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Research article

Dynamics and optimal control for an HIV/AIDS model with VCT and PrEP treatments among MSM

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Abstract: Considering the important role of Voluntary counseling and testing (VCT) and Pre-exposure prophylaxis (PrEP) in controlling the spread of Acquired Immunodeficiency Syndrome (AIDS), in this paper, we formulate a Human Immunodeficiency Virus (HIV)/AIDS model among men who have sex with men (MSM) to quantitatively describe the impact of VCT and PrEP treatments on the transmission dynamics. First, we analyze the dynamic properties of the model, including proving the global asymptotic stability of the disease-free equilibrium and the uniform persistence of the disease. Then, we explore the optimal control problem of the model using the Pontryagin maximum principle. Finally, numerical simulations and a sensitivity analysis are conducted. The results indicate that enhancing VCT awareness among susceptible and infected individuals and promoting PrEP treatment among susceptible individuals can significantly reduce the number of HIV/AIDS cases. These efforts could include strengthening knowledge dissemination, organizing knowledge contests, and establishing platforms for AIDS education. Therefore, actively promoting VCT awareness and PrEP treatment education is essential to control the spread of HIV/AIDS.

Keywords: dynamic model; VCT awareness; uniform persistence; optimal control
Mathematics Subject Classification: 34D23, 92D20, 92D30

1. Introduction

Acquired Immunodeficiency Syndrome (AIDS) is a malignant infectious disease with a high mortality rate caused by the Human Immunodeficiency Virus (HIV), a retrovirus that damages and

destroys the human body by infecting immune cells [1]. Since the first global case of AIDS that was reported in the United States in June 1981, the disease has rapidly spread to more than 100 countries and regions around the world [2]. According to data reported by the World Health Organization (WHO), by the end of 2021, an estimated 38.4 million people were living with HIV, and HIV/AIDS remains a major global public health issue [3]. It has been 38 years since China reported its first imported case of HIV in 1985. Currently, the mode of HIV transmission in China has gradually shifted from blood and drug use to sexual transmission, particularly among MSM [4]. The risk of HIV infection in MSM is 28 times higher than that of ordinary men [5], and the proportion of newly reported cases of HIV infections among men of the same sex in China has increased from 2.5% in 2005 to 23.3% in 2020 [6].

AIDS is incurable at the current level of medical care. However, if it is found early, and timely detection and standard treatment are carried out, then the life span of AIDS infected people can be close to that of normal people. Therefore, it is more important to identify and confirm the infection status at the earliest time. However, according to the new AIDS self inspection guide issued by the WHO, 40% (more than 14 million) of infected people are still ignorant of their situation. HIV virus testing shows that nearly 80% of HIV infected adults in sub Saharan Africa do not know their own situation, and more than 90% do not know whether their partners are infected with HIV [7,8]. Voluntary counseling and testing (VCT) is an internationally recognized, effective, and important prevention and care strategy to control the transmission of HIV, which provides opportunities for primary prevention (i.e., preventing HIV negative people from infection) and secondary prevention (i.e., avoiding the disease progression of infected people by providing early medical care and psychosocial support) [9]. Since HIV testing is the only way to know about HIV infection, in recent years, the WHO has proposed to expand the AIDS VCT, which can effectively reduce the incidence rate and mortality of AIDS related diseases.

In addition, Pre-exposure prophylaxis (PrEP) is a novel biological prevention method used by HIV negative individuals to reduce the risk of infection by taking antiviral drugs to block the virus transmission before engaging in high-risk sexual behavior [10, 11]. The consensus among Chinese HIV PrEP medication experts points out that using antiviral drugs for PrEP in MSM can effectively block the transmission of HIV [12]. The study reported that the willingness rate to accept MSM PrEP in China is about 72.4%, which indicaties a relatively high overall willingness. However, due to the late implementation of PrEP in China, there are still some high-risk groups with an insufficient understanding of PrEP, and the willingness to accept PrEP and related factors are also complex and variable [13].

Mathematical modeling has been widely applied to explore the transmission laws of infectious diseases [14, 15]. The research on the transmission mechanism of HIV/AIDS and the prediction of the disease development pattern is also abundant (e.g., ordinary differential equation (ODE) models [16–18], partial differential equation (PDE) models [19, 20], SDE models [21, 22] and the associated references). In [16], the authors proposed a HIV/AIDS mathematical model with PrEP to study the effect of PrEP on the dynamics of the model. In order to understand the adjoint effect of PrEP and antiretroviral therapy (ART) treatments on the HIV transmission for MSM population, Shen et al. [18] verified that a high PrEP coverage and earlier ART are expected to provide the greatest benefit among MSM in San Francisco. In addition, considering the important role of spacial heterogeneity in the epidemiological compartment model, Wu et al. [19] incorporated spatial heterogeneity and

ART into a nonlocal dispersal HIV/AIDS epidemic model and studied the adjoint effect of spatial heterogeneity and ART treatments on the dynamics of HIV/AIDS in China. Additionally, Wang et al. [20] divided HIV infectious people into three classes based on the CD4+ T cell count and investigated the synthetic effect of PrEP and ART on HIV transmission among MSM in a heterogenous environment. Moreover, in order to study the effect of environmental interference on the HIV/AIDS transmission, Liu proposed a higher order stochastically perturbed Susceptible-Infectious-Chronic-AIDS (SICA) epidemic model given the sufficient conditions for the extinction of the disease and the existence of an ergodic stationary distribution [22]. Zhai and Wei established a stochastic HIV/AIDS model that involved the susceptible with protection awareness within a total population and ART treatments among MSM [21].

Although the above-mentioned research works has considered many factors (e.g., PrEP, ART, spatial heterogeneity, white noise, and so on) that affect the spread of HIV/AIDS and achieved rich theoretical results. To the best of our knowledge, few HIV/AIDS epidemic models have been proposed to study the adjoint effect of VCT and PrEP treatments, alongside an optimal control on HIV infections among MSM group. The corresponding flow diagram is shown in Figure 1.

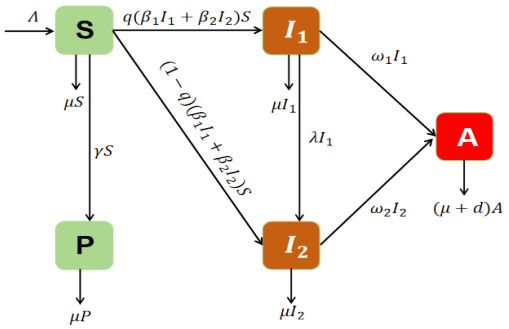

Figure 1. The final scale of the disease under the different control variable.

With these consideration, we can finally construct the following the HIV/AIDS epidemic model among MSM:

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \beta_1 I_1 S - \beta_2 I_2 S - \mu S - \gamma S, \\ \dfrac{dI_1}{dt} = q(\beta_1 I_1 S + \beta_2 I_2 S) - \lambda I_1 - \omega_1 I_1 - \mu I_1, \\ \dfrac{dI_2}{dt} = (1-q)(\beta_1 I_1 S + \beta_2 I_2 S) + \lambda I_1 - \omega_2 I_2 - \mu I_2, \\ \dfrac{dA}{dt} = \omega_1 I_1 + \omega_2 I_2 - \mu A - dA, \\ \dfrac{dP}{dt} = \gamma S - \mu P, \end{cases} \tag{1.1}$$

where the total MSM populations are divided into five epidemiological classes: Susceptible individuals (S), infected individuals without VCT awareness (I_1), infected individuals with VCT awareness (I_2), individuals with AIDS (A), individuals with PrEP treatment (P). The main infection routes are as follows: (i) The susceptible individuals are infected by infected individuals without

VCT awareness and infected individuals with VCT awareness at a rate β_1 and β_2, respectively, and become infected individuals; (ii) infected individuals enter the infectious without a VCT awareness compartment with a proportion p and the infectious with a VCT awareness compartment with a proportion $(1-q)$, $q \in [0,1]$; (iii) both infected individuals without VCT awareness and infected individuals with VCT awareness develop into AIDS patients at the rate ω_1 and ω_2, respectively; (iv) infected individuals without VCT awareness convert to infected individuals with VCT awareness at the rate λ; (v) the susceptible individuals take PrEP treatment at the rate γ. In addition, Λ and d are the recruitment rate of the susceptible individuals and the rate of disease-related death at the stage AIDS, respectively, and u is the natural mortality rate for all individuals. To make things not too complicated, we use the simplest growth term for MSM populations and assume the following:

- The interaction among susceptible individuals, infected individuals without VCT awareness, and infected individuals with VCT awareness fulfills the bilinear infection mechanism;
- Patients entering the AIDS stage will not have high-risk sexual behaviors with susceptible individuals because they have a good understanding of their own infection status and a good sense of consciousness.

The goal of this paper is to provide a comprehensive analysis of the dynamics of the model (1.1), and aims to study the effect of multiple transmission pathways, PrEP treatment, and VCT treatment on the disease spread. We shall solve the following basic problems: (i) Is the solution of model (1.1) positive and bounded; (ii) how to characterize the mutual effects of the PrEP treatment rate and the VCT coverage rate on the basic reproduction number; (iii) what are the threshold dynamics (including disease extinction and persistence) that rely on the basic reproduction number; and (iv) is there an optimal control strategy for PrEP and VCT treatments?

The structure of this paper is as follows: In Section 2, we present some preliminary results to solve the basic problem (1); in Section 3, the basic reproduction number is computed using the next-generation matrix method; we further analyze the threshold dynamics of the model in Section 4; in Section 5, we formulate an optimal control problem, thereby defining the control variables, objectives, and constraints; in Section 6, we perform numerical simulations to verify the theoretical results obtained in this paper and investigate what actions should be taken for disease control and prevention; and the paper concludes with a brief summary.

2. Preliminary results

Lemma 1. *For any initial value* $(S(0), I_1(0), I_2(0), A(0), P(0)) \in R^5_+$*, system* (1.1) *has a unique global positive solution for all* $t \in [0, \infty)$.

Proof. Let

$$L(t) = \min\{S(t), I_1(t), I_2(t), A(t), P(t)\};$$

then, $L(0) > 0$ for any $t \geq 0$. Suppose there exists a $t_1 > 0$ such that $L(t_1) = 0$ and $L(t) > 0$ for all $t \in [0, t_1)$. If $L(t_1) = S(t)$, then $I_1(t) \geq 0, I_2(t) \geq 0, A(t) \geq 0$, and $P(t) \geq 0$ for all $t \in [0, t_1]$. According to the first equation in system (1.1), we have

$$\frac{dS}{dt} \geq -\beta_1 I_1 S - \beta_2 I_2 S - (\mu + \gamma)S, \text{ for all } t \in [0, t_1];$$

thus,

$$0 = S(t_1) \geq S(0)e^{-\int_0^{t_1}[\beta_1 I_1+\beta_2 I_2+(\mu+\gamma)]dt} > 0.$$

There is a contradiction here. Thus, $S(t) > 0$ for all $t \geq 0$. Similarity, we can prove $I_1(t) > 0, I_2(t) > 0, A(t) > 0, P(t) > 0$ for all $t \geq 0$. □

Lemma 2. *The following feasible region*

$$\Omega = \left\{S(t), I_1(t), I_2(t), A(t), P(t) \in R_+^5 : 0 \leq N(t) \leq \frac{\Lambda}{\mu},\ 0 \leq P(t) \leq \frac{\Lambda\gamma}{\mu^2}\right\}$$

is positively invariant set for system (1.1) *with the initial values* $(S(0), I_1(0), I_2(0), A(0), P(0)) \in R_+^5$.

Proof. Denote $N(t) = S(t) + I_1(t) + I_2(t) + A(t)$. According to the first four equations of system (1.1), we have the following:

$$\frac{dN}{dt} = \Lambda - dN - \gamma S - dA \leq \Lambda - \mu N.$$

Furthermore, we have the following:

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

By the fifth equation of system (1.1), we have the following:

$$\frac{dP}{dt} = \gamma S - \mu P \leq \frac{\Lambda\gamma}{\mu} - \mu P.$$

Similarity, we get

$$\limsup_{t\to\infty} P(t) \leq \frac{\Lambda\gamma}{\mu^2},$$

which completes the proof.

□

3. Basic reproduction number

The basic reproduction number R_0 is a core concept in studying epidemic models, which is crucial to understand the transmission dynamics of diseases and to develop effective prevention and control strategies. If $R_0 > 1$, then it means that the disease may continue to spread in the population and control measures need to be taken to reduce the transmission speed; if $R_0 < 1$, then the disease may resolve on its own. In addition, studying the basic reproduction number R_0 can help international health organizations and governments develop coordinated international response strategies. By sharing and comparing the estimate values R_0 from different regions, we can better understand the transmission patterns and risks of the diseases on a global scale, and promote cross-border cooperation and resource sharing. Therefore, in this subsection, we first obtain the basic reproduction number R_0 by using the next generation matrix method [23], and prove the global asymptotic stability of the disease-free equilibrium of system (1.1) by constructing a Lyapunov function.

First, it is easy to find that system (1.1) always exists in the disease-free equilibrium as follows:

$$E^0 = (S^0, 0, 0, 0, P^0), \text{ where } S^0 = \frac{\Lambda}{\mu+\gamma}, \quad P^0 = \frac{\Lambda\gamma}{\mu(\mu+\gamma)}.$$

For system (1.1), we define the following:

$$\mathcal{F} = \begin{pmatrix} q(\beta_1 I_1 S + \beta_2 I_2 S) \\ (1-q)(\beta_1 I_1 S + \beta_2 I_2 S) \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (\lambda+\omega_1+\mu)I_1 \\ -\lambda I_1 + \omega_2 I_2 + \mu I_2 \end{pmatrix}.$$

Then,

$$\mathbf{F} = \begin{pmatrix} q\beta_1 S^0 & q\beta_2 S^0 \\ (1-q)\beta_1 S^0 & (1-q)\beta_2 S^0 \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} \lambda+\omega_1+\mu & 0 \\ -\lambda & \omega_2+\mu \end{pmatrix}.$$

Thus, we have the following:

$$\mathbf{FV^{-1}} = \begin{pmatrix} \frac{q\beta_1(\omega_2+\mu)+q\beta_2\lambda}{(\lambda+\omega_1+\mu)(\omega_2+\mu)}S^0 & \frac{q\beta_2}{\omega_2+\mu}S^0 \\ \frac{(1-q)\beta_1(\omega_2+\mu)+(1-q)\beta_2\lambda}{(\lambda+\omega_1+\mu)(\omega_2+\mu)}S^0 & \frac{(1-q)\beta_2}{\omega_2+\mu}S^0 \end{pmatrix}.$$

By the next generation matrix method [23], the basic reproduction number R_0 is defined as the spectral radius (dominant eigenvalue) of the matrix $\mathbf{FV^{-1}}$, which is denoted by $\rho(\mathbf{FV}^{-1})$. That is,

$$\begin{aligned} R_0 =& \rho(\mathbf{FV}^{-1}) \\ =& \frac{q\beta_1\Lambda(\omega_2+\mu) + q\beta_2\Lambda\lambda + (1-q)\beta_2\Lambda(\lambda+\omega_1+\mu)}{(\lambda+\omega_1+\mu)(\omega_2+\mu)(\mu+\gamma)}. \end{aligned}$$

Second, we discuss the local asymptotic stability of the disease-free equilibrium E^0.

Theorem 1. *If $R_0 < 1$, then the disease-free equilibrium E^0 of system (1.1) is locally asymptotically stable.*

Proof. The stability of the disease-free equilibrium E^0 of system (1.1) is determined by the eigenvalues of the following Jacobian matrix:

$$\mathbf{J}(E^0) = \begin{pmatrix} -\mu-\gamma & -\frac{\beta_1\Lambda}{\mu+\gamma} & -\frac{\beta_2\Lambda}{\mu+\gamma} & 0 & 0 \\ 0 & \frac{q\beta_1\Lambda}{\mu+\gamma} - \lambda - \omega_1 - \mu & \frac{q\beta_2\Lambda}{\mu+\gamma} & 0 & 0 \\ 0 & \frac{(1-q)\beta_1\Lambda}{\mu+\gamma} + \lambda & \frac{(1-q)\beta_2\Lambda}{\mu+\gamma} - \omega_2 - \mu & 0 & 0 \\ 0 & \omega_1 & \omega_2 & -\mu-d & 0 \\ \gamma & 0 & 0 & 0 & -\mu \end{pmatrix}.$$

Through a calculation, the eigenvalues of the above matrix are given by the following:

$$\xi_1 = -\mu, \quad \xi_2 = -(\mu+\gamma), \quad \xi_3 = -(\mu+d), \quad \xi_{4,5} = \frac{-b \pm \sqrt{b^2-4c}}{2},$$

where

$$\begin{aligned} b &= \lambda+\omega_1+\omega_2+2\mu) - \frac{q\beta_1\Lambda}{\mu+\gamma} - \frac{(1-q)\beta_2\Lambda}{\mu+\gamma}, \\ c &= (\lambda+\omega_1+\mu)(\omega_2+\mu) - (\lambda+\omega_1+\mu)\frac{(1-q)\beta_2\Lambda}{\mu+\gamma} - (\omega_2+\mu)\frac{q\beta_1\Lambda}{\mu+\gamma} - \frac{q\lambda\beta_2\Lambda}{\mu+\gamma}. \end{aligned}$$

By $R_0 < 1$, it is easy to verify that $b > 0$ and $c > 0$. From the Routh-Hurwitz criterion [24], the characteristic roots of the above characteristic equation are all negative real numbers. Thus, the disease-free equilibrium E^0 of system (1.1) is locally asymptotically stable. □

4. Threshold dynamics

In this section, we use basic reproduction number to discuss the dynamic behavior of the system (1.1), including stability and uniform persistence.

4.1. Stability of disease-free equilibrium

Next, we discuss the global asymptotic stability of the disease-free equilibrium E^0.

Theorem 2. *If $R_0 \leq 1$, then the disease-free equilibrium E^0 of system (1.1) is globally asymptotically stable.*

Proof. Let $\mathbf{y} = (I_1, I_2)^T$, and

$$\bar{\mathbf{F}} = \begin{pmatrix} q\beta_1\frac{\Lambda}{\mu+\gamma} & q\beta_2\frac{\Lambda}{\mu+\gamma} \\ (1-q)\beta_1\frac{\Lambda}{\mu+\gamma} & (1-q)\beta_2\frac{\Lambda}{\mu+\gamma} \end{pmatrix}, \quad \bar{\mathbf{V}} = \begin{pmatrix} \lambda+\omega_1+\mu & 0 \\ -\lambda & \omega_2+\mu \end{pmatrix}.$$

Then, system (1.1) satisfies the inequality $\frac{d\mathbf{y}}{dt} \leq (\bar{\mathbf{F}} - \bar{\mathbf{V}})\mathbf{y}$ in Ω.

Because $R_0 = \rho(\bar{\mathbf{F}}\bar{\mathbf{V}}^{-1}) = \rho(\bar{\mathbf{V}}^{-1}\bar{\mathbf{F}})$, thus $\bar{\mathbf{F}}\bar{\mathbf{V}}^{-1}\bar{\mathbf{F}} = \bar{\mathbf{R}}_\mathbf{0}\bar{\mathbf{F}}$; here, $\bar{\mathbf{R}}_\mathbf{0}$ is a diagonal matrix composed of eigenvalues.

Define the Lyapunov function $L = \bar{\mathbf{F}}\bar{\mathbf{V}}^{-1}\mathbf{y}$ by taking the derivative as follows:

$$\frac{dL}{dt} = \bar{\mathbf{F}}\bar{\mathbf{V}}^{-1}\frac{d\mathbf{y}}{dt} \leq \bar{\mathbf{F}}\bar{\mathbf{V}}^{-1}(\bar{\mathbf{F}} - \bar{\mathbf{V}})\mathbf{y} = (\bar{\mathbf{R}}_\mathbf{0} - 1)\bar{\mathbf{F}}\mathbf{y}.$$

Therefore, $dL/dt \leq 0$ when $R_0 \leq 1$. At this time, $\bar{\mathbf{F}}\mathbf{y} = 0$ when $dL/dt = 0$, which means that $I_1 = I_2 = 0, S = \Lambda/(\mu+\gamma)$, that is, the maximum invariant set of system (1.1) is the single point set $\{(\Lambda/(\mu+\gamma), 0, 0)\}$ when $dL/dt = 0$. According to the Lasalle's invariant principle [25], E^0 is globally asymptotically stable on Ω. □

4.2. The existence of endemic equilibrium and uniform persistence of system

In this subsection, we prove the existence and uniqueness of the endemic equilibrium of system (1.1), and discuss the uniform persistence of system (1.1). First, we assume that system (1.1) has an endemic equilibrium $E^* = (S^*, I_1^*, I_2^*, A^*, P^*)$, which satisfies the following equations:

$$\begin{cases} \Lambda - \beta_1 I_1^* S^* - \beta_2 I_2^* S^* - \mu S^* - \gamma S^* = 0, \\ q(\beta_1 I_1^* S^* + \beta_2 I_2^* S^*) - \lambda I_1^* - \omega_1 I_1^* - \mu I_1^* = 0, \\ (1-q)(\beta_1 I_1^* S^* + \beta_2 I_2^* S^*) + \lambda I_1^* - \omega_2 I_2^* - \mu I_2^* = 0, \\ \omega_1 I_1^* + \omega_2 I_2^* - \mu A^* - dA^* = 0, \\ \gamma S^* - \mu P^* = 0. \end{cases} \tag{4.1}$$

According to the second and third equations of system (4.1),

$$S^* = \frac{(\lambda+\omega_1+\mu)(\omega_2+\mu)}{q[\beta_1(\omega_2+\mu)+\beta_2\lambda] + (\lambda+\omega_1+\mu)(1-q)\beta_2} = \frac{\Lambda}{(\mu+\gamma)R_0}.$$

By adding the first three equations of system (4.1) together, we have the following:

$$\Lambda - (\mu + \gamma)S^* - (\omega_1 + \mu)I_1^* - (\omega_2 + \mu)I_2^* = 0.$$

According to the second equation of (4.1), we obtain the following:

$$\Lambda - \frac{\Lambda(\mu + \gamma)}{(\mu + \gamma)R_0} = (\omega_1 + \mu)I_1^* + (\omega_2 + \mu)\frac{I_1^*[(1 - q)\beta_1 S^* + \lambda]}{(\omega_2 + \mu) - (1 - q)\beta_2 S^*}. \tag{4.2}$$

Because

$$(\omega_2 + \mu) - (1 - q)\beta_2 S^* = (\omega_2 + \mu)\left\{1 - \frac{(1 - q)\beta_2(\lambda + \omega_1 + \mu)}{q[\beta_1(\omega_2 + \mu) + \beta_2\lambda] + (1 - q)\beta_2(\lambda + \omega_1 + \mu)}\right\} > 0,$$

from Eq (4.2), we have

$$I_1^* = \frac{\Lambda(1 - \frac{1}{R_0})}{(\omega_1 + \mu) + (\omega_2 + \mu)\frac{\left[(1-q)\beta_1 \frac{\Lambda}{(\mu+\gamma)R_0} + \lambda\right]}{(\omega_2+\mu)-(1-q)\beta_2 \frac{\Lambda}{(\mu+\gamma)R_0}}}.$$

To summarize, system (1.1) has a unique endemic equilibrium $E^* = (S^*, I_1^*, I_2^*, A^*, P^*)$ when $R_0 > 1$, and the details are as follows:

$$S^* = \frac{\Lambda}{(\mu + \gamma)R_0}, \qquad I_1^* = \frac{\Lambda(1 - \frac{1}{R_0})}{(\omega_1 + \mu) + (\omega_2 + \mu)\frac{\left[(1-q)\beta_1 \frac{\Lambda}{(\mu+\gamma)R_0} + \lambda\right]}{(\omega_2+\mu)-(1-q)\beta_2 \frac{\Lambda}{(\mu+\gamma)R_0}}},$$

$$I_2^* = \frac{(1 - q)\beta_1 S^* + \lambda}{(\omega_2 + \mu) - (1 - q)\beta_2 S^*} \times \frac{\Lambda(1 - \frac{1}{R_0})}{(\omega_1 + \mu) + (\omega_2 + \mu)\frac{\left[(1-q)\beta_1 \frac{\Lambda}{(\mu+\gamma)R_0} + \lambda\right]}{(\omega_2+\mu)-(1-q)\beta_2 \frac{\Lambda}{(\mu+\gamma)R_0}}},$$

$$A^* = \frac{\omega_1 I_1^* + \omega_2 I_2^*}{\mu + d}, \qquad P^* = \frac{\Lambda\gamma}{\mu(\mu + \gamma)R_0}.$$

Next, we discuss the uniform persistence of system (1.1).

Theorem 3. *If $R_0 > 1$, then system (1.1) is uniformly persistent, that is, there exist a positive constant ϵ such that for the initial value $S(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, A(0) \geq 0, P(0) \geq 0$, the solutions $(S(t), I_1, I_2(t), A(t), P(t))$ of system (1.1) satisfies*

$$\liminf_{t\to\infty} I_1(t) \geq \epsilon, \liminf_{t\to\infty} I_2(t) \geq \epsilon, \liminf_{t\to\infty} A(t) \geq \epsilon.$$

Proof. Define

$$\mathbf{X} = \left\{(S, I_1, I_2, A, P)|S \geq 0, I_1 \geq 0, I_2 \geq 0, A \geq 0, P \geq 0, 0 \leq S + I_1 + I_2 + A \leq \frac{\Lambda}{\mu}, 0 \leq P \leq \frac{\Lambda\gamma}{\mu^2}\right\},$$

$$\mathbf{X_0} = \{(S, I_1, I_2, A, P)|I_1 > 0, I_2 > 0, A > 0\},$$

$$\partial\mathbf{X_0} = \mathbf{X}\backslash\mathbf{X_0}.$$

Then, it is only necessary to prove that system (1.1) is uniformly persistent with respect to $(\mathbf{X_0}, \partial\mathbf{X_0})$.

Obviously, $\mathbf{X}$ and $\mathbf{X_0}$ are a positive invariant set, and $\partial\mathbf{X_0}$ is closed on $\mathbf{X}$. System (1.1) is point dissipative. Define

$$M_\partial = \{(S(0), I_1(0), I_2(0), A(0), P(0))|S(t), I_1(t), I_2(t), A(t), P(t) \in \partial\mathbf{X_0}, \text{for any } t \geq 0\}.$$

We prove that

$$M_\partial = \{(S(0), 0, 0, 0, P(0))|S(0) \geq 0, P(0) \geq 0\}.$$

Since $\{(S(0), 0, 0, 0, P(0))|S(0) \geq 0, P(0) \geq 0\} \subseteq M_\partial$ is true, we only need to prove

$$M_\partial \subseteq \{(S(0), 0, 0, 0, P(0))|S(0) \geq 0, P(0) \geq 0\}.$$

Let $(S(0), I_1(0), I_2(0), A(0), P(0)) \in M_\partial$; then, we prove $I_1(t) = 0, I_2(t) = 0, A(t) = 0$ when $t \geq 0$. Assume there exist $t_0 \geq 0$, such that one of the following hold true:

$$(i)\ I_1(t) > 0, \qquad (ii)\ I_2(t) > 0, \qquad (iii)\ A(t) > 0.$$

For any $t \geq t_0$, if (i) is true, then we solve system (1.1) as follows:

$$\begin{aligned} I_1(t) &\geq I_1(t_0)e^{-(\lambda+\omega_1+\mu)(t-t_0)} > 0, \\ I_2(t) &\geq I_2(t_0)e^{-(\omega_2+\mu)(t-t_0)} \geq 0, \\ A(t) &\geq A(t_0)e^{-(\mu+d)(t-t_0)} \geq 0. \end{aligned}$$

Thus, $(S(t), I_1(t), I_2(t), A(t), P(t)) \notin \partial\mathbf{X_0}$ for any $t \geq t_0$, which contradicts $(S(0), I_1(0), I_2(0), A(0), P(0)) \in M_\partial$. Similarly, this proves (ii) and (iii).

Obviously, there is only one equilibrium E_0 in M_∂. From the above conclusion, it follows that E_0 is a single point set in $\mathbf{X}$, and $\mathbf{W^s}(E_0) \cap \mathbf{X_0} = \varnothing$. Every orbital in M_∂ tends to E_0, and E_0 is non-cyclic in M_∂. According to [26, Theorem 4.6], system (1.1) is uniformly persistent with respect to $(\mathbf{X_0}, \partial\mathbf{X_0})$. □

5. The optimal control problems

In this section, we prove the existence of the optimal solution. First, we use three control variables-$u_1(t)$, $u_2(t)$ and $u_3(t)$-to reduce the number of HIV/AIDS individuals. $u_1(t)$ represents the control variable that enhances the PrEP treatment. $u_2(t)$ represents the control variable that enhances the effect of VCT awareness education during the susceptible period. $u_3(t)$ represents the control variable that enhances the effect of VCT awareness education during the infected period. First, define the control set

$$U = \{u(t) = (u_1(t), u_2(t), u_3(t)) :\ u_i(t) \text{ is measurable},\ 0 \leq u_i(t) \leq 1, t \in [0, T], i = 1, 2, 3\},$$

where T is the end time of implementing the controls. Then, the optimal control model is as follows:

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \beta_1 I_1 S - \beta_2 I_2 S - \mu S - (1 + u_1)\gamma S, \\ \dfrac{dI_1}{dt} = (1 - u_2)q(\beta_1 I_1 S + \beta_2 I_2 S) - \lambda I_1 - \omega_1 I_1 - \mu I_1 - u_3 I_1(t), \\ \dfrac{dI_2}{dt} = (1 - q)(\beta_1 I_1 S + \beta_2 I_2 S) + \lambda I_1 - \omega_2 I_2 - \mu I_2 + u_3 I_1(t), \\ \dfrac{dA}{dt} = \omega_1 I_1 + \omega_2 I_2 - \mu A - dA, \\ \dfrac{dP}{dt} = (1 + u_1)\gamma S - \mu P. \end{cases} \tag{5.1}$$

The initial conditions satisfy the following:

$$S(0) \geq 0,\ I_1(0) \geq 0,\ I_2(0) \geq 0,\ A(0) \geq 0,\ P(0) \geq 0. \tag{5.2}$$

Thus, we define the objective function as follows:

$$J(u_1(t), u_2(t), u_3(t)) = \int_0^t [S(t) + I_1(t) + I_2(t) + \frac{1}{2}(c_1u_1^2(t) + c_2u_2^2(t) + c_3u_3^2(t))]dt, \tag{5.3}$$

where, c_1, c_2 and c_3 represent the weight of the treatment costs.

5.1. The existence of optimal control pairs

In this subsection, we study the existence of optimal control pairs in system (1.1) using the results of Fleming and Rishel [27].

Theorem 4. *For the objective function $J(u)$, it has a optimal solution $u^* = \{u_1^*, u_2^*, u_3^*\} \in U$, such that*

$$J(u^*) = J(u_1^*, u_2^*, u_3^*) = \min_{u \in U} J(u_1, u_2, u_3).$$

Proof. In order to prove the existence of optimal control, the following conditions must be met:
(a) The control variables and the related state variables are non negative values;
(b) the control set U is convex and closed;
(c) the right side of system (5.1) is bounded, which is a linear function of the control variable and the state variable;
(d) the integrand of the objective function $J(u)$ on U is convex;
(e) there exist constants $b_1 > 0, b_2 > 0$, and $\alpha > 1$ such that the integrant of the objective functional

$$L(t, u_1, u_2, u_3) = S(t) + I_1(t) + I_2(t) + \frac{1}{2}(c_1u_1^2(t) + c_2u_2^2(t) + c_3u_3^2(t))$$

satisfies

$$L(t, u_1, u_2, u_3) \geq b_1[u_1^2(t) + u_2^2(t) + u_3^2(t)]^{\frac{\alpha}{2}} - b_2.$$

Due to the non-negative values of the control and state variables, the control variable $u_1(t), u_2(t), u_3(t) \in U$ is convex closed. At the same time, the boundedness of the optimal system determines the compactness required for the existence of the optimal control. Therefore, $L(t, u_1, u_2, u_3)$ is convex on the control set U, that is, the conditions (a), (b), and (d) are satisfied.

Additionally, because

$$\frac{dS}{dt} \leq \Lambda,\ \frac{dI_1}{dt} \leq (1 - u_2)q(\beta_1 I_1 S + \beta_2 I_2 S),\ \frac{dI_2}{dt} \leq (1 - q)(\beta_1 I_1 S + \beta_2 I_2 S) + \lambda I_1 + u_3 I_1,$$
$$\frac{dA}{dt} \leq \omega_1 I_1 + \omega_2 I_2,\ \frac{dP}{dt} \leq (1 + u_1)\gamma S,$$

condition (c) is satisfied.

For condition (e), there exist $b_1 = \min\{\frac{c_1}{2}, \frac{c_2}{2}, \frac{c_3}{2}\}, b_2 \in R^+, \alpha = 2$ such that

$$L(t, u_1, u_2, u_3) \geq b_1[u_1^2(t) + u_2^2(t) + u_3^2(t)]^{\frac{\alpha}{2}} - b_2,$$

which proves the existence of the optimal control. □

5.2. Characterization of optimal controls

The Pontryagin maximum principle [28] is applied to determine the exact formula for optimal control of $u_1(t)$ and $u_2(t)$. In order to find the optimal solution, we construct the following Lagrange function L and Hamiltonian function H:

$$L(t, \Phi, u) = S(t) + I_1(t) + I_2(t) + \frac{1}{2}(c_1 u_1^2(t) + c_2 u_2^2(t) + c_3 u_3^2(t)),$$

$$\begin{aligned} H(t, \Phi, u) = {} & S(t) + I_1(t) + I_2(t) + \frac{1}{2}c_1 u_1^2(t) + \frac{1}{2}c_2 u_2^2(t) + \frac{1}{2}c_3 u_3^2(t) \\ & + \lambda_S \cdot [\Lambda - \beta_1 I_1 S - \beta_2 I_2 S - \mu S - (1+u_1)\gamma S] \\ & + \lambda_{I_1} \cdot [(1-u_2)q(\beta_1 I_1 S + \beta_2 I_2 S) - \lambda I_1 - \omega_1 I_1 - \mu I_1 - u_3(t) I_1] \\ & + \lambda_{I_2} \cdot [(1-q)(\beta_1 I_1 S + \beta_2 I_2 S) + \lambda I_1 - \omega_2 I_2 - \mu I_2 + u_3 I_1] \\ & + \lambda_A \cdot [\omega_1 I_1 + \omega_2 I_2 - \mu A - dA] \\ & + \lambda_P \cdot [(1+u_1)\gamma S - \mu P], \end{aligned}$$

where $\lambda_S, \lambda_{I_1}, \lambda_{I_2}, \lambda_A$, and λ_P are the associated adjoints for the states S, I_1, I_2, A, and P, respectively. By taking the appropriate partial derivatives of the Hamiltonian function with respect to the relevant state variables and control variables, the adjoint equations are obtained.

Theorem 5. *If there are optimal control pairs (u_1^*, u_2^*, u_3^*) and solutions $S^*(t), I_1^*(t), I_2^*(t), A^*(t), P^*(t)$ of the corresponding state system* (5.1) *that minimizes the objective functional $J(u_1^*, u_2^*, u_3^*)$ on U, then there exist adjoint variables $\lambda_S, \lambda_{I_1}, \lambda_{I_2}, \lambda_A, \lambda_P$ that satisfy*

$$-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial i},$$

with the terminal conditions

$$\lambda_i(T) = 0, \ i = S, I_1, I_2, A, P.$$

The optimality conditions is

$$\frac{\partial H}{\partial u_j} = 0, \ j = 1, 2, 3.$$

Furthermore, (u_1^*, u_2^*, u_3^*) can be obtained by the following equations:

$$\begin{aligned} u_1^* &= \min\left\{1, \ \max\{0, \frac{(\lambda_S - \lambda_P)\gamma S^*}{c_1}\}\right\}, \\ u_2^* &= \min\left\{1, \ \max\{0, \frac{\lambda_{I_1} q(\beta_1 S^* I_1^* + \beta_2 S^* I_2^*)}{c_2}\}\right\}, \\ u_3^* &= \min\left\{1, \ \max\{0, \frac{(\lambda_{I_1} - \lambda_{I_2}) I_1^*}{c_3}\}\right\}. \end{aligned}$$

Proof. According to the existence of the optimal control solution based on Pontryagin's maximum principle [28], we obtain the adjoint system, which can be written as follows:

$$\frac{d\lambda_S}{dt} = -\frac{\partial H}{\partial S} = -1 + \lambda_S \cdot [\beta_1 I_1^* + \beta_2 I_2^* + \mu + (1+u_1)\gamma] - \lambda_{I_1} \cdot (1-u_2) q(\beta_1 I_1^* + \beta_2 I_2^*) - \lambda I_2 \cdot (1-q)$$

$$
\begin{aligned}
&(\beta_1 I_1^* + \beta_2 I_2^*) - \lambda_P \cdot (1+u_1)\gamma,\\
\frac{d\lambda_{I_1}}{dt} = -\frac{\partial H}{\partial I_1} &= -1 + \lambda_S \cdot \beta_1 S^* - \lambda_{I_1} \cdot [(1-u_2)q\beta_1 S^* - \lambda - \omega_1 - \mu - u_3] - \lambda_{I_2} \cdot [(1-q)\beta_1 S^* + \lambda + u_3]\\
&\quad - \lambda_A \cdot \omega_1,\\
\frac{d\lambda_{I_2}}{dt} = -\frac{\partial H}{\partial I_2} &= -1 + \lambda_S \cdot \beta_2 S^* - \lambda_{I_1} \cdot (1-u_2)q\beta_2 S^* - \lambda_{I_2} \cdot [(1-q)\beta_2 S^* - \omega_2 - \mu] - \lambda_A \cdot \omega_2,\\
\frac{d\lambda_A}{dt} = -\frac{\partial H}{\partial A} &= \lambda_A(\mu + d),\\
\frac{d\lambda_P}{dt} = -\frac{\partial H}{\partial P} &= \lambda_P \mu.
\end{aligned}
$$

In addition, we obtain the following optimal conditions by the differential Hamiltonian:

$$
\frac{\partial H}{\partial u_1} = c_1 u_1^*(t) - \lambda_S \cdot \gamma S^* + \lambda_P \cdot \gamma S^*, \quad \frac{\partial H}{\partial u_2} = c_2 u_2^*(t) - \lambda_{I_1} q(\beta_1 S^* I_1^* + \beta_2 S^* I_2^*) = 0,
$$

$$
\frac{\partial H}{\partial u_3} = c_3 u_3^*(t) - \lambda_{I_1} \cdot I_1^* + \lambda_{I_2} \cdot I_1^*.
$$

Thus, there are

$$
u_1^* = \frac{(\lambda_S - \lambda_P)\gamma S^*}{c_1}, \quad u_2^* = \frac{\lambda_{I_1} q(\beta_1 S^* I_1^* + \beta_2 S^* I_2^*)}{c_2}, \quad u_3^* = \frac{(\lambda_{I_1} - \lambda_{I_2}) I_1^*}{c_3}.
$$

Moreover, because the control variables satisfy $0 \le u_i \le 1, i = 1, 2, 3$, the optimal control is as follows:

$$
\begin{cases}
u_1^* = \min\left\{1,\ \max\{0, \dfrac{(\lambda_S - \lambda_P)\gamma S^*}{c_1}\}\right\},\\
u_2^* = \min\left\{1,\ \max\{0, \dfrac{\lambda_{I_1} q(\beta_1 S^* I_1^* + \beta_2 S^* I_2^*)}{c_2}\}\right\},\\
u_3^* = \min\left\{1,\ \max\{0, \dfrac{(\lambda_{I_1} - \lambda_{I_2}) I_1^*}{c_3}\}\right\}.
\end{cases}
$$

□

6. Numerical simulations

We will divide the numerical simulation into two parts. In the first section, we mainly verify the theoretical results in the paper. In the second section, we study the influence of different parameters on the number of patients, and then analyze the influence of different control measures on the final scale of the disease.

First, we select the parameters in Table 1.

Table 1. Values of all parameters in model (1.1).

Parameter	Value	Parameter	Value
Λ	10	β_1	0.003
β_2	0.00012	γ	0.1
λ	0.15	ω_1	0.15
ω_2	0.1	d	0.5
μ	0.0143	q	0.65

6.1. Numerical verification of the theorem

In this subsection, according to the Table 1, we change the parameters to get different R_0 study the trend of the solutions.

First, we take the parameters of Table 1. In this case, we calculate $R_0 = 0.6034$. The solutions of model (1.1) are shown in Figure 2. This corresponds to the result described in Theorem (2).

Second, we take the parameters $\beta_1 = 0.012$, $\beta_2 = 0.001$, $\lambda = 0.15$, $\omega_1 = 0.1$, and $\omega_2 = 0.05$ alongside other parameters shown in Table 1 . In this case, we calculate $R_0 = 2.9564$. The solutions of model (1.1) are shown in Figure 3. This corresponds to the result described in Theorem (3).

6.2. The sensitivity analysis of basic regeneration number

In this subsection, we focus on the sensitivity analysis of the influence of different parameters on the basic regeneration number R_0. From Figure 4, it can be seen that the R_0 value can only be reduced by reducing β_1 or β_2 and increasing γ. Reducing β_1 means reducing the transmission power of individuals without VCT awareness, while reducing β_2 means reducing the transmission power of individuals with VCT awareness. This indicates that HIV transmission can be reduced by providing awareness education to susceptible and infected individuals (e.g., strengthening publicity, organizing activities, setting up AIDS Study Day, organizing knowledge competitions, etc., to guide the public to form a correct understanding). Improving γ means doing a better job in promoting and implementing PrEP treatments. In summary, we can better control diseases through the above measures.

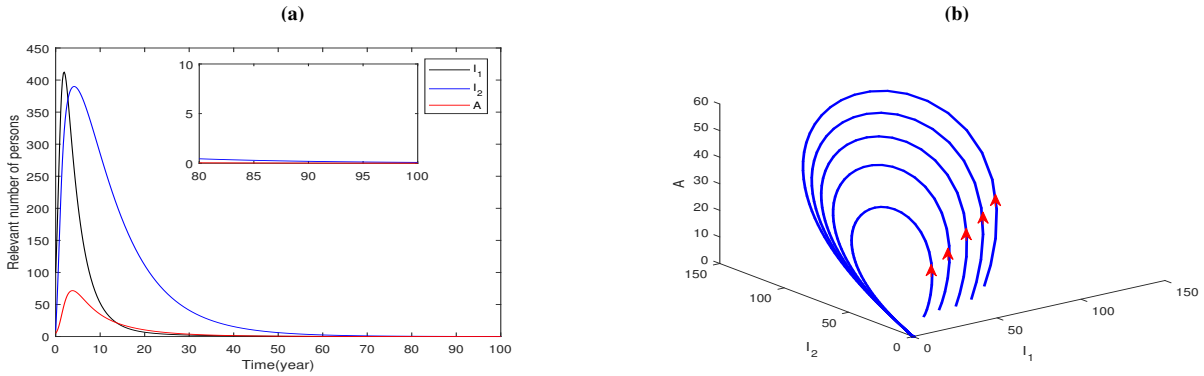

Figure 2. The global asymptotically stability of the disease-free equilibrium E^0.

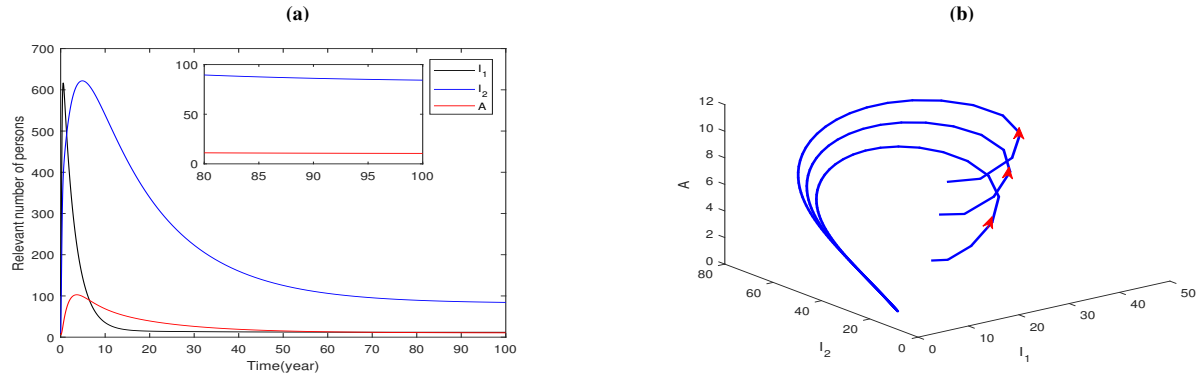

Figure 3. The persistence of system (1.1).

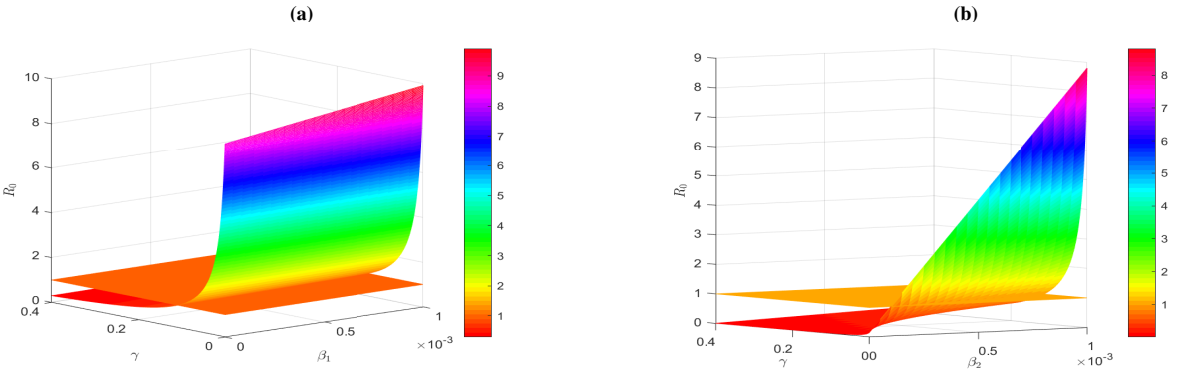

Figure 4. The sensitivity analysis of basic regeneration number R_0.

6.3. The final scale of the infected and AIDS under different parameters

In this subsection, we conduct some numerical simulations to analyze the parameters sensitivity of I_1, I_2, A, which can lead us to get some practical conclusions about the influence of different control measures on the final scale of the infected and AIDS.

First, we analyze the situation during the extinction of the disease.

In Figure 5(a), it shows the final scale of the infected individuals without VCT awareness under the different parameter values λ. As the rates of the infected receiving VCT awareness increase, the peak size of the infected individuals I_1 is reduced. Additionally, in Figure 5(b), as the infected receiving VCT awareness rates increase, the peak size of the infected I_2 is increased. However, from Figure 5(c), we can see that the peak size of AIDS patients eventually decreases with the increase of λ. This indicates that strengthening the awareness education for infected individuals can help control the number of AIDS infections.

Figure 6(a) shows the final scale of the infected individuals without VCT awareness under the different parameter values q. As the rates of the susceptible individuals who do not receive VCT awareness increase, the peak size of the infected individuals I_1 is increased. In Figure 6(b), as the rates of the susceptible individuals who do not receive VCT awareness increase, the peak size of the infected I_2 is decreased. However, from Figure 6(c), we can see that the peak size of AIDS patients eventually increases with the increase of q. This means that VCT awareness education in susceptible individuals is also necessary.

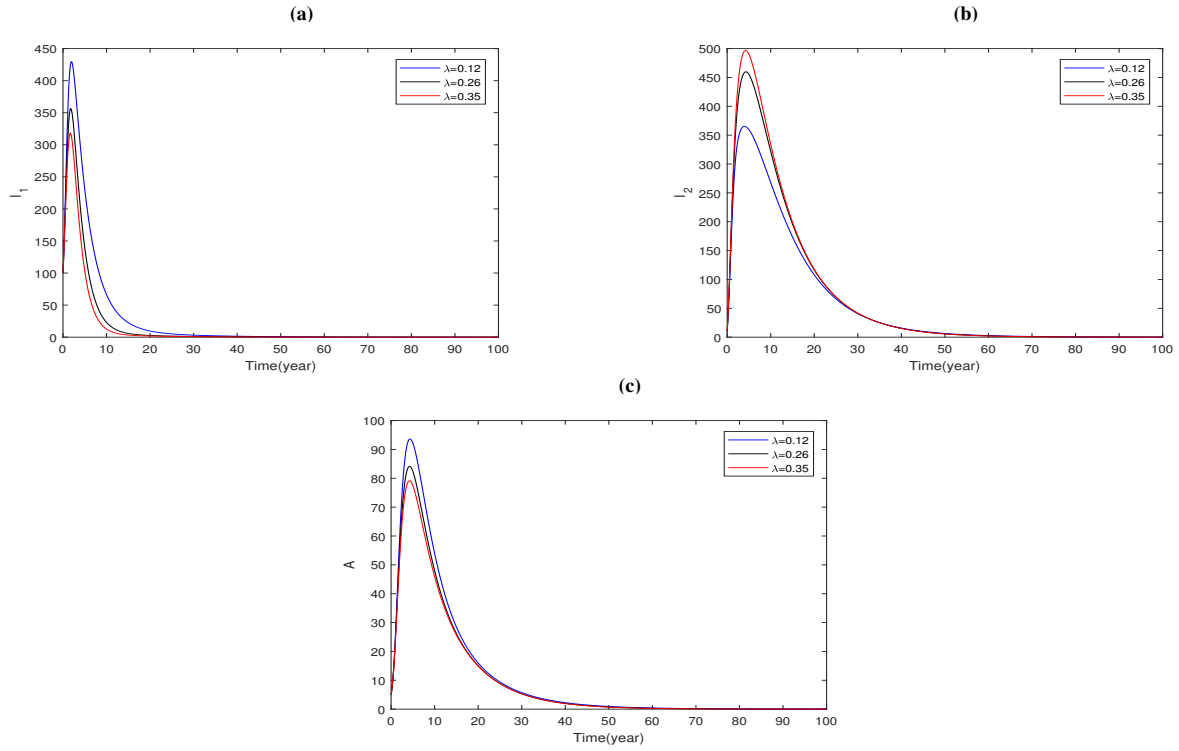

Figure 5. The final scale of the disease under the different parameter values λ.

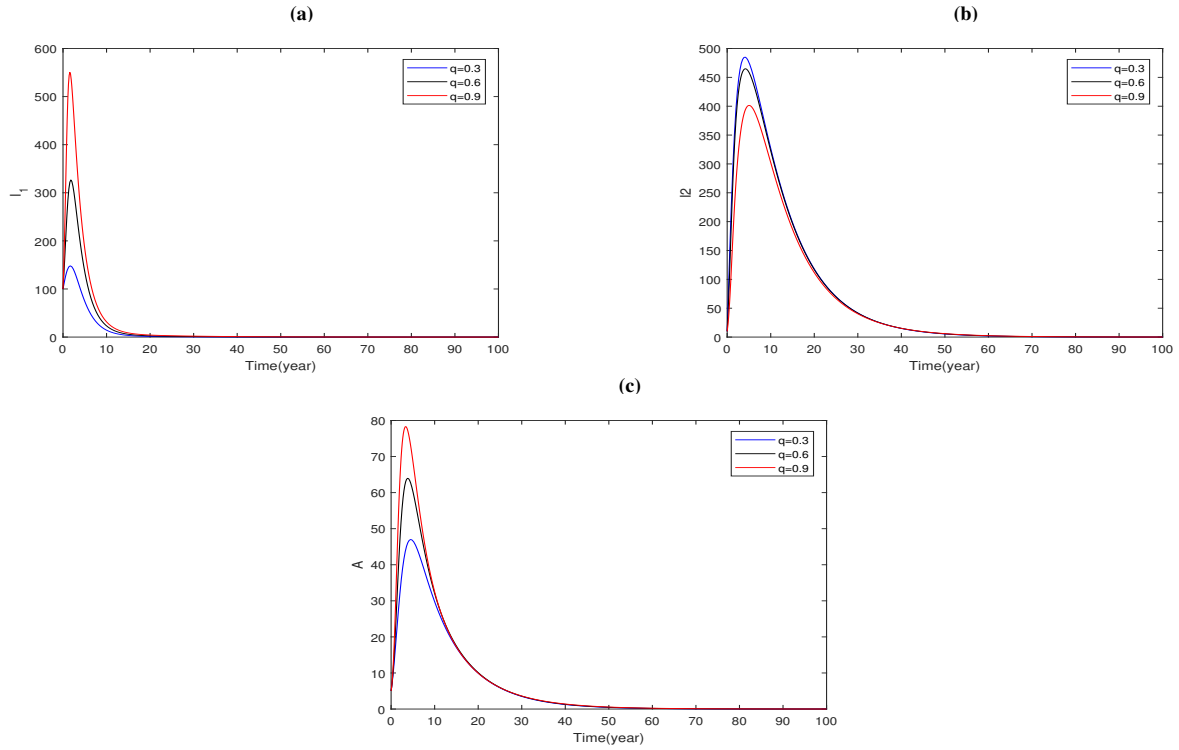

Figure 6. The final scale of the disease under the different parameter values q.

The following is the situation when the disease is uniformly persistent.

Figures 7 and 8 indicate that the impact trend on the infection is basically consistent when the disease is uniformly persistent, regardless if it is parameter λ or parameter q. This further proves the importance of VCT awareness education to control the spread of AIDS. More interestingly, if these measures are continuously strengthened, then they can also play a role in the later stages of infection, as seen from the figures. Additionally, this indicates that controlling the spread of HIV should be a systematic and long-term project.

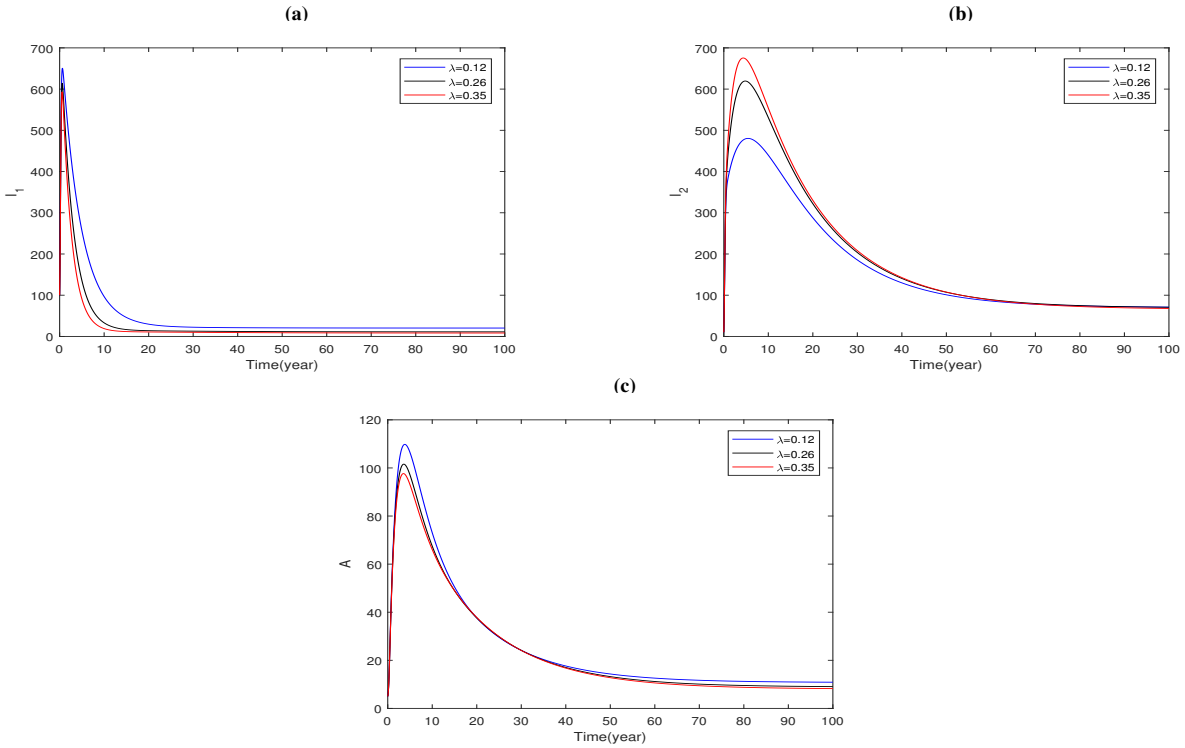

Figure 7. The final scale of the disease under the different parameter values λ.

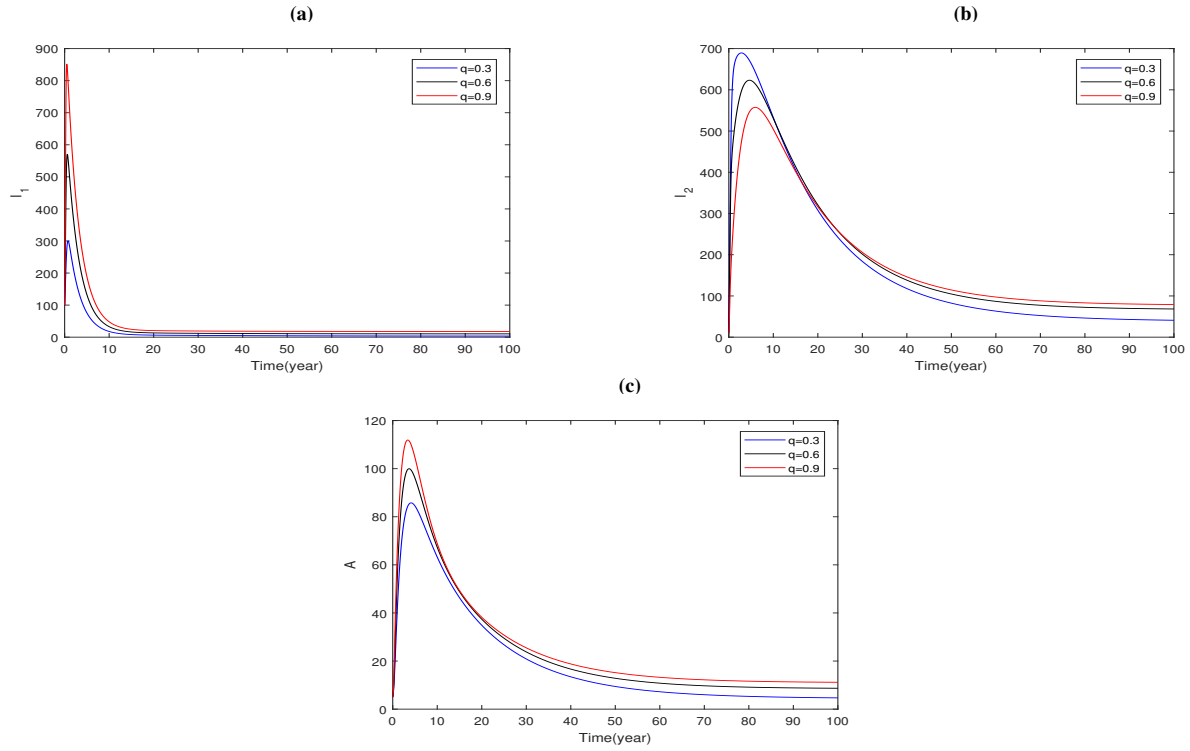

Figure 8. The final scale of the disease under the different parameter values q.

6.4. Numerical simulation of the optimal control problems

In this section, we conduct numerical simulations of the optimal control problems and discuss the changes in the number of infected individuals in the system with or without the control variables.

When the disease is extinct, Figure 9 shows the impact of changes in the different control variables on the number of infected individuals. When only the control variable u_2 increases, the peak number of infected and HIV/AIDS individuals decreases, which indicates that strengthening VCT awareness education during the susceptible period is effective. For example, one can establish a AIDS knowledge learning platform, and reward those who have an excellent academic performance, which can drive more people to participate in learning. When only the control variable u_1, u_3 increases, the peak number I_1 decreases, I_2 increases, and A decreases. This indicates that strengthening PrEP treatment and enhancing VCT awareness education during infection is effective. This can encourage people living with HIV to participate in the exchange meeting and strengthen their determination to defeat the virus. When the control variables u_1, u_2, and u_3 increase, the peak number I_1 decreases, I_2 increases, but the decrease in A is more significant than the situation without strengthening u_2. The increase in the control variables u_1, u_2, and u_3 are effective in preventing HIV/AIDS.

When the disease is persistent, Figure 10 shows the impact of different control variables on the number of infections. The trend of change is similar to when the disease is extinct. Thus, the increase in the control variables u_1, u_2, and u_3 are effective in preventing HIV/AIDS.

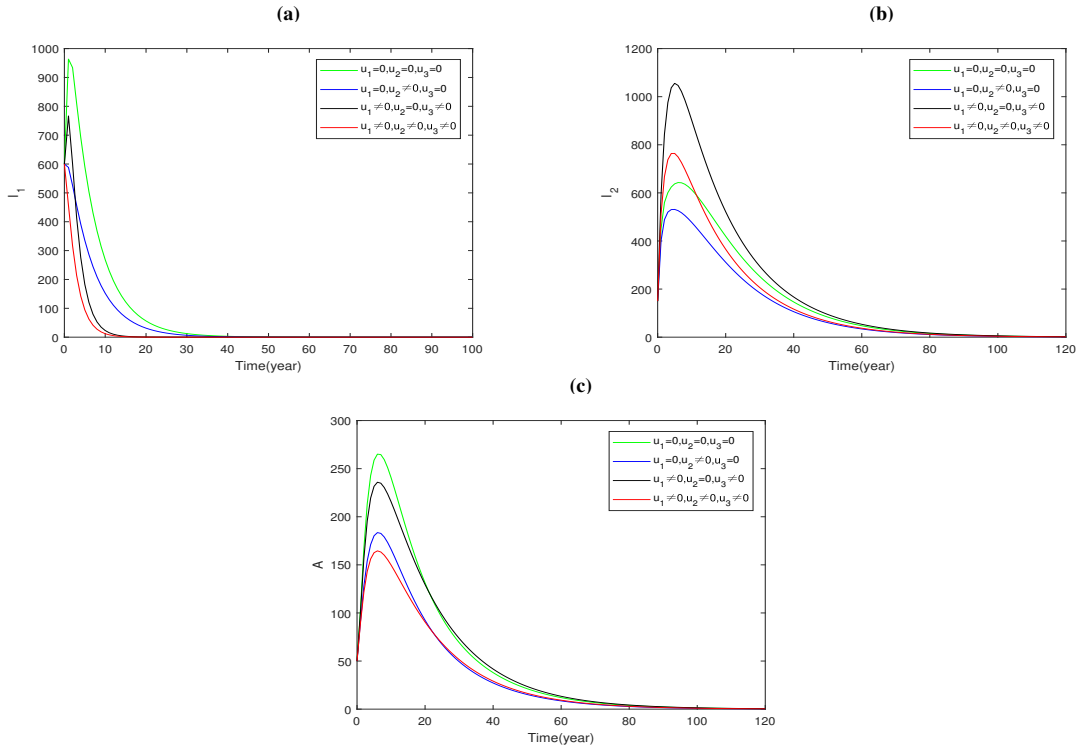

Figure 9. The final scale of the disease under the different control variable.

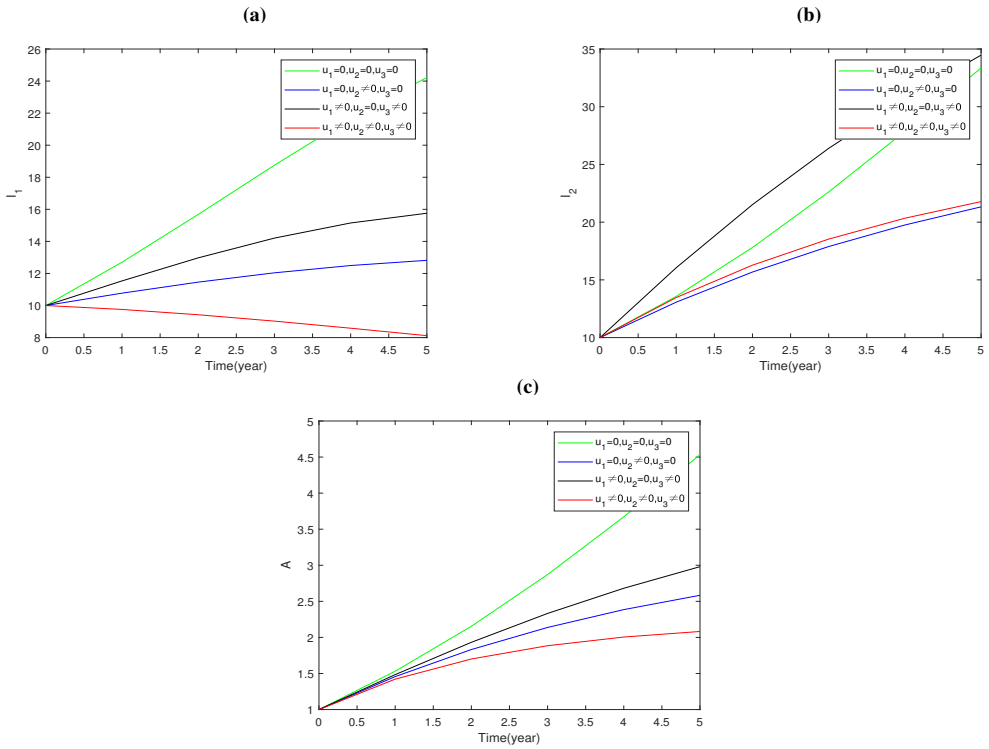

Figure 10. The final scale of the disease under the different control variable.

7. Conclusions

We analyzed the dynamic behavior of the model of HIV/AIDS with an awareness classification. From the theoretical analysis perspective, the main dynamic properties of the model were proven, including the global asymptotic stability of the disease-free equilibrium and the uniform persistence of diseases. At the same time, the optimal control problem of the model was analyzed, and the necessity of strengthening VCT awareness education and PrEP treatments was found. In the numerical simulation, we validated the theoretical results of this article and discussed the impact of three control variables on HIV/AIDS. The results indicate that strengthening VCT awareness in susceptible and infected individuals and promoting PrEP treatments in susceptible individuals can play roles in reducing HIV/AIDS patients. For example, one can strengthen the knowledge publicity, set up AIDS study days, organize knowledge contests, establish AIDS knowledge learning platforms, and reward those who have an excellent academic performance, which can drive more people to participate in learning. Therefore, we need to further enhance VCT awareness education and PrEP treatment for HIV/AIDS prevention and control.

Author contributions

Tingting Zheng: Writing-original draft, methodology, conceptualization; Yunqiang Yuan: Formal analysis, software; Yantao Luo: Writing-review & editing, conceptualization. All authors have read and approved the final version of the manuscript for publication.

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

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