Contemporary Mathematics
http://ojs.wiserpub.com/index.php/CM/

Research Article

Dynamics of HIV-1 and HTLV-1 Coinfection with both CTL and Antibody Responses

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Received: 6 November 2023; **Revised:** 28 November 2024; **Accepted:** 28 November 2023

Abstract: Human immunodeficiency virus type 1 (HIV-1) and human T-lymphotropic virus type 1 (HTLV-1) coinfection models with simply an antibody response or a CTL response have been the subject of several recent investigations. Nevertheless, no prior research has been done on the dynamics of HIV-1 and HTLV-1 coinfection under the influence of both CTL and antibody responses. Thus, the primary objective of this paper is to formulate and examine a mathematical framework for analyzing the intricate dynamics of coinfection between HIV-1 and HTLV-1 under the influence of both CTL and antibody responses. While CTLs are thought to destroy HTLV-1-infected cells, antibodies neutralize free HIV-1 particles. We prove that the model is well-posed and it admits eight equilibria. The stability and existence of the equilibria are precisely controlled by eight threshold parameters $\Re_i$, $i = 1, 2, ..., 8$. By formulating suitable Lyapunov functions and applying LaSalle's invariance principle, we show the global asymptotic stability for all equilibria. To demonstrate the theoretical results, we conduct numerical simulations. We look at how the antibody and CTL responses affect the dynamical behavior of HIV-1/HTLV-1 coinfection. Although the parameters of antibody and CTL responses have no effect on the basic reproduction ratio of HIV-1 single-infection ($\Re_1$) and HTLV-1 single-infection ($\Re_2$), it has been demonstrated that viral coinfection can be inhibited by immunological activation of antibody and CTL responses. This could potentially facilitate the advancement of therapeutic approaches for HIV-1 and HTLV-1, which have the capability to enhance the HIV-1-specific antibody and HTLV-1-specific CTL reactions.

Keywords: HIV-1/HTLV-1 coinfection, global stability, Lyapunov function, immune response

MSC: 01A01, 22B22, 31K13

1. Introduction

Human immunodeficiency virus type 1 (HIV-1) and human T-lymphotropic virus type 1 (HTLV-1) are two retroviruses that target $CD4^+$T cells, which is vital component of the adaptive immune system. HIV-1 eventually results in the development of acquired immunodeficiency syndrome (AIDS). HTLV-1-related illnesses comprise tropical spastic, paraparesis/HTLV-1 associated myelopathy (TSP/HAM), adult T-cell leukemia/lymphoma (ATLL), uveitis and infective dermatitis [1, 2]. The most significant means of HIV-1 and HTLV-1 transmissions are sharing needles, contaminated body fluids, and sexual contact. Breastfeeding is another way that HTLV-1 may spread [2]. The coinfection of HIV-1 and

DOI: https://doi.org/10.37256/cm.5220243870

HTLV-1 has been widely documented during the past ten years. Simultaneous infection by the two viruses has been found to impact pathogenic development and determine the course of related chronic disorders [3].

The human body's defensive mechanism is the immune system. This tool aids in the prevention of infections and illnesses. The immune system's function is to combat foreign objects, such as cancer cells and germs like bacteria, viruses, fungus, and parasites. Traditionally, there have been two categories of immune responses identified: the innate response, which is characterized by quick engagement but temporary and low specificity, and the adaptive response, which is characterized by delayed but specificity and permits the formation of immunological memory. The two primary components of the adaptive immune response are B cells and cytotoxic T lymphocytes (CTLs). Viral-infected cells are eliminated by CTLs, and B cells produce antibodies to combat and eradicate the viruses.

Our knowledge of the dynamic interactions that take place between human viruses, target cells, and immune response has improved thanks to mathematical models of within-host viral infection. The following biological parameters can be estimated using analytical and numerical analysis of viral infection models: (i) the half-lives of the virus and infected cell, as well as viral production; (ii) the efficacies of various antiviral drugs; (iii) the intensity of immune system responses; and (iv) long-term disease progression prediction.

Nowak and Bangham [4] formulated the fundamental model of HIV-1 single-infection, which is now used to explain the within-host dynamics of numerous other viruses. Three populations interact in this model, uninfected $CD4^+$T cells, infected $CD4^+$T cells, and free HIV-1 particles. A multitude of mathematical models have been developed to include several biological aspects, including latent reservoirs [5–7], time delay [8, 9], pharmacological treatments [9, 10], antibody response [11], CTL response [4], and reaction-diffusion [12], into the fundamental HIV-1 model. Numerous studies have modeled the dynamics of within-host HTLV-1 mono-infection while accounting for many factors, including: (i) latently HTLV-1-infected cells and leukemia cells [13, 14], (ii) CTL response [15, 16], (iii) time delay [17, 18], and (iv) reaction-diffusion [19].

There are people with coinfections with HIV-1 and HTLV-1 in a number of global geographic locations, including South America, Brazil, Europe, Mozambique, Japan and the Caribbean [20]. Recently, mathematical model for within-host HIV-1 and HTLV-1 coinfection with CTL response have been developed as [21, 22]:

$$\dot{X} = \overbrace{\lambda}^{\text{Production of uninfected CD4}^+\text{T cells}} - \overbrace{\gamma X}^{\text{Natural death}} - \overbrace{\phi_1 XV}^{\text{HIV-1 infectious transmission}} - \overbrace{\phi_2 XW}^{\text{HTLV-1 infectious transmission}}, \tag{1}$$

$$\dot{Y} = \overbrace{\phi_1 XV}^{\text{HIV-1 infectious transmission}} - \overbrace{\kappa Y}^{\text{Natural death}} - \overbrace{\tau SY}^{\text{Killing of HIV-1-infected cells via HIV-1-specific CTLs}}, \tag{2}$$

$$\dot{V} = \overbrace{\rho Y}^{\text{Production of HIV-1}} - \overbrace{\sigma V}^{\text{Natural death}}, \tag{3}$$

$$\dot{W} = \overbrace{\phi_2 XW}^{\text{HTLV-1 infectious transmission}} - \overbrace{\alpha W}^{\text{Natural death}} - \overbrace{\delta UW}^{\text{Killing of HTLV-1-infected cells via HTLV-1-specific CTLs}}, \tag{4}$$

$$\dot{S} = \overbrace{\varsigma SY}^{\text{Stimulation of HIV-1-specific CTLs}} - \overbrace{\upsilon S}^{\text{Natural death}}, \tag{5}$$

$$\dot{U} = \overbrace{\rho UW}^{\text{Stimulation of HTLV-1-specific CTLs}} - \overbrace{\varepsilon U}^{\text{Natural death}}, \tag{6}$$

where $X = X(t)$, $Y = Y(t)$, $V = V(t)$, $W = W(t)$, $S = S(t)$ and $U = U(t)$ are the concentrations of uninfected CD4$^+$T cells, HIV-1-infected cells, free HIV-1 particles, HTLV-1-infected cells, HIV-1-specific CTLs and HTLV-1-specific CTLs, respectively, at time t. The antibody response was includied in HIV-1 and HTLV-1 coinfection model as [23]:

$$\dot{X} = \lambda - \gamma X - \phi_1 XV - \phi_2 XW, \tag{7}$$

$$\dot{Y} = \phi_1 XV - \kappa Y,$$

$$\dot{V} = \rho Y - \sigma V - \theta VZ,$$

$$\dot{W} = \phi_2 XW - \alpha W,$$

$$\dot{Z} = \xi VZ - \mu Z. \tag{8}$$

where $Z = Z(t)$ is the concentration of HIV-1-specific antibodies. The HIV-1-specific antibodies are stimulated and die at rates ξVZ and μZ, respectively. The free HIV-1 particles are neutralized via antibodies at rate θVZ.

It was shown that model (1)–(6), examined in [21, 22], took into account the CTL response but disregarded the antibody response. However, model (7)–(8), which was examined in [23], ignored the CTL immunological response in favor of the antibody response. Modeling the co-infection of HIV-1 and HTLV-1 with both CTL and antibody responses has never been done previously. Thus, the purpose of this work is to develop and examine an HIV-1/HTLV-1 coinfection model that includes both CTL and antibody responses. We first look into the fundamental characteristics of the system, then we find all equilibria and discusses their existence and global stability. We construct suitable Lyapunov functions and use LaSalle's invariance principle (LIP) to investigate the global asymptotic stability of all equilibria. We use numerical simulations to demonstrate the theoretical findings. Finally, we discuss the obtained results.

Our suggested approach could be helpful in simulating various coinfections with viruses, as SARS-CoV-2/dengue/zika [24], SARS-CoV-2/HBV [25] and SARS-CoV-2/influenza [26].

2. HIV-1/HTLV-1 model with CTL and antibody responses

HIV-1/HTLV-1 infection model with CTL and antibody responses can be written as:

$$\dot{X} = \lambda - \gamma X - \phi_1 XV - \phi_2 XW, \tag{9}$$

$$\dot{Y} = \phi_1 XV - \kappa Y - \tau SY, \tag{10}$$

$$\dot{V} = \rho Y - \sigma V - \theta VZ, \tag{11}$$

$$\dot{W} = \phi_2 XW - \alpha W - \delta UW, \tag{12}$$

$$\dot{Z} = \xi VZ - \mu Z, \tag{13}$$

$$\dot{S} = \varsigma SY - \upsilon S, \tag{14}$$

$$\dot{U} = \rho UW - \varepsilon U. \tag{15}$$

We point out that model (9)–(15) leads to the model given in [21] when the antibody reaction is ignored, whereas it leads to the model given in [23] when the CTL response is ignored.

This model admits fourteen equilibrium points and studying the stability analysis of all equilibria will be too long. To simplify the model we consider two immune responses for HIV-1/HTLV-1 coinfection, HTLV-1-specific CTLs and HIV-1-specific antibodies. The effect of HIV-1-specific CTLs in killing the HIV-1-infected cells may be included in the parameter κ. As a result we study the following model:

$$\dot{X} = \lambda - \gamma X - \phi_1 XV - \phi_2 XW, \tag{16}$$

$$\dot{Y} = \phi_1 XV - \kappa Y, \tag{17}$$

$$\dot{V} = \rho Y - \sigma V - \theta VZ, \tag{18}$$

$$\dot{W} = \phi_2 XW - \alpha W - \delta UW, \tag{19}$$

$$\dot{Z} = \xi VZ - \mu Z, \tag{20}$$

$$\dot{U} = \rho UW - \varepsilon U. \tag{21}$$

A schematic representation of the model in (16)–(21) is illustrated in Figure 1. The basic and global properties of (16)–(21) will be invetigated in the next sections.

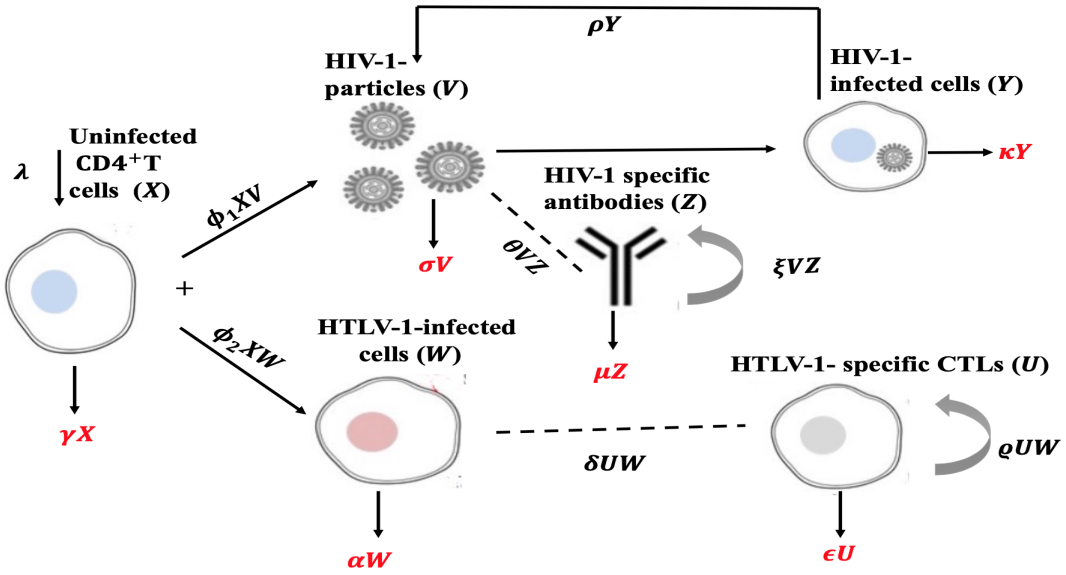


Figure 1. The illustrative diagram of the dynamics at a within-host HIV-1/HTLV-1 coinfection.

3. Basic results

3.1 *Nonnegativity and boundedness*

This subsection proves the nonnegativity and boundedness of the solutions of system (16)–(21).

Lemma 1. *The solution* $(X(t),Y(t),V(t),W(t),Z(t),U(t))$ *of system (16)–(21) are nonnegative and bounded.*

Proof. From system (16)–(21) we get

$$\dot{X}\,|_{X=0}=\lambda>0,\quad \dot{Y}\,|_{Y=0}=\phi_1 XV\geq 0 \text{ for any } X,V\geq 0,$$

$$\dot{V}\,|_{V=0}=\rho Y\geq 0 \text{ for any } Y\geq 0,$$

$$\dot{W}\,|_{W=0}=0,\qquad \dot{Z}\,|_{Z=0}=0,\quad \dot{U}\,|_{U=0}=0.$$

Hence, $(X(t),Y(t),V(t),W(t),Z(t),U(t))\in\mathbb{R}^6_{\geq 0}$ for all $t\geq 0$ when $(X(0),Y(0),V(0),W(0),Z(0),U(0))\in\mathbb{R}^6_{\geq 0}$ (see Proposition B.7 of [27]). Hence, X,Y,V,W,Z and U are nonnegative.

Now, we prove the boundedness of X, Y, V, W, Z and U. We define

$$\Psi=X+Y+\frac{\kappa}{2\rho}V+W+\frac{\kappa\theta}{2\rho\xi}Z+\frac{\delta}{\rho}U.$$

Then, we have

$$\dot{\Psi} = \lambda - \gamma X - \phi_1 XV - \phi_2 XW + \phi_1 XV - \kappa Y + \frac{\kappa}{2\rho}\left[\rho Y - \sigma V - \theta VZ\right]$$

$$+ \phi_2 XW - \alpha W - \delta UW + \frac{\kappa\theta}{2\rho\xi}\left[\xi VZ - \mu Z\right] + \frac{\delta}{\rho}\left[\rho UW - \varepsilon U\right]$$

$$= \lambda - \gamma X - \frac{\kappa}{2}Y - \frac{\sigma\kappa}{2\rho}V - \alpha W - \frac{\kappa\theta\mu}{2\rho\xi}Z - \frac{\varepsilon\delta}{\rho}U$$

$$\leq \lambda - \eta\left[X + Y + \frac{\kappa}{2\rho}V + W + \frac{\kappa\theta}{2\rho\xi}Z + \frac{\delta}{\rho}U\right]$$

$$= \lambda - \eta\Psi,$$

where $\eta = \min\{\gamma, \frac{\kappa}{2}, \sigma, \alpha, \mu, \varepsilon\}$. If $\Psi(0) \leq \omega_1$, then, $0 \leq \Psi(t) \leq \omega_1$ for all $t \geq 0$, where $\omega_1 = \frac{\lambda}{\eta}$. Since $X, Y, V, W, Z, U \geq 0$, then $0 \leq X(t), Y(t), W(t) \leq \omega_1, 0 \leq V(t) \leq \omega_2, 0 \leq Z(t) \leq \omega_3, 0 \leq U(t) \leq \omega_4$ if $X(0) + Y(0) + \frac{\kappa}{2\rho}V(0) + W(0) + \frac{\kappa\theta}{2\rho\xi}Z(0) + \frac{\delta}{\rho}U(0) \leq \omega_1$, where $\omega_2 = \frac{2\rho}{\kappa}\omega_1$, $\omega_3 = \frac{2\rho\xi}{\kappa\theta}\omega_1$, and $\omega_4 = \frac{\rho}{\delta}\omega_1$. □

3.2 *Threshold parameters and equilibria*

Here, we find all equilibria of model (16)–(21) as well as the threshold parameters that guarantee the existence of the model's equilibria. An equilibrium point $\Xi = (X, Y, V, W, Z, U)$ satisfies:

$$0 = \lambda - \gamma X - \phi_1 XV - \phi_2 XW, \tag{22}$$

$$0 = \phi_1 XV - \kappa Y, \tag{23}$$

$$0 = \rho Y - \sigma V - \theta VZ, \tag{24}$$

$$0 = \phi_2 XW - \alpha W - \delta UW, \tag{25}$$

$$0 = \xi VZ - \mu Z, \tag{26}$$

$$0 = \rho UW - \varepsilon U. \tag{27}$$

Solving Eqs. (22)–(27), we obtain eight equilibria as follows:
(i) The uninfected equilibrium, $\Xi_0 = (X_0, 0, 0, 0, 0, 0)$, where $X_0 = \lambda/\gamma$.
(ii) HIV-1 single-infection equilibrium without antibody response $\Xi_1 = (X_1, Y_1, V_1, 0, 0, 0)$, where

$$X_1 = \frac{\kappa\sigma}{\rho\phi_1}, \quad Y_1 = \frac{\gamma\sigma}{\rho\phi_1}\left(\frac{X_0\rho\phi_1}{\kappa\sigma} - 1\right), \quad V_1 = \frac{\gamma}{\phi_1}\left(\frac{X_0\rho\phi_1}{\kappa\sigma} - 1\right).$$

Hence, $Y_1 > 0$ and $V_1 > 0$ when

$$\frac{X_0\rho\phi_1}{\kappa\sigma} > 1.$$

We define the basic HIV-1 single-infection reproductive ratio as:

$$\Re_1 = \frac{X_0\rho\phi_1}{\kappa\sigma}.$$

The determination of whether an HIV-1 single-infection can be confirmed is contingent upon the parameter $\Re_1$. Thus, we can write

$$X_1 = \frac{X_0}{\Re_1},\ \ Y_1 = \frac{\gamma\sigma}{\rho\phi_1}(\Re_1 - 1),\ \ V_1 = \frac{\gamma}{\phi_1}(\Re_1 - 1).$$

Consequently, Ξ_1 exists if $\Re_1 > 1$.

(iii) HTLV-1 single-infection equilibrium without CTL response, $\Xi_2 = (X_2, 0, 0, W_2, 0, 0)$, where

$$X_2 = \frac{\alpha}{\phi_2},\ \ W_2 = \frac{\gamma}{\phi_2}\left(\frac{X_0\phi_2}{\alpha} - 1\right).$$

Hence, $W_2 > 0$ when

$$\frac{X_0\phi_2}{\alpha} > 1.$$

We define the basic HTLV-1-infection reproductive ratio as:

$$\Re_2 = \frac{X_0\phi_2}{\alpha}.$$

The determination of whether an HTLV-1 single-infection can be confirmed is contingent upon the parameter $\Re_2$. Thus, we can write

$$X_2 = \frac{X_0}{\Re_2},\ \ W_2 = \frac{\gamma}{\phi_2}(\Re_2 - 1).$$

Therefore, Ξ_2 exists if $\Re_2 > 1$.

(iv) HIV-1 single-infection equilibrium with stimulated HIV-1-specific antibody response, $\Xi_3 = (X_3, Y_3, V_3, 0, Z_3, 0)$, where

$$X_3 = \frac{\lambda\xi}{\gamma\xi + \mu\phi_1},\quad Y_3 = \frac{\lambda\phi_1\mu}{\kappa(\gamma\xi + \mu\phi_1)},$$

$$V_3 = \frac{\mu}{\xi},\quad Z_3 = \frac{\sigma}{\theta}\left[\frac{\lambda\xi\rho\phi_1}{\kappa\sigma(\gamma\xi + \mu\phi_1)} - 1\right].$$

We note that Ξ_3 exists when

$$\frac{\lambda\xi\rho\phi_1}{\kappa\sigma(\gamma\xi + \mu\phi_1)} > 1.$$

The HIV-1-specific antibody activation ratio in case of HIV-1 single-infection is:

$$\Re_3 = \frac{\lambda\xi\rho\phi_1}{\kappa\sigma(\gamma\xi+\mu\phi_1)}.$$

Thus, $Z_3 = \frac{\sigma}{\theta}(\Re_3 - 1)$.

The activation of HIV-1-specific antibody response in the absence of HTLV-1 infection is contingent upon the parameter $\Re_3$.

(v) HTLV-1 single-infection equilibrium with stimulated HTLV-1-specific CTL response, $\Xi_4 = (X_4, 0, 0, W_4, 0, U_4)$, where

$$X_4 = \frac{\lambda\rho}{\gamma\rho+\varepsilon\phi_2}, \quad W_4 = \frac{\varepsilon}{\rho}, \quad U_4 = \frac{\alpha}{\delta}\left(\frac{\lambda\rho\phi_2}{\alpha(\gamma\rho+\varepsilon\phi_2)} - 1\right).$$

Clearly Ξ_4 exists if

$$\frac{\lambda\rho\phi_2}{\alpha(\gamma\rho+\varepsilon\phi_2)} > 1.$$

The HTLV-1-specific CTL activation ratio for HTLV-1 single-infection is:

$$\Re_4 = \frac{\lambda\rho\phi_2}{\alpha(\gamma\rho+\varepsilon\phi_2)}.$$

Thus, $U_4 = \frac{\alpha}{\delta}(\Re_4 - 1)$. The activation of HTLV-1-specific CTL response in the absence of HIV-1 infection is contingent upon the parameter $\Re_4$.

(vi) HIV-1/HTLV-1 coinfection equilibrium with only stimulated HIV-1-specific antibody response, $\Xi_5 = (X_5, Y_5, V_5, W_5, Z_5, 0)$, where

$$X_5 = \frac{\alpha}{\phi_2} = X_2, \quad Y_5 = \frac{\alpha\mu\phi_1}{\kappa\xi\phi_2}, \quad V_5 = \frac{\mu}{\xi} = V_3,$$

$$W_5 = \frac{\gamma\xi+\mu\phi_1}{\xi\phi_2}\left(\frac{\lambda\xi\phi_2}{\alpha(\gamma\xi+\mu\phi_1)} - 1\right),$$

$$Z_5 = \frac{\sigma}{\theta}\left(\frac{\alpha\rho\phi_1}{\sigma\kappa\phi_2} - 1\right) = \frac{\sigma}{\theta}(\Re_1/\Re_2 - 1).$$

Obviously Ξ_5 exists if,

$$\frac{\Re_1}{\Re_2} > 1 \text{ and } \frac{\lambda\xi\phi_2}{\alpha(\gamma\xi+\mu\phi_1)} > 1.$$

The HTLV-1 infection reproductive ratio in the presence of HIV-1 infection is:

$$\Re_5 = \frac{\lambda\xi\phi_2}{\alpha(\gamma\xi+\mu\phi_1)}.$$

The parameter $\Re_5$ locates the potential for coinfection of HTLV-1 in patients already infected with HIV-1. Hence,

$$W_5 = \frac{\gamma\xi+\mu\phi_1}{\xi\phi_2}(\Re_5 - 1),$$

and then Ξ_5 exists if $\Re_1/\Re_2 > 1$ and $\Re_5 > 1$.

(vii) HIV-1/HTLV-1 coinfection equilibrium with only stimulated HTLV-1-specific CTL response, $\Xi_6 = (X_6, Y_6, V_6, W_6, 0, U_6)$, where

$$X_6 = \frac{\kappa\sigma}{\rho\phi_1}, \quad Y_6 = \frac{\sigma(\gamma\rho + \varepsilon\phi_2)}{\rho\rho\phi_1}\left(\frac{\lambda\rho\rho\phi_1}{\kappa\sigma(\gamma\rho + \varepsilon\phi_2)} - 1\right),$$

$$V_6 = \frac{\gamma\rho + \varepsilon\phi_2}{\rho\phi_1}\left(\frac{\lambda\rho\rho\phi_1}{\kappa\sigma(\gamma\rho + \varepsilon\phi_2)} - 1\right), \quad W_6 = \frac{\varepsilon}{\rho} = W_4,$$

$$U_6 = \frac{\alpha}{\delta}\left(\frac{\sigma\kappa\phi_2}{\alpha\rho\phi_1} - 1\right) = \frac{\alpha}{\delta}\left(\Re_2/\Re_1 - 1\right).$$

We note that Ξ_6 exists when

$$\frac{\Re_2}{\Re_1} > 1 \text{ and } \frac{\lambda\rho\rho\phi_1}{\kappa\sigma(\gamma\rho + \varepsilon\phi_2)} > 1.$$

We define the HIV-1 infection reproductive ratio in the context of HTLV-1 infection as:

$$\Re_6 = \frac{\lambda\rho\rho\phi_1}{\kappa\sigma(\gamma\rho + \varepsilon\phi_2)}.$$

Thus,

$$Y_6 = \frac{\sigma(\gamma\rho + \varepsilon\phi_2)}{\rho\rho\phi_1}\left(\Re_6 - 1\right), \quad V_6 = \frac{(\gamma\rho + \varepsilon\phi_2)}{\rho\phi_1}\left(\Re_6 - 1\right).$$

The parameter $\Re_6$ locates the potential for coinfection of HIV-1 in patients already infected with HTLV-1.

(viii) HIV-1/HTLV-1 coinfection equilibrium with stimulated both HIV-1-specific antibody and HTLV-1-specific CTL responses $\Xi_7 = (X_7, Y_7, V_7, W_7, Z_7, U_7)$, where

$$X_7 = \frac{\lambda\xi\rho}{\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2}, \quad Y_7 = \frac{\lambda\mu\rho\phi_1}{\kappa(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)},$$

$$V_7 = \frac{\mu}{\xi} = V_3, \quad W_7 = \frac{\varepsilon}{\rho} = W_6 = W_4,$$

$$Z_7 = \frac{\sigma}{\theta}\left(\frac{\lambda\xi\rho\rho\phi_1}{\kappa\sigma(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)} - 1\right),$$

$$U_7 = \frac{\alpha}{\delta}\left(\frac{\lambda\xi\rho\phi_2}{\alpha(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)} - 1\right).$$

It is obvious that Ξ_7 exists when

$$\frac{\lambda\xi\rho\rho\phi_1}{\kappa\sigma(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)} > 1, \quad \frac{\lambda\xi\rho\phi_2}{\alpha(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)} > 1.$$

Now we define $\Re_7$ and $\Re_8$ as:

$$\Re_7 = \frac{\lambda\xi\rho\rho\phi_1}{\kappa\sigma(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)}, \quad \Re_8 = \frac{\lambda\xi\rho\phi_2}{\alpha(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)},$$

where $\Re_7$ is the HIV-1-specific antibody activation ratio in case of HIV-1/HTLV-1 coinfection, and $\Re_8$ is the HTLV-1-specific CTL activation ratio in case of HIV-1/HTLV-1 coinfection. Hence, $U_7 = \frac{\alpha}{\delta}(\Re_8 - 1)$ and $Z_7 = \frac{\sigma}{\theta}(\Re_7 - 1)$. As a consequence, the equilibrium Ξ_7 exists when $\Re_7 > 1$ and $\Re_8 > 1$.

To sum up, we have eight threshold parameters which locate the existence of the model's equilibria as given below:

$$\Re_1 = \frac{X_0\rho\phi_1}{\kappa\sigma}, \quad \Re_2 = \frac{X_0\phi_2}{\alpha}, \quad \Re_3 = \frac{\lambda\xi\rho\phi_1}{\kappa\sigma(\gamma\xi + \mu\phi_1)},$$

$$\Re_4 = \frac{\lambda\rho\phi_2}{\alpha(\gamma\rho + \varepsilon\phi_2)}, \quad \Re_5 = \frac{\lambda\xi\phi_2}{\alpha(\gamma\xi + \mu\phi_1)}, \quad \Re_6 = \frac{\lambda\rho\rho\phi_1}{\kappa\sigma(\gamma\rho + \varepsilon\phi_2)},$$

$$\Re_7 = \frac{\lambda\xi\rho\rho\phi_1}{\kappa\sigma(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)}, \quad \Re_8 = \frac{\lambda\xi\rho\phi_2}{\alpha(\gamma\xi\rho + \mu\rho\phi_1 + \varepsilon\xi\phi_2)}. \tag{28}$$

4. Global stability analysis

This section formulates Lyapunov function and uses LIP to establish the global asymptotic stability of equilibria. We follow the method presented in [7, 21, 23]. We'll use the arithmetic and geometric means inequality below.:

$$\frac{g_1 + g_2 + \ldots + g_n}{n} \geq \sqrt[n]{g_1 g_2 \cdots g_n}, \quad g_i \geq 0, i = 1, 2, \ldots, n. \tag{29}$$

Define

$$\Omega_i = \left\{ (X, Y, V, W, Z, U) : \frac{d\Lambda_i}{dt} = 0 \right\}, \quad i = 0, 1, 2, \ldots, 7.$$

and $\tilde{\Omega}_i$ be the greatest subset of Ω_i that is invariant. Moreover, we use the function

$$F(\varkappa) = \varkappa - 1 - \ln\varkappa.$$

According to the following findings, independent of the beginning conditions (any illness phases), both HIV-1 and HTLV-1 infections are projected to die out when $\Re_1 \leq 1$ and $\Re_2 \leq 1$.

Theorem 1. *Suppose that* $\Re_1 \leq 1$ *and* $\Re_2 \leq 1$*, then* Ξ_0 *is globally asymptotically stable (G.A.S).*

Proof. Define

$$\Lambda_0 = X_0 F\left(\frac{X}{X_0}\right) + Y + \frac{\kappa}{\rho}V + W + \frac{\kappa\theta}{\rho\xi}Z + \frac{\delta}{\rho}U.$$

We note that, $\Lambda_0 > 0$ for all $X, Y, V, W, Z, U > 0$, and $\Lambda_0(X_0, 0, 0, 0, 0, 0) = 0$. We calculate $\frac{d\Lambda_0}{dt}$ along the solutions of model (16)–(21) as:

$$\frac{d\Lambda_0}{dt} = \left(1 - \frac{X_0}{X}\right)\dot{X} + \dot{Y} + \frac{\kappa}{\rho}\dot{V} + \dot{W} + \frac{\kappa\theta}{\rho\xi}\dot{Z} + \frac{\delta}{\rho}\dot{U}$$

$$= \left(1 - \frac{X_0}{X}\right)(\lambda - \gamma X - \phi_1 XV - \phi_2 XW) + \phi_1 XV - \kappa Y + \frac{\kappa}{\rho}(\rho Y - \sigma V - \theta VZ)$$

$$+ \phi_2 XW - \alpha W - \delta UW + \frac{\kappa\theta}{\rho\xi}(\xi VZ - \mu Z) + \frac{\delta}{\rho}(\rho UW - \varepsilon U)$$

$$= \left(1 - \frac{X_0}{X}\right)(\lambda - \gamma X) + \phi_1 X_0 V + \phi_2 X_0 W - \frac{\kappa\sigma}{\rho}V - \alpha W - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\delta\varepsilon}{\rho}U.$$

Using $\lambda = \gamma X_0$, we get:

$$\frac{d\Lambda_0}{dt} = -\frac{\gamma(X - X_0)^2}{X} + \frac{\kappa\sigma}{\rho}(\Re_1 - 1)V + \alpha(\Re_2 - 1)W - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\delta\varepsilon}{\rho}U.$$

Since $\Re_1 \leq 1$ and $\Re_2 \leq 1$, then $\frac{d\Lambda_0}{dt} \leq 0$ for any $X, V, W, Z, U > 0$. In addition $\frac{d\Lambda_0}{dt} = 0$ when $X = X_0$ and $V = W = Z = U = 0$. Solutions of system (16)–(21) are attracted to to $\tilde{\Omega}_0$ [28]. Since $\tilde{\Omega}_0$ is invariant with respect to (16), on $\tilde{\Omega}_0$, we have from Eq. (18)

$$0 = \dot{V} = \rho Y \Longrightarrow Y(t) = 0, \text{ for all } t.$$

Therefore, $\tilde{\Omega}_0 = \{\Xi_0\}$ and applying LIP [29], we obtain that Ξ_0 is G.A.S. □

Based on the results below, it can be concluded that regardless of initial states, HIV-1 single-infection with unstimulated antibody response is always established when $\Re_1 > 1$, $\Re_2/\Re_1 \leq 1$ and $\Re_3 \leq 1$.

Theorem 2. *If* $\Re_1 > 1$, $\Re_2/\Re_1 \leq 1$ *and* $\Re_3 \leq 1$, *then* Ξ_1 *is G.A.S.*

Proof. Define Λ_1 as:

$$\Lambda_1 = X_1 F\left(\frac{X}{X_1}\right) + Y_1 F\left(\frac{Y}{Y_1}\right) + \frac{\kappa}{\rho}V_1 F\left(\frac{V}{V_1}\right) + W + \frac{\kappa\theta}{\rho\xi}Z + \frac{\delta}{\rho}U.$$

We calculate $\frac{d\Lambda_1}{dt}$ as:

$$\frac{d\Lambda_1}{dt} = \left(1-\frac{X_1}{X}\right)\dot{X} + \left(1-\frac{Y_1}{Y}\right)\dot{Y} + \frac{\kappa}{\rho}\left(1-\frac{V_1}{V}\right)\dot{V}$$

$$+\dot{W} + \frac{\kappa\theta}{\rho\xi}\dot{Z} + \frac{\delta}{\rho}\dot{U}$$

$$= \left(1-\frac{X_1}{X}\right)(\lambda - \gamma X - \phi_1 XV - \phi_2 XW) + \left(1-\frac{Y_1}{Y}\right)(\phi_1 XV - \kappa Y)$$

$$+\frac{\kappa}{\rho}\left(1-\frac{V_1}{V}\right)(\rho Y - \sigma V - \theta VZ) + \phi_2 XW - \alpha W - \delta UW$$

$$+\frac{\kappa\theta}{\rho\xi}(\xi VZ - \mu Z) + \frac{\delta}{\rho}(\rho UW - \varepsilon U). \tag{30}$$

Simplifying Eq. (30) as:

$$\frac{d\Lambda_1}{dt} = \left(1-\frac{X_1}{X}\right)(\lambda - \gamma X) + \phi_1 X_1 V + \phi_2 X_1 W - \phi_1 XV\frac{Y_1}{Y} + \kappa Y_1 - \frac{\kappa\sigma}{\rho}V$$

$$-\kappa Y\frac{V_1}{V} + \frac{\kappa\sigma}{\rho}V_1 + \frac{\kappa\theta}{\rho}V_1 Z - \alpha W - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\delta\varepsilon}{\rho}U.$$

Using the equilibrium conditions for Ξ_1:

$$\lambda = \gamma X_1 + \phi_1 X_1 V_1, \quad \phi_1 X_1 V_1 = \kappa Y_1, \quad Y_1 = \frac{\sigma}{\rho}V_1,$$

we obtain

$$\frac{d\Lambda_1}{dt} = \left(1-\frac{X_1}{X}\right)(\gamma X_1 - \gamma X) + 3\phi_1 X_1 V_1 - \phi_1 X_1 V_1\frac{X_1}{X} - \phi_1 X_1 V_1\frac{Y_1 XV}{YX_1V_1}$$

$$-\phi_1 X_1 V_1\frac{V_1 Y}{VY_1} + \alpha\left(\frac{\sigma\kappa\phi_2}{\alpha\rho\phi_1} - 1\right)W + \frac{\kappa\theta\mu}{\rho\xi}\left(\frac{\xi}{\mu}V_1 - 1\right)Z - \frac{\delta\varepsilon}{\rho}U. \tag{31}$$

Then, collecting terms of (31), we get

$$\frac{d\Lambda_1}{dt} = -\frac{\gamma(X-X_1)^2}{X} + \phi_1 X_1 V_1\left(3 - \frac{X_1}{X} - \frac{Y_1 XV}{YX_1V_1} - \frac{V_1 Y}{VY_1}\right)$$

$$+\alpha(\Re_2/\Re_1 - 1)W + \frac{\kappa\theta(\gamma\xi + \mu\phi_1)}{\rho\phi_1\xi}(\Re_3 - 1)Z - \frac{\delta\varepsilon}{\rho}U.$$

Using inequality (29), we get

$$3-\frac{X_1}{X}-\frac{Y_1XV}{YX_1V_1}-\frac{V_1Y}{VY_1}\leq 0.$$

Since $\Re_2/\Re_1 \leq 1$ and $\Re_3 \leq 1$, then $\frac{d\Lambda_1}{dt} \leq 0$ for all $X,Y,V,W,Z,U > 0$. Moreover, $\frac{d\Lambda_1}{dt} = 0$ when $X = X_1$, $Y = Y_1$, $V = V_1$ and $W = Z = U = 0$. Therefore, $\tilde{\Omega}_1 = \{\Xi_1\}$. Applying LIP, we obtain Ξ_1 is G.A.S. □

The findings below suggest that HTLV-1 single-infection with unstimulated CTL response is always established when $\Re_2 > 1$, $\Re_1/\Re_2 \leq 1$ and $\Re_4 \leq 1$, independent of initial conditions.

Theorem 3. *Let* $\Re_2 > 1$, $\Re_1/\Re_2 \leq 1$ *and* $\Re_4 \leq 1$, *then* Ξ_2 *is G.A.S.*

Proof. Consider

$$\Lambda_2 = X_2 F\left(\frac{X}{X_2}\right)+Y+\frac{\kappa}{\rho}V+W_2 F\left(\frac{W}{W_2}\right)+\frac{\kappa\theta}{\rho\xi}Z+\frac{\delta}{\rho}U.$$

We calculate $\frac{d\Lambda_2}{dt}$ as:

$$\frac{d\Lambda_2}{dt} = \left(1-\frac{X_2}{X}\right)\dot{X}+\dot{Y}+\frac{\kappa}{\rho}\dot{V}+\left(1-\frac{W_2}{W}\right)\dot{W}+\frac{\kappa\theta}{\rho\xi}\dot{Z}+\frac{\delta}{\rho}\dot{U}$$

$$= \left(1-\frac{X_2}{X}\right)(\lambda-\gamma X-\phi_1 XV-\phi_2 XW)+\phi_1 XV-\kappa Y$$

$$+\frac{\kappa}{\rho}(\rho Y-\sigma V-\theta VZ)+\left(1-\frac{W_2}{W}\right)(\phi_2 XW-\alpha W-\delta UW)$$

$$+\frac{\kappa\theta}{\rho\xi}(\xi VZ-\mu Z)+\frac{\delta}{\rho}(\rho UW-\varepsilon U). \tag{32}$$

Then simplifying Eq. (32) as:

$$\frac{d\Lambda_2}{dt} = \left(1-\frac{X_2}{X}\right)(\lambda-\gamma X)+\phi_1 X_2 V+\phi_2 X_2 W-\frac{\kappa\sigma}{\rho}V-\alpha W$$

$$-\phi_2 XW_2+\alpha W_2+\delta UW_2-\frac{\kappa\theta\mu}{\rho\xi}Z-\frac{\delta\varepsilon}{\rho}U.$$

Using the equilibrium conditions for Ξ_2:

$$\lambda = \gamma X_2+\phi_2 X_2 W_2, \quad \phi_2 X_2 W_2 = \alpha W_2,$$

we obtain

$$\frac{d\Lambda_2}{dt} = \left(1-\frac{X_2}{X}\right)(\gamma X_2 - \gamma X) + 2\phi_2 X_2 W_2 - \phi_2 X_2 W_2 \frac{X_2}{X} - \phi_2 X_2 W_2 \frac{X}{X_2}$$

$$+ \frac{\kappa\sigma}{\rho}(\frac{\phi_1\rho}{\kappa\sigma}X_2 - 1)V - \frac{\kappa\theta\mu}{\rho\xi}Z + \frac{\delta\varepsilon}{\rho}\left(\frac{\rho}{\varepsilon}W_2 - 1\right)U$$

$$= -\frac{\gamma(X-X_2)^2}{X} + \phi_2 X_2 W_2\left(2 - \frac{X_2}{X} - \frac{X}{X_2}\right) + \frac{\kappa\sigma}{\rho}(\Re_1/\Re_2 - 1)V$$

$$- \frac{\kappa\theta\mu}{\rho\xi}Z + \frac{\delta(\gamma\rho+\varepsilon\phi_2)}{\rho\phi_2}(\Re_4 - 1)U$$

$$= -\frac{(X-X_2)^2}{X}(\gamma + \phi_2 W_2) + \frac{\kappa\sigma}{\rho}(\Re_1/\Re_2 - 1)V - \frac{\kappa\theta\mu}{\rho\xi}Z + \frac{\delta(\gamma\rho+\varepsilon\phi_2)}{\rho\phi_2}(\Re_4 - 1)U.$$

Since $\Re_1/\Re_2 \leq 1$ and $\Re_4 \leq 1$, then $\frac{d\Lambda_2}{dt} \leq 0$ for all $X,V,Z,U > 0$. In addition, $\frac{d\Lambda_2}{dt} = 0$ when $X = X_2$ and $V = Z = U = 0$. Solutions of system (16)-(21) converge to $\tilde{\Omega}_2$ where $V = 0$ and $X = X_2$. Thus, $\dot{V} = 0$, $\dot{X} = 0$ and Eqs. (16), (18) provide

$$0 = \dot{X} = \lambda - \gamma X_2 - \phi_2 X_2 W \Longrightarrow W(t) = W_2, \text{ for all } t,$$

$$0 = \dot{V} = \rho Y \Longrightarrow Y(t) = 0, \text{ for all } t.$$

Therefore, $\tilde{\Omega}_2 = \{\Xi_2\}$. Applying LIP, we get Ξ_2 is G.A.S. □

The following finding demonstrates that, regardless of the starting points, an HIV-1 single-infection with an active antibody response is always founded when $\Re_3 > 1$ and $\Re_5 \leq 1$.

Theorem 4. *Suppose that* $\Re_3 > 1$ *and* $\Re_5 \leq 1$*, then* Ξ_3 *is G.A.S.*

Proof. Define

$$\Lambda_3 = X_3 F\left(\frac{X}{X_3}\right) + Y_3 F\left(\frac{Y}{Y_3}\right) + \frac{\kappa}{\rho}V_3 F\left(\frac{V}{V_3}\right) + W + \frac{\kappa\theta}{\rho\xi}Z_3 F\left(\frac{Z}{Z_3}\right) + \frac{\delta}{\rho}U.$$

We calculate $\frac{d\Lambda_3}{dt}$ as:

$$\frac{d\Lambda_3}{dt} = \left(1-\frac{X_3}{X}\right)\dot{X} + \left(1-\frac{Y_3}{Y}\right)\dot{Y} + \frac{\kappa}{\rho}\left(1-\frac{V_3}{V}\right)\dot{V} + \dot{W}$$

$$+ \frac{\kappa\theta}{\rho\xi}\left(1-\frac{Z_3}{Z}\right)\dot{Z} + \frac{\delta}{\rho}\dot{U}$$

$$= \left(1-\frac{X_3}{X}\right)(\lambda - \gamma X - \phi_1 XV - \phi_2 XW) + \left(1-\frac{Y_3}{Y}\right)(\phi_1 XV - \kappa Y)$$

$$+ \frac{\kappa}{\rho}\left(1-\frac{V_3}{V}\right)(\rho Y - \sigma V - \theta VZ) + \phi_2 XW - \alpha W - \delta UW$$

$$+ \frac{\kappa\theta}{\rho\xi}\left(1-\frac{Z_3}{Z}\right)(\xi VZ - \mu Z) + \frac{\delta}{\rho}(\rho UW - \varepsilon U). \tag{33}$$

Collecting terms as:

$$\frac{d\Lambda_3}{dt} = \left(1-\frac{X_3}{X}\right)(\lambda - \gamma X) + \phi_1 X_3 V + \phi_2 X_3 W - \phi_1 XV\frac{Y_3}{Y} + \kappa Y_3 - \frac{\kappa\sigma}{\rho}V - \kappa Y\frac{V_3}{V}$$

$$+ \frac{\kappa\sigma}{\rho}V_3 + \frac{\kappa\theta}{\rho}V_3 Z - \alpha W - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\kappa\theta}{\rho}Z_3 V + \frac{\kappa\theta\mu}{\rho\xi}Z_3 - \frac{\delta\varepsilon}{\rho}U.$$

Using the equilibrium conditions for Ξ_3:

$$\lambda = \gamma X_3 + \phi_1 X_3 V_3, \quad \phi_1 X_3 V_3 = \kappa Y_3, \quad Y_3 = \frac{\sigma}{\rho}V_3 + \frac{\theta}{\rho}V_3 Z_3, \quad V_3 = \frac{\mu}{\xi},$$

and collecting terms we obtain

$$\frac{d\Lambda_3}{dt} = \left(1-\frac{X_3}{X}\right)(\gamma X_3 - \gamma X) + 3\phi_1 X_3 V_3 - \phi_1 X_3 V_3\frac{X_3}{X} - \phi_1 X_3 V_3\frac{Y_3 XV}{YX_3V_3}$$

$$- \phi_1 X_3 V_3\frac{V_3 Y}{VY_3} + \alpha\left(\frac{\lambda\xi\phi_2}{\alpha(\gamma\xi + \mu\phi_1)} - 1\right)W - \frac{\delta\varepsilon}{\rho}U$$

$$= -\frac{\gamma(X-X_3)^2}{X} + \phi_1 X_3 V_3\left(3 - \frac{X_3}{X} - \frac{Y_3 XV}{YX_3V_3} - \frac{V_3 Y}{VY_3}\right) + \alpha(\Re_5 - 1)W - \frac{\delta\varepsilon}{\rho}U.$$

Using inequality (29) and since $\Re_5 \leq 1$, we get $\frac{d\Lambda_3}{dt} \leq 0$ for any $X, Y, V, W, U > 0$. In addition, $\frac{d\Lambda_3}{dt} = 0$ when $X = X_3$, $Y = Y_3$, $V = V_3$ and $W = U = 0$. Trajectories of system (16)-(21) attracted to $\tilde{\Omega}_3$ where $Y = Y_3$ and $V = V_3$. Then $\dot{V} = 0$, and Eq. (18) provide

$$0 = \dot{V} = \rho Y_3 - \sigma V_3 - \theta V_3 Z \Longrightarrow Z(t) = Z_3, \text{ for any } t.$$

Therefore, $\tilde{\Omega}_3 = \{\Xi_3\}$. Applying LIP, yields Ξ_3 is G.A.S. □

The following finding demonstrates that, regardless of the starting points, an HTLV-1 single-infection with stimulated CTL response is always founded when $\Re_4 > 1$ and $\Re_6 \leq 1$.

Theorem 5. *Suppose that* $\Re_4 > 1$ *and* $\Re_6 \leq 1$*, then* Ξ_4 *is G.A.S.*

Proof. Consider

$$\Lambda_4 = X_4 F\left(\frac{X}{X_4}\right) + Y + \frac{\kappa}{\rho}V + W_4 F\left(\frac{W}{W_4}\right) + \frac{\kappa\theta}{\rho\xi}Z + \frac{\delta}{\rho}U_4 F\left(\frac{U}{U_4}\right).$$

Calculating $\frac{d\Lambda_4}{dt}$ as:

$$\begin{aligned}
\frac{d\Lambda_4}{dt} &= \left(1 - \frac{X_4}{X}\right)\dot{X} + \dot{Y} + \frac{\kappa}{\rho}\dot{V} + \left(1 - \frac{W_4}{W}\right)\dot{W} \\
&+ \frac{\kappa\theta}{\rho\xi}\dot{Z} + \frac{\delta}{\rho}\left(1 - \frac{U_4}{U}\right)\dot{U} \\
&= \left(1 - \frac{X_4}{X}\right)(\lambda - \gamma X - \phi_1 XV - \phi_2 XW) + \phi_1 XV - \kappa Y \\
&+ \frac{\kappa}{\rho}(\rho Y - \sigma V - \theta VZ) + \left(1 - \frac{W_4}{W}\right)(\phi_2 XW - \alpha W - \delta UW) \\
&+ \frac{\kappa\theta}{\rho\xi}(\xi VZ - \mu Z) + \frac{\delta}{\rho}\left(1 - \frac{U_4}{U}\right)(\rho UW - \varepsilon U). \qquad (34)
\end{aligned}$$

Eq. (34) can be written as:

$$\begin{aligned}
\frac{d\Lambda_4}{dt} &= \left(1 - \frac{X_4}{X}\right)(\lambda - \gamma X) + \phi_1 X_4 V + \phi_2 X_4 W - \frac{\kappa\sigma}{\rho}V - \alpha W \\
&- \phi_2 XW_4 + \alpha W_4 + \delta UW_4 - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\delta\varepsilon}{\rho}U - \delta U_4 W + \frac{\delta\varepsilon}{\rho}U_4.
\end{aligned}$$

Using the equilibrium conditions for Ξ_4:

$$\lambda = \gamma X_4 + \phi_2 X_4 W_4, \quad \phi_2 X_4 W_4 = \alpha W_4 + \delta U_4 W_4, \quad W_4 = \frac{\varepsilon}{\rho},$$

we obtain

$$\frac{d\Lambda_4}{dt} = \left(1-\frac{X_4}{X}\right)(\gamma X_4 - \gamma X) + 2\phi_2 X_4 W_4 - \phi_2 X_4 W_4 \frac{X_4}{X} - \phi_2 X_4 W_4 \frac{X}{X_4}$$

$$+ \frac{\kappa\sigma}{\rho}\left[\frac{\lambda\rho\rho\phi_1}{\kappa\sigma(\gamma\rho+\varepsilon\phi_2)} - 1\right]V - \frac{\kappa\theta\mu}{\rho\xi}Z$$

$$= -\frac{\gamma(X-X_4)^2}{X} + \phi_2 X_4 W_4\left(2 - \frac{X_4}{X} - \frac{X}{X_4}\right) + \frac{\kappa\sigma}{\rho}(\Re_6 - 1)V - \frac{\kappa\theta\mu}{\rho\xi}Z$$

$$= -\frac{(\gamma+\phi_2 W_4)(X-X_4)^2}{X} + \frac{\kappa\sigma}{\rho}(\Re_6 - 1)V - \frac{\kappa\theta\mu}{\rho\xi}Z.$$

Since $\Re_6 \leq 1$, then $\frac{d\Lambda_4}{dt} \leq 0$ for all $X,V,Z > 0$. Further, $\frac{d\Lambda_4}{dt} = 0$ when $X = X_4$, $V = 0$ and $Z = 0$. System's solutions converge to $\tilde{\Omega}_4$ where $X = X_4, V = 0$ and $Z = 0$, Then $\dot{X} = 0$, $\dot{V} = 0$ and Eqs. (16), (18) imply

$$0 = \dot{X} = \lambda - \gamma X_4 - \phi_2 X_4 W \Longrightarrow W(t) = W_4, \text{ for any } t,$$

$$0 = \dot{V} = \rho Y \Longrightarrow Y(t) = 0, \text{ for any } t.$$

Since $W = W_4$, then $\dot{W} = 0$ and Eq. (19) implies

$$0 = \dot{W} = \phi_2 X_4 W_4 - \alpha W_4 - \delta U W_4 \Longrightarrow U(t) = U_4, \text{ for any } t.$$

Therefore, $\tilde{\Omega}_4 = \{\Xi_4\}$ and by applying LIP, we get Ξ_4 is G.A.S. □

Regardless of the initial states, the following result indicates that, the HIV-1 and HTLV-1 coinfection with solely stimulated HIV-1-specific antibodies is always founded when $\Re_5 > 1$, $\Re_1/\Re_2 > 1$ and $\Re_8 \leq 1$.

Theorem 6. *If* $\Re_5 > 1$, $\Re_1/\Re_2 > 1$ *and* $\Re_8 \leq 1$, *then* Ξ_5 *is G.A.S.*

Proof. Define

$$\Lambda_5 = X_5 F\left(\frac{X}{X_5}\right) + Y_5 F\left(\frac{Y}{Y_5}\right) + \frac{\kappa}{\rho}V_5 F\left(\frac{V}{V_5}\right) + W_5 F\left(\frac{W}{W_5}\right) + \frac{\kappa\theta}{\rho\xi}Z_5 F\left(\frac{Z}{Z_5}\right) + \frac{\delta}{\rho}U.$$

Calculating $\frac{d\Lambda_5}{dt}$ as:

$$\frac{d\Lambda_5}{dt} = \left(1-\frac{X_5}{X}\right)\dot{X} + \left(1-\frac{Y_5}{Y}\right)\dot{Y} + \frac{\kappa}{\rho}\left(1-\frac{V_5}{V}\right)\dot{V}$$

$$+\left(1-\frac{W_5}{W}\right)\dot{W} + \frac{\kappa\theta}{\rho\xi}\left(1-\frac{Z_5}{Z}\right)\dot{Z} + \frac{\delta}{\rho}\dot{U}$$

$$= \left(1-\frac{X_5}{X}\right)(\lambda-\gamma X-\phi_1 XV-\phi_2 XW) + \left(1-\frac{Y_5}{Y}\right)(\phi_1 XV-\kappa Y)$$

$$+\frac{\kappa}{\rho}\left(1-\frac{V_5}{V}\right)(\rho Y-\sigma V-\theta VZ) + \left(1-\frac{W_5}{W}\right)(\phi_2 XW-\alpha W-\delta UW)$$

$$+\frac{\kappa\theta}{\rho\xi}\left(1-\frac{Z_5}{Z}\right)(\xi VZ-\mu Z) + \frac{\delta}{\rho}(\rho UW-\varepsilon U). \tag{35}$$

Eq. (35) can be simplified as:

$$\frac{d\Lambda_5}{dt} = \left(1-\frac{X_5}{X}\right)(\lambda-\gamma X) + \phi_1 X_5 V + \phi_2 X_5 W - \phi_1 XV\frac{Y_5}{Y} + \kappa Y_5$$

$$-\frac{\kappa\sigma}{\rho}V - \kappa Y\frac{V_5}{V} + \frac{\kappa\sigma}{\rho}V_5 + \frac{\kappa\theta}{\rho}V_5 Z - \alpha W - \phi_2 XW_5 + \alpha W_5$$

$$+\delta UW_5 - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\kappa\theta}{\rho}Z_5 V + \frac{\kappa\theta\mu}{\rho\xi}Z_5 - \frac{\delta\varepsilon}{\rho}U.$$

Using the equilibrium conditions for Ξ_5:

$$\lambda = \gamma X_5 + \phi_1 X_5 V_5 + \phi_2 X_5 W_5, \quad \phi_1 X_5 V_5 = \kappa Y_5,$$

$$\phi_2 X_5 W_5 = \alpha W_5, \quad Y_5 = \frac{\sigma}{\rho}V_5 + \frac{\theta}{\rho}V_5 Z_5, \quad V_5 = \frac{\mu}{\xi},$$

and collecting terms we obtain

$$\frac{d\Lambda_5}{dt} = \left(1-\frac{X_5}{X}\right)(\gamma X_5 - \gamma X) + 3\phi_1 X_5 V_5 + 2\phi_2 X_5 W_5 - \phi_1 X_5 V_5 \frac{X_5}{X} - \phi_2 X_5 W_5 \frac{X_5}{X}$$

$$- \phi_1 X_5 V_5 \frac{Y_5 X V}{Y X_5 V_5} - \phi_1 X_5 V_5 \frac{V_5 Y}{V Y_5} - \phi_2 X_5 W_5 \frac{X}{X_5} + \frac{\delta\varepsilon}{\rho}\left(\frac{\rho}{\varepsilon} W_5 - 1\right) U$$

$$= -\frac{\gamma(X-X_5)^2}{X} + \phi_1 X_5 V_5 \left(3 - \frac{X_5}{X} - \frac{Y_5 X V}{Y X_5 V_5} - \frac{V_5 Y}{V Y_5}\right)$$

$$+ \phi_2 X_5 W_5 \left(2 - \frac{X_5}{X} - \frac{X}{X_5}\right) + \frac{\delta(\phi_1 \mu\rho + \phi_2 \varepsilon\xi + \gamma\xi\rho)}{\xi\rho\phi_2}(\Re_8 - 1) U$$

$$= -\frac{(\gamma + \phi_2 W_5)(X-X_5)^2}{X} + \phi_1 X_5 V_5 \left(3 - \frac{X_5}{X} - \frac{Y_5 X V}{Y X_5 V_5} - \frac{V_5 Y}{V Y_5}\right)$$

$$+ \frac{\delta(\phi_1 \mu\rho + \phi_2 \varepsilon\xi + \gamma\xi\rho)}{\xi\rho\phi_2}(\Re_8 - 1) U.$$

Since $\Re_8 \leq 1$, then inequality (29) gives $\frac{d\Lambda_5}{dt} \leq 0$ for any $X, Y, V, U > 0$. In addition, $\frac{d\Lambda_5}{dt} = 0$ when $X = X_5$, $Y = Y_5$, $V = V_5$ and $U = 0$. Trajectories of system (16)-(21) tend to $\tilde{\Omega}_5$ where $X = X_5$ and $V = V_5$. Then $\dot{X} = 0$, $\dot{V} = 0$ and Eqs. (16), (18) provide

$$0 = \dot{X} = \lambda - \gamma X_5 - \phi_1 X_5 V_5 - \phi_2 X_5 W \Longrightarrow W(t) = W_5, \text{ for any } t,$$

$$0 = \dot{V} = \rho Y_5 - \sigma V_5 - \theta V_5 Z \Longrightarrow Z(t) = Z_5, \text{ for any } t.$$

Thus, $\tilde{\Omega}_5 = \{\Xi_5\}$ and LIP implies that Ξ_5 is G.A.S. □

Regardless of the initial states, the following result indicates that, the HIV-1 and HTLV-1 coinfection with solely stimulated HTLV-1-specific CTL is always founded when $\Re_6 > 1$, $\Re_2/\Re_1 > 1$ and $\Re_7 \leq 1$.

Theorem 7. *If* $\Re_6 > 1$, $\Re_2/\Re_1 > 1$ *and* $\Re_7 \leq 1$, *then* Ξ_6 *is G.A.S.*

Proof. Consider

$$\Lambda_6 = X_6 F\left(\frac{X}{X_6}\right) + Y_6 F\left(\frac{Y}{Y_6}\right) + \frac{\kappa}{\rho} V_6 F\left(\frac{V}{V_6}\right) + W_6 F\left(\frac{W}{W_6}\right) + \frac{\kappa\theta}{\rho\xi} Z + \frac{\delta}{\rho} U_6 F\left(\frac{U}{U_6}\right).$$

Calculating $\frac{d\Lambda_6}{dt}$ as:

$$\frac{d\Lambda_6}{dt} = \left(1-\frac{X_6}{X}\right)\dot{X} + \left(1-\frac{Y_6}{Y}\right)\dot{Y} + \frac{\kappa}{\rho}\left(1-\frac{V_6}{V}\right)\dot{V}$$

$$+\left(1-\frac{W_6}{W}\right)\dot{W} + \frac{\kappa\theta}{\rho\xi}\dot{Z} + \frac{\delta}{\rho}\left(1-\frac{U_6}{U}\right)\dot{U}$$

$$= \left(1-\frac{X_6}{X}\right)(\lambda-\gamma X-\phi_1 XV-\phi_2 XW) + \left(1-\frac{Y_6}{Y}\right)(\phi_1 XV-\kappa Y)$$

$$+\frac{\kappa}{\rho}\left(1-\frac{V_6}{V}\right)(\rho Y-\sigma V-\theta VZ) + \left(1-\frac{W_6}{W}\right)(\phi_2 XW-\alpha W-\delta UW)$$

$$+\frac{\kappa\theta}{\rho\xi}(\xi VZ-\mu Z) + \frac{\delta}{\rho}\left(1-\frac{U_6}{U}\right)(\rho UW-\varepsilon U). \tag{36}$$

Collecting terms as:

$$\frac{d\Lambda_6}{dt} = \left(1-\frac{X_6}{X}\right)(\lambda-\gamma X) + \phi_1 X_6 V + \phi_2 X_6 W - \phi_1 XV\frac{Y_6}{Y} + \kappa Y_6$$

$$-\frac{\kappa\sigma}{\rho}V - \kappa Y\frac{V_6}{V} + \frac{\kappa\sigma}{\rho}V_6 + \frac{\kappa\theta}{\rho}V_6 Z - \alpha W - \phi_2 XW_6 + \alpha W_6$$

$$+\delta UW_6 - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\delta\varepsilon}{\rho}U - \delta U_6 W + \frac{\delta\varepsilon}{\rho}U_6.$$

Using the equilibrium conditions for Ξ_6:

$$\lambda = \gamma X_6 + \phi_1 X_6 V_6 + \phi_2 X_6 W_6, \quad \phi_1 X_6 V_6 = \kappa Y_6,$$

$$\phi_2 X_6 W_6 = \alpha W_6 + \delta U_6 W_6, \quad Y_6 = \frac{\sigma}{\rho}V_6, \quad W_6 = \frac{\varepsilon}{\rho},$$

and collecting terms we obtain

$$\frac{d\Lambda_6}{dt} = \left(1-\frac{X_6}{X}\right)(\gamma X_6 - \gamma X) + 3\phi_1 X_6 V_6 + 2\phi_2 X_6 W_6 - \phi_1 X_6 V_6 \frac{X_6}{X} - \phi_2 X_6 W_6 \frac{X_6}{X}$$

$$- \phi_1 X_6 V_6 \frac{Y_6 X V}{Y X_6 V_6} - \phi_2 X_6 W_6 \frac{X}{X_6} - \phi_1 X_6 V_6 \frac{V_6 Y}{V Y_6} + \frac{\kappa\theta\mu}{\rho\xi}\left(\frac{\xi}{\mu}V_6 - 1\right) Z$$

$$= -\frac{\gamma(X-X_6)^2}{X} + \phi_1 X_6 V_6 \left(3 - \frac{X_6}{X} - \frac{Y_6 X V}{Y X_6 V_6} - \frac{V_6 Y}{V Y_6}\right)$$

$$+ \phi_2 X_6 W_6 \left(2 - \frac{X_6}{X} - \frac{X}{X_6}\right) + \frac{\kappa\theta(\phi_1\mu\rho + \phi_2\varepsilon\xi + \gamma\xi\rho)}{\xi\rho\rho\phi_1}(\Re_7 - 1) Z$$

$$= -\frac{(\gamma + \phi_2 W_6)(X-X_6)^2}{X} + \phi_1 X_6 V_6 \left(3 - \frac{X_6}{X} - \frac{Y_6 X V}{Y X_6 V_6} - \frac{V_6 Y}{V Y_6}\right)$$

$$+ \frac{\kappa\theta(\phi_1\mu\rho + \phi_2\varepsilon\xi + \gamma\xi\rho)}{\xi\rho\rho\phi_1}(\Re_7 - 1) Z.$$

Since $\Re_7 \leq 1$, then inequality (29) gives $\frac{d\Lambda_6}{dt} \leq 0$ for any $X, Y, V, Z > 0$. Moreover, $\frac{d\Lambda_6}{dt} = 0$ when $X = X_6, Y = Y_6, V = V_6$, and $Z = 0$. System's solutions attracted to $\tilde{\Omega}_6$ which has $X = X_6$. Then $\dot{X} = 0$, Eq. (16) provides

$$0 = \dot{X} = \lambda - \gamma X_6 - \phi_1 X_6 V_6 - \phi_2 X_6 W \Longrightarrow W(t) = W_6, \text{ for any } t.$$

Since $W = W_6$, then $\dot{W} = 0$, Eq. (19) implies

$$0 = \dot{W} = \phi_2 X_6 W_6 - \alpha W_6 - \delta U W_6 \Longrightarrow U(t) = U_6, \text{ for any } t.$$

Consequently, $\tilde{\Omega}_6 = \{\Xi_6\}$ by using LIP, we get Ξ_6 is G.A.S. □

The ensuing finding implies that, regardless of the starting points, the HIV-1 and HTLV-1 coinfection with stimulated both HIV-1-specific antibody and HTLV-1-specific CTL responses is always established when $\Re_7 > 1$ and $\Re_8 > 1$.

Theorem 8. *If* $\Re_7 > 1$ *and* $\Re_8 > 1$*, then* Ξ_7 *is G.A.S.*

Proof. Define

$$\Lambda_7 = X_7 F\left(\frac{X}{X_7}\right) + Y_7 F\left(\frac{Y}{Y_7}\right) + \frac{\kappa}{\rho} V_7 F\left(\frac{V}{V_7}\right) + W_7 F\left(\frac{W}{W_7}\right) + \frac{\kappa\theta}{\rho\xi} Z_7 F\left(\frac{Z}{Z_7}\right) + \frac{\delta}{\rho} U_7 F\left(\frac{U}{U_7}\right).$$

Calculating $\frac{d\Lambda_7}{dt}$ as:

$$\frac{d\Lambda_7}{dt} = \left(1-\frac{X_7}{X}\right)\dot{X} + \left(1-\frac{Y_7}{Y}\right)\dot{Y} + \frac{\kappa}{\rho}\left(1-\frac{V_7}{V}\right)\dot{Z}$$

$$+\left(1-\frac{W_7}{W}\right)\dot{W} + \frac{\kappa\theta}{\rho\xi}\left(1-\frac{Z_7}{Z}\right)\dot{Z} + \frac{\delta}{\rho}\left(1-\frac{U_7}{U}\right)\dot{U}$$

$$= \left(1-\frac{X_7}{X}\right)(\lambda-\gamma X-\phi_1 XV-\phi_2 XW) + \left(1-\frac{Y_7}{Y}\right)(\phi_1 XV-\kappa Y)$$

$$+\frac{\kappa}{\rho}\left(1-\frac{V_7}{V}\right)(\rho Y-\sigma V-\theta VZ) + \left(1-\frac{W_7}{W}\right)(\phi_2 XW-\alpha W-\delta UW)$$

$$+\frac{\kappa\theta}{\rho\xi}\left(1-\frac{Z_7}{Z}\right)(\xi VZ-\mu Z) + \frac{\delta}{\rho}\left(1-\frac{U_7}{U}\right)(\rho UW-\varepsilon U). \tag{37}$$

Collecting terms as:

$$\frac{d\Lambda_7}{dt} = \left(1-\frac{X_7}{X}\right)(\lambda-\gamma X) + \phi_1 X_7 V + \phi_2 X_7 W - \phi_1 XV\frac{Y_7}{Y} + \kappa Y_7$$

$$-\frac{\kappa\sigma}{\rho}V - \kappa Y\frac{V_7}{V} + \frac{\kappa\sigma}{\rho}V_7 + \frac{\kappa\theta}{\rho}V_7 Z - \alpha W - \phi_2 XW_7 + \alpha W_7$$

$$+\delta UW_7 - \frac{\kappa\theta\mu}{\rho\xi}Z - \frac{\kappa\theta}{\rho}Z_7 V + \frac{\kappa\theta\mu}{\rho\xi}Z_7 - \frac{\delta\varepsilon}{\rho}U - \delta U_7 W + \frac{\delta\varepsilon}{\rho}U_7.$$

Using the equilibrium conditions for Ξ_7:

$$\lambda = \gamma X_7 + \phi_1 X_7 V_7 + \phi_2 X_7 W_7, \quad \phi_1 X_7 V_7 = \kappa Y_7,$$

$$\phi_2 X_7 W_7 = \alpha W_7 + \delta U_7 W_7, \quad Y_7 = \frac{\sigma}{\rho}V_7 + \frac{\theta}{\rho}V_7 Z_7, \; V_7 = \frac{\mu}{\xi}, \quad W_7 = \frac{\varepsilon}{\rho},$$

and collecting terms we obtain

$$\frac{d\Lambda_7}{dt} = \left(1-\frac{X_7}{X}\right)(\gamma X_7 - \gamma X) + 3\phi_1 X_7 V_7 + 2\phi_2 X_7 W_7 - \phi_1 X_7 V_7 \frac{X_7}{X} - \phi_2 X_7 W_7 \frac{X_7}{X}$$

$$- \phi_1 X_7 V_7 \frac{Y_7 X V}{Y X_7 V_7} - \phi_1 X_7 V_7 \frac{V_7 Y}{V Y_7} - \phi_2 X_7 W_7 \frac{X}{X_7}$$

$$= -\frac{\gamma(X-X_7)^2}{X} + \phi_1 X_7 V_7 \left(3 - \frac{X_7}{X} - \frac{Y_7 X V}{Y X_7 V_7} - \frac{V_7 Y}{V Y_7}\right) + \phi_2 X_7 W_7 \left(2 - \frac{X_7}{X} - \frac{X}{X_7}\right)$$

$$= -\frac{(\gamma+\phi_2 W_7)(X-X_7)^2}{X} + \phi_1 X_7 V_7 \left(3 - \frac{X_7}{X} - \frac{Y_7 X V}{Y X_7 V_7} - \frac{V_7 Y}{V Y_7}\right).$$

Using inequality (29), we obtain $\frac{d\Lambda_7}{dt} \leq 0$ for all $X, Y, V > 0$. Moreover $\frac{d\Lambda_7}{dt} = 0$ when $X = X_7$, $Y = Y_7$ and $V = V_7$. System' solutions attacked to $\tilde{\Omega}_7$ which has $X = X_7, Y = Y_7$ and $V = V_7$. Then $\dot{X} = 0$, $\dot{V} = 0$ and Eqs. (16), (18) imply

$$0 = \dot{X} = \lambda - \gamma X_7 - \phi_1 X_7 V_7 - \phi_2 X_7 W \Longrightarrow W(t) = W_7, \text{ for any } t,$$

$$0 = \dot{V} = \rho Y_7 - \sigma V_7 - \theta V_7 Z \Longrightarrow Z(t) = Z_7, \text{ for any } t.$$

Since $W = W_7$, then $\dot{W} = 0$ and Eq. (19) implies

$$0 = \dot{W} = \phi_2 X_7 W_7 - \alpha W_7 - \delta U W_7 \Longrightarrow U(t) = U_6, \text{ for any } t.$$

Hence, $\tilde{\Omega}_7 = \{\Xi_7\}$ and LIP yields that Ξ_7 is G.A.S. □

Now we able to summarize the existence and stability conditions of the model's equilibria (see Table 1):

Table 1. Existence and stability conditions .

Equilibrium	Existence conditions	Stability conditions
$\Xi_0 = (X_0, 0, 0, 0, 0, 0)$	None	$\Re_1 \leq 1$ and $\Re_2 \leq 1$
$\Xi_1 = (X_1, Y_1, V_1, 0, 0, 0)$	$\Re_1 > 1$	$\Re_1 > 1$, $\frac{\Re_2}{\Re_1} \leq 1$ and $\Re_3 \leq 1$
$\Xi_2 = (X_2, 0, 0, W_2, 0, 0)$	$\Re_2 > 1$	$\Re_2 > 1$, $\frac{\Re_1}{\Re_2} \leq 1$ and $\Re_4 \leq 1$
$\Xi_3 = (X_3, Y_3, V_3, 0, Z_3, 0)$	$\Re_3 > 1$	$\Re_3 > 1$ and $\Re_5 \leq 1$
$\Xi_4 = (X_4, 0, 0, W_4, 0, U_4)$	$\Re_4 > 1$	$\Re_4 > 1$ and $\Re_6 \leq 1$
$\Xi_5 = (X_5, Y_5, V_5, W_5, Z_5, 0)$	$\Re_5 > 1$ and $\frac{\Re_1}{\Re_2} > 1$	$\Re_5 > 1$, $\frac{\Re_1}{\Re_2} > 1$ and $\Re_8 \leq 1$
$\Xi_6 = (X_6, Y_6, V_6, W_6, 0, U_6)$	$\Re_6 > 1$ and $\frac{\Re_2}{\Re_1} > 1$	$\Re_6 > 1$, $\frac{\Re_2}{\Re_1} > 1$ and $\Re_7 \leq 1$
$\Xi_7 = (X_7, Y_7, V_7, W_7, Z_7, U_7)$	$\Re_7 > 1$ and $\Re_8 > 1$	$\Re_7 > 1$ and $\Re_8 > 1$

5. Comparison study

In this section we show the significance of incorporating HIV-1-specific antibody and HTLV-1-specific CTL responses in our proposed model. Model (7)–(8), which was investigated in [23], is the result of our model when the CTL immune response is disregarded. Here, the model includes just five equilibria (for further information, see [23]).

System (16)–(21) without CTL and antibody immune responses becomes:

$$\begin{cases} \dot{X} = \lambda - \gamma X - \phi_1 XV - \phi_2 XW, \\ \dot{Y} = \phi_1 XV - \kappa Y, \\ \dot{V} = \rho Y - \sigma V, \\ \dot{W} = \phi_2 XW - \alpha W. \end{cases} \tag{38}$$

This model has only three equilibria:

- Uninfected equilibrium, $\breve{\Xi}_0 = (\breve{X}_0, 0, 0, 0)$, where HIV-1 and HTLV-1 are cleared,
- HIV-1 single-infection equilibrium, $\breve{\Xi}_1 = (\breve{X}_1, \breve{Y}_1, \breve{V}_1, 0)$, where the HTLV-1 is prevented,
- HTLV-1 single-infection equilibrium, $\breve{\Xi}_2 = (\breve{X}_2, 0, 0, \breve{W}_2)$, where the HIV-1 is prevented, where $\breve{X}_i = X_i$, $i = 0, 1, 2$, $\breve{Y}_1 = Y_1$, $\breve{V}_1 = V_1$ and $\breve{W}_2 = W_2$. Section 3 defines two thresholds, $\Re_1$ and $\Re_2$, which determine the existence of these three equilibria.

As a result, in the absence of antibody and CTL responses, there will be a competition between HTLV-1 and HIV-1 on consuming resources of target cells, $CD4^+T$ cells. There is only one kind of virus that can survive with the highest basic reproduction ratio. The scenario of HTLV-1 and HIV-1 cohabitation appears in our suggested model. This scenario is more plausible since HTLV-1 and HIV-1 are often present and cannot currently be eliminated from the body by medication. This scenario might be interpreted as follows: cohabitation of HTLV-1 and HIV-1 is possible because antibody and CTL immune responses inhibit viral development, which also suppresses competition between HTLV-1 and HIV-1 [30].

Lastly, our model leads to the HIV-1 single-infection scenario given in [4] in the absence of HTLV-1 infection and immune response as follows:

$$\begin{cases} \dot{X} = \lambda - \gamma X - \phi_1 XV, \\ \dot{Y} = \phi_1 XV - \kappa Y, \\ \dot{V} = \rho Y - \sigma V. \end{cases} \tag{39}$$

System (39) has only two equilibria:

(I) Uninfected equilibrium, $\tilde{\Xi}_0 = (\frac{\lambda}{\gamma}, 0, 0)$, where the HIV-1 particles are cleared,

(II) HIV-1 single-infection equilibrium, $\tilde{\Xi}_1 = (\frac{\lambda}{\gamma\Re_1}, \frac{\gamma\sigma}{\rho\phi_1}(\Re_1 - 1), \frac{\gamma}{\phi_1}(\Re_1 - 1))$, where the HIV-1 is chronic.

6. Numerical simulations

In this section, we conduct numerical simulation for model (16)–(21) to illustrate the theoretical findings. We demonstrate the effect of HIV-1-specific antibody and HTLV-1-specific CTL responses on the HIV-1/HTLV-1 coinfection dynamics. MATLAB's ode45 solver will be used to numerically solve the ODEs system (16)–(21). The values of model's parameters are listed in Table 2.

Table 2. Model parameters.

Parameter	Value	Source	Parameter	Value	Source
λ	10	[31, 32]	θ	0.8	[33, 34]
γ	0.01	[35, 36]	α	0.2	[19, 17, 18]
ϕ_1	Varied	Assumed	δ	0.2	[33]
ϕ_2	Varied	Assumed	ξ	Varied	Assumed
κ	0.5	[37, 9], [10]	μ	0.1	[33]
ρ	5	[33, 21]	ρ	Varied	Assumed
σ	2	[33, 21]	ε	0.1	[15, 38]

6.1 *Stability of the equilibria*

In order to demonstrate the global stability of the system's equilibria, we demonstrate that the system's solutions attracted to one of its equilibria regardless of the initial conditions. So, we use three distinct initials as follows:

$$\text{C1} : (X(0),Y(0),V(0),W(0),Z(0),U(0)) = (500,1.5,5,30,3,2),$$

$$\text{C2} : (X(0),Y(0),V(0),W(0),Z(0),U(0)) = (400,1,2,20,2,1.5),$$

$$\text{C3} : (X(0),Y(0),V(0),W(0),Z(0),U(0)) = (300,0.5,1.5,10,1,0.5).$$

Here, we select the values of ϕ_1, ϕ_2, ξ and ρ as:

State 1 (Stability of Ξ_0): $\phi_1 = 0.0001$, $\phi_2 = 0.0001$, $\xi = 0.3$ and $\rho = 0.2$. These values give $\Re_1 = 0.5 < 1$ and $\Re_2 = 0.5 < 1$. Figure 2 demonstrates that for all starting values, the trajectories lead to the equilibrium $\Xi_0 = (1000,0,0,0,0,0)$. This demonstrates that based on Theorem 1, Ξ_0 is G.A.S. In this state, both HIV-1 and HTLV-1 will be eventually cleared.

State 2 (Stability of Ξ_1): $\phi_1 = 0.0005$, $\phi_2 = 0.0001$, $\xi = 0.001$ and $\rho = 0.02$. This gives $\Re_2 = 0.5 < 1 < 2.5 = \Re_1$, $\Re_3 = 0.4167 < 1$ and hence $\Re_2/\Re_1 = 0.2 < 1$. The numerical results show that, $\Xi_1 = (400,12,30,0,0,0)$ exists. Figure 3 clearly demonstrates that the trajectories eventually trend to Ξ_1 for all initials, which is consistent with Theorem 2. This case simulates an HIV-1 single-infection but without antibody response.

State 3 (Stability of Ξ_2): $\phi_1 = 0.0001$, $\phi_2 = 0.0007$, $\xi = 0.001$ and $\rho = 0.002$. These values give $\Re_1 = 0.5 < 1 < 3.5 = \Re_2$, $\Re_4 = 0.7778 < 1$ and then $\Re_1/\Re_2 = 0.1429 < 1$. The equilibrium point Ξ_2 exists with $\Xi_2 = (285.71,0,0,35.71,0,0)$. Figure 4 shows that, for all initials, the trajectories tend to Ξ_2, which is consistent with Theorem 3. This state represents an HTLV-1 single-infection but without CTL response.

State 4 (Stability of Ξ_3): $\phi_1 = 0.001$, $\phi_2 = 0.00003$, $\xi = 0.01$ and $\rho = 0.002$. With such selection we obtain $\Re_3 = 2.5 > 1$ and $\Re_5 = 0.075 < 1$. The equilibrium point Ξ_3 exists with $\Xi_3 = (500,10,10,0,3.75,0)$. Figure 5 clearly demonstrates that the trajectories eventually trend to Ξ_3 for all initials, which is consistent with Theorem 4. Once an HIV-1 single infection has been achieved, an HIV-1-specific antibody has been induced.

State 5 (Stability of Ξ_4): $\phi_1 = 0.0003$, $\phi_2 = 0.0009$, $\xi = 0.05$ and $\rho = 0.003$. With such selection we obtain $\Re_4 = 1.125 > 1$ and $\Re_6 = 0.375 < 1$. Thus, Ξ_4 exists with $\Xi_4 = (250,0,0,33.33,0,0.13)$. Figure 6 shows that, for all initials, the trajectories tend to Ξ_4 which is consistent with Theorem 5. It is possible to achieve the case of HTLV-1 single-infection with activated HTLV-1-specific CTL.

State 6 (Stability of Ξ_5): $\phi_1 = 0.0006$, $\phi_2 = 0.0005$, $\xi = 0.1$ and $\rho = 0.0005$. This gives $\Re_5 = 2.3585 > 1$, $\Re_8 = 0.226 < 1$ and $\Re_1/\Re_2 = 1.2 > 1$. The numerical results show that, $\Xi_5 = (400, 0.48, 1, 28.8, 0.5, 0)$ exists. Figure 7 clearly demonstrates that the trajectories eventually trend to Ξ_5 for all initials, which agrees with Theorem 6. Hence, a coinfection with HIV-1 and HTLV-1 is attained where only HIV-1-specific antibody is stimulated.

State 7 (Stability of Ξ_6): $\phi_1 = 0.0005$, $\phi_2 = 0.0007$, $\xi = 0.001$ and $\rho = 0.08$. With such selection we have $\Re_6 = 2.2989 > 1, \Re_7 = 0.4107 < 1$ and $\Re_2/\Re_1 = 1.4 > 1$. We find that, the equilibrium $\Xi_6 = (400, 11.3, 28.25, 1.25, 0, 0.4)$ exists. Figure 8 shows that, for all initials, the trajectories tend to Ξ_6 which is consistent with Theorem 7. The patient will only have active HTLV-1-specific CTL when they reach the case of coinfection with HIV-1 and HTLV-1.

State 8 (Stability of Ξ_7): $\phi_1 = 0.006$, $\phi_2 = 0.007$, $\xi = 0.01$ and $\rho = 0.05$. This selection gives $\Re_7 = 3.5714 > 1$ and $\Re_8 = 4.1667 > 1$. Figure 9 shows that $\Xi_7 = (119.05, 14.29, 10, 2, 6.43, 3.17)$ exists and for all initials. The trajectories of the system attacked to Ξ_7 and this is in line with Theorem 8. In this stage of the coinfection, both HTLV-1-specific CTL and HIV-1-specific antibodies are at work.

To obtain further verification, we conduct an investigation the local stability of the system's equilibria. We calculate the Jacobian matrix $J = J(X, Y, V, W, Z, U)$ of system (16)–(21) as:

$$J = \begin{pmatrix} -(\gamma + \phi_1 V + \phi_2 W) & 0 & -\phi_1 X & -\phi_2 X & 0 & 0 \\ \phi_1 V & -\kappa & \phi_1 X & 0 & 0 & 0 \\ 0 & \rho & -\theta Z - \sigma & 0 & -\theta V & 0 \\ \phi_2 W & 0 & 0 & -(\alpha + \delta U) + \phi_2 X & 0 & -\delta W \\ 0 & 0 & \xi Z & 0 & -\mu + \xi V & 0 \\ 0 & 0 & 0 & \rho U & 0 & -\varepsilon + \rho W \end{pmatrix}. \tag{40}$$

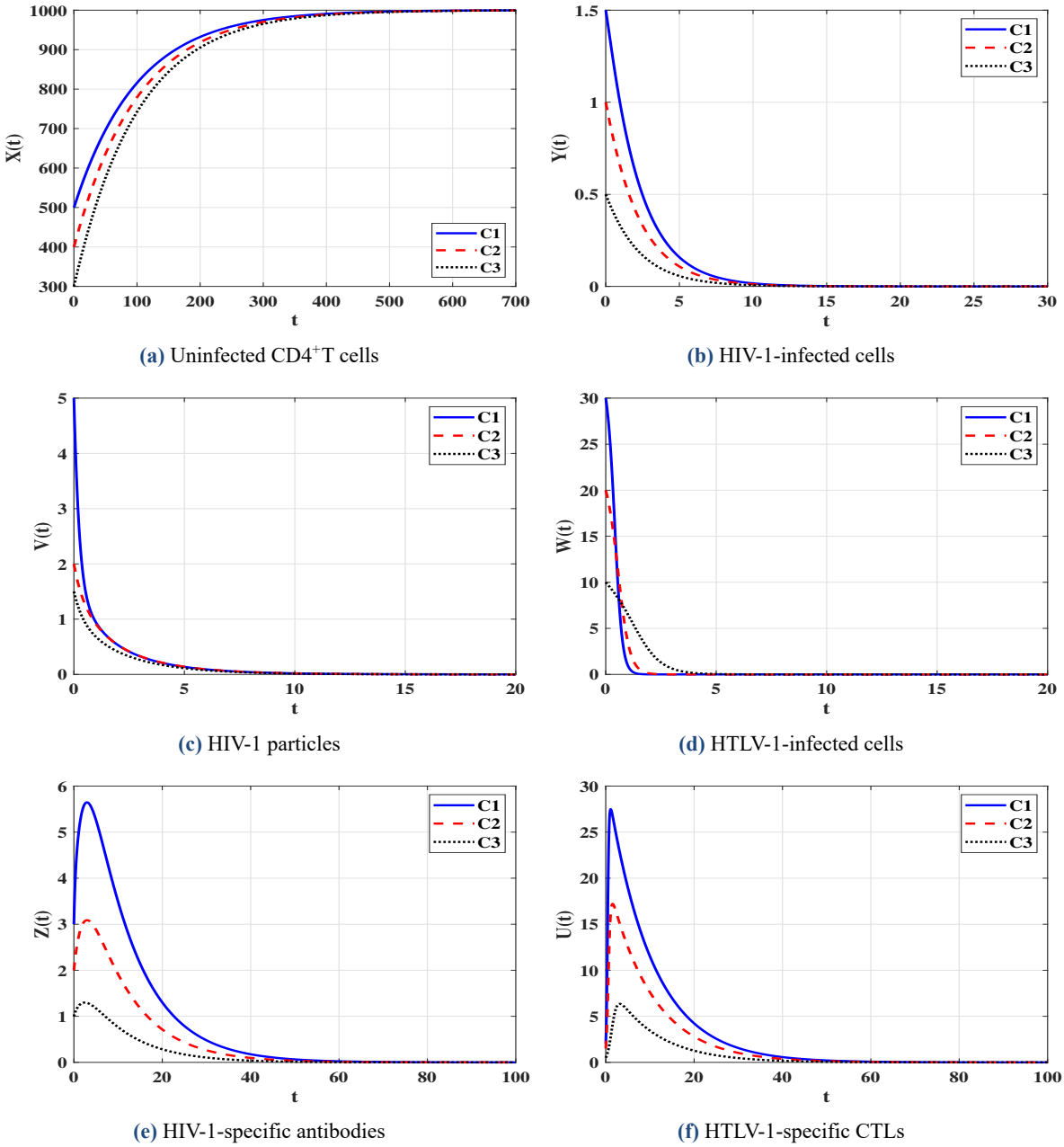
At each equilibrium we find the eigenvalues λ_j, $j = 1, 2, ..., 7$ of J. An equilibrium is locally stable if $\mathrm{Re}(\lambda_j) < 0$. Choosing the parameters ϕ_1, ϕ_2, ξ and ρ from states 1–8, we proceed to compute all equilibria that are nonnegative along with their corresponding eigenvalues. The real components of the eigenvalues, the nonnegative equilibria, and the stability of the equilibrium point were all shown in Table 3. It was observed that the local stability characteristics were in accordance with the global stability analysis.

Table 3. Local stability of nonnegative equilibria Ξ_j, $j = 0, 1, ..., 7$.

State	The equilibria	$\mathrm{Re}(\lambda_j)$ $j = 0, 1, 2, ..., 7$	Stability
1	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(-2.28, -0.22, -0.1, -0.1, -0.1, -0.01)$	stable
2	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(-3, 0.5, -0.1, -0.1, -0.1, -0.01)$	unstable
	$\Xi_1 = (400, 12, 30, 0, 0, 0)$	$(-2.5, -0.16, -0.1, -0.01, -0.01, -0.07)$	stable
3	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(-2.28, 0.5, -0.22, -0.1, -0.1, -0.01)$	unstable
	$\Xi_2 = (285.71, 0, 0, 35.71, 0, 0)$	$(-2.09, -0.41, -0.1, -0.02, -0.02, -0.03)$	unstable
			stable
4	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(-3.61, 1.11, -0.17, -0.1, -0.1, -0.01)$	unstable
	$\Xi_1 = (200, 16, 40, 0, 0, 0)$	$(-2.51, 0.3, -0.19, -0.02, -0.02, -0.1)$	unstable
	$\Xi_3 = (500, 10, 10, 0, 3.75, 0)$	$(-5.45, -0.19, -0.03, -0.03, -0.1, -0.02)$	stable
5	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(-2.69, 0.7, 0.19, -0.1, -0.1, -0.01)$	unstable
	$\Xi_1 = (666.67, 6.67, 16.67, 0, 0, 0)$	$(-2.5, 0.73, 0.4, -0.1, -0.007, -0.007)$	unstable
	$\Xi_2 = (222.22, 0, 0, 38.89, 0, 0)$	$(-2.2, -0.3, -0.1, -0.02, -0.02, 0.02)$	unstable
	$\Xi_3 = (943.4, 1.13, 2, 0, 1.04, 0)$	$(-3.31, 0.65, -0.01, -0.01, -0.1, -0.01)$	unstable
	$\Xi_4 = (250, 0, 0, 33.33, 0, 0.13)$	$(-2.22, -0.28, -0.1, -0.01, -0.01, -0.01)$	stable
6	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(-3.14, 0.64, 0.3, -0.1, -0.1, -0.01)$	unstable
	$\Xi_1 = (333.33, 13.33, 33.33, 0, 0, 0)$	$(3.23, -2.5, -0.1, -0.01, -0.01, -0.03)$	unstable
	$\Xi_2 = (400, 0, 0, 30, 0, 0)$	$(-2.58, -0.1, -0.09, 0.08, -0.01, -0.01)$	unstable
	$\Xi_3 = (943.4, 1.13, 1, 0, 4.58, 0)$	$(-6.11, 0.27, -0.03, -0.03, -0.1, -0.01)$	unstable
	$\Xi_5 = (400, 0.48, 1, 28.8, 0.5, 0)$	$(-2.89, -0.09, -0.007, -0.007, -0.01, -0.01)$	stable
7	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(-3, 0.5, 0.5, -0.1, -0.1, -0.01)$	unstable
	$\Xi_1 = (400, 12, 30, 0, 0, 0)$	$(-2.5, -0.1, 0.08, -0.01, -0.01, -0.07)$	unstable
	$\Xi_2 = (285.71, 0, 0, 35.71, 0, 0)$	$(2.76, -2.38, -0.12, -0.1, -0.02, -0.02)$	unstable
	$\Xi_4 = (919.54, 0, 0, 1.25, 0, 2.22)$	$(-2.94, 0.44, -0.0002, -0.0002, -0.1, -0.01)$	unstable
	$\Xi_6 = (400, 11.3, 28.25, 1.25, 0, 0.4)$	$(-2.5, -0.002, -0.002, -0.01, -0.01, -0.07)$	stable

Table 3. *Cont.*

8	$\Xi_0 = (1000, 0, 0, 0, 0, 0)$	$(6.8, -6.78, 4.28, -0.1, -0.1, -0.01)$	unstable
	$\Xi_1 = (33.33, 19.33, 48.33, 0, 0, 0)$	$(-2.55, 0.38, -0.12, -0.12, -0.1, 0.03)$	unstable
	$\Xi_2 = (28.57, 0, 0, 48.57, 0, 0)$	$(-2.44, 2.33, -0.18, -0.18, -0.1, -0.06)$	unstable
	$\Xi_3 = (142.86, 17.14, 10, 0, 8.21, 0)$	$(-9.002, 0.8, -0.05, -0.05, -0.1, -0.04)$	unstable
	$\Xi_4 = (416.67, 0, 0, 2, 0, 13.58)$	$(-4.86, 2.36, -0.001, -0.001, -0.1, -0.02)$	unstable
	$\Xi_6 = (33.33, 18.4, 46, 2, 0, 0.17)$	$(-2.55, 0.36, -0.13, -0.13, -0.0005, -0.0005)$	unstable
	$\Xi_7 = (119.05, 14.29, 10, 2, 6.43, 3.17)$	$(-7.58, -0.03, -0.03, -0.03, -0.03, -0.04)$	stable



(a) Uninfected CD4$^+$T cells

(b) HIV-1-infected cells

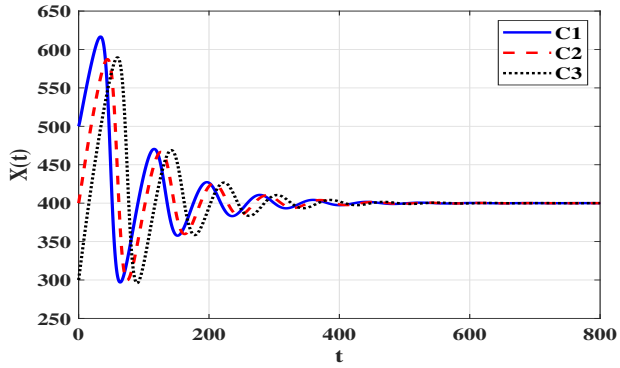
(c) HIV-1 particles

(d) HTLV-1-infected cells

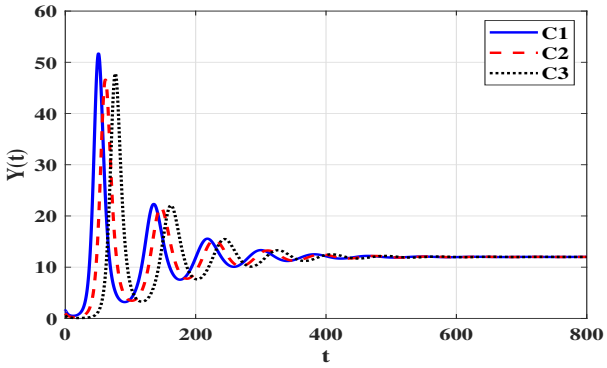
(e) HIV-1-specific antibodies

(f) HTLV-1-specific CTLs

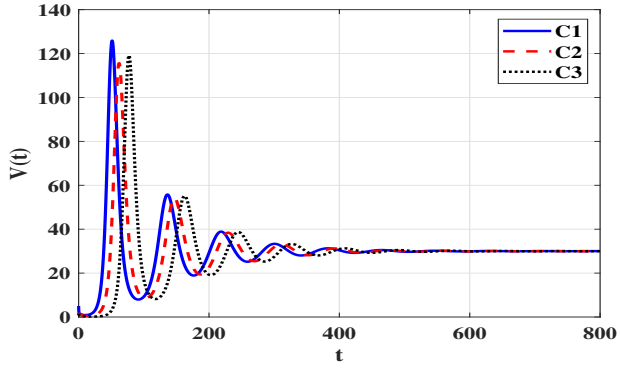
Figure 2. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_0 = (1000, 0, 0, 0, 0, 0)$ when $\Re_1 \leq 1$ and $\Re_2 \leq 1$ (State 1).



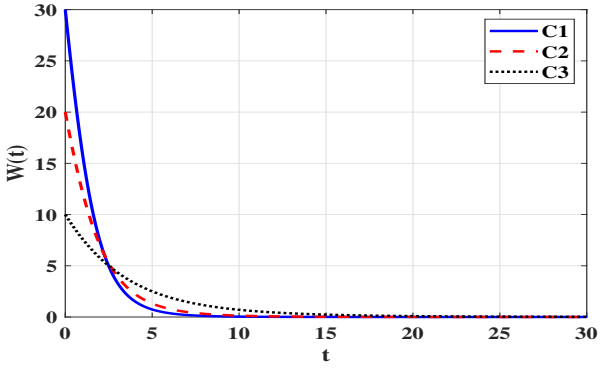
(a) Uninfected CD4$^+$T cells



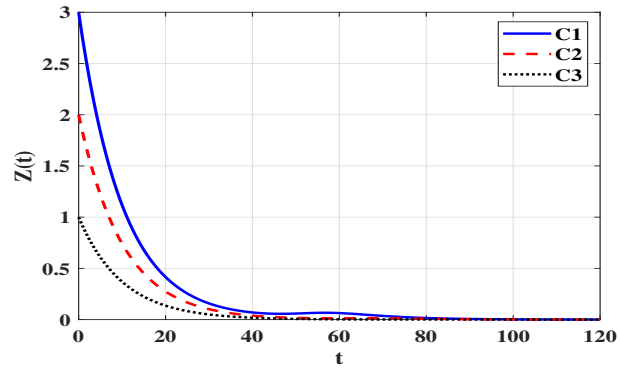
(b) HIV-1-infected cells



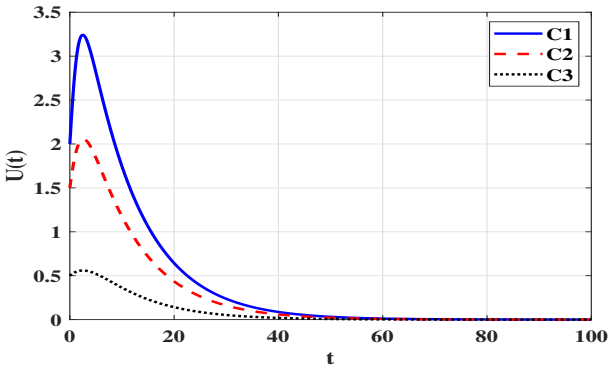
(c) HIV-1 particles



(d) HTLV-1-infected cells

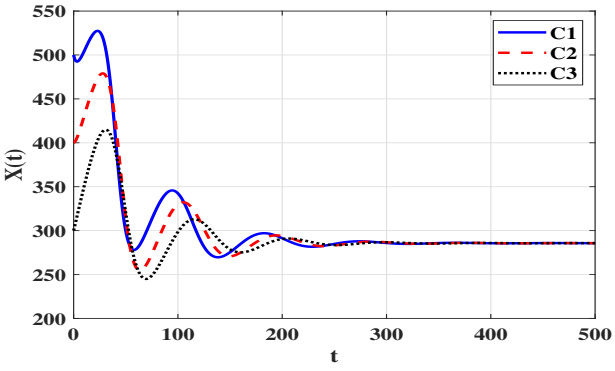


(e) HIV-1-specific antibodies

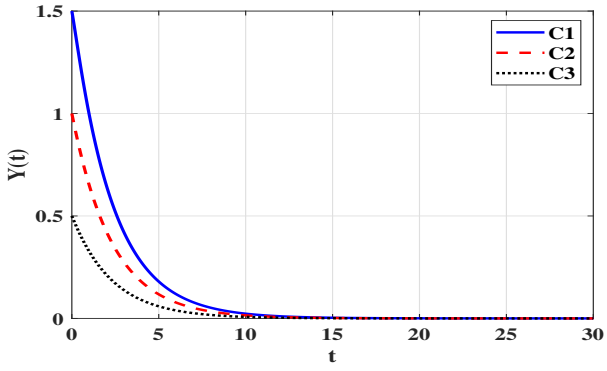


(f) HTLV-1-specific CTLs

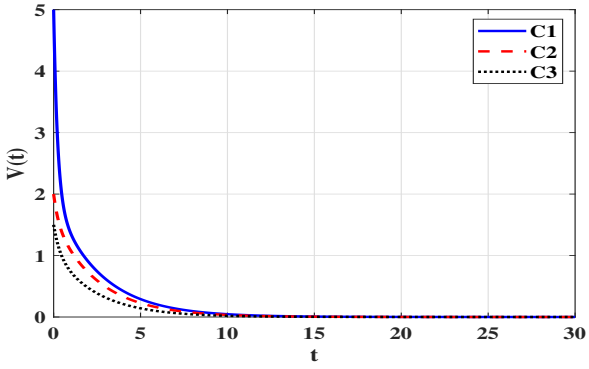
Figure 3. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_1 = (400, 12, 30, 0, 0, 0)$ when $\Re_1 > 1$, $\Re_2/\Re_1 \leq 1$ and $\Re_3 \leq 1$ (State 2).



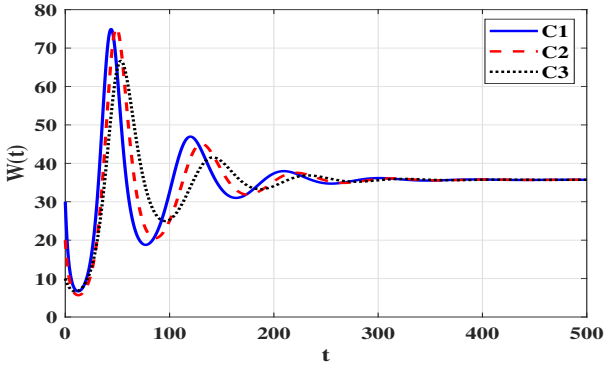
(a) Uninfected CD4$^+$T cells



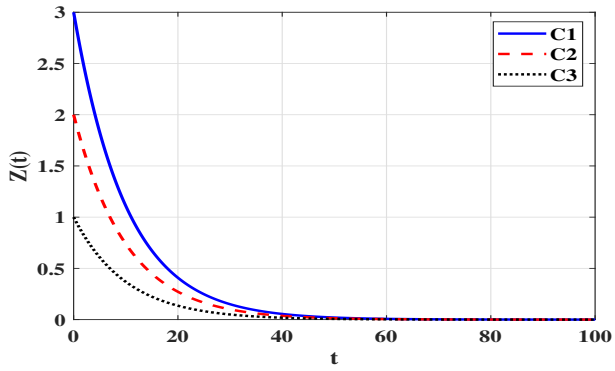
(b) HIV-1-infected cells



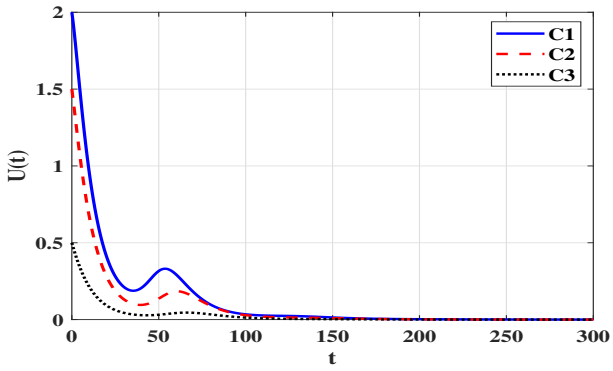
(c) HIV-1 particles



(d) HTLV-1-infected cells

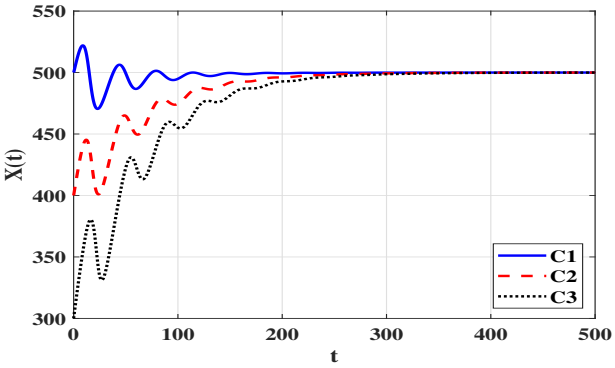


(e) HIV-1-specific antibodies

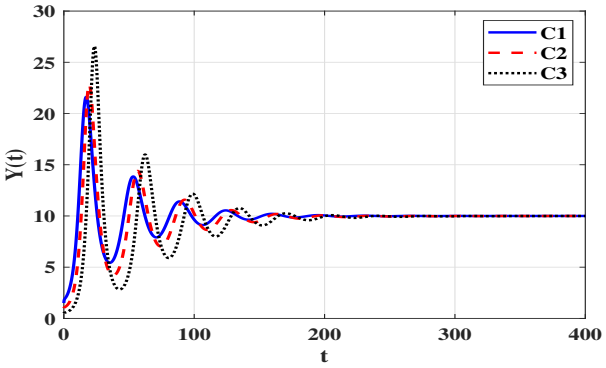


(f) HTLV-1-specific CTLs

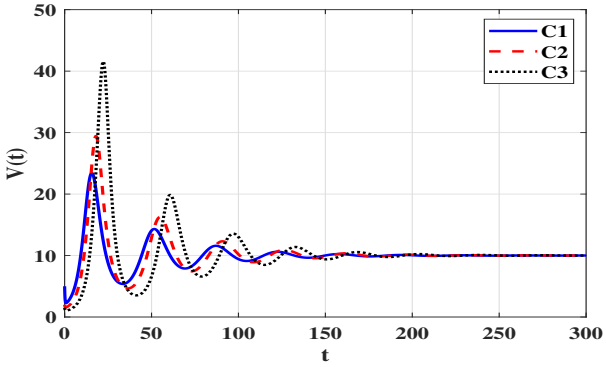
Figure 4. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_2 = (285.71, 0, 0, 35.71, 0, 0)$ when $\Re_2 > 1$, $\Re_1/\Re_2 \leq 1$ and $\Re_4 \leq 1$ (State 3).



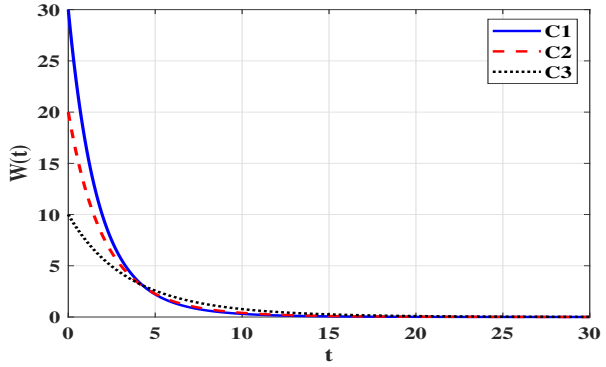
(a) Uninfected CD4$^+$T cells



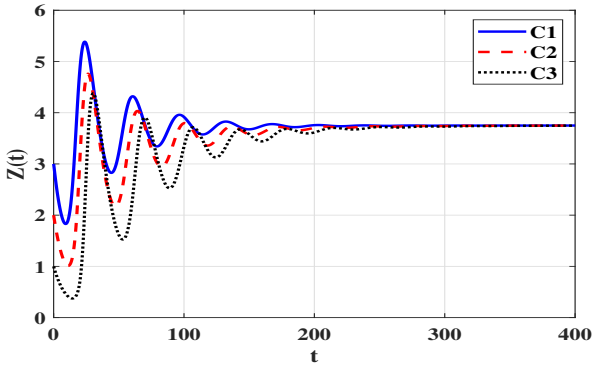
(b) HIV-1-infected cells



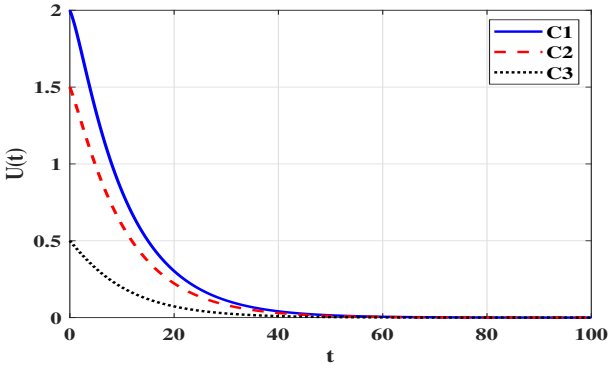
(c) HIV-1 particles



(d) HTLV-1-infected cells

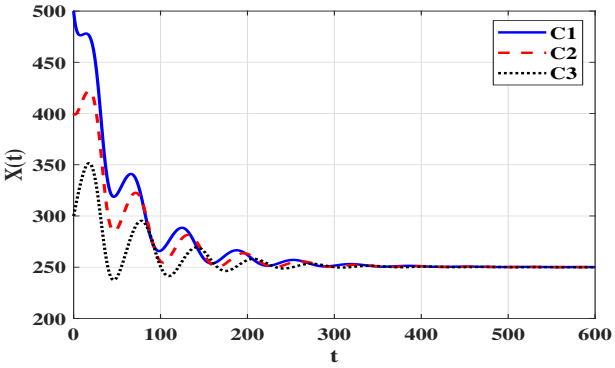


(e) HIV-1-specific antibodies

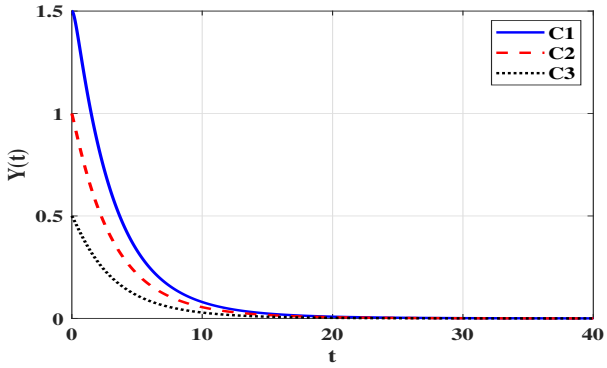


(f) HTLV-1-specific CTLs

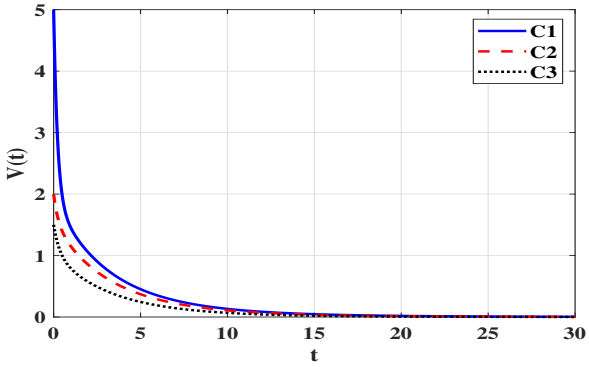
Figure 5. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_3 = (500, 10, 10, 0, 3.75, 0)$ when $\Re_3 > 1$ and $\Re_5 \leq 1$ (State 4).



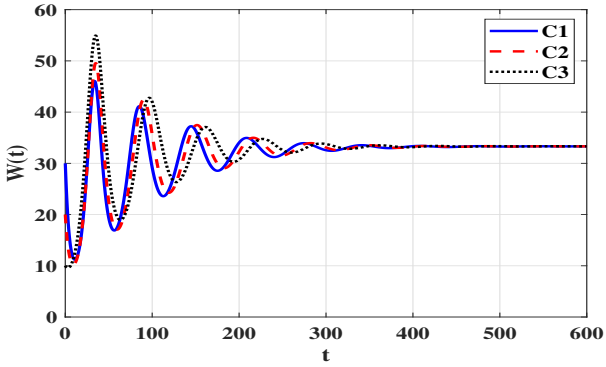
(a) Uninfected CD4$^+$T cells



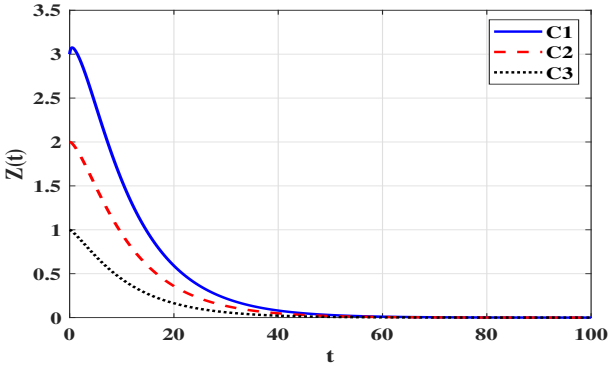
(b) HIV-1-infected cells



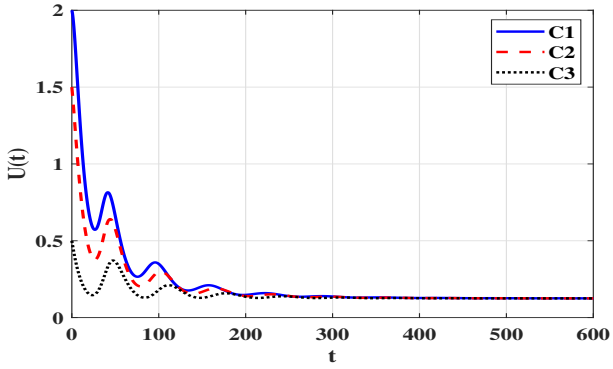
(c) HIV-1 particles



(d) HTLV-1-infected cells

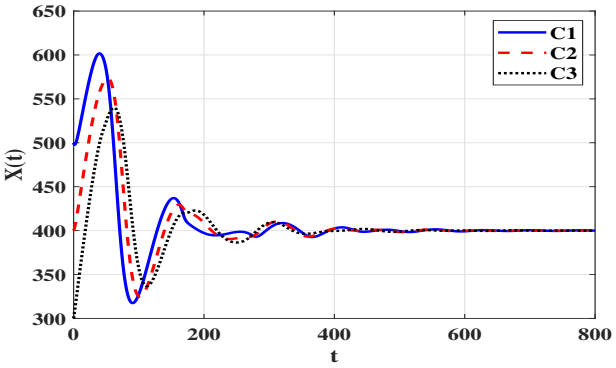


(e) HIV-1-specific antibodies

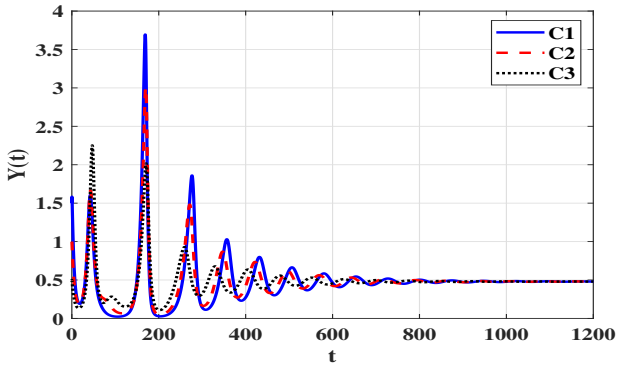


(f) HTLV-1-specific CTLs

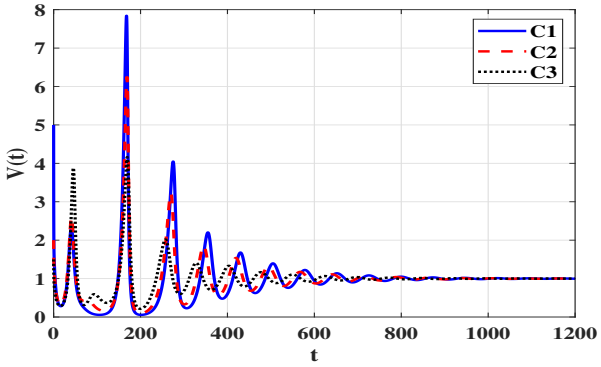
Figure 6. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_4 = (250, 0, 0, 33.33, 0, 0.13)$ when $\Re_4 > 1$ and $\Re_6 \leq 1$ (State 5).



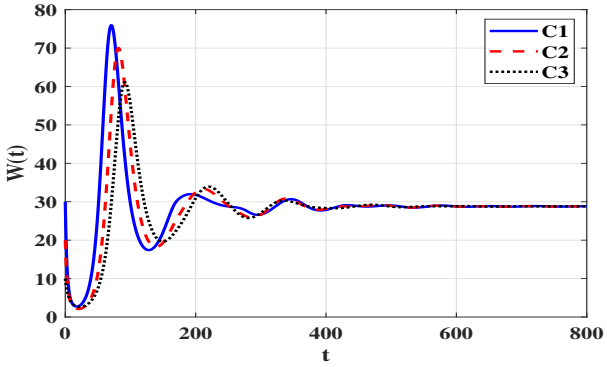
(a) Uninfected CD4$^+$T cells



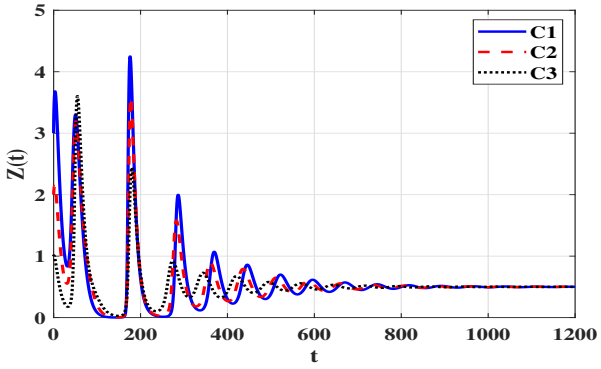
(b) HIV-1-infected cells



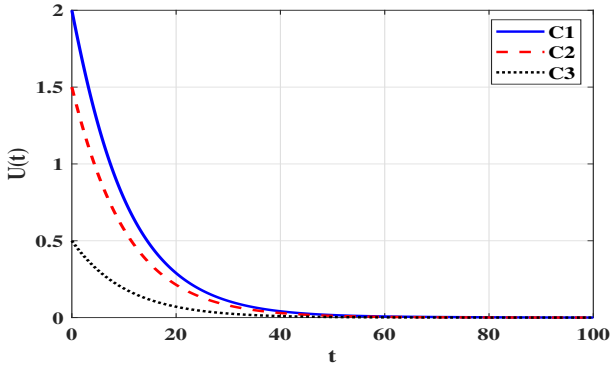
(c) HIV-1 particles



(d) HTLV-1-infected cells

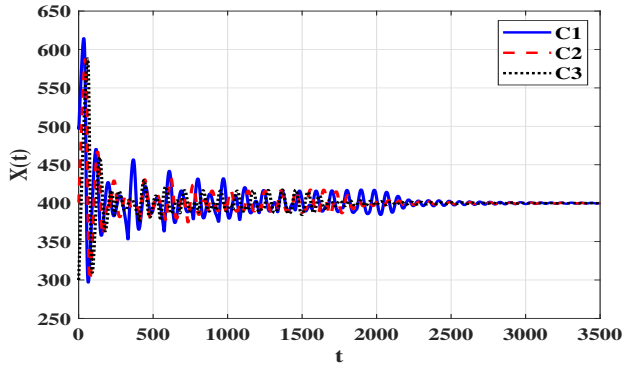


(e) HIV-1-specific antibodies

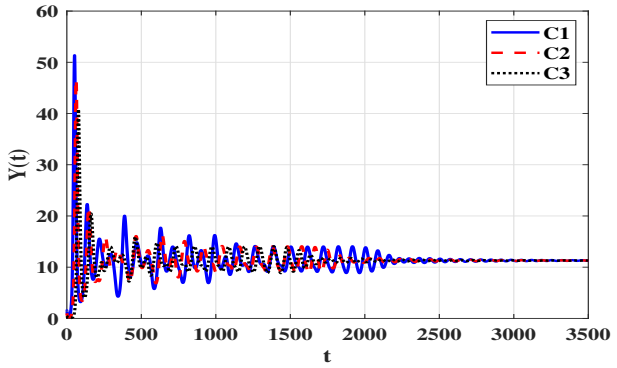


(f) HTLV-1-specific CTLs

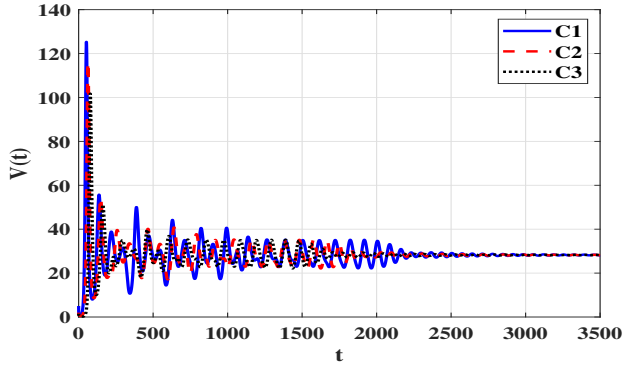
Figure 7. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_5 = (400, 0.48, 1, 28.8, 0.5, 0)$ when $\Re_5 > 1, \Re_1/\Re_2 > 1$ and $\Re_8 \leq 1$ (State 6).



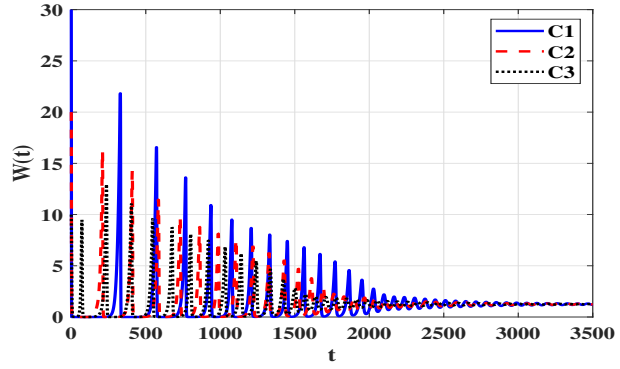
(a) Uninfected CD4$^+$T cells



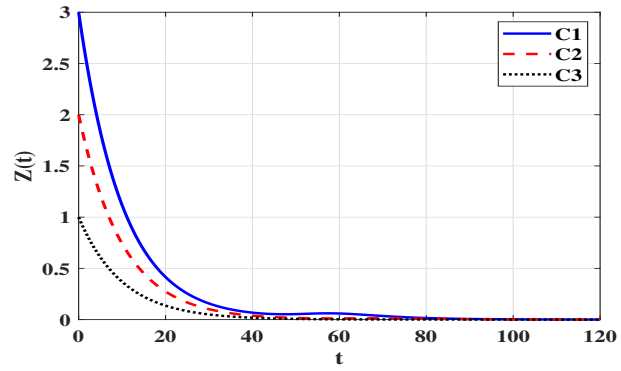
(b) HIV-1-infected cells



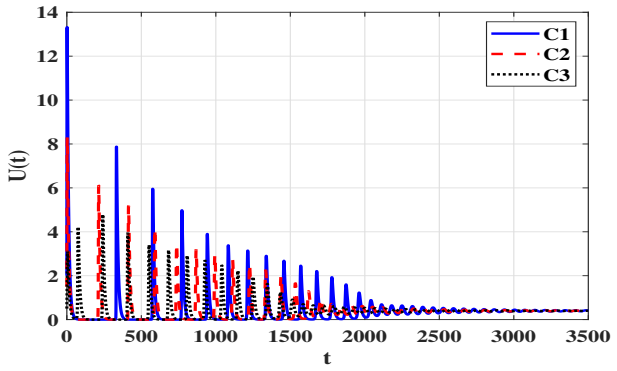
(c) HIV-1 particles



(d) HTLV-1-infected cells

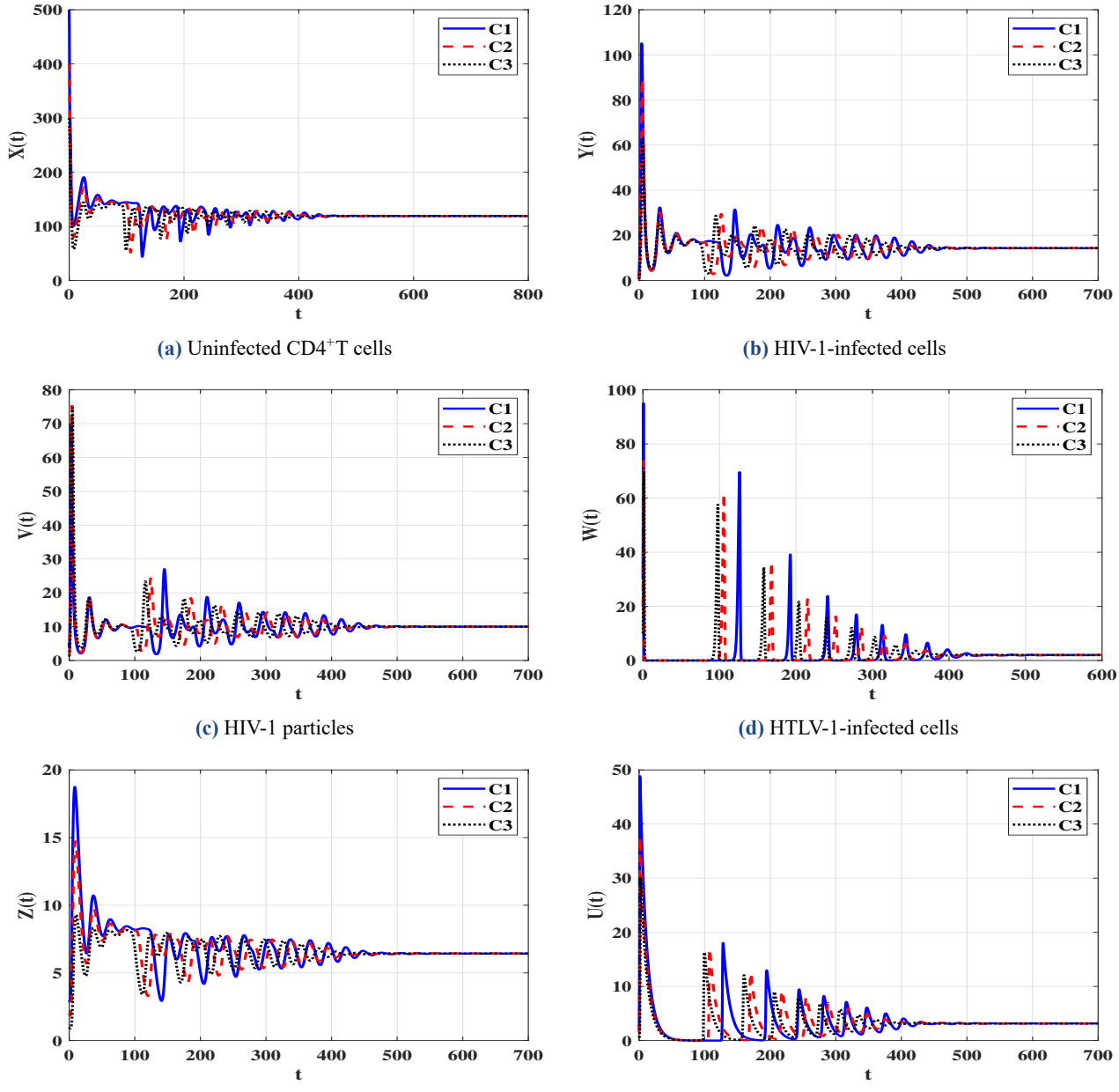


(e) HIV-1-specific antibodies



(f) HTLV-1-specific CTLs

Figure 8. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_6 = (400, 11.3, 28.25, 1.25, 0, 0.4)$ when $\Re_6 > 1, \Re_2/\Re_1 > 1$ and $\Re_7 \leq 1$ (State 7).



(a) Uninfected CD4$^+$T cells

(b) HIV-1-infected cells

(c) HIV-1 particles

(d) HTLV-1-infected cells

(e) HIV-1-specific antibodies

(f) HTLV-1-specific CTLs

Figure 9. Solutions of model (16)–(21) with C1–C3 converge to $\Xi_7 = (119.05, 14.29, 10, 2, 6.43, 3.17)$ when $\Re_7 > 1$ and $\Re_8 > 1$ (State 8).

6.2 *Impact of CTL and antibody responses on the system's behavior*

This subsection addresses the effect of stimulated rate constants of HIV-1-specific antibody and HTLV-1-specific CTL responses, (ρ and ξ) on the dynamics of system (16)-(21). We fix the parameters $\phi_1 = 0.01$ and $\phi_2 = 0.03$ and vary the parameter ρ and ξ as:

B1: $\rho = 0.01,\ \ \xi = 0.05,$

B2: $\rho = 0.04,\ \ \xi = 0.07,$

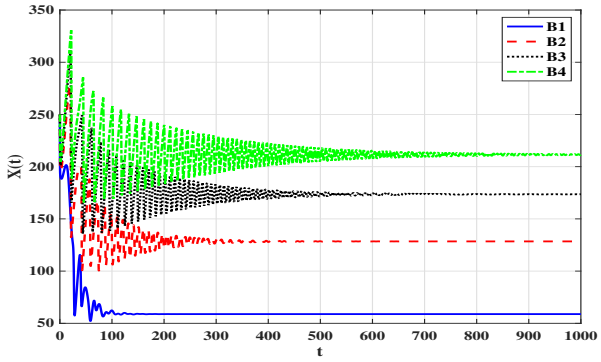
B3: $\rho = 0.07,\ \ \xi = 0.09,$

B4: $\rho = 0.1,\ \ \xi = 0.11.$

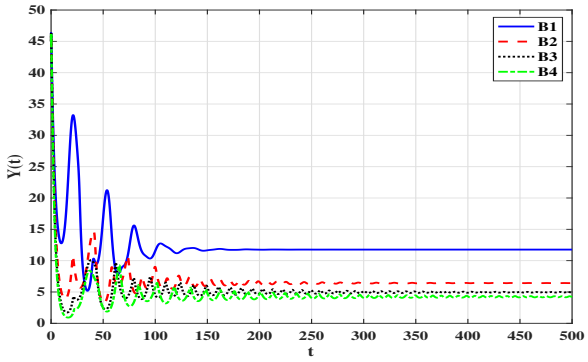
Additionally, we take into account the initial condition:

$$\text{C4}: (X(0), Y(0), V(0), W(0), Z(0), U(0)) = (250, 45, 35, 45, 40, 50).$$

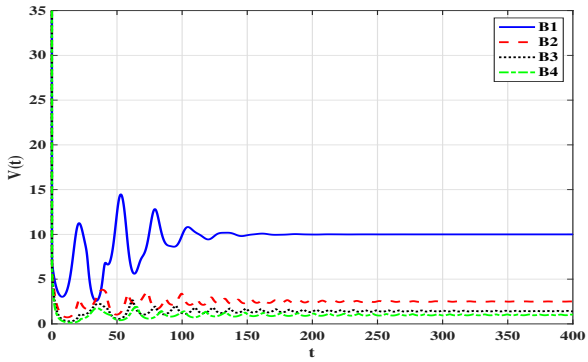
The impact of HIV-1-specific antibody and HTLV-1-specific CTL responses on the system's solutions can be seen in Figure 10. We observe that, as ρ and ξ are increased, the concentrations of uninfected CD4$^+$T cells, HIV-1-specific antibody and HTLV-1-specific CTL are increased, while concentrations of HIV-1 infected cells, HTLV-1 infected cells and HIV-1 particles are decreased. Therefore, antibody and CTL responses can control the HIV-1/HTLV-1 coinfection. Note that, $\Re_1$ and $\Re_2$ do not depend on ρ and ξ, therefore Ξ_0 can not be reached by increasing ρ and ξ. This might contribute to the development of treatments for HIV-1 and HTLV-1 with the potential to boost HIV-1-specific antibody and HTLV-1-specific CTL responses.



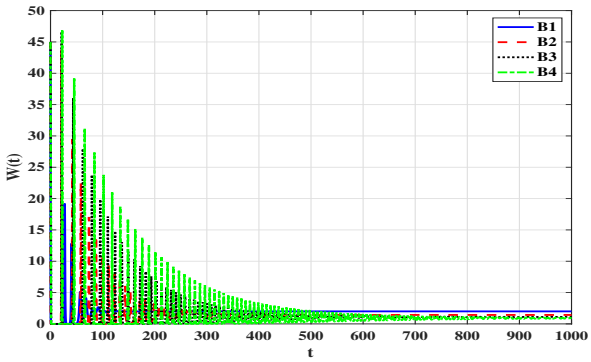
(a) Uninfected CD4$^+$T cells



(b) HIV-1-infected cells

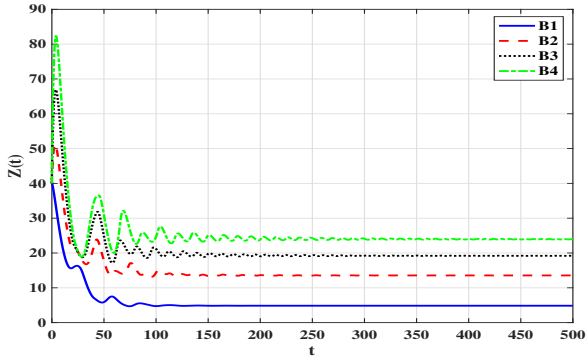


(c) HIV-1 particles

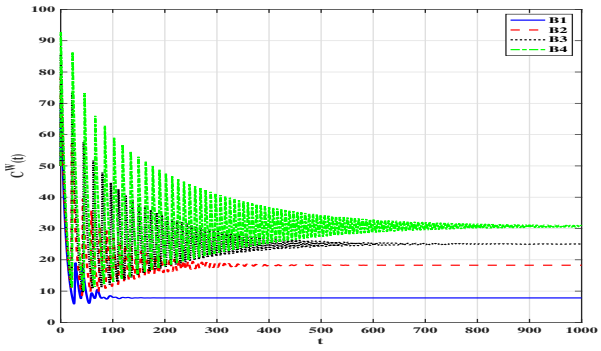


(d) HTLV-1-infected cells

Figure 10. *Cont.*



(e) HIV-1-specific antibodies



(f) HTLV-1-specific CTLs

Figure 10. Solutions of model (16)–(21) under the impact of adaptive immune response.

7. Discussion

We presented a mathematical model that accurately captures the dynamics of coinfection between HIV-1 and HTLV-1 in this study. The model included both CTL and antibody responses. The fundamental and global properties of the model were examined in our study. The model has eight equilibrium points as the following:

- Uninfected equilibrium (Ξ_0) which is usually exists. In addition, Ξ_0 is G.A.S when $\Re_1 \leq 1$, $\Re_2 \leq 1$ and unstable otherwise. In this state, the concentration of HIV-1 and HTLV-1 infections cells eventually converges to 0 and the HIV-1 and HTLV-1 patient will recover.

- The HIV-1 single-infection equilibrium without immune response (Ξ_1) exists when $\Re_1 > 1$. Futher Ξ_1 is G.A.S when $\Re_1 > 1$, $\frac{\Re_2}{\Re_1} \leq 1$ and $\Re_3 \leq 1$. In this state, the only HIV-1 infection is there, but the immune system is not responding.

- The HTLV-1 single-infection equilibrium without immune response (Ξ_2) exists when $\Re_2 > 1$ and it is G.A.S when $\Re_2 > 1$, $\frac{\Re_1}{\Re_2} \leq 1$ and $\Re_4 \leq 1$. In this state, the only HTLV-1 infection is there, but the immune system is not responding.

- The HIV-1 single-infection equilibrium with only HIV-1-specific antibody response (Ξ_3) exists when $\Re_3 > 1$. Moreover, Ξ_3 is G.A.S when $\Re_3 > 1$ and $\Re_5 \leq 1$. For this case, the body has enough number of HIV-1 viruses (i.e., $V > \mu/\xi$) which trigger the HIV-1-specific antibody response.

- The HTLV-1 single-infection equilibrium with only HTLV-1-specific CTL response (Ξ_4) exists when $\Re_4 > 1$. Moreover, Ξ_4 is G.A.S when $\Re_4 > 1$ and $\Re_6 \leq 1$. For this case, the body has enough number of HTLV-1 infected cells (i.e., $W > \varepsilon/\rho$) which trigger the HTLV-1-specific CTL response.

- The HIV-1/HTLV-1 coinfection equilibrium with only HIV-1-specific antibody response (Ξ_5) exists when $\Re_5 > 1$ and $\frac{\Re_1}{\Re_2} > 1$. Moreover, Ξ_5 is G.A.S when $\Re_5 > 1$, $\frac{\Re_1}{\Re_2} > 1$ and $\Re_8 \leq 1$. For this case, the body has enough number of HIV-1 viruses (i.e., $V > \mu/\xi$) which trigger the HIV-1-specific antibody response. But, the number of HTLV-1-infected cells still not enough to activate the HTLV-1-specific CTL response (i.e., $W \leq \varepsilon/\rho$).

- The HIV-1/HTLV-1 coinfection equilibrium with only HTLV-1-specific CTL response (Ξ_6) exists when $\Re_6 > 1$ and $\frac{\Re_2}{\Re_1} > 1$. Moreover, Ξ_6 is G.A.S when $\Re_6 > 1$, $\frac{\Re_2}{\Re_1} > 1$ and $\Re_7 \leq 1$. For this case, the body has enough number of HTLV-1 infected cells (i.e., $W > \varepsilon/\rho$) which trigger HTLV-1-specific CTL response. But, the number of HIV-1 viruses still not enough to activate the HIV-1-specific antibody response (i.e., $V \leq \mu/\xi$).

- The HIV-1/HTLV-1 coinfection equilibrium with HIV-1-specific antibody and HTLV-1-specific CTL responses (Ξ_7) exists and is G.A.S when $\Re_7 > 1$ and $\Re_8 > 1$. For this case, the concentrations of HIV-1 viruses and HTLV-1 infected cells are high enough to trigger the HIV-1-specific antibody and HTLV-1-specific CTL responses (i.e., $V > \mu/\xi$ **and** $W > \varepsilon/\rho$).

Our study's primary shortcoming is that we were unable to estimate the model's parameter values using actual data from HIV-1/HTLV-1 coinfected individuals. The reason for this is that although there may be real data for people who are infected with either HIV-1 or HTLV-1, real data for co-infection with both HIV-1 and HTLV-1 is harder to come by.

8. Conclusion

Mathematical models are commonly employed in order to comprehend the intricate dynamics of biological systems. Several several recent investigations were devoted for modeling HIV-1 and HTLV-1 coinfection with either antibody response or CTL response. In the current research work we developed an HIV-1/HTLV-1 coinfection model accounting for both antibody and CTL responses. We began by displaying the fundamental properties of the solutions, nonnegativity and boundedness. Eight threshold parameters ($\Re_1$-$\Re_8$) that we determined fully define whether the model's equilibria exist and are globally stable. The global asymptotic stability of each and every equilibria was demonstrated using the Lyapunov approach. We used numerical methods to solve the model and then visually displayed the outcomes. The theoretical and numerical results agreed, as we discovered. We discussed the effect of adaptive immunity on the HIV-1/HTLV-1 coinfection dynamics. We examined the effects of antibody and CTL responses on the HIV-1/HTLV-1 coinfection dynamical behavior. We showed that viral coinfection may be suppressed by immunological activation of antibody and CTL responses, even though the parameters of these responses have no influence on the basic reproduction ratio of HIV-1 single-infection ($\Re_1$) and HTLV-1 single-infection ($\Re_2$). This might make it easier to develop treatment strategies for HIV-1 and HTLV-1, which can improve the production of HIV-1-specific antibodies and CTL responses.

In our proposed model we assumed that the uninfected CD4$^+$T cells are produced with a constant rate λ. Indeed, in some publications (see e.g., [10, 31, 39–41]), the uninfected CD4+T cells are assumed to be proliferated at a logistic growth rate $\varpi X\left(1-\frac{X}{X_{\max}}\right)$, where where ϖ is the rate of growth and $X_{\max}$ is the maximum capacity of uninfected CD4$^+$T cells in the human body. Our suggested model, which accounts for the uninfected CD4$^+$T cells' logistic growth rate, results in:

$$\dot{X} = \lambda + \varpi X\left(1-\frac{X}{X_{\max}}\right) - \gamma X - \phi_1 XV - \phi_2 XW,$$

$$\dot{Y} = \phi_1 XV - \kappa Y,$$

$$\dot{V} = \rho Y - \sigma V - \theta VZ,$$

$$\dot{W} = \phi_2 XW - \alpha W - \delta UW,$$

$$\dot{Z} = \xi VZ - \mu Z,$$

$$\dot{U} = \rho UW - \varepsilon U.$$

We leave this concept for future study since it requires more research.

There are other methods to expand our proposed model, such by integrating: (i) reaction diffusion [42], (ii) memory effect [43, 44], (iii) stochastic effect [44, 45] and (iv) time delay [8, 40]. Future studies might also focus on including the effects of vaccines and antiviral drugs into the model. Additionally, we wish to compare the results with patient data that has been infected.

Confilict of interest

The authors declare no conflicts of interest.

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