t) \in \mathbb{R}_+^5 : 0 < S_u < \varepsilon_1\}, \\ D_2 &= \{X(t) \in \mathbb{R}_+^5 : 0 < S_a < \varepsilon_1^2, S_u \geq \varepsilon_1\}, \\ D_3 &= \{X(t) \in \mathbb{R}_+^5 : 0 < I < \varepsilon_1\}, \\ D_4 &= \{X(t) \in \mathbb{R}_+^5 : 0 < C < \varepsilon_1^2, I \geq \varepsilon_1\}, \\ D_5 &= \{X(t) \in \mathbb{R}_+^5 : 0 < A < \varepsilon_1^2, I \geq \varepsilon_1\}, \\ D_6 &= \{X(t) \in \mathbb{R}_+^5 : S_u \geq 1/\varepsilon_1\}, \\ D_7 &= \{X(t) \in \mathbb{R}_+^5 : S_a \geq 1/\varepsilon_1^2\}, \\ D_8 &= \{X(t) \in \mathbb{R}_+^5 : I \geq 1/\varepsilon_1\}, \\ D_9 &= \{X(t) \in \mathbb{R}_+^5 : C \geq 1/\varepsilon_1^2\}, \\ D_{10} &= \{X(t) \in \mathbb{R}_+^5 : A \geq 1/\varepsilon_1^2\}. \end{aligned}$$

We next discuss each case as follows:

Case 1. When $X(t) \in D_1$, according to (4.1), (4.9), (4.10), we can get

$$\mathcal{L}V \leq -\frac{\Lambda}{S_u} + F \leq -\frac{\Lambda}{\varepsilon_1} + F \leq -1.$$

Case 2. When $X(t) \in D_2$, according to (4.1), (4.9), (4.11), we can get

$$\mathcal{L}V \leq -\frac{\alpha S_u}{S_a} + F \leq -\frac{\alpha}{\varepsilon_1} + F \leq -1.$$

Case 3. When $X(t) \in D_3$, according to (4.1), (4.9), (4.12), we can get

$$\begin{aligned}\mathcal{L}V &\leq -3M \left(\sqrt[3]{R_0^s} - 1 \right) + [M\beta^* + (2 - \varepsilon)\beta]I + F - e \\ &\leq -3M \left(\sqrt[3]{R_0^s} - 1 \right) + [M\beta^* + (2 - \varepsilon)\beta]\varepsilon_1 + F - e \\ &\leq -1.\end{aligned}$$

Case 4. When $X(t) \in D_4$, according to (4.1), (4.9), (4.13), we can get

$$\mathcal{L}V \leq -\frac{\rho I}{C} + F \leq -\frac{\rho}{\varepsilon_1} + F \leq -1.$$

Case 5. When $X(t) \in D_5$, according to (4.1), (4.9), (4.14), we can get

$$\mathcal{L}V \leq -\frac{\gamma I}{A} + F \leq -\frac{\gamma}{\varepsilon_1} + F \leq -1.$$

Case 6. When $X(t) \in D_6$, according to (4.1), (4.9), (4.15), we can get

$$\begin{aligned}\mathcal{L}V &\leq -\frac{1}{2}(m+1)\hat{M}S_u^{m+1} + F \\ &\leq -\frac{1}{2}(m+1)\hat{M}\frac{1}{\varepsilon_1^{m+1}} + F \leq -1.\end{aligned}$$

Case 7. When $X(t) \in D_7$, according to (4.1), (4.9), (4.16), we can get

$$\begin{aligned}\mathcal{L}V &\leq -\frac{1}{2}(m+1)\hat{M}S_a^{m+1} + F \\ &\leq -\frac{1}{2}(m+1)\hat{M}\frac{1}{\varepsilon_1^{2m+2}} + F \leq -1.\end{aligned}$$

Case 8. When $X(t) \in D_8$, according to (4.1), (4.9), (4.17), we can get

$$\begin{aligned}\mathcal{L}V &\leq -\frac{1}{2}(m+1)\hat{M}I^{m+1} + (\hat{M}\beta^* + (2-\varepsilon)\beta)I + F - e \\ &\leq -\frac{1}{4}(m+1)\hat{M}I^{m+1} + F \\ &\leq -\frac{1}{4}(m+1)\hat{M}\frac{1}{\varepsilon_1^{m+1}} + F \leq -1.\end{aligned}$$

Case 9. When $X(t) \in D_9$, according to (4.1), (4.9), (4.16), we can get

$$\begin{aligned}\mathcal{L}V &\leq -\frac{1}{2}(m+1)\hat{M}C^{m+1} + F \\ &\leq -\frac{1}{2}(m+1)\hat{M}\frac{1}{\varepsilon_1^{2m+2}} + F \leq -1.\end{aligned}$$

Case 10. When $X(t) \in D_{10}$, according to (4.1), (4.9), (4.16), we can get

$$\begin{aligned}\mathcal{L}V &\leq -\frac{1}{2}(m+1)\hat{M}A^{m+1} + F \\ &\leq -\frac{1}{2}(m+1)\hat{M}\frac{1}{\varepsilon_1^{2m+2}} + F \leq -1.\end{aligned}$$

Therefore, we get $\mathcal{L}V \leq -1$ as $X(t) \in \mathbb{R}_+^5 \setminus H$. So condition (ii) of Lemma 3.1 is performed. The proof is complete.

5. Extinction

In this section, we will establish the sufficient conditions for the extinction of infectious disease HIV/AIDS. Denote

$$\langle S_u(t) \rangle = \frac{1}{t} \int_0^t S_u(s) ds, \quad \langle S_a(t) \rangle = \frac{1}{t} \int_0^t S_a(s) ds.$$

Theorem 5.1 Suppose that $\mu > (\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2 \vee \sigma_4^2 \vee \sigma_5^2)/2$, for any initial value $X_0 \in \mathbb{R}_+^5$, if

$$R_0^e = \frac{\beta [\mu + (1 - \varepsilon) \alpha] \Lambda}{\mu(\mu + \alpha)(\mu + \frac{1}{3}\hat{\sigma})} < 1,$$

with

$$\hat{\sigma} = \left(\delta + \frac{1}{2}\sigma_5^2 \right) \wedge \frac{1}{2}\sigma_3^2 \wedge \frac{1}{2}\sigma_4^2,$$

then HIV/AIDS will become extinct, and the solution of model (2.3) satisfies:

$$\lim_{t \rightarrow \infty} \frac{1}{t} \ln(I(t) + C(t) + A(t)) < 0.$$

Moreover,

$$\lim_{t \rightarrow \infty} \langle S_u(t) \rangle = \frac{\Lambda}{\mu + a}, \quad \lim_{t \rightarrow \infty} \langle S_a(t) \rangle = \frac{\alpha \Lambda}{\mu(\mu + a)}.$$

$$\lim_{t \rightarrow \infty} I(t) = 0, \quad \lim_{t \rightarrow \infty} C(t) = 0, \quad \lim_{t \rightarrow \infty} A(t) = 0.$$

Proof It is easy to check that

$$dS_u(t) \leq [\Lambda - (\alpha + \mu) S_u(t)] dt + \sigma_1 S_u(t) dB_1(t), \quad (5.1)$$

$$dS_a(t) \leq [\alpha S_u(t) - \mu S_a(t)] dt + \sigma_2 S_a(t) dB_2(t). \quad (5.2)$$

By Lemma 3.2 and Lemma 3.3, after integration, we have

$$\lim_{t \rightarrow \infty} \frac{1}{t} (S_u(t) - S_u(0)) \leq \lim_{t \rightarrow \infty} \left[\Lambda - (\alpha + \mu) \langle S_u(t) \rangle + \frac{\sigma_1}{t} \int_0^t S_u(s) dB_1(s) \right],$$

which further shows

$$\lim_{t \rightarrow \infty} \langle S_u(t) \rangle \leq \frac{\Lambda}{\mu + a}. \quad (5.3)$$

By the similar argument, we get

$$\lim_{t \rightarrow \infty} \frac{1}{t} (S_a(t) - S_a(0)) \leq \lim_{t \rightarrow \infty} \left[\alpha \langle S_u(t) \rangle - \mu \langle S_a(t) \rangle + \frac{\sigma_2}{t} \int_0^t S_a(s) dB_2(s) \right],$$

which thus implies

$$\lim_{t \rightarrow \infty} \langle S_a(t) \rangle \leq \frac{\alpha \Lambda}{\mu(\mu + a)}. \quad (5.4)$$

Here we define $P(X(t)) = I + C + A$, so Itô's formula gives that

$$d \ln P(X) = w dt + \frac{1}{P(X)} [\sigma_3 I dB_3(t) + \sigma_4 C dB_4(t) + \sigma_5 A dB_5(t)], \quad (5.5)$$

with

$$\begin{aligned} w = & \frac{1}{P(X)} (\beta I (S_u + (1 - \varepsilon) S_a) - \mu P(X) - \delta A) \\ & - \frac{1}{2P^2(X)} (\sigma_3^2 I^2 + \sigma_4^2 C^2 + \sigma_5^2 A^2). \end{aligned}$$

Due to the facts that

$$-\frac{\delta A}{P(X)} \leq -\frac{\delta A^2}{P^2(X)}, \quad \frac{I}{P(X)} \leq 1, \quad \frac{C}{P(X)} \leq 1, \quad \frac{A}{P(X)} \leq 1,$$

then (5.5) is simplified as

$$\begin{aligned}
 w &\leq \beta (S_u + (1 - \varepsilon) S_a) - \mu - \frac{\delta A^2}{P^2(X)} - \frac{1}{2P^2(X)} (\sigma_3^2 I^2 + \sigma_4^2 C^2 + \sigma_5^2 A^2) \\
 &= \beta (S_u + (1 - \varepsilon) S_a) - \mu - \frac{1}{P^2(X)} \left(\frac{1}{2} \sigma_3^2 I^2 + \frac{1}{2} \sigma_4^2 C^2 + \left(\delta + \frac{1}{2} \sigma_5^2 \right) A^2 \right) \\
 &\leq \beta (S_u + (1 - \varepsilon) S_a) - \mu - \frac{I^2 + C^2 + A^2}{P^2(X)} \hat{\sigma} \\
 &\leq \beta (S_u + (1 - \varepsilon) S_a) - \mu - \frac{1}{3} \hat{\sigma}. \tag{5.6}
 \end{aligned}$$

The integration on (5.6) gives that

$$\begin{aligned}
 \frac{1}{t} (\ln P(X) - \ln P(X(0))) &\leq \beta \langle S_u(t) \rangle + (1 - \varepsilon) \langle S_a(t) \rangle - \mu - \frac{1}{3} \hat{\sigma} \\
 &\quad + \frac{\sigma_3}{t} B_3(t) + \frac{\sigma_4}{t} B_4(t) + \frac{\sigma_5}{t} B_5(t). \tag{5.7}
 \end{aligned}$$

Applying the strong law of numbers, we get

$$\lim_{t \rightarrow \infty} \frac{B_i(t)}{t} = 0 \quad (i = 3, 4, 5). \tag{5.8}$$

When $t \rightarrow \infty$, by (5.3), (5.4), (5.8) and $R_0^e < 1$, (5.6) can be simplified as

$$\begin{aligned}
 \limsup_{t \rightarrow \infty} \frac{\ln P(X)}{t} &\leq \beta \frac{\Lambda}{\mu + a} + (1 - \varepsilon) \frac{\alpha \Lambda}{\mu (\mu + a)} - \mu - \frac{1}{3} \hat{\sigma} \\
 &= (R_0^e - 1) \left(\mu + \frac{1}{3} \hat{\sigma} \right) < 0.
 \end{aligned}$$

Therefore, we derive

$$\lim_{t \rightarrow \infty} I(t) = 0, \quad \lim_{t \rightarrow \infty} C(t) = 0, \quad \lim_{t \rightarrow \infty} A(t) = 0. \tag{5.9}$$

Furthermore, we consider

$$N = \Lambda - \mu N - \delta A + \sigma_1 S_u + \sigma_2 S_a + \sigma_3 I + \sigma_4 C + \sigma_5 A,$$

the integration implies that

$$\begin{aligned} & \frac{N(t) - N(0)}{t} \\ &= \Lambda - \mu (\langle S_u(t) \rangle + \langle S_a(t) \rangle + \langle I(t) \rangle + \langle C(t) \rangle + \langle A(t) \rangle) - \delta \langle A(t) \rangle \\ &+ \frac{\sigma_1}{t} \int_0^t S_u(s) dB_1(s) + \frac{\sigma_2}{t} \int_0^t S_a(s) dB_2(s) + \frac{\sigma_3}{t} \int_0^t I(s) dB_3(s) \\ &+ \frac{\sigma_4}{t} \int_0^t C(s) dB_4(s) + \frac{\sigma_5}{t} \int_0^t A(s) dB_5(s). \end{aligned}$$

By Lemma 3.2 and Lemma 3.3, together with (5.3), (5.4) and (5.9), the following expressions are obtained:

$$\lim_{t \rightarrow \infty} \langle S_u(t) \rangle = \frac{\Lambda}{\mu + a}, \quad \lim_{t \rightarrow \infty} \langle S_a(t) \rangle = \frac{\alpha \Lambda}{\mu(\mu + a)}.$$

Thus, the proof of Theorem 5.1 is complete.

6. Examples and numerical simulations

We take the parameters in this section from Fatmawati et al. [8] except for the values for β and σ . We further govern the positive preserving truncated Euler-Maruyama method (also referred as PPTM) in [35] to simulate the long-term properties of the solution. Let

$$X(t_k) = (S_u(t_k), S_a(t_k), I(t_k), C(t_k), A(t_k)), k = 0, 1, 2, \dots$$

be the discrete solution of model (2.3) with $t_k = k\Delta$, then the corresponding discretization equations are written as

$$\begin{aligned}
 S_u(t_{k+1}) &= S_u(t_k) + (\Lambda + f_{11} + f_{12})\Delta + g_1\sqrt{\Delta}, \\
 S_a(t_{k+1}) &= S_a(t_k) + (f_{21} + f_{22})\Delta + g_2\sqrt{\Delta}, \\
 I(t_{k+1}) &= I(t_k) + (f_{31} + f_{32})\Delta + g_3\sqrt{\Delta}, \\
 C(t_{k+1}) &= C(t_k) + f_{41}\Delta + g_4\sqrt{\Delta}, \\
 A(t_{k+1}) &= A(t_k) + f_{51}\Delta + g_5\sqrt{\Delta},
 \end{aligned} \tag{6.1}$$

for $k = 0, 1, 2, 3, \dots$, and

$$\begin{aligned}
 f_{11} &= -(\alpha + \mu)\hat{\pi}_0(S_u(t_k)), \quad f_{12} = -\beta\hat{\pi}_0(S_u(t_k)I(t_k)), \\
 f_{21} &= \alpha\hat{\pi}_0(S_u(t_k)) - \mu\hat{\pi}_0(S_a(t_k)), \\
 f_{22} &= -(1 - \varepsilon)\beta\hat{\pi}_0(S_a(t_k)I(t_k)), \\
 f_{31} &= \eta\hat{\pi}_0(C(t_k)) + \nu\hat{\pi}_0(A(t_k)) - (\rho + \gamma + \mu)\hat{\pi}_0(I(t_k)), \\
 f_{32} &= \beta\hat{\pi}_0(I(t_k)[\hat{\pi}_0(S_u(t_k)) + (1 - \varepsilon)\hat{\pi}_0(S_a(t_k))]), \\
 f_{41} &= \rho\hat{\pi}_0(I(t_k)), \quad f_{51} = \gamma\hat{\pi}_0(I(t_k)), \\
 g_1 &= \sigma r_{1,k}\hat{\pi}_0(S_u(t_k)), \quad g_2 = \sigma r_{2,k}\hat{\pi}_0(S_a(t_k)), \quad g_3 = \sigma r_{3,k}\hat{\pi}_0(I(t_k)), \\
 g_4 &= \sigma r_{4,k}\hat{\pi}_0(C(t_k)), \quad g_5 = \sigma r_{5,k}\hat{\pi}_0(A(t_k)),
 \end{aligned} \tag{6.2}$$

where Δ is the stepsize and $r_{i,k}$ ($i = 1, 2, 3, 4, 5$ and $k = 0, 1, 2, 3, \dots$) are independent random variables with the normal distribution $\mathcal{N}(0, 1)$ and the function $\hat{\pi}_0(u)$ is defined as $\hat{\pi}_0(u) = 0 \vee u$. So, the generalized discretization

equations can be denoted as:

$$X(t_{k+1}) = X(t_k) + [f_1(X(t_k)) + f_2(X(t_k))]\Delta + g(X(t_k))\Delta B_k$$

where

$$\begin{aligned} f_1 &= (\Lambda + f_{11}, f_{21}, f_{31}, f_{41}, f_{51})^T, f_2 = (f_{12}, f_{22}, f_{32}, 0, 0)^T, \\ g &= (g_1, g_2, g_3, g_4, g_5)^T. \end{aligned}$$

We define a strictly increasing function $z : \mathbb{R}_+ \rightarrow \mathbb{R}_+$ by $z(u) = u$ for $u \geq 1$, which gives the inverse function of $z^{-1} : [1, \infty) \rightarrow \mathbb{R}_+$ with the form $z^{-1}(u) = u$ for $u \geq 1$. We also define a strictly decreasing function $h : (0, 1] \rightarrow [1, \infty)$ by $h(\Delta) = \hat{h}\Delta^{-\frac{1}{4}}$ with $\hat{h} = 1 \vee z(1) \vee |X(0)|$ and $|X(0)| = \sqrt{S_u^2(0) + S_a^2(0) + I^2(0) + C^2(0) + A^2(0)}$.

Define

$$\pi_\Delta(X) = (|X| \wedge z^{-1}(h(\Delta))) \frac{X}{|X|},$$

and

$$\hat{\pi}_\Delta(X) = (\Delta \vee S_u, \Delta \vee S_a, \Delta \vee I, \Delta \vee C, \Delta \vee A)^T.$$

Let $\bar{X}_\Delta(t_k)$ be the intermediate step in order to get nonnegative preserving truncated Euler-Maruyama (NPTEM) solution $X_\Delta(t_k)$, and $\bar{X}_\Delta(0) = X_\Delta(0) = X(0)$ be the initial value, then the generalized discretization equation of NPTEM is then defined by

$$\bar{X}_\Delta(t_{k+1}) = \bar{X}_\Delta(t_k) + [f_1(\bar{X}_\Delta(t_k)) + f_2(X_\Delta(t_k))]\Delta + g(\bar{X}_\Delta(t_k))\Delta B_k, \quad (6.3)$$

$$X_\Delta(t_{k+1}) = \hat{\pi}_0(\pi_\Delta(\bar{X}_\Delta(t_{k+1}))), \quad (6.4)$$

for $k = 0, 1, 2, \dots$, where $\Delta B_k = B(t_{k+1}) - B(t_k)$, and then we extend the definition of $X_\Delta(\cdot)$ from the grid points t_k to the whole $t \geq 0$ by defining

$$X_\Delta(t) = X_\Delta(t_k) \quad \text{for } t \in [t_k, t_{k+1}), k = 0, 1, 2, \dots \quad (6.5)$$

Together with (6.3) and (6.5), the PPTEM solution is consequently derived by

$$X_\Delta^+(t_{k+1}) = \hat{\pi}_\Delta(\pi_\Delta(\bar{X}(t_{k+1}))), k = 0, 1, 2, \dots$$

Next, we present the simulations in both Indonesia and China by using of PPTEM and predict the development and prevalence of HIV/AIDS for next five decades.

Example 6.1

We firstly study the epidemics of HIV/AIDS in Indonesia. We choose the initial values $S_u(0) = 129789089$, $S_a(0) = 100000000$, $I(0) = 7195$, $C(0) = 0$, $A(0) = 3716$ of total population 229800000, the stepsize $\Delta = 10^{-2}$, and let

$$\Lambda = \frac{229800000}{67.39}, \beta = \frac{0.3465}{229800000}, \mu = \frac{1}{67.39},$$

and other parameters be

$$\alpha = 0.2351, \varepsilon = 0.3243, \eta = 0.2059, v = 0.7661,$$

$$\gamma = 0.1882, \rho = 0.00036523, \delta = 0.7012.$$

By Theorem 4.1, the stochastic index is $R_0^s \approx 2.075 > 1$ as $\sigma_i (i = 1, 2, 3, 4, 5) = 0.05$, so HIV/AIDS is persistent in a long run (see the left of Figure 6.1). The population size in each compartment of the stochastic model (2.3) fluctuates

around the endemic equilibrium point

$$P^* = (12267874, 85638867, 18584806, 30748, 2359917).$$

Furthermore, the solution of model (2.3) has a unique stationary distribution, which is ergodic when $R_0^s \approx 2.2676 > 1$ for $\sigma_i = 0.01$ ($i = 1, 2, 3, 4, 5$) and $T = 40000, 60000, 80000$ respectively, the population size in each compartment is presented on the right of Figure 6.1.

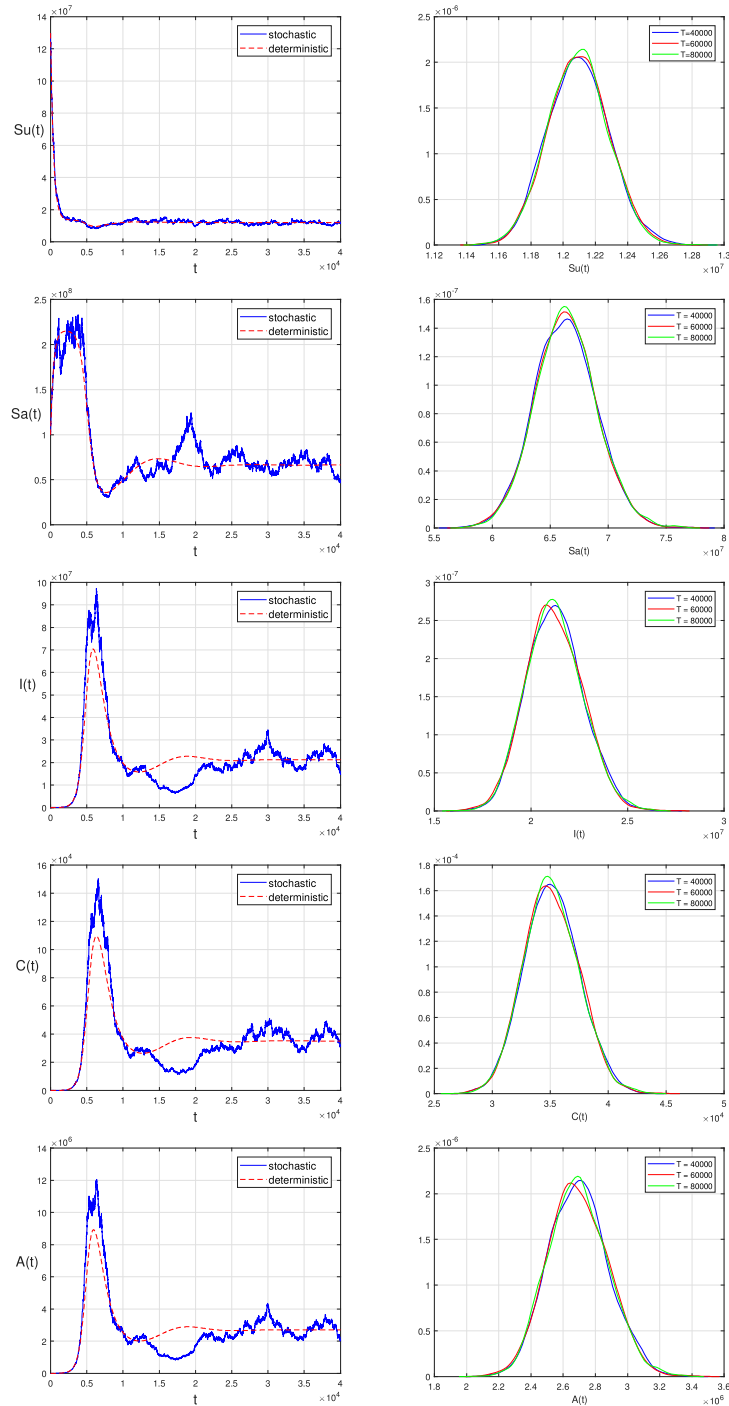


Figure 6.1 The persistence and stationary distribution of S_u, S_a, I, C, A in model (2.3)

We set $\beta = \frac{0.1065}{229800000}$ and keep the remaining parameters and initial values same with those in Figure 6.1. So, $\hat{R}_0^e \approx 0.072 < 1$ and

$$0.0148 \approx \mu > 0.5(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2 \vee \sigma_4^2 \vee \sigma_5^2) = 0.00125.$$

By Theorem 5.1, HIV/AIDS is extinct in Figure 6.2.

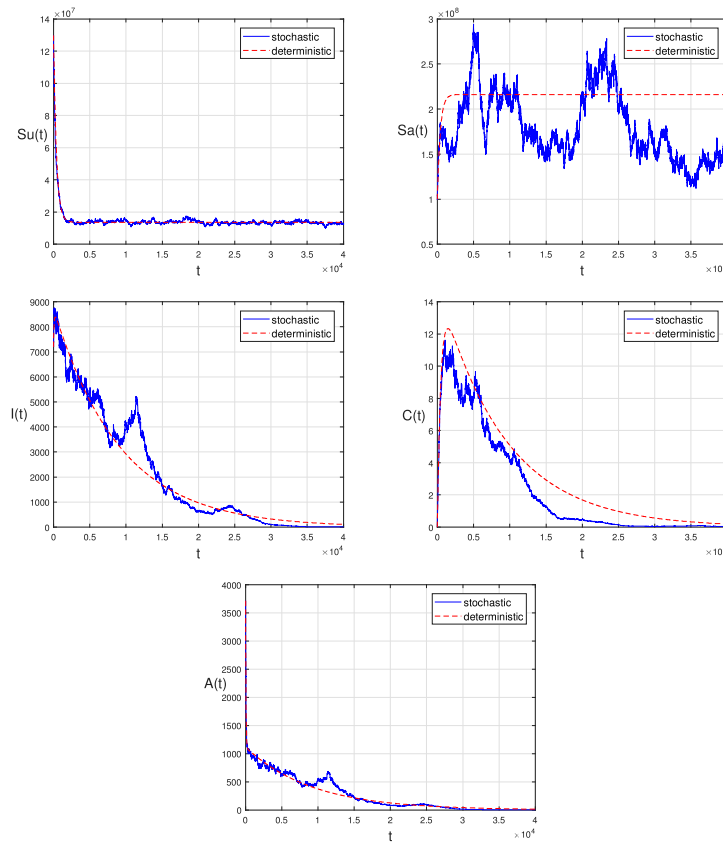


Figure 6.2 The extinction of HIV/AIDS

Next, we discuss the impacts of the parameter ε , and let $\sigma_i (i = 1, 2, 3, 4, 5) = 0.02$ and other parameters and the initial values be same with those in

Figure 6.1. We observe that HIV/AIDS is persistent as ε increases, while the population size of the infected decreases significantly in Figure 6.2 Therefore, the enhancement of ε is of significant importance for the prevention and control of HIV/AIDS.

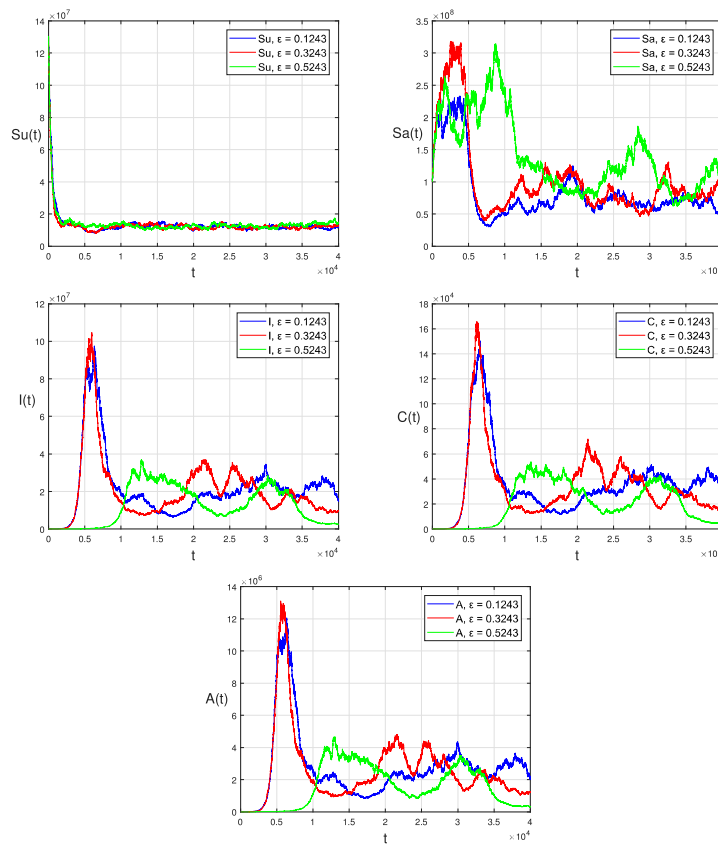


Figure 6.3 The impact of ε

Example 6.2.

We perform the numerical simulations on the spread of HIV/AIDS in China for next five decades, and provide some suggestions for the epidemics of MSM population. Since the population size is 1376460000 for the year

2014, and the average life span of the population is 76.34 [39], we assume that the natural growth rate, the natural case-fatality rate and the infection rate for the population are respectively

$$\lambda = \frac{1376460000}{76.34}, \mu = \frac{1}{76.34}, \beta = \frac{0.71}{1376460000}.$$

By the data in 2014 in Zhao et al.[36], the number of the individuals with HIV/AIDS is 500579, and the number of the individuals with ART is 295358, therefore we assume that

$$S_u = 1088230000, S_a = 288230000, I = 153193, C = 295358, A = 52128$$

and choose $\Delta = 10^{-2}$ and $\alpha = 0.13, \varepsilon = 0.5, \eta = 0.18, v = 0.72, \gamma = 0.14, \rho = 0.82, \delta = 0.42$ as other parameters for simulation.

Firstly, we collect the data for the individuals with HIV/AIDS from the year 2014 to 2020 in China in Zhao et al.[36] (year 2014-2018), Liu et al.[37] (year 2019) and He [38] (year 2020). We thus adopt Runge Kutta method to fit the parameters, and the simulation with fitted parameters and the data are shown on the left of Figure 6.4. By Theorem 4.1, we derive that $R_0^s = 2.8942 > 1$ as $\sigma_i = 0.05 (i = 1, 2, 3, 4, 5)$. Further, we govern the data in 2020 as the initial values to preform the simulations for next 5 decades in China as presented on the right of Figure 6.4. It is easy to observe that although the spread of HIV/AIDS in China is running in a low epidemic level, there still exists a risk of the exponential growth for HIV/AIDS control.

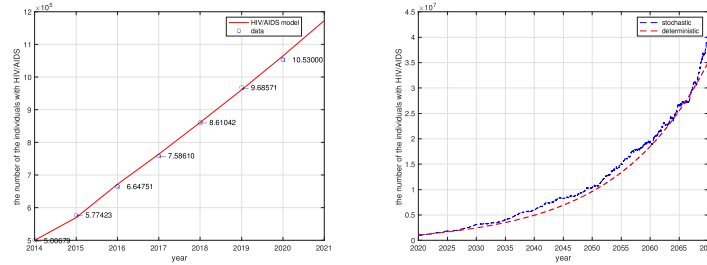


Figure 6.4 Data fitting for the individuals with HIV/AIDS from 2014 to 2020, and prediction of HIV/AIDS for next 5 decades in China

Next, we discuss the impacts of main parameters to the control of HIV/AIDS in China. The extensive publicity and detailed publicity on HIV/AIDS are two important ways to prevent and control the spread of HIV/AIDS in China. In practice, the extensive publicity improves the number of the individuals having protection awareness from S_u to S_a by varying α , which further reveals that less impact on the transmission of HIV/AIDS occurs as α increases (see the left in Figure 6.5). Meanwhile, the detailed publicity presents more details such as taking post-exposure prophylaxis (PEP) within 72 hours to avoid infection, which further suppresses the number of the individuals with HIV/AIDS as ε increases (see the right in Figure 6.5).

Therefore, the extensive publicity and the detailed publicity including lessons and lectures of AIDS in universities and communities to the targeted population play significant roles to prevent the spread of HIV/AIDS. Meanwhile, the prompt and continuous antiretroviral therapy (ART) after being infected is helpful to each individual with HIV/AIDS. Figure 6.6 demonstrates the simulations with distinct values of η , which also verify that ART suppresses the rapid growth of the individuals with HIV/AIDS as $\eta = 0.08$.

We also notice that the conversion rates γ and v affect the long-term epi-

demics of HIV/AIDS in China. More precisely, the period that the individuals with HIV/AIDS see the doctors and get checked declines (γ increases), then the number of the individuals with HIV/AIDS decreases. Meanwhile, the period that the individuals with AIDS stay at hospital declines (v increases), so the number of the individuals with HIV increases as presented in Figure 6.7.

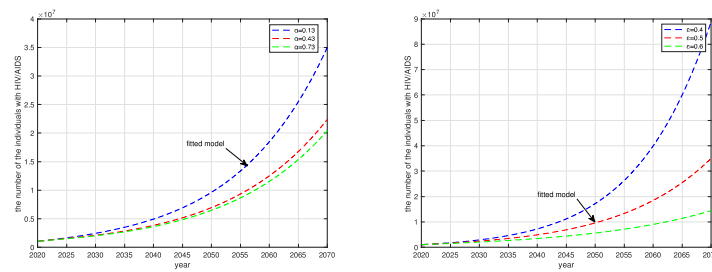


Figure 6.5 Impacts of the extensive publicity and detailed publicity on HIV/AIDS

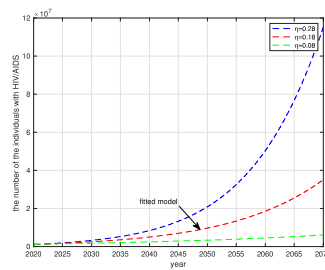
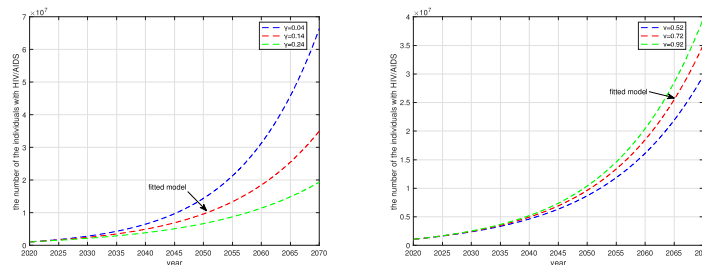


Figure 6.6 Impact of the continuous ART

Figure 6.7 Impacts of γ and v on HIV/AIDS

7. Conclusions and discussions

We propose a stochastic epidemic model with the bilinear incidence rate and show the existence of a global positive solution. By constructing Lyapunov functions, we also show that the stochastic model has an ergodic stationary distribution when $R_0^s > 1$. Moreover, the sufficient condition $R_0^e < 1$ for the extinction of HIV/AIDS is obtained. The corresponding simulations verify that the numbers of the individuals in Indonesia and in China decrease when the detailed publicity and the continuous ART are governed to prevent and control the spread of HIV/AIDS. Therefore, we suggest that all countries should enhance the systematic and detailed publicity on AIDS, which are of significant importance to control the growth of HIV/AIDS. For instance, taking the PEP within 72 hours and receiving prompt ART treatment after being infected are effective measurements for the elimination of AIDS by 2030.

Compared with the conclusions derived by Fatmawati et al. [8], the disease-free equilibrium point P_0 attracts the solution of (2.3) under condition $R_0^e < 1$ (see Theorem 5.1), which also means that HIV/AIDS becomes extinct as the intensities of the white noises increase. Meanwhile, the solu-

tion of (2.3) fluctuates around the endemic equilibrium P^* , and the solution has a unique ergodic stationary distribution when $R_0^s > 1$ (see Theorem 4.1).

We also point out that the expressions for R_0^s and R_0^e are two distinct indices for indicating the prevalence of HIV/AIDS. When the intensities of the white noises disappear, R_0^s turns into R_0 in (2.2). Theoretically, model (2.3) has a smaller index for the persistence of HIV/AIDS than that of model (2.1). The long-term properties of model (2.3) are quite different from the results in Fatmawati et al. [8].

CRedit authorship contribution statement

Xuanpei Zhai: Modelling, Data curation, Writing - original draft. **Wen-shuang Li:** Software, Visualization, Writing - original draft. **Fengying Wei:** Methodology, Validation, Writing - review & editing. **Xuerong Mao:** Supervision, Methodology, Writing - review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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