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Global stability of a double-delay HIV dynamical model with curative mechanism, absorption effect, and Beddington–DeAngelis functional response*

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Abstract. This study examines the stability of a modified mathematical model for HIV infection, including two time delays. The novel aspect is that both the absorption effect and the cure rate are incorporated into the model with the Beddington–DeAngelis (BD) functional response. The global asymptotic stability of the infection-free equilibrium and uniform persistence of the model are established. To corroborate the theoretical outcomes, the numerical simulations are illustrated. Numerical simulations reveal how the two time delays, cure rate, and BD functional response influence the eradication of the disease.

Keywords: HIV infection, global stability, uniform persistence, time delay.

1 Introduction

Virus models within hosts have been studied in several pieces of literature over the last few decades using ordinary differential equations, partial differential equations, and fractional differential equations (see, for example, [6, 10, 22, 28–30], and the references therein),

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which have aided our understanding of virus–host cell interactions. The human immunodeficiency virus (HIV), a potentially fatal infection, causes acquired immunodeficiency syndrome, an infectious disease that damages the human immune system and poses a serious threat to people's lives. The fundamental mathematical model discussed by Nowak and Bangham in [22] for HIV-1 infection dynamics is of the form:

$$\begin{aligned} \dot{x}(t) &= \lambda - \beta x(t)v(t) - dx(t), \\ \dot{y}(t) &= \beta x(t)v(t) - py(t), \\ \dot{v}(t) &= ky(t) - uv(t). \end{aligned} \tag{1}$$

Here the concentrations of uninfected cells (or target cells), infected cells, and free virus particles are denoted by $x(t)$, $y(t)$, and $v(t)$. Uninfected cells are generated at a rate λ, die at a rate d, and become infected at rate β per virion per target cell. Besides, p is the death rate of infected cells, k is the average number of free virus particles generated by an infected cell, and u is the removal rate of virus particles. All parameters are expected to be constants that seem to be positive, and the units are mentioned in Table 1.

Model (1) assumes that once a virus makes contact with a target cell, the cell starts creating more viral particles. In actuality, there is a time delay between viral entry into a target cell and the production of additional virus particles from the same target cell. In view of this, more realistic models in epidemiology, virology, and immunology have been presented that account for the time delay utilizing a system of nonlinear delay differential equations (DDEs) (see, for example, [2, 5, 12, 27, 31–34] and the references therein).

Model (1) is based on the mass-action principle implying that the infection rate per host or virus remains constant. Hence, this concept is inadequate to properly explain the cellular infection process; authors have suggested nonlinear incidence rates. For instance, Huang et al. [16] have incorporated intracellular time delay with the BD functional response into their models and described stability. When pathogens are absorbed into susceptible cells, the concentration of those pathogens in the blood volume is decreased. This phenomenon is defined as the absorption effect in biology [25]. Consequently, Xu [33] have discussed the global stability of an HIV model with an absorption effect and intracellular time delay. In view of this, the authors in [23] have included the BD functional response into the model in [33] and discussed global stability.

Moreover, there exists a time lag, known as the maturation time delay, which takes for a recently generated virus to mature and become infectious [25]. As a consequence, authors in [25] have incorporated the absorption effect with both intracellular and maturation time delays into their model and determined the stability properties under some conditions. Another advancement in infectious disease modeling is the "curative" mechanisms of infected cells. Under the treatments, a portion of infected cells becomes uninfected at a specific rate by shedding all covalently closed circular DNA from the nucleus [15]. Mathematically, the term $\delta y(t)$ leaves the infected class and engages the susceptible class. As a consequence, some mathematical analysis of virus models with the cure rate has been developed (see, for example, [15], and the references therein). Authors in [24] recently examined the stability of an HIV model with two distinct time lags, incorporating absorption effects and cure rates under a saturated infection rate.

Encouraged by the works that have been done in the literature, we propose a mathematical model with the BD functional response, curative mechanism, absorption effect, and two time lags, given by the following system of delay differential equations:

$$\begin{aligned}
\dot{x}(t) &= \lambda - \frac{\beta x(t)v(t)}{1 + ax(t) + bv(t)} - dx(t) + \delta y(t),\\
\dot{y}(t) &= \frac{\mathrm{e}^{-m_1\tau_1}\beta x(t-\tau_1)v(t-\tau_1)}{1 + ax(t-\tau_1) + bv(t-\tau_1)} - (p+\delta)y(t),\\
\dot{v}(t) &= k\mathrm{e}^{-m_2\tau_2}y(t-\tau_2) - uv(t) - \frac{\beta x(t)v(t)}{1 + ax(t) + bv(t)}.
\end{aligned} \tag{2}$$

In model (2), $a, b > 0$. Thus, $\tau_1 \geqslant 0$ indicates the time gap between viral entry into a susceptible cell, getting infected by it, and producing a new virus from it, which denotes the intracellular time delay. Furthermore, $\tau_2 \geqslant 0$ represents the maturation time delay. The terms $\mathrm{e}^{-m_1\tau_1}$ and $\mathrm{e}^{-m_2\tau_2}$ refer to the probability of survival over time intervals $[t-\tau_1,\, t]$ and $[t-\tau_2,\, t]$, respectively, where $m_1, m_2 > 0$. Realistically, m_1 may differ from p. Other terms have the same biological meaning as model (1) described above. We analyze the global stability of model (2) by considering some analysis methods that have been described in [9, 11].

This paper has been structured in the following way. In Section 2, we establish the nonnegativity, boundedness of solutions, the basic reproduction number, and the existence of possible equilibrium for model (2). The global stability of infection-free equilibrium is studied in Section 3. In Section 4, we examine the uniform persistence of model (2). The numerical simulations are performed in Section 5 to validate our theoretical results. Finally, the paper concludes with a brief discussion in Section 6.

2 Preliminary analysis

First, we establish the nonnegativity and the boundedness of solutions of model (2). Let $\zeta = \max\{\tau_1, \tau_2\}$, and let $C = C([-\zeta, 0], \mathbb{R}^3_+)$ be the Banach space of continuous functions mapping the interval $[-\zeta, 0]$ into $\mathbb{R}^3_+$ with $\mathbb{R}_+ = [0, \infty)$.

We use the norm

$$\|\chi\| = \max_{-\zeta \leqslant \theta \leqslant 0} \big|\chi(\varrho)\big|$$

for elements of the space $C([-\zeta, 0], \mathbb{R}^3_+)$.

The initial conditions for model (2) take the form

$$x(\varrho) = \chi_1(\varrho) \geqslant 0, \quad y(\varrho) = \chi_2(\varrho) \geqslant 0, \quad v(\varrho) = \chi_3(\varrho) \geqslant 0, \quad \varrho \in [-\zeta, 0], \tag{3}$$

where $\chi = (\chi_1, \chi_2, \chi_3)^{\mathrm{T}} \in C$.

Now we are going to consider the following theorem.

Theorem 1. *All solutions of model* (2) *with initial conditions* (3) *exist, are unique, nonnegative on* $[0, \infty)$*, and ultimately bounded.*

Proof. Note that C is closed in C. Define

$$\Upsilon(\chi)=\begin{pmatrix}\Upsilon_1(\chi)\\ \Upsilon_2(\chi)\\ \Upsilon_3(\chi)\end{pmatrix}=\begin{pmatrix}\lambda-\frac{\beta\chi_1(0)\chi_3(0)}{1+a\chi_1(0)+b\chi_3(0)}-d\chi_1(0)+\delta\chi_2(0)\\ \frac{\mathrm{e}^{-m_1\tau_1}\beta\chi_1(-\tau_1)\chi_3(-\tau_1)}{1+a\chi_1(-\tau_1)+b\chi_3(-\tau_1)}-(p+\delta)\chi_2(0)\\ \mathrm{e}^{-m_2\tau_2}k\chi_2(-\tau_2)-u\chi_3(0)-\frac{\beta\chi_1(0)\chi_3(0)}{1+a\chi_1(0)+b\chi_3(0)}\end{pmatrix},$$

where $\Upsilon(\chi)$ is continuous for any given $\chi\in C$. Moreover, on each compact set of C, $\Upsilon(\chi)$ is Lipschitz in χ. According to the existence and uniqueness theorem of solutions of DDEs (see, for example, [17]), for any $\chi\in C$, the solution $(x(t),y(t),v(t))^{\mathrm{T}}$ of model (2) is unique on its maximal interval $[0,\Upsilon_\chi)$ of existence. Then by [26, Thm. 5.2.1], the solution $(x(t),y(t),v(t))^{\mathrm{T}}$ is nonnegative on $[0,\Upsilon_\chi)$ because for any $\chi\in C$ with $\chi_i(0)=0$, $\Upsilon_i(\chi)\geqslant 0$, $i=1,2,3$.

Next, we are going to study the boundedness of solutions of model (2) on $[0,\Upsilon_\chi)$. Define

$$\aleph(t)=x(t)+y(t)+l(t),$$

where

$$l(t)=\beta\int\limits_{t-\tau_1}^{t}\mathrm{e}^{-m_1(t-s)}\frac{x(s)v(s)}{1+ax(s)+bv(s)}\,\mathrm{d}s.$$

By taking into account the time derivative of $\aleph(t)$ along solutions of model (2), we have

$$\dot{\aleph}(t)=\lambda-dx(t)-py(t)-m_1l(t)\leqslant\lambda-\eta\aleph(t),$$

where $\eta=\min\{d,p,m_1\}$. It implies that $\aleph(t)$ is bounded from the comparison principle. Thus, $x(t)$ and $y(t)$ are bounded. Then it holds that $v(t)$ is also bounded. Hence, it follows that $\Upsilon_\chi=\infty$ by the well-known continuation theorem in [13, Thm. 12.2.4]. Therefore,

$$\limsup_{t\to\infty}\aleph(t)\leqslant\frac{\lambda}{\eta}\quad\text{and}\quad\limsup_{t\to\infty}v(t)\leqslant\frac{\lambda k\mathrm{e}^{-m_2\tau_2}}{u\eta}.\tag{4}$$

This completes the proof. □

Model (2) always has the infection-free equilibrium $\mathcal{E}_0(x_0,0,0)$, where $x_0=\lambda/d$. By considering the approach in [35], we have

$$\mathbb{F}=\begin{pmatrix}0&\frac{\beta x_0\mathrm{e}^{-m_1\tau_1}}{1+ax_0}\\0&0\end{pmatrix},\qquad\mathbb{V}=\begin{pmatrix}p+\delta&0\\-k\mathrm{e}^{-m_2\tau_2}&u+\frac{\beta x_0}{1+ax_0}\end{pmatrix}.$$

Consequently, we derive the basic reproduction number $R_0=\rho(\mathbb{F}\mathbb{V}^{-1})$ (spectral radius of the next generation matrix) for model (2) as

$$R_0=\frac{\lambda\beta k\mathrm{e}^{-m_1\tau_1-m_2\tau_2}}{(p+\delta)(\lambda\beta+ud+au\lambda)}.\tag{5}$$

In general, R_0 is crucial in determining whether or not viruses clean out over time.

Thus, model (2) has a chronic infection equilibrium $\mathcal{E}^*(x^*, y^*, v^*)$ when $R_0 > 1$, where

$$v^* = \frac{\gamma(p+\delta)(\lambda\beta + ud + au\lambda)(R_0 - 1)}{u\{bd(p+\delta)\gamma + (p+\delta-\delta \mathrm{e}^{-m_1\tau_1})[\beta k \mathrm{e}^{-m_1\tau_1 - m_2\tau_2} - (\beta + au)(p+\delta)]\}},$$
$$x^* = \frac{u(p+\delta)(1+bv^*)}{\beta\gamma - au(p+\delta)}, \qquad y^* = \frac{uv^*\mathrm{e}^{-m_1\tau_1}}{\gamma}.$$

Here $\gamma = k\mathrm{e}^{-m_1\tau_1 - m_2\tau_2} - (p+\delta)$.

3 Global stability analysis of the infection free equilibrium

This section addresses the global stability of $\mathcal{E}_0$. For $R_0 < 1$, it is not hard to prove that $\mathcal{E}_0$ is locally asymptotically stable (see [24, 25]). Thus, we merely show the global attractiveness of $\mathcal{E}_0$ when $R_0 < 1$. Let $\zeta = \max\{\tau_1, \tau_2\}$, and let $\varkappa_t = (x_t, y_t, v_t)^{\mathrm{T}}$ be the solution of model (2) with any $\chi \in C$, which is defined as $\varkappa_t(\varrho) = \varkappa(t+\varrho)$, $\varrho \in [-\zeta, 0]$. It is easy to see that $\varkappa_t$ is bounded from Theorem 1, and that for all $t > 0$, $x(t) > 0$. Then we can obtain that the ω-limit set $\omega(\chi)$ is a compact subset of C for model (2). Now, we consider the following theorem.

Theorem 2. *If $R_0 < 1$ and $d \leqslant \min\{p, m_1\}$, the infection-free equilibrium $\mathcal{E}_0$ is globally asymptotically stable in C.*

Proof. Define a functional $\mathcal{V}_1$ on C of the form

$$\mathcal{V}_1 = \frac{\mathrm{e}^{m_1\tau_1}}{\beta}\chi_2(0) + \frac{(p+\delta)\mathrm{e}^{m_1\tau_1+m_2\tau_2}}{k\beta}\chi_3(0) + \int_{-\tau_1}^{0} \frac{\chi_1(\varrho)\chi_3(\varrho)\,\mathrm{d}\varrho}{1 + a\chi_1(\varrho) + b\chi_3(\varrho)}$$
$$+ \frac{(p+\delta)\mathrm{e}^{m_1\tau_1+m_2\tau_2}}{\beta}\int_{-\tau_2}^{0} \chi_2(\varrho)\,\mathrm{d}\varrho.$$

Then, by calculating the derivative of $\mathcal{V}_1$ along the solution $\varkappa_t$ for $t \geqslant 0$,

$$\dot{\mathcal{V}}_1 = \frac{\mathrm{e}^{m_1\tau_1}}{\beta}\dot{y}(t) + \frac{(p+\delta)\mathrm{e}^{m_1\tau_1+m_2\tau_2}}{k\beta}\dot{v}(t) + \frac{\mathrm{d}}{\mathrm{d}t}\int_{-\tau_1}^{0} \frac{x(t+\varrho)v(t+\varrho)}{1 + ax(t+\varrho) + bv(t+\varrho)}\,\mathrm{d}\varrho$$
$$+ \frac{(p+\delta)\mathrm{e}^{m_1\tau_1+m_2\tau_2}}{\beta}\frac{\mathrm{d}}{\mathrm{d}t}\int_{-\tau_2}^{0} y(t+\varrho)\,\mathrm{d}\varrho,$$
$$\dot{\mathcal{V}}_1 = \frac{x(t)v(t)}{1+ax(t)+bv(t)}\left[1 - \frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}u(p+\delta)(1+ax(t)+bv(t))}{k\beta x(t)} - \frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}(p+\delta)}{k}\right]$$

$$\leqslant \frac{x(t)v(t)}{1+ax(t)+bv(t)}\left[1-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}u(p+\delta)(1+ax(t))}{k\beta x(t)}-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}(p+\delta)}{k}\right]$$
$$=\frac{x(t)v(t)}{1+ax(t)+bv(t)}g(t),$$

where

$$g(t)=1-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}u(p+\delta)(1+ax(t))}{k\beta x(t)}-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}(p+\delta)}{k}.$$

It follows that

$$\limsup_{t\to\infty} g(t)\leqslant 1-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}u(p+\delta)}{k\beta}\left(\frac{1}{\limsup_{t\to\infty}x(t)}+a\right)-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}(p+\delta)}{k}.$$

From (4), if $d\leqslant\min\{p,m_1\}$, it follows that $\limsup_{t\to\infty}x(t)\leqslant x_0=\lambda/d$. Then

$$\limsup_{t\to\infty} g(t)\leqslant 1-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}u(p+\delta)(1+ax_0)}{k\beta x_0}-\frac{\mathrm{e}^{m_1\tau_1+m_2\tau_2}(p+\delta)}{k}.$$

Then

$$\limsup_{t\to\infty} g(t)\leqslant 1-\frac{1}{R_0}<\frac{1}{2}\left(1-\frac{1}{R_0}\right)<0\quad\text{for } R_0<1.$$

Hence, for $t\geqslant\mathfrak{T}$, there exists $\mathfrak{T}=\mathfrak{T}(\chi)$, and the following holds:

$$g(t)\leqslant\frac{1}{2}\left(1-\frac{1}{R_0}\right).$$

Thus, if $R_0<1$ and $t\geqslant\mathfrak{T}$,

$$\dot{\mathcal{V}}_1\leqslant\frac{x(t)v(t)}{1+ax(t)+bv(t)}g(t)\leqslant 0. \tag{6}$$

Thus, $\mathcal{V}_1$ is a Lyapunov functional on $\{\varkappa_t(\chi),\,t\geqslant\mathfrak{T}\}$.

We now investigate whether $\liminf_{t\to\infty}x(t)>0$. From the first equation of model (2) it follows that

$$\dot{x}(t)\geqslant\lambda-\frac{\beta x(t)v(t)}{1+ax(t)+bv(t)}-dx(t).$$

From (4) we obtain

$$\liminf_{t\to\infty}x(t)>\frac{\lambda}{d+\frac{\beta\lambda k\mathrm{e}^{-m_2\tau_2}}{\eta u}}. \tag{7}$$

Hence, $\liminf_{t\to\infty}x(t)>0$.

Clearly, $\dot{\mathcal{V}}_1(\sigma)=0$ for any $\sigma\in\omega(\chi)$ from [8, Cor. 2.1]. Next, to examine the global attractiveness of equilibrium $\mathcal{E}_0$, we exhibit that $\omega(\chi)=\{\mathcal{E}_0\}$. Let $\varkappa_t$ be the solution of model (2) with any $\sigma\in\omega(\chi)$. Consequently, for all $t\in\mathbb{R}$, $\varkappa_t\in\omega(\chi)$ by the invariance of $\omega(\chi)$. Thus, by [8, Thm. 3.2], (6), and (7), for all $t\in\mathbb{R}$, we have $v(t)=0$. It follows from model (2) and the invariance of $\omega(\chi)$ that for all $t\in\mathbb{R}$, $y(t)=0$ and $x(t)=x_0$. Hence, this implies that $\omega(\chi)=\{\mathcal{E}_0\}$. □

Now, we use an alternative approach to demonstrate the global stability of $\mathcal{E}_0$ state. For our convenience, let us define

$$R_1 = \frac{\beta\lambda k \mathrm{e}^{-m_2\tau_2}}{pu(d+a\lambda)}.$$

Then we consider

$$R_0 - R_1 = -\lambda\beta k \mathrm{e}^{-m_2\tau_2}\left[\frac{pu(d+a\lambda)(1-\mathrm{e}^{-m_1\tau_1})+p\lambda\beta+\delta(\lambda\beta+ud+au\lambda)}{(p+\delta)(\lambda\beta+ud+au\lambda)pu(d+a\lambda)}\right].$$

Clearly, $1-\mathrm{e}^{-m_1\tau_1} \geqslant 0$ for all $\tau_1 \geqslant 0$. Therefore, $R_0 - R_1 < 0$, which implies that $R_0 < R_1$.

Define

$$P_1 = \{\chi \in C\colon\ \chi_1 > 0\},$$

and we consider the following theorem.

Theorem 3. *If $R_1 \leqslant 1$, the infection-free equilibrium $\mathcal{E}_0$ is globally asymptotically stable in C.*

Proof. Define a functional $\mathcal{V}_2$ on P_1 of the following form:

$$\begin{aligned}\mathcal{V}_2 = {}& \chi_1(0) - x_0 - x_0 \int_{x_0}^{\chi_1(0)} \frac{(1+as)ds}{(1+ax_0)s} + \chi_2(0) + \frac{p\mathrm{e}^{m_2\tau_2}}{k}\chi_3(0) \\ & + \mathrm{e}^{-m_1\tau_1}\int_{-\tau_1}^{0} \frac{\beta\phi_1(\varrho)\phi_3(\varrho)\,\mathrm{d}\varrho}{1+a\phi_1(\varrho)+b\phi_3(\varrho)} + p\int_{-\tau_2}^{0}\phi_2(\varrho)\,\mathrm{d}\varrho. \end{aligned} \tag{8}$$

Then, by calculating the derivative of $\mathcal{V}_2$ along the solution $\varkappa_t$ for $t > 0$,

$$\begin{aligned}\dot{\mathcal{V}}_2 = {}& \frac{-d(x(t)-x_0)^2}{x(t)(1+ax_0)} - \frac{\beta x(t)v(t)(1-\mathrm{e}^{-m_1\tau_1})}{1+ax(t)+bv(t)} - \frac{(1+ax(t))x_0\delta y(t)}{x(t)(1+ax_0)} \\ & + \frac{1+ax(t)}{1+ax_0}\frac{\beta x_0 v(t)}{1+ax(t)+bv(t)} - \frac{p\mathrm{e}^{m_2\tau_2}}{k}uv(t) - \frac{p\mathrm{e}^{m_2\tau_2}}{k}\frac{\beta x(t)v(t)}{1+ax(t)+bv(t)}.\end{aligned}$$

Now, we consider

$$\begin{aligned}& \frac{1+ax(t)}{1+ax_0}\frac{\beta x_0 v(t)}{1+ax(t)+bv(t)} - \frac{p\mathrm{e}^{m_2\tau_2}}{k}uv(t) \\ & \quad \leqslant \frac{\beta x_0 v(t)}{1+ax_0} - \frac{p}{k\mathrm{e}^{-m_2\tau_2}}uv(t) = \left(\frac{\beta\lambda}{d+a\lambda} - \frac{pu}{k\mathrm{e}^{-m_2\tau_2}}\right)v(t) \\ & \quad = \frac{puv(t)}{k\mathrm{e}^{-m_2\tau_2}}(R_1 - 1).\end{aligned}$$

Then we have

$$\begin{aligned}\dot{\mathcal{V}}_2 \leqslant {}& \frac{-d(x(t)-x_0)^2}{x(t)(1+ax_0)} - \frac{\beta x(t)v(t)(1-\mathrm{e}^{-m_1\tau_1})}{1+ax(t)+bv(t)} + \frac{puv(t)}{k\mathrm{e}^{-m_2\tau_2}}(R_1-1) \\ & - \frac{(1+ax(t))x_0\delta y(t)}{x(t)(1+ax_0)} - \frac{p\mathrm{e}^{m_2\tau_2}}{k}\frac{\beta x(t)v(t)}{1+ax(t)+bv(t)} \leqslant 0.\end{aligned} \tag{9}$$

Clearly, $\dot{\mathcal{V}}_2(\sigma) = 0$ for any $\sigma \in \omega(\chi)$ from [8, Cor. 2.1]. Next, to derive the global attractiveness of equilibrium $\mathcal{E}_0$, we show that $\omega(\chi) = \{\mathcal{E}_0\}$. Let $\varkappa_t$ be the solution of model (2) with any $\sigma \in \omega(\chi)$. Consequently, for all $t \in \mathbb{R}$, $\varkappa_t \in \omega(\chi)$ by the invariance of $\omega(\chi)$. Thus, by [8, Thm. 3.2], (8), and (9), we have $x(t) = x_0$ and $y(t) = v(t) = 0$ for all $t \in \mathbb{R}$. Hence, this implies that $\omega(\chi) = \{\mathcal{E}_0\}$. Therefore, if $R_1 \leqslant 1$, $\mathcal{E}_0$ is globally asymptotically stable. □

4 Uniform persistence

Now, we focus on the uniform persistence of model (2) following the definitions in [1, 3, 14] and some analysis methods in [9, 18–20, 31]. Model (2) is said to be uniformly persistent if there exists a constant $\varpi > 0$ such that any solution $(x(t), y(t), v(t))^{\mathrm{T}}$ with initial conditions given by (2) fulfills

$$\liminf_{t\to\infty} x(t) \geqslant \varpi, \qquad \liminf_{t\to\infty} y(t) \geqslant \varpi, \qquad \liminf_{t\to\infty} v(t) \geqslant \varpi.$$

We denote

$$C^0 = \big\{\chi \in C,\ \chi_2(0) + \chi_3(0) > 0\big\},$$
$$\partial C = C \setminus C^0 = \big\{\chi \in C,\ \chi_2(0) = 0,\ \chi_3(0) = 0\big\},$$

to obtain the uniform persistence of model (2),

For $t \geqslant 0$, let $\mathcal{P}(t)$ denote the family of solution operators corresponding to model (2). Define the ω-limit set as follows:

$$\omega(x) := \big\{y \in C\colon \exists \text{ a sequence } t_n \to \infty \text{ with } \mathcal{P}(t_n)x \to y \text{ as } n \to \infty\big\},$$

and

$$\mathcal{M}_\partial = \big\{\chi \in \partial C\colon\ P(t)\chi \text{ satisfies model (2), and } P(t)\chi \in \partial C\ \ \forall t \geqslant 0\big\}.$$

Let $(x(t), y(t), v(t))^{\mathrm{T}} = (x(t,\chi), y(t,\chi), v(t,\chi))^{\mathrm{T}}$ be the solutions of model (2) with any $\chi \in C^0$. It is not difficult to see that both C^0 and M_∂ are positively invariant under $\mathcal{P}(t)$, and $\mathcal{M}_\partial \subseteq \partial C$. Furthermore, we have $(x(t), y(t), v(t))^{\mathrm{T}} \gg \mathbf{0}$ for all $t > 0$.

Now we consider the following Lemma.

Lemma 1. *If $R_0 > 1$, for any $\chi \in C^0$, there exists a $\xi \in (0, x_0)$, which satisfies*

$$\limsup_{t\to\infty} \big\|\mathcal{P}(t)\chi - \mathcal{E}_0\big\| \geqslant \xi.$$

Proof. Assume, by contradiction, that for all $\xi \in (0, x_0)$, there exists $\psi \in C^0$ such that

$$\limsup_{t\to\infty} \big\|\mathcal{P}(t)\psi - \mathcal{E}_0\big\| < \xi,$$

which implies that there exists $\mathfrak{T}_0 = \mathfrak{T}_0(\epsilon, \psi) > 0$ such that

$$x_0 - \xi < x(t) < x_0 + \xi, \quad y(t) < \xi, \quad v(t) < \xi, \quad t \geqslant \mathfrak{T}_0.$$

Thus, for chosen ξ, $t \geqslant \mathfrak{T}_0 + \tau_1$, and $t \geqslant \mathfrak{T}_0 + \tau_2$, it follows from model (2) that

$$\begin{aligned}
\dot{y}(t) &\geqslant \frac{\beta \mathrm{e}^{-m_1\tau_1}(x_0 - \xi)v(t-\tau_1)}{1 + a(x_0 - \xi) + b\xi} - (p+\delta)y(t),\\
\dot{v}(t) &\geqslant k\mathrm{e}^{-m_2\tau_2}y(t-\tau_2) - uv(t) - \beta(x_0+\xi)v(t).
\end{aligned}$$

Let us consider the following linear cooperative system:

$$\begin{aligned}
\dot{\varkappa}_1(t) &= \frac{\beta \mathrm{e}^{-m_1\tau_1}(x_0 - \xi)\varkappa_2(t-\tau_1)}{1 + a(x_0 - \xi) + b\xi} - (p+\delta)\varkappa_1(t),\\
\dot{\varkappa}_2(t) &= k\mathrm{e}^{-m_2\tau_2}\varkappa_1(t-\tau_2) - u\varkappa_2(t) - \beta(x_0+\xi)\varkappa_2(t).
\end{aligned} \tag{10}$$

Then consider the following Jacobian matrix of system (10):

$$\mathcal{J} = \begin{pmatrix} -(p+\delta) & \frac{\beta \mathrm{e}^{-(m_1+\mu)\tau_1}(x_0-\xi)}{1+a(x_0-\xi)+b\xi} \\ k\mathrm{e}^{-(m_2+\mu)\tau_2} & -(u+\beta(x_0+\xi)) \end{pmatrix},$$

where μ must be any root of $\det(\mu I - \mathcal{J}) = 0$. Then, by considering the characteristic equation of system (10), we have

$$\begin{aligned}
&\mu^2 + \mu\big[p+\delta+u+\beta(x_0+\xi)\big] + (p+\delta)\big[u+\beta(x_0+\xi)\big]\\
&\quad - \frac{\beta k \mathrm{e}^{-m_1\tau_1 - m_2\tau_2 - \mu(\tau_1+\tau_2)}(x_0-\xi)}{1+a(x_0-\xi)+b\xi} = 0.
\end{aligned} \tag{11}$$

Furthermore, if $R_0 > 1$, Eq. (11) has a principal eigenvalue $\mu_0(\xi) > 0$. Let $\zeta = \max\{\tau_1, \tau_2\}$, and let the corresponding positive right eigenvector associated with $\mu_0(\xi)$ be $\varkappa_s = (\varkappa_y, \varkappa_v)^{\mathrm{T}}$ for model (2). Then, by [26, Thm. 5.5.1], it follows that there is a principal eigenvalue $\mu_0(\xi)$ associated with a strictly positive eigenvector $\varkappa_s$, and a solution $(\varkappa_1(t), \varkappa_2(t))^{\mathrm{T}} = l\mathrm{e}^{\mu_0(\xi)t}\varkappa_s$ for any given $l > 0$ of system (10). This implies that $\lim_{t\to\infty}(\varkappa_1(t), \varkappa_2(t))^{\mathrm{T}} = (\infty, \infty)^{\mathrm{T}}$. Since $y(\mathfrak{T}_0 + \zeta) > 0$ and $v(\mathfrak{T}_0 + \zeta) > 0$, by choosing $l > 0$ such that

$$\big(y(\mathfrak{T}_0+\zeta), v(\mathfrak{T}_0+\zeta)\big)^{\mathrm{T}} \geqslant \big(\varkappa_1(\mathfrak{T}_0+\zeta), \varkappa_2(\mathfrak{T}_0+\zeta)\big)^{\mathrm{T}},$$

it follows from [26, Thm. 5.1.1] that $(y(t), v(t))^{\mathrm{T}} \geqslant l\mathrm{e}^{\mu_0(\xi)t}\varkappa_s$ for all $t \geqslant \mathfrak{T}_0 + \zeta$. Consequently, $\lim_{t\to\infty} y(t) = \infty$ and $\lim_{t\to\infty} v(t) = \infty$, which confirms that $y(t)$ and $v(t)$ are unbounded, contradicting the assumption. □

Theorem 4. *Model* (2) *is uniformly persistent in* C^0*, provided that* $R_0 > 1$.

Proof. We easily obtain that C^0 is a positively invariant set under $\mathcal{P}(t)$ and that for $t \geqslant \zeta$, $\mathcal{P}(t)$ is compact. Theorem 1 implies that $\mathcal{P}(t)$ is point dissipative in C. Since $\mathcal{M}_\partial \subseteq \partial C$ and by the first equation of model (2), we obtain that $\lim_{t\to\infty} x(t, \chi) = x_0$. Consequently, it follows that $\omega(\chi) = \{\mathcal{E}_0\}$, and $\cup_{\chi\in\mathcal{M}_\partial}\omega(\chi) = \{\mathcal{E}_0\}$ for any $\chi \in \mathcal{M}_\partial$. Let $\mathcal{W}^s(\mathcal{E}_0)$ be the stable set of $\mathcal{E}_0$ for $\mathcal{P}(t)$. Lemma 1 implies that $\{\mathcal{E}_0\}$ is an isolated invariant set in C and that $\mathcal{W}^s(\mathcal{E}_0) \cap C^0 = \emptyset$. Moreover, no orbit connects $\{\mathcal{E}_0\}$ to itself in ∂C, which implies that $\{\mathcal{E}_0\}$ cannot form a cycle in ∂C. Therefore, $\mathcal{P}(t)$ is uniformly persistent with respect to $(C^0, \partial C)$ by [14, Thm. 4.1] (see also [17, Thm. 8.2.4]). □

5 Numerical simulations

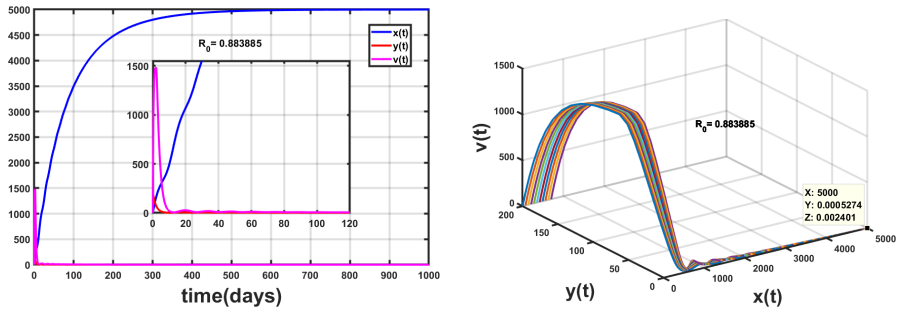
This section includes various numerical simulations using "dde23" in MATLAB to validate the theoretical results obtained in Section 3. The values in Table 1 are utilized to demonstrate how stable the equilibrium solutions of model (2) with a connection to R_0. In our simulations, $x(t)$, $y(t)$, and $v(t)$ represent the cell concentrations of uninfected cells (or target cells), infected cells, and free virus particles on day t in cells per milliliter.

Taking $\tau_1 = 10$ and $\tau_2 = 2$, we calculate $R_0 = 0.883885 < 1$. Based on Theorem 2, we conclude that $\mathcal{E}_0(5000, 0, 0)$ is globally asymptotically stable, which is numerically shown by Fig. 1. Here $d = 0.02 < 0.3 = \min\{p, m_1\}$. Similarly, according to Theorem 3, we can numerically verify that for $\tau_1 = 2$ and $\tau_2 = 12$, when $R_0 = 0.178453 < R_1 = 0.555563 < 1$, and for $\tau_1 = 1$ and $\tau_2 = 10.53057$, when $R_0 = 0.43359 < R_1 = 1$, $\mathcal{E}_0(5000, 0, 0)$ is globally asymptotically stable.

In Fig. 2, we numerically depict the chronic infection equilibrium's global stability, which is difficult to discuss utilizing an appropriate Lyapunov functional. For $\tau_1 = 1$ and $\tau_2 = 2$, when $R_0 = 13.151969 > 1$, chronic infection equilibrium $\mathcal{E}^*(432.43, 134.65, 977.48)$ is globally asymptotically stable.

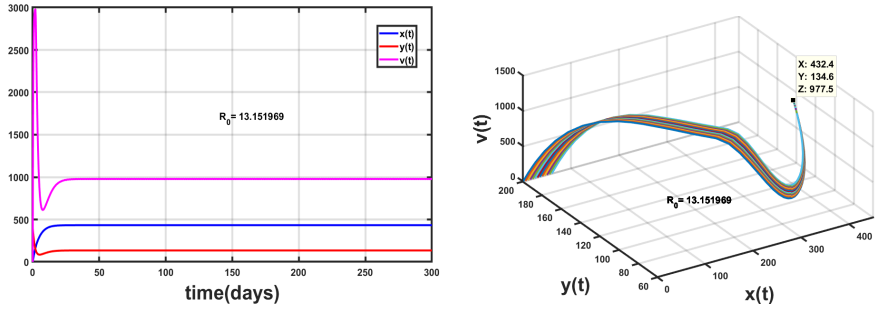
Table 1. Parameter values used in the numerical simulations.

Parameter	Value	Source
λ	100 cells ml^{-1} day^{-1}	[23]
β	0.45 ml cells^{-1} day^{-1}	[23]
d	0.02 day^{-1}	[23]
δ	0.01 day^{-1}	[15]
p	0.5 day^{-1}	[23]
k	50 day^{-1}	[23]
u	3 day^{-1}	[15]
a	0.222 ml cells^{-1}	[23]
b	2 ml cells^{-1}	[23]
m_1	0.3 day^{-1}	[32]
m_2	0.4 day^{-1}	[32]



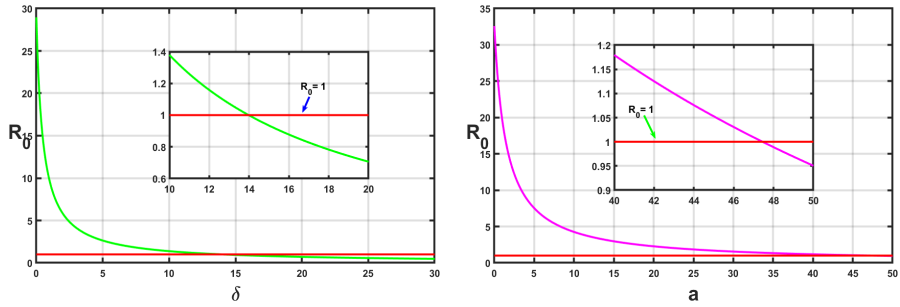
(a) (b)

Figure 1. Stability of $\mathcal{E}_0(5000, 0, 0)$ for $\tau_1 = 10$ and $\tau_2 = 2$ of model (2): (a) dynamical behavior of $x(t)$, $y(t)$, and $v(t)$ versus time; (b) 3D plot of $x(t)$, $y(t)$, and $v(t)$ at the infection-free equilibrium state.



(a) (b)

Figure 2. Stability of $\mathcal{E}^*(432.43, 134.65, 977.48)$ for $\tau_1 = 1$ and $\tau_2 = 2$ of model (2): (a) dynamical behavior of $x(t)$, $y(t)$, and $v(t)$ versus time; (b) 3D plot of $x(t), y(t)$, and $v(t)$ at the chronic infection equilibrium state.



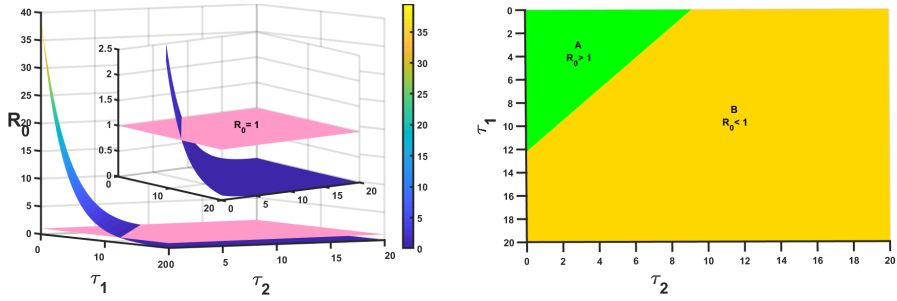
(a) (b)

Figure 3. The relationship between the basic reproduction number R_0 versus cure rate δ and BD constant a of model (2): (a) the effect of δ on the R_0; (b) the effect of a on the R_0.

In Fig. 3, both plots (a) and (b) show the behavior of R_0 for different δ and a values, respectively. The red line represents $R_0 = 1$ in both plots. We can see that, as the cure rate δ and the constant a in the BD infection rate increase, the basic reproduction number R_0 drops below the $R_0 = 1$ red line in both plots.

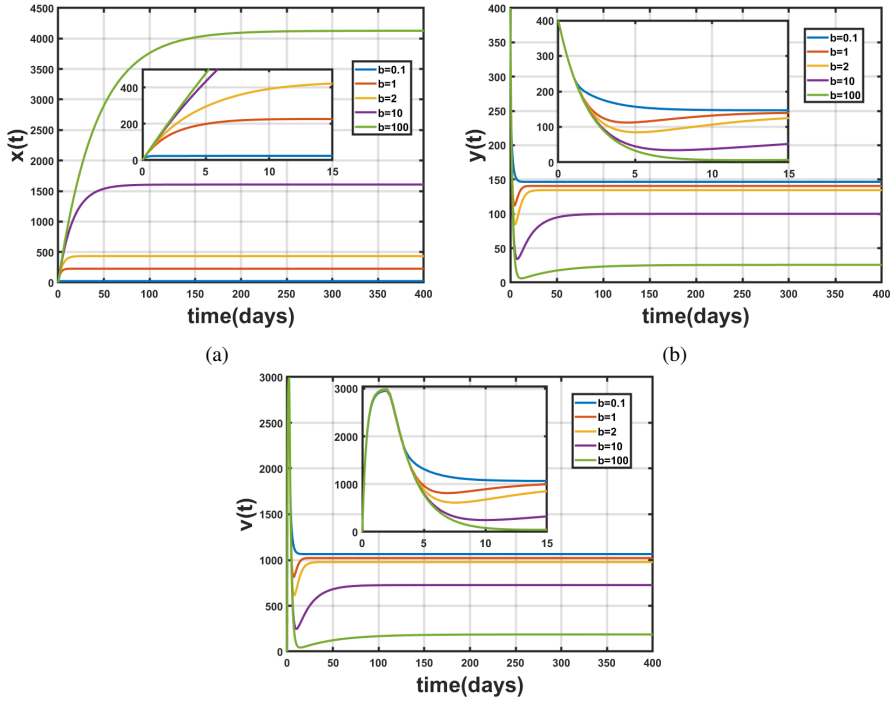
Figure 4 exhibits the relationship between R_0 and two time delays (τ_1, τ_2) in the 3D space. Here we only change the values of τ_1 and τ_2. In Fig. 4(a), the pink color plane represents the R_0 as 1. When the two lags increase, a point moves from above to below across the pink plane. In Fig. 4(b), a point in the green plane moves to a point in the yellow plane as the two delays rise. This indicates that both intracellular and maturation time delays can alter the HIV dynamics.

Next, we use various values of the BD constant b to understand the behavior of $x(t)$, $y(t)$, and $v(t)$ over the time range from 0 to 400 days. When we raise the constant b in model (2), we observe that the concentrations of infected cells and virus decrease, whereas the number of uninfected cells increases (see Fig. 5). This result indicates that the BD infection rate constant b also influences the dynamic of model (2).



(a) (b)

Figure 4. (a) The behavior of R_0 with respect to two time delays of model (2). (b) The sign region of $R_0 - 1$, which means that $R_0 - 1$ is greater than zero in region A, less than zero in region B, and equal to zero at the border of regions A and B.

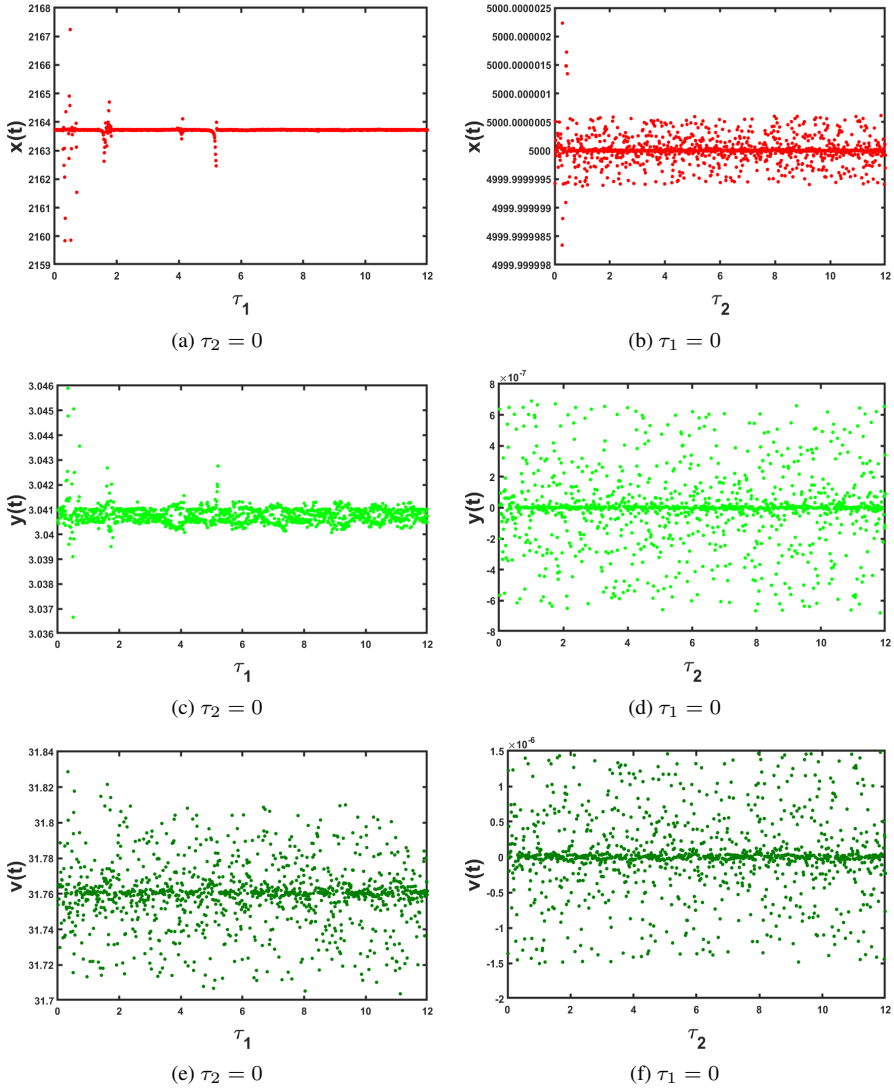


(a) (b)

(c)

Figure 5. The influence of BD constant b for $\mathcal{E}^*$ of model (2) when $\tau_1 = 1, \tau_2 = 2$: (a) the dynamic behavior of uninfected cells with varying b values; (b) the dynamic behavior of infected cells with varying b values; (c) the dynamic behavior of virus particles with varying b values.

Additionally, Fig. 6 elucidates the dynamical behavior of uninfected, infected, and viral cells in the absence of one delay. In this figure, we consider the delay axes spanning



(a) $\tau_2 = 0$ (b) $\tau_1 = 0$

(c) $\tau_2 = 0$ (d) $\tau_1 = 0$

(e) $\tau_2 = 0$ (f) $\tau_1 = 0$

Figure 6. The dynamical behavior of (a)–(b) infected cells $x(t)$, (c)–(d) uninfected cells $y(t)$, and (e)–(f) virus particles $v(t)$.

from 0 to 12. In Figs. 6(a), 6(c), and 6(e), we fixed $\tau_2 = 0$, and in Figs. 6(b), 6(d), and 6(f), we fixed $\tau_1 = 0$. The other parameters stay unchanged. We observe that even a single delay changes from a little amount; the concentration of $x(t)$, $y(t)$, and $v(t)$ is changing. Therefore, both the intracellular and maturation time delays of model (2) play nonnegligible roles in the HIV dynamics inside the body. Finally, from Figs. 3–6 we can conclude that cure rate δ, BD constants a and b, and the two delays have a huge impact on eradicating the disease and can be useful in developing HIV treatment strategies.

6 Discussion

Throughout our work, we studied a delayed HIV dynamic model that included both absorption effect and cure rate. We have incorporated the BD rate into model (2) to improve its medical comprehension. Using some analysis methods that have been discussed in [9, 11], we demonstrated that if $R_0 < 1$ and $d \leqslant \min\{p, m_1\}$, $\mathcal{E}_0$ is globally asymptotically stable (see Fig. 1), indicating that the virus is unable to exacerbate the disease and will eventually become extinct with the help of treatments or the immune system. Regarding the extra condition that we obtain in Theorem 2, biologically, if the minimum value of the death rate of infected cells and the removal rate of infected cells during the time interval τ_2 is greater than or equal to the death rate of target cells, then the disease will be eradicated. Furthermore, as an alternative approach, we defined R_1, which is always greater than R_0, and derived that if $R_1 \leqslant 1$, the infection-free equilibrium is globally asymptotically stable in Theorem 3.

On the other hand, it is difficult to determine an effective Lyapunov functional for global asymptotic stability in $\mathcal{E}^*$ because developing a suitable Lyapunov functional to examine the global properties of DDEs is an immensely challenging task. Hence, it is difficult to demonstrate the global stability analysis of the chronic infection equilibrium analytically by utilizing an appropriate Lyapunov functional. We have illustrated it numerically (see Fig. 2). If $R_0 > 1$, $\mathcal{E}^*$ should become globally asymptotically stable, implying that no medicine or immune system can prevent a host from becoming infected. Hence, R_0 is directly involved in determining whether the virus in the host will persist or die. As an alternative method for ensuring the global stability of $\mathcal{E}^*$, we established that model (2) exhibits uniform persistence when $R_0 > 1$, as stated in Theorem 4.

Meanwhile, we can see from the straightforward formula (5) that R_0 can be reduced by increasing τ_1 and τ_2, as R_0 depends on the delay through factors of $\mathrm{e}^{-m_1\tau_1}$ and $\mathrm{e}^{-m_2\tau_2}$. Consequently, m_1, m_2, τ_1, and τ_2 are significant factors in determining whether or not a disease can be eradicated. Therefore, by increasing the intracellular and maturation time delays, R_0 can potentially be reduced below unity by appropriately adjusting the values of m_1 and m_2. Figure 4 depicts the immense influence of two time lags on eradicating the disease. We exhibit the dynamical behavior of uninfected, infected, and virus cells for fixed values $\tau_2 = 0$ (see Figs. 6(a), 6(c), 6(e)) and $\tau_1 = 0$ (see Figs. 6(b), 6(d), 6(f)), respectively, for a better understanding of the influence of time delays. Besides, if we increase the cure rate δ and the constants a and b in BD incidence, we can easily control the disease (see Figs. 3 and 5). Subsequently, through this research, we found that both the intracellular and maturation time delays, the cure rate, and the BD functional response have a significant impact on the HIV dynamics inside the body. Hence, our results contribute to a deeper knowledge of viral pathology and may assist in improving novel treatment strategies to control viral infections. Conversely, the parameters can be increased or decreased only within certain limits due to biological constraints.

Model (2) assumes that the death or decay of the intermediate population infected cells occurs at a constant per capita rate m_2, leading to the exponential survival factor $\mathrm{e}^{-m_2\tau_2}$. This is a simplification. In biological reality, death rates are often not constant; they may be age-dependent (i.e., depending on how long a cell has been infected), density-

dependent, or influenced by other factors like immune responses not explicitly modeled here. The model does not account for a programmed cell death (apoptosis) timeline or variations in cell lifespans. Additionally, model (2) employs fixed, discrete delays τ_1 and τ_2. This assumes that every biological process (for example, the time to produce a virion from an infected cell) takes exactly the same amount of time. In reality, these processes exhibit significant variability and are better represented by a distribution of delays. For instance, the time for a virus to replicate and bud from a cell can vary. Using a fixed delay is a mathematical convenience that may obfuscate the dynamics induced by this heterogeneity. In a biological host, the interactions between cells and viruses are often highly localized. Spatial structure (for example, diffusion, cell-to-cell transmission) may lead to different dynamics (like traveling waves of infection) that this type of model fails to capture.

Additionally, the current cure rate focuses on eradicating the viral reservoir (killing infected cells), which would be better represented by an enhanced loss term for infected cells rather than a gain term for target cells. Infected cells that are exposed to protease inhibitors create noninfectious virions. However, virions that were produced previous to the medication therapy are still infectious. One type of virus particle is unaffected by protease inhibitors, while the other is affected. Authors in [21] integrated these forms of virus particles and employed models that did not include the curative mechanism of infected cells.

In the meantime, we discover that our study incorporates some previous works. When $\delta = 0$, our model (2) converges into the model discussed in [25]. Authors in [24] considered the saturated infection rate, which is similar to our model when $a = 0$. In both cases, they considered $m_1 = p$ and $m_2 = u$. Realistically, they are not equal. We can clearly see the influence of a and δ for the HIV dynamics from Fig. 3. When we consider the general incidence rate function (see, for example, [4, 7], and the references therein) in model (2), there is also room for improvement. This will be explored in the future.

Conflicts of interest. The authors declare no conflicts of interest.

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