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Analysis of spatio-temporal HIV-AIDS model

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Abstract: The aims of this paper is to study the HIV-AIDS model in space and time. This paper is the continuation of study in [6], where the fixed controls of highly antiretroviral and immunotherapy are considered for the interaction between susceptible and infected CD4⁺T cell. The equilibrium points of disease-free and endemic, positivity, boundedness and basic reproduction number of dynamical system are provided in the standard ways. For the local stability, the Fourier series is firstly employed to obtain the Jacobian matrix which is then used for further analysis of stability. Moreover, the classical numerical scheme of non-standard finite difference (NSFD) is applied to approximate our model. The stability, positivity, and consistency of numerical scheme are very important in the numerical analysis. At the last section of numerical analysis, we provide the experiments of our model numerically by varying values for the parameters of treatment HAART and Immunotherapy. We can conclude that the combinations of HAART and Immunotherapy at once is the most efficient in decreasing the infected CD4⁺T cells and the treatment of immunotherapy is more effective than the treatment of HAART. Finally, our dynamical system is eligible to predict the spread of HIV-AIDS based on the validation results with the actual data by using least square technique.

Keywords: HIV-AIDS model; non-standard finite difference; highly active antiretroviral therapy; immunotherapy; basic reproduction number; least square technique; disease transmissions.

AMS (2020) Subject Classification: 35A01, 35B40.

1 Introduction

103 Human Immunodeficiency Virus (HIV) infectious disease presents a significant obstacle to public health professionals in both developing and developed countries [11, 34, 32]. According to the United Nations for AIDS (UNAIDS) reports, 37.9 million people worldwide had HIV as of the end of 2018, and approximately one million people died each year because of infection's symptoms of HIV. In 2030, UNAIDS makes some plan to eliminate the disease. The retrovirus HIV can attack the immune's system of human CD4 cells [18, 16]. Moreover, HIV keeps attacking further CD4 cells continuously if the infected individuals are left untreated. The worst thing, the HIV infection will be in the most critical level, i.e., in stage of Acquired Immunodeficiency Syndrome

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(AIDS). In general, AIDS takes about 2 to 10 years reach its maximal stage [33]. During that stage, the ability of immune's system is much too insufficient to counter infectious diseases such as tuberculosis [5, 45], cryptococcal meningitis [36, 35], cryptosporidiosis [29], as well as cancers. In general, HIV are transmitted through bad behavior of sexual, and blood's transfusion. The infection's symptoms of HIV involve muscle aches, swollen lymph nodes, fever, and weight loss. Most infected individuals of HIV do not show the obvious the infection's symptoms. Because the infection's symptoms are possibility associated with other indications, lab testing is the appropriate manner to monitor the HIV stage. Around 8.1 million of infected individuals are generally unaware of their HIV status. People are discouraged from testing for HIV because of factors such as social stigma, discrimination, and the high cost of health care [40]. HIV-1 and HIV-2 are two distinct strains of the virus. Moreover, several more genetically different subgroups of HIV exist in HIV-1 and HIV-2. Besides that, HIV-1 was more infective than HIV-2, which is primarily discovered in West Africa.

At the present moment, there is no vaccine to eliminate the virus because of the HIV's ability to mutate. Moreover, AntiRetroviral Therapy (ART) has ability to reduce the infected individuals and prevent the disease from spreading. The life expectancy of HIV patients totally depends on the level of infection. The average survival rate for an HIV-infected individual without treatment is between 9 and 11 years, but appropriate treatment with ART can increase the patient's average lifespan to more than 10 years after the onset of AIDS [33]. Moreover, the UN-AIDS has a plan to eliminate AIDS from the planet in the last year 2030. In the last year 2020, 90 percent of HIV infected individuals will be aware of HIV status. In 2018, the deaths caused by AIDS diseases are confirmed to be increased 36.9 percent in Pakistan. According to UNAIDS, 86 percent of HIV-infected individuals living in Pakistan are unaware of their HIV status, and only 10 percent are knowledgeable of their HIV status. Having shared of needles between many users of injection drugs (IDUs), reduced contraceptive use rate, unmonitored transfusion, and a lack of resources to monitor HIV-positive individuals are all factors that contribute to the spread of infectious disease HIV in Pakistan. Government officials are requiring intersectoral and organized actions to eliminate HIV infection from the country, such as unrestricted access to HIV laboratory equipment, the ART's accessibility for infected individuals of HIV, and full adherence to testing prior to transfusion of blood and the utilization for disinfected equipment in health facilities and barbers. It is critical to raise the general public's level of awareness.

Many infectious diseases are transmitted through unrestricted population interaction. The medical staff recommends isolation or quarantine for the infected person. Many infectious disease models do not account for this critical factor. Because of this, simple mathematical models were unsuitable for disease dynamic behavior. We further adapted the dynamical system by adding the diffusion terms and two treatments of HAART and Immunotherapy in the continuous system to account for this omitted aspect in a simple system of differential equations HIV-AIDS. Moreover, the case of dynamical system HIV-1 under the delay factor was studied in [27], where this model is futher studied as in [1]. Other studies of HIV-AIDS model under the delay factor can be found in [22, 23, 7, 8, 21, 37, 42]. The media can play a significant role for understanding in population referring to HIV/AIDS infectious disease in this digital era, by encouraging the individuals to take precautions through regards to infectious disease. As a consequence, social media sites are powerful and efficient equipment that can be utilized to inform the public regarding preventing infection as well as contagious diseases including HIV/AIDS [26]. Complex dynamical behaviors of a simple unified SIR and HIV disease model was addressed in [46]. Moreover, the SIR epidemic model with various basic reproduction

numbers was studied in [17]. Based on the HIV and TSWV data, the SIR epidemic model was verified. The analytical solution provides an accurate approximation of both the experimental and clinical data. As a consequence, they can make the argument that their proposed solution is helpful for exhibiting the influence of epidemics and the fundamental factual information controlling the transmission of infectious diseases. Moreover, the transmission of infectious diseases can be more actually prevented and monitored with approaches that rely on their proposed solution. A nonlinear fractional order epidemic model was proposed and evaluated in [31] for HIV transmission with extended compartment particularly regarding exposed class to the basic SIR epidemic model. They as well established a fractional optimal control problem condition for such proposed model. It was used to solve the fractional optimal control problem related to control techniques including such contraceptive use in the exposed class, treatment for aware infectives, disease awareness between some of unaware infectives, and behavioral change for susceptible. The result revealed that control measures significantly enhance the age limit and life quality of HIV patients while somehow significantly decreasing the number of HIV/AIDS patients throughout epidemic.

Through with a mathematical model, efforts made to prevent and treat HIV/AIDS may also be analyzed. For instance, the global asymptotical stability of new fractional order HIV/AIDS models was evaluated in [44] with treatment compartment and switching parameters, and the results showed that if the threshold value is lower than one, the disease can potentially be released; the impact of trying to rehabilitate treatments on the control of HIV/AIDS spread in prisons was examined in [9], as well as the results indicated that the use of treatment methods for drug abuse removal can decrease HIV/AIDS transmission. Another of the HIV/AIDS drug treatments, HAART (highly active antiretroviral therapy), commonly keeps failing because of the emergence of drug-resistant virus [13]. As a result, Chang created a mathematical model of the probability of the occurrence of drug-resistant virus species based on the trajectories of the state variables of the HIV infection dynamic model. The stability analysis of ART in HIV/AIDS treatment dynamics was discussed in [10, 43, 41].

The organizing of this paper consists of four sections. In Section 1, we introduce the study of HIV-AIDS and also the last study for dynamical system of HIV-AIDS model. Our proposed model is provided in Section 2 by conducting two treatments of HAART and Immunotherapy. Moreover, the equilibria, basic reproduction number, positivity, boundedness, and stability of dynamical system are also presented in this section. Section 3 provides the numerical analysis of our proposed model involving discretization step, stability, positivity, and consistency of numerical scheme. Finally, the conclusion is given in Section 4.

2 Mathematical model

We consider the following HIV-AIDS model with two treatment of highly active antiretroviral therapy (HAART) and immunotherapy:

$$\begin{aligned} \frac{dU}{dt} &= \lambda - dU - (1 - u_1)\beta UV - u_2 U, \\ \frac{dV}{dt} &= (1 - u_1)\beta UV - (s + a)V, \\ \frac{dW}{dt} &= sV - bW + u_2 U, \end{aligned} \tag{2.1}$$

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where the parameters λ , β , d , a , s , and b , represent the rate of production for healthy CD4⁺T cells, infection rate, natural mortality rate of healthy CD4⁺T cells, natural mortality rate of infected CD4⁺T cells, recovery rate after getting infected, and natural mortality rate of recovered CD4⁺T cells respectively. Moreover, u_1 and u_2 represent the fixed parameters for highly active antiretroviral therapy (HAART) and Immunotherapy respectively, where $0 \leq (u_1, u_2) \leq 1$. We consider that the HIV/AIDS can transmit over the time as well as the space, then the dynamical system (2.1) can be transformed into:

$$\begin{aligned}\frac{\partial U}{\partial t} &= D_1 \frac{\partial^2 U}{\partial x^2} + \lambda - dU - (1 - u_1)\beta UV - u_2 U, \\ \frac{\partial V}{\partial t} &= D_2 \frac{\partial^2 V}{\partial x^2} + (1 - u_1)\beta UV - (s + a)V, \\ \frac{\partial W}{\partial t} &= D_3 \frac{\partial^2 W}{\partial x^2} + sV - bW + u_2 U,\end{aligned}\tag{2.2}$$

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for $(x, t) \in \Omega \times [0, +\infty)$, the initial data,

$$U(x, 0) = U_0, \quad V(x, 0) = V_0, \quad W(x, 0) = W_0, \quad x \in \Omega,\tag{2.3}$$

and the no-flux homogeneous Neumann boundary conditions

$$\frac{\partial U(x, t)}{\partial n} = \frac{\partial V(x, t)}{\partial n} = \frac{\partial W(x, t)}{\partial n} = 0, \quad (x, t) \in \Sigma_T = \partial\Omega \times [0, T],\tag{2.4}$$

where n is the Euclidean space.

2.1 Equilibria of dynamical system

This section provides the equilibria points and reproduction number \mathcal{R}_0 . In epidemiology, the basic reproduction number \mathcal{R}_0 of an infection can be thought of as the number of cases produced by one case, on average during its infectious period, in an uninfected population [19]. This basic reproduction number is helpful to ensure that infectious disease can transmit or not in the population. If $\mathcal{R}_0 < 1$ the infection will stop in the long term. Moreover, if $\mathcal{R}_0 > 1$ the infection has the ability to transmit in the population. In general, if the \mathcal{R}_0 value is greater, then the control of epidemic is more difficult.

Theorem 1. For all positive parameters, if $\mathcal{R}_0 < 1$ then the dynamical system (2.1) provides the disease-free equilibrium point $\mathcal{E}_0 = \left(\frac{\lambda}{d+u_2}, 0, \frac{\lambda u_2}{b(d+u_2)} \right)$. Moreover, the endemic equilibrium point $\mathcal{R}_0 > 1$ provides $\mathcal{E}_1 = \left(\frac{s+a}{(1-u_1)\beta}, \frac{u_2+d}{(1-u_1)\beta}(\mathcal{R}_0 - 1), \frac{s(u_2+d)}{b\beta(1-u_1)}(\mathcal{R}_0 - 1) + \frac{(s+a)u_2}{b\beta(1-u_1)} \right)$, where the basic reproduction number is defined as $\mathcal{R}_0 = \frac{\lambda\beta(1-u_1)}{(s+a)(d+u_2)}$.

Proof. The equilibrium points are obtained by considering $\frac{dU}{dt} = \frac{dV}{dt} = \frac{dW}{dt} = 0$ in the Eq. (2.1),

$$\begin{aligned}0 &= \lambda - dU - (1 - u_1)\beta UV - u_2 U, \\ 0 &= (1 - u_1)\beta UV - (s + a)V, \\ 0 &= sV - bW + u_2 U.\end{aligned}\tag{2.5}$$

30 In the steady states, there are disease free and endemic equilibrium points. Then, we consider $V = 0$ in Eq. (2.5) to provide for both the disease free and endemic equilibrium points of this HIV-AIDS model:

$$\mathcal{E}_0 = (U^0, V^0, W^0) = \left(\frac{\lambda}{d + u_2}, 0, \frac{\lambda u_2}{b(d + u_2)} \right), \quad (2.6)$$

and

$$\mathcal{E}_1 = (U^*, V^*, W^*), \quad (2.7)$$

where

$$\begin{aligned} U^* &= \frac{s + a}{(1 - u_1)\beta}, \\ V^* &= \frac{u_2 + d}{(1 - u_1)\beta}(\mathcal{R}_0 - 1), \\ W^* &= \frac{s(u_2 + d)}{b\beta(1 - u_1)}(\mathcal{R}_0 - 1) + \frac{(s + a)u_2}{b\beta(1 - u_1)}. \end{aligned}$$

The reproduction number \mathcal{R}_0 can be obtained from (2.1)₂ at the disease free equilibrium point \mathcal{E}_0 and when $D_1 = D_2 = D_3 = 0$, then we have

$$\begin{aligned} \frac{dV}{dt} &= (1 - u_1)\beta U^0 V^0 - (s + a)V^0 > 0, \\ (1 - u_1)\beta U^0 &> (s + a), \\ \frac{\lambda\beta(1 - u_1)}{(s + a)(d + u_2)} &> 1 \rightarrow \mathcal{R}_0 > 1. \end{aligned} \quad (2.8)$$

□

2.2 Positivity and boundedness of dynamical system

8 **Theorem 2.** Suppose that $U(x, t)$, $V(x, t)$, and $W(x, t)$ be the solution of dynamical system (2.2) satisfying the initial data (2.3) and boundary conditions (2.4). Then, for $(x, t) \in \Omega \times [0, +\infty)$, $U(x, t)$, $V(x, t)$, and $W(x, t)$ are uniformly bounded, i.e.,

$$\limsup_{x \in \Omega, t \rightarrow +\infty} (U, V, W)(x, t) \leq \frac{\lambda}{a + b + d}.$$

6 *Proof.* Let $\mathcal{P}(x, t) = (U + V + W)(x, t)$ be the total population. Then, by differentiating it in t , one has

$$\begin{aligned} \frac{\partial \mathcal{P}(x, t)}{\partial t} &= \frac{\partial U(x, t)}{\partial t} + \frac{\partial V(x, t)}{\partial t} + \frac{\partial W(x, t)}{\partial t} \\ &= (D_1 + D_2 + D_3) \frac{\partial^2 \mathcal{P}(x, t)}{\partial x^2} + \lambda - (a + b + d)\mathcal{P}(x, t). \end{aligned}$$

Hence, one can derive

$$\limsup_{x \in \Omega, t \rightarrow +\infty} \mathcal{P}(x, t) = \frac{\lambda}{a + b + d}.$$

We notice that $\mathcal{P}(x, t) = (U + V + W)(x, t)$, then we have

$$\limsup_{x \in \Omega, t \rightarrow +\infty} U(x, t) \leq \limsup_{x \in \Omega, t \rightarrow +\infty} \mathcal{P}(x, t) = \frac{\lambda}{a + b + d}.$$

The similar ways are employed into $V(x, t)$ and $W(x, t)$. Therefore, $U(x, t)$, $V(x, t)$, and $W(x, t)$ are uniformly bounded for $(x, t) \in \Omega \times [0, +\infty)$. \square

Theorem 3. Suppose that $U(x, t)$, $V(x, t)$, and $W(x, t)$ be the solution of dynamical system (2.2) satisfying the initial data (2.3) and boundary conditions (2.4). Then, for $(x, t) \in \Omega \times [0, +\infty)$, $U(x, t)$, $V(x, t)$, and $W(x, t)$ are non-negative.

Proof. By ignoring the diffusion terms, it follows from (2.2)₁, one has

$$\frac{dU(t)}{dt} + (d + (1 - u_1)\beta V) U(t) = \lambda.$$

We further integrate the above equation in t to get

$$\begin{aligned} U(t^*) &= \left(U(0) + \int_0^{t^*} \exp \left(\int_0^t \lambda (d + (1 - u_1)\beta V(\tau)) d\tau \right) \right) \\ &\quad \exp \left(\int_0^{t^*} (d + (1 - u_1)\beta V(\tau)) d\tau \right)^{-1} \\ &\geq 0, \end{aligned}$$

for $t^* = \sup \{t \geq 0 : (U, V, W)(t) \geq 0\} \in [0, t]$. The similar arguments are employed to provide $(V, W)(t) \geq 0$. \square

2.3 Stability of dynamical system

In the next study, we provide the analysis of local and global stability for the disease-free and endemic equilibrium points as stated in the following theorems.

Theorem 4. Suppose $\mathcal{R}_0 < 1$. Then, we can show that the equilibrium point \mathcal{E}_0 ⁷ is locally asymptotically stable.

Proof. The local stability of dynamical system with the diffusion terms can be established by the strategy as introduced in ([12, 4]) by defining the following formula

$$\begin{aligned} U(x, t) &= \sum_k U_k e^{\phi t} \sin(kx), \\ V(x, t) &= \sum_k V_k e^{\phi t} \sin(kx), \\ W(x, t) &= \sum_k W_k e^{\phi t} \sin(kx). \end{aligned} \tag{2.9}$$

Then, we substitute Eq. (2.9) into (2.2) to get

$$\begin{aligned} \sum_k (\mathcal{P}_{11} - D_1 k^2 - \phi) U_k + \sum_k \mathcal{P}_{12} V_k + \sum_k \mathcal{P}_{13} W_k &= 0, \\ \sum_k \mathcal{P}_{21} U_k + \sum_k (\mathcal{P}_{22} - D_2 k^2 - \phi) V_k + \sum_k \mathcal{P}_{23} W_k &= 0, \\ \sum_k \mathcal{P}_{31} U_k + \sum_k \mathcal{P}_{32} V_k + \sum_k (\mathcal{P}_{33} - D_3 k^2 - \phi) W_k &= 0. \end{aligned} \quad (2.10)$$

It follows from (2.10), one has the following matrix

$$J = \begin{pmatrix} \mathcal{P}_{11} - D_1 k^2 - \phi & \mathcal{P}_{12} & \mathcal{P}_{13} \\ \mathcal{P}_{21} & \mathcal{P}_{22} - D_2 k^2 - \phi & \mathcal{P}_{23} \\ \mathcal{P}_{31} & \mathcal{P}_{32} & \mathcal{P}_{33} - D_3 k^2 - \phi \end{pmatrix} \quad (2.11)$$

where

$$\begin{aligned} \mathcal{P}_{11} &= -(d + (1 - u_1)\beta V + u_2), \quad \mathcal{P}_{12} = -(1 - u_1)\beta U, \quad \mathcal{P}_{13} = 0, \\ \mathcal{P}_{21} &= (1 - u_1)\beta V, \quad \mathcal{P}_{22} = -((s + a) - (1 - u_1)\beta U), \quad \mathcal{P}_{23} = 0, \\ \mathcal{P}_{31} &= u_2, \quad \mathcal{P}_{32} = s, \quad \mathcal{P}_{33} = -b. \end{aligned}$$

We substitute the disease-free equilibrium point \mathcal{E}_0 into (2.11), then one can derive

$$J(\mathcal{E}_0) = \begin{pmatrix} -(d + u_2 + D_1 k^2 + \phi) & -(1 - u_1)\beta U^0 & 0 \\ 0 & -((s + a) - (1 - u_1)\beta U^0 + D_2 k^2 + \phi) & 0 \\ u_2 & s & -(b + D_3 k^2 + \phi) \end{pmatrix}. \quad (2.12)$$

Applying the formula $|J(\mathcal{E}_0) - \lambda I_d| = 0$ into (2.12), then the eigenvalues satisfy the following characteristic equation

$$\begin{aligned} &(-(b + D_3 k^2 + \phi) - \lambda)(-(d + u_2 + D_1 k^2 + \phi) - \lambda) \\ &(-((s + a) - (1 - u_1)\beta U^0 + D_2 k^2 + \phi) - \lambda) = 0. \end{aligned} \quad (2.13)$$

It follows from (2.13), we notice that the eigenvalues $\lambda_1 = -(b + D_3 k^2 + \phi) < 0$ and $\lambda_2 = -(d + u_2 + D_1 k^2 + \phi) < 0$. Then, we only need to make sure that $\lambda_3 = -((s + a) - (1 - u_1)\beta U^0 + D_2 k^2 + \phi) < 0$. By substituting U^0 in Theorem 1, one has

$$\frac{\lambda\beta(1 - u_1)}{d + u_2} - (s + a) - D_2 k^2 - \phi = (s + a)(\mathcal{R}_0 - 1) - D_2 k^2 - \phi.$$

Then, we can conclude that $\lambda_3 < 0$ only if $\mathcal{R}_0 < 1$. □

Theorem 5. Suppose $\mathcal{R}_0 > 1$. Then, we can show that the equilibrium point \mathcal{E}_1 is locally asymptotically stable. 51

Proof. By employing the similar ways as in Theorem 4. At the endemic equilibrium point \mathcal{E}_1 , one has

$$J(\mathcal{E}_1) = \begin{pmatrix} \mathcal{A} & -(1 - u_1)\beta U^* & 0 \\ (1 - u_1)\beta V^* & \mathcal{B} & 0 \\ u_2 & s & \mathcal{C} \end{pmatrix}. \quad (2.14)$$

where

$$\begin{aligned}\mathcal{A} &= -(d + (1 - u_1)\beta V^* + u_2 + D_1 k^2 + \phi), \\ \mathcal{B} &= -((s + a) - (1 - u_1)\beta U^* + D_2 k^2 + \phi), \\ \mathcal{C} &= -(b + D_3 k^2 + \phi).\end{aligned}$$

The formula $|J(\mathcal{E}_1) - \lambda I_d| = 0$ is employed into (2.14), then the characteristic equation provides the following eigenvalues

$$\begin{aligned}\lambda_1 &= -(b + D_3 k^2 + \phi) < 0, \\ \lambda_2 &= \frac{-(a + d + (1 - u_1)\beta(V^* - U^*) + u_2 + (D_1 + D_2)k^2 + 2\phi) - \sqrt{\mathcal{D}}}{2}, \\ \lambda_3 &= \frac{-(a + d + (1 - u_1)\beta(V^* - U^*) + u_2 + (D_1 + D_2)k^2 + 2\phi) + \sqrt{\mathcal{D}}}{2},\end{aligned}$$

where

$$\begin{aligned}\mathcal{D} &= (a + d + (1 - u_1)\beta(V^* - U^*) + u_2 + (D_1 + D_2)k^2 + 2\phi)^2 \\ &\quad - 4[(d + (1 - u_1)\beta V^* + u_2 + D_1 k^2 + \phi)((s + a) - (1 - u_1)\beta U^* + D_2 k^2 + \phi) \\ &\quad + (1 - u_1)\beta V^*(1 - u_1)\beta U^*].\end{aligned}$$

Then $\mathcal{D} > 0$. This condition gives $\lambda_2 < 0$. We notice that

$$\begin{aligned}\mathcal{D} &< (a + d + (1 - u_1)\beta(V^* - U^*) + u_2 + (D_1 + D_2)k^2 + 2\phi)^2 \\ &\quad - 4[(d + (1 - u_1)\beta V^* + u_2 + D_1 k^2 + \phi)((s + a) - (1 - u_1)\beta U^* + D_2 k^2 + \phi)] \\ &\quad - 4[(1 - u_1)^2 \beta^2 V^* U^*] \\ &< (a + d + (1 - u_1)\beta(V^* - U^*) + u_2 + (D_1 + D_2)k^2 + 2\phi)^2.\end{aligned}$$

Therefore,

$$\begin{aligned}\lambda_3 &< \frac{-(a + d + (1 - u_1)\beta(V^* - U^*) + u_2 + (D_1 + D_2)k^2 + 2\phi)}{2} \\ &\quad + \frac{\sqrt{(a + d + (1 - u_1)\beta(V^* - U^*) + u_2 + (D_1 + D_2)k^2 + 2\phi)^2}}{2} = 0,\end{aligned}$$

giving $\lambda_3 < 0$. Then, we can conclude that all eigen values satisfy Theorem 5. \square

Theorem 6. Suppose $\mathcal{R}_0 < 1$. Then, we can show that the equilibrium point \mathcal{E}_0 is globally asymptotically stable. 68

Proof. We consider the following Lyapunov function

$$L_{\mathcal{E}_0}(t) = \int_{\Omega} \left[U(x, t) - U^0 - U^0 \ln \frac{U(x, t)}{U^0} + \frac{V^2(x, t)}{2} \right] dx.$$

Then, differentiating the above equation in t and substituting the dynamical system (2.2) into the result, one can derive

$$\begin{aligned} \frac{dL_{\mathcal{E}_0}(t)}{dt} &= \int_{\Omega} \left[\left(1 - \frac{U^0}{U(x,t)} \right) \frac{\partial U(x,t)}{\partial t} + V(x,t) \frac{\partial V(x,t)}{\partial t} \right] dx \\ &= \int_{\Omega} \left[\left(1 - \frac{U^0}{U(x,t)} \right) (D_1 \Delta U(x,t) + \lambda - (d+u_2)U(x,t) - (1-u_1)\beta U(x,t)V(x,t)) \right] dx \\ &\quad + \int_{\Omega} [D_2 V(x,t) \Delta V(x,t) + (1-u_1)\beta U(x,t)V^2(x,t) - (s+a)V^2(x,t)] dx. \end{aligned} \tag{2.15}$$

By employing the equilibrium points as in Theorem 1 into (2.15), then one has

$$\begin{aligned} \frac{dL_{\mathcal{E}_0}(t)}{dt} &\leq \int_{\Omega} \left[\left(1 - \frac{U^0}{U(x,t)} \right) (D_1 \Delta U(x,t) + (d+u_2)U^0 - (d+u_2)U(x,t)) \right] dx \\ &\quad + \int_{\Omega} \left[D_2 V(x,t) \Delta V(x,t) + \frac{(1-u_1)\lambda\beta}{d+u_2} V^2(x,t) - (s+a)V^2(x,t) \right] dx. \end{aligned} \tag{2.16}$$

We notice that

$$\begin{aligned} 0 &= \int_{\partial\Omega} \frac{U_0}{U(x,t)} \nabla U(x,t) \cdot n \, dx = \int_{\Omega} \operatorname{div} \left(\frac{U^0}{U(x,t)} \nabla U(x,t) \right) dx \\ &= \int_{\Omega} \left(\frac{U^0}{U(x,t)} \Delta U(x,t) - U^0 \frac{|\nabla U(x,t)|^2}{U^2(x,t)} \right), \end{aligned} \tag{2.17}$$

$$\begin{aligned} 0 &= \int_{\partial\Omega} V(x,t) \nabla V(x,t) \cdot n \, dx = \int_{\Omega} \operatorname{div} (V(x,t) \nabla V(x,t)) dx \\ &\equiv \int_{\Omega} (V(x,t) \Delta V(x,t) + |\nabla V(x,t)|^2), \end{aligned}$$

and

$$\int_{\Omega} \Delta U(x,t) \, dx = \int_{\partial\Omega} \frac{\partial U(x,t)}{\partial n} \, dx = 0, \quad \int_{\Omega} \Delta V(x,t) \, dx = \int_{\partial\Omega} \frac{\partial V(x,t)}{\partial n} \, dx = 0, \tag{2.18}$$

where the divergence theorem and boundary conditions in (2.4) have been applied. Substituting Eqs. (2.17) and (2.18) into Eq. (2.16), then one has

$$\begin{aligned} \frac{dL_{\mathcal{E}_0}(t)}{dt} &\leq -(d+u_2) \int_{\Omega} \frac{(U-U^0)^2}{U(x,t)} \, dx - (s+a) \int_{\Omega} (1-\mathcal{R}_0)V^2(x,t) \, dx \\ &\quad - D_1 U^0 \int_{\Omega} \frac{|\nabla U(x,t)|^2}{U^2(x,t)} \, dx - \int_{\Omega} |V(x,t)|^2 \, dx \\ &\leq -C \int_{\Omega} ((U(x,t) - U^0)^2 + (V(x,t) - 0)^2 + |\nabla U(x,t)|^2 + |\nabla V(x,t)|^2) \, dx. \end{aligned} \tag{2.19}$$

We can conclude that $\frac{dL_{\mathcal{E}_0}(t)}{dt} \leq 0$ if $\mathcal{R}_0 < 1$. Moreover, it follows from the following Lemma,

Lemma 1. Assume that $\psi, \varphi \in C^1([c_1, \infty))$, $\varphi \geq 0$ and ψ is bounded from below. Then, the conditions of $\psi'(t) \leq -c_2\varphi$ and $\varphi'(t) \leq C$ in $[c_1, \infty)$ imply that $\lim_{t \rightarrow \infty} \varphi = 0$ for some constants c_1, c_2, C ,

where the detailed proof of this Lemma can be seen in [28]. Then, we have

$$\lim_{t \rightarrow \infty} \int_{\Omega} [(U - U^0)^2 + (V - 0)^2 + |\nabla U|^2 + |\nabla I|^2] dx = 0.$$

Moreover, we apply the following Poincaré inequality

$$\int_{\Omega} c_1 |U - \bar{U}|^2 \leq |\nabla U|^2 dx, \quad \int_{\Omega} c_2 |V - \bar{V}|^2 \leq |\nabla V|^2 dx,$$

to get

$$\lim_{t \rightarrow \infty} \int_{\Omega} [(U - \bar{U})^2 + (V - \bar{V})^2] dx = 0, \quad (2.20)$$

where $\bar{U} = \frac{1}{|\Omega|} \int_{\Omega} U(x, t) dx$ and $\bar{V} = \frac{1}{|\Omega|} \int_{\Omega} V(x, t) dx$. Hence,

$$\begin{aligned} |\Omega|(\bar{U} - U^0)^2 &= \int_{\Omega} [\bar{U} - U(x, t) + U(x, t) - U^0]^2 dx \\ &\stackrel{6}{\leq} \int_{\Omega} [\bar{U} - U(x, t)]^2 dx + \int_{\Omega} [U(x, t) - U^0]^2 dx \\ |\Omega|(\bar{V} - 0)^2 &= \int_{\Omega} [\bar{V} - V(x, t) + V(x, t) - 0]^2 dx \\ &\stackrel{2}{\leq} \int_{\Omega} [\bar{V} - V(x, t)]^2 dx + \int_{\Omega} [V(x, t) - 0]^2 dx. \end{aligned} \quad (2.21)$$

Therefore, one has $\bar{U} \rightarrow U^0$ as $t \rightarrow \infty$ and $\bar{V} \rightarrow 0$ as $t \rightarrow \infty$. It follows from Theorem 2, the dynamical system (2.2) is bounded. Moreover, one has

$$\lim_{n \rightarrow \infty} \|U(\cdot, t_n) - F_1(\cdot)\|_{C^2(\Omega)} = 0, \quad \lim_{n \rightarrow \infty} \|V(\cdot, t_n) - F_2(\cdot)\|_{C^2(\Omega)} = 0,$$

for a subsequence t_n , and non-negative functions $F_1, F_2 \in C^2(\Omega)$. By employing (2.20)-(2.21), one provides $F_1 \equiv U^0$ and $F_2 \equiv 0$. Therefore,

$$\lim_{n \rightarrow \infty} \|U(\cdot, t_n) - U^0\|_{C^2(\Omega)} = 0, \quad \lim_{n \rightarrow \infty} \|V(\cdot, t_n) - 0\|_{C^2(\Omega)} = 0.$$

□

Theorem 7. Suppose $\mathcal{R}_0 > 1$. Then, we can show that the equilibrium point \mathcal{E}_1 is globally asymptotically stable.

Proof. Firstly, we provide the following Lyapunov function

$$L_{\mathcal{E}_1}(t) = \int_{\Omega} \left[U(x, t) - U^* - U^* \ln \frac{U(x, t)}{U^*} + V(x, t) - V^* - V^* \ln \frac{V(x, t)}{V^*} \right] dx.$$

Then, differentiating the above equation in t and substituting the dynamical system (2.2) into the result, one can derive

$$\begin{aligned}
\frac{dL_{\mathcal{E}_1}(t)}{dt} &= \int_{\Omega} \left[\left(1 - \frac{U^*}{U(x,t)} \right) \frac{\partial U(x,t)}{\partial t} + \left(1 - \frac{V^*}{V(x,t)} \right) \frac{\partial V(x,t)}{\partial t} \right] dx \\
&= \int_{\Omega} \left[\left(1 - \frac{U^*}{U(x,t)} \right) (\lambda - (d+u_2)U(x,t) - (1-u_1)\beta U(x,t)V(x,t)) \right] dx \\
&\quad + \int_{\Omega} \left[\left(1 - \frac{V^*}{V(x,t)} \right) ((1-u_1)\beta U(x,t)V(x,t) - (s+a)V(x,t)) \right] dx \\
&\quad + \int_{\Omega} \left(1 - \frac{U^*}{U(x,t)} \right) D_1 \Delta U(x,t) + \int_{\Omega} \left(1 - \frac{V^*}{V(x,t)} \right) D_2 \Delta V(x,t). \tag{2.22}
\end{aligned}$$

By conducting the equilibrium points in Theorem 1, divergen theorem and boundary conditions (2.4) as in (2.17)-(2.18) into (2.22) and $\lambda = (d+u_2)U^* + (s+a)V^*$, then one has

$$\begin{aligned}
\frac{dL_{\mathcal{E}_1}(t)}{dt} &\leq -(d+u_2) \int_{\Omega} \frac{(U(x,t) - U^*)^2}{U(x,t)} dx - (s+a) \int_{\Omega} \frac{(V(x,t) - V^*)^2}{V(x,t)} \\
&\quad - D_1 U^* \int_{\Omega} \frac{|\nabla U(x,t)|^2}{U^2(x,t)} dx - D_2 V^* \int_{\Omega} \frac{|\nabla V(x,t)|^2}{V^2(x,t)} dx. \tag{2.23}
\end{aligned}$$

According to the equilibrium point $(s+a)V^* = (1-u_1)\beta U^*V^*$, then (2.23) becomes

$$\begin{aligned}
\frac{dL_{\mathcal{E}_1}(t)}{dt} &\leq -(d+u_2) \int_{\Omega} \frac{(U(x,t) - U^*)^2}{U(x,t)} dx - (1-u_1)\beta U^* \int_{\Omega} \frac{(V(x,t) - V^*)^2}{V(x,t)} \\
&\quad - D_1 U^* \int_{\Omega} \frac{|\nabla U(x,t)|^2}{U^2(x,t)} dx - D_2 V^* \int_{\Omega} \frac{|\nabla V(x,t)|^2}{V^2(x,t)} dx \\
&\leq -CG(t), \tag{2.24}
\end{aligned}$$

From the previous results we can see that $\frac{dL_{\mathcal{E}_1}(t)}{dt} \leq -CG(t)$. According to Lemma 1, one has

$$\lim_{t \rightarrow \infty} \int_{\Omega} [(U - U^*)^2 + (V - V^*)^2 + |\nabla U|^2 + |\nabla V|^2] dx = 0. \tag{56}$$

Applying the following Poincaré inequality

$$\int_{\Omega} r_1 |U - \bar{U}|^2 \leq |\nabla U|^2 dx, \quad \int_{\Omega} r_2 |V - \bar{V}|^2 \leq |\nabla V|^2 dx, \tag{86}$$

then one has

$$\lim_{t \rightarrow \infty} \int_{\Omega} (U - \bar{U})^2 dx = 0, \quad \lim_{t \rightarrow \infty} \int_{\Omega} (V - \bar{V})^2 dx = 0, \tag{2.25}$$

where $\bar{U} = \frac{1}{|\Omega|} \int_{\Omega} U(x, t) dx$ and $\bar{V} = \frac{1}{|\Omega|} \int_{\Omega} V(x, t) dx$. Hence,

$$\begin{aligned} |\Omega|(\bar{U} - U^*)^2 &= \int_{\Omega} [\bar{U} - U(x, t) + U(x, t) - U^*]^2 dx \\ &\leq \int_{\Omega} [\bar{U} - U(x, t)] dx + \int_{\Omega} [U(x, t) - U^*] dx \end{aligned} \quad (2.26)$$

$$\begin{aligned} |\Omega|(\bar{V} - V^*)^2 &= \int_{\Omega} [\bar{V} - V(x, t) + V(x, t) - V^*]^2 dx \\ &\leq \int_{\Omega} [\bar{V} - V(x, t)] dx + \int_{\Omega} [V(x, t) - V^*] dx. \end{aligned}$$

Therefore, one has $\bar{U} \rightarrow U^*$ as $t \rightarrow \infty$ and $\bar{V} \rightarrow V^*$ as $t \rightarrow \infty$. Moreover, one has

$$\lim_{n \rightarrow \infty} \|U(\cdot, t_n) - H_1(\cdot)\|_{C^2(\Omega)} = 0, \quad \lim_{n \rightarrow \infty} \|V(\cdot, t_n) - H_2(\cdot)\|_{C^2(\Omega)} = 0,$$

for a subsequence t_n , and non-negative functions $H_1, H_2 \in C^2(\Omega)$. By employing (2.25)-(2.26), one provides $H_1 \equiv U^*$ and $H_2 \equiv V^*$. Therefore,

$$\lim_{n \rightarrow \infty} \|U(\cdot, t_n) - U^*\|_{C^2(\Omega)} = 0, \quad \lim_{n \rightarrow \infty} \|V(\cdot, t_n) - V^*\|_{C^2(\Omega)} = 0.$$

□

3 Numerical analysis

The physical phenomenon can be represented into the system of differential equations to provide the further studies. The dynamical system involving the diffusion terms are more complicated to provide the analytical solutions. Therefore, the role of numerical technique is required in this case to establish the numerical solutions. The numerical techniques are employed to investigate the model's behavior. Although these techniques do not provide an analytical solution to the model, they do assist us in studying the physical phenomenon of the model. Moreover, in mathematical epidemiology, a numerical technique with meaningful properties such as positivity, consistency, and population boundedness is required. For this purpose, we apply the non-standard finite difference providing the physical behavior of mathematical epidemiology.

3.1 Discretization step

In this paper, the non-standard finite difference scheme is employed for studying the behavior of our model. R. E. Mickens firstly introduced the non-standard finite difference (NSFD) in 1989. The NSFD ensures the model's positivity and boundedness, which are essential properties of the state variables. The numerical techniques provide the alternative to approximate the solutions of dynamical systems for both linear and nonlinear differential equations [3, 14, 20]. We firstly transform our continuous dynamical system into the discrete formulation. Moreover, the Taylor's series is the most effective method to provide the approximations. We now consider maximum values of space and time namely M and N . Moreover, the partitions $lb = x_0 < x_1 < x_2 < \dots < x_M = ub$ and $0 = t_0 < t_1 < t_2 < \dots < t_N = T$ respectively with the step size of space

$h = \frac{lb-ub}{M}$ and step size of time $k = \frac{T}{N}$ are the discretization results of the spatial interval of space $[lb, ub]$ over the time $[0, T]$. Moreover, $x_j = jh$ and $t_m = mk$ are the points of partitions, where $j \in [0, M]$ and $m \in [0, N]$. At the points of partitions $(x_j, t_m) = (jh, mk)$, we suppose that U_j^m , V_j^m , and W_j^m denote the approximations of $U(x, t)$, $V(x, t)$, and $W(x, t)$ respectively. We firstly define the forward and central finite difference for first derivative in time and second derivative in space respectively as shown below.

$$\begin{aligned}\frac{\partial \mathcal{K}}{\partial t} &= \frac{\mathcal{K}_j^{m+1} - \mathcal{K}_j^m}{\Delta t}, \\ \frac{\partial^2 \mathcal{K}}{\partial x^2} &= \frac{\mathcal{K}_{j-1}^{m+1} - 2\mathcal{K}_j^{m+1} + \mathcal{K}_{j+1}^{m+1}}{(\Delta x)^2}.\end{aligned}$$

We further employ the discretization steps to the compartment U in (2.2)₁ is:

$$\begin{aligned}\frac{U_j^{m+1} - U_j^m}{\Delta t} &= D_1 \frac{U_{j-1}^{m+1} - 2U_j^{m+1} + U_{j+1}^{m+1}}{(\Delta x)^2} + \lambda - dU_j^{m+1} \\ &\quad - (1 - u_1)\beta U_j^{m+1} V_j^m - u_2 U_j^{m+1}, \\ U_j^{m+1} - U_j^m &= \frac{D_1 \Delta t}{(\Delta x)^2} \left(U_{j-1}^{m+1} - 2U_j^{m+1} + U_{j+1}^{m+1} \right) + \Delta t \lambda - \Delta t d U_j^{m+1} \\ &\quad - \Delta t (1 - u_1) \beta U_j^{m+1} V_j^m - \Delta t u_2 U_j^{m+1}.\end{aligned}$$

$$-\nu_1 U_{j-1}^{m+1} + (1 + 2\nu_1 + \Delta t d + \Delta t (1 - u_1) \beta V_j^m + \Delta t u_2) U_j^{m+1} - \nu_1 U_{j+1}^{m+1} = U_j^m + \Delta t \lambda, \quad (3.1)$$

where

$$\nu_1 = \frac{D_1 \Delta t}{(\Delta x)^2}.$$

The similar ways for the compartment V in (2.2)₂, one has:

$$\begin{aligned}\frac{V_j^{m+1} - V_j^m}{\Delta t} &= D_2 \frac{V_{j-1}^{m+1} - 2V_j^{m+1} + V_{j+1}^{m+1}}{(\Delta x)^2} - (s + a) V_j^{m+1} \\ &\quad + (1 - u_1) \beta U_j^m V_j^{m+1}, \\ V_j^{m+1} - V_j^m &= \frac{D_2 \Delta t}{(\Delta x)^2} \left(V_{j-1}^{m+1} - 2V_j^{m+1} + V_{j+1}^{m+1} \right) - \Delta t (s + a) V_j^{m+1} \\ &\quad + \Delta t (1 - u_1) \beta U_j^m V_j^{m+1}.\end{aligned}$$

$$-\nu_2 V_{j-1}^{m+1} + (1 + 2\nu_2 + \Delta t (s + a) - \Delta t (1 - u_1) \beta U_j^m) V_j^{m+1} - \nu_2 V_{j+1}^{m+1} = V_j^m, \quad (3.2)$$

where

$$\nu_2 = \frac{D_2 \Delta t}{(\Delta x)^2}.$$

Moreover, the compartment W in (2.2)₃ is:

$$\begin{aligned} \frac{W_j^{m+1} - W_j^m}{\Delta t} &= D_3 \frac{W_{j-1}^{m+1} - 2W_j^{m+1} + W_{j+1}^{m+1}}{(\Delta x)^2} - bW_j^{m+1} + sV_j^m + u_2U_j^m \\ W_j^{m+1} - W_j^m &= \frac{D_3\Delta t}{(\Delta x)^2} \left(W_{j-1}^{m+1} - 2W_j^{m+1} + W_{j+1}^{m+1} \right) - \Delta tbW_j^{m+1} + \Delta tsV_j^m + \Delta tu_2U_j^m. \\ -\nu_3W_{j-1}^{m+1} + (1 + 2\nu_3 + \Delta tb)W_j^{m+1} - \nu_3W_{j+1}^{m+1} &= W_j^m + \Delta tsV_j^m + \Delta tu_2U_j^m, \end{aligned} \quad (3.3)$$

where

$$\nu_3 = \frac{D_3\Delta t}{(\Delta x)^2}.$$

3.2 Stability of numerical scheme

The main concern in approximations of dynamical system is the increase of round-off errors. In other words, the small changes of initial conditions can affect the results significantly. Moreover, if the difference between approximate and exact solutions is small, then such approximations is stable. Then, Von-Nuemann method [39, 30, 15, 25, 2] is addressed to study the stability criterion of the NSFD. The Von-Neumann method provides the characteristics of stability for the numerical results of proposed model. For this purpose, we provide the following Fourier series to break down the numerical error in the results of HIV-AIDS model. Then, the following Fourier series are substituted into Eq. (3.1)

$$\begin{aligned} U_j^m &= \mathcal{G}_u^m e^{i\phi j h}, \\ U_j^{m+1} &= \mathcal{G}_u^{m+1} e^{i\phi j h}, \\ U_{j-1}^{m+1} &= \mathcal{G}_u^{m+1} e^{i\phi(j-1)h}, \\ U_{j+1}^{m+1} &= \mathcal{G}_u^{m+1} e^{i\phi(j+1)h}, \end{aligned}$$

to get

$$\begin{aligned} -\nu_1U_{j-1}^{m+1} + (1 + 2\nu_1 + \Delta td + \Delta t(1 - u_1)\beta V_j^m + \Delta tu_2)U_j^{m+1} - \nu_1U_{j+1}^{m+1} &= U_j^m + \Delta t\lambda, \\ -\nu_1\mathcal{G}_u^{m+1}e^{i\phi(j-1)h} + (1 + 2\nu_1 + \Delta td + \Delta t(1 - u_1)\beta V_j^m + \Delta tu_2)\mathcal{G}_u^{m+1}e^{i\phi j h} \\ -\nu_1\mathcal{G}_u^{m+1}e^{i\phi(j+1)h} &= \mathcal{G}_u^m e^{i\phi j h}. \end{aligned}$$

The above equation is divided by $\mathcal{G}_u^m e^{i\phi j h}$ for both sides, then one has:

$$\begin{aligned} -\nu_1\mathcal{G}_u e^{-i\phi h} + (1 + 2\nu_1 + \Delta td + \Delta t(1 - u_1)\beta V_j^m + \Delta tu_2)\mathcal{G}_u - \nu_1\mathcal{G}_u e^{i\phi h} &= 1, \\ -\nu_1\mathcal{G}_u \left(e^{-i\phi h} + e^{i\phi h} \right) + (1 + 2\nu_1 + \Delta td + \Delta t(1 - u_1)\beta V_j^m + \Delta tu_2)\mathcal{G}_u &= 1, \\ -2\nu_1\mathcal{G}_u \cos(\phi h) + (1 + 2\nu_1 + \Delta td + \Delta t(1 - u_1)\beta V_j^m + \Delta tu_2)\mathcal{G}_u &= 1, \\ \left(-2\nu_1 + 4\nu_1 \sin^2 \left(\frac{\phi h}{2} \right) + 1 + 2\nu_1 + \Delta td + \Delta t(1 - u_1)\beta V_j^m + \Delta tu_2 \right) \mathcal{G}_u &= 1. \end{aligned}$$

$$|\mathcal{G}_u| = \left| \frac{1}{4\nu_1 \sin^2\left(\frac{\phi h}{2}\right) + 1 + \Delta t(d + u_2) + \Delta t(1 - u_1)\beta V_j^m} \right| < 1 \quad (3.4)$$

We further employ the same strategy for the Eq. (3.2) by substituting:

$$\begin{aligned} V_j^m &= \mathcal{G}_v^m e^{i\phi j h}, \\ V_j^{m+1} &= \mathcal{G}_v^{m+1} e^{i\phi j h}, \\ V_{j-1}^{m+1} &= \mathcal{G}_v^{m+1} e^{i\phi(j-1)h}, \\ V_{j+1}^{m+1} &= \mathcal{G}_v^{m+1} e^{i\phi(j+1)h}. \end{aligned}$$

Then one has:

$$\begin{aligned} -\nu_2 V_{j-1}^{m+1} + (1 + 2\nu_2 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m) V_j^{m+1} - \nu_2 V_{j+1}^{m+1} &= V_j^m, \\ -\nu_2 \mathcal{G}_v^{m+1} e^{i\phi(j-1)h} + (1 + 2\nu_2 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m) \mathcal{G}_v^{m+1} e^{i\phi j h} \\ -\nu_2 \mathcal{G}_v^{m+1} e^{i\phi(j+1)h} &= \mathcal{G}_v^m e^{i\phi j h}. \end{aligned}$$

For the simplification, we further divide the above equation by $\mathcal{G}_v^m e^{i\phi j h}$ for both sides to obtain:

$$\begin{aligned} -\nu_2 \mathcal{G}_v e^{-i\phi h} + (1 + 2\nu_2 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m) \mathcal{G}_v - \nu_2 \mathcal{G}_v e^{i\phi h} &= 1, \\ -\nu_2 \mathcal{G}_v (e^{-i\phi h} + e^{i\phi h}) + (1 + 2\nu_2 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m) \mathcal{G}_v &= 1, \\ -2\nu_2 \mathcal{G}_v \cos(\phi h) + (1 + 2\nu_2 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m) \mathcal{G}_v &= 1, \\ \left(-2\nu_2 + 4\nu_2 \sin^2\left(\frac{\phi h}{2}\right) + 1 + 2\nu_2 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m \right) \mathcal{G}_v &= 1. \end{aligned}$$

$$|\mathcal{G}_v| = \left| \frac{1}{4\nu_2 \sin^2\left(\frac{\phi h}{2}\right) + 1 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m} \right| < 1 \quad (3.5)$$

Moreover, by substituting the following equation:

$$\begin{aligned} W_j^m &= \mathcal{G}_w^m e^{i\phi j h}, \\ W_j^{m+1} &= \mathcal{G}_w^{m+1} e^{i\phi j h}, \\ W_{j-1}^{m+1} &= \mathcal{G}_w^{m+1} e^{i\phi(j-1)h}, \\ W_{j+1}^{m+1} &= \mathcal{G}_w^{m+1} e^{i\phi(j+1)h}, \end{aligned}$$

into Eq. (3.3), one can derive:

$$\begin{aligned} & -\nu_3 W_{j-1}^{m+1} + (1 + 2\nu_3 + \Delta tb) W_j^{m+1} - \nu_3 W_{j+1}^{m+1} = W_j^m + \Delta ts V_j^m, \\ & -\nu_3 \mathcal{G}_w^{m+1} e^{i\phi(j-1)h} + (1 + 2\nu_3 + \Delta tb) \mathcal{G}_w^{m+1} e^{i\phi j h} - \nu_3 \mathcal{G}_w^{m+1} e^{i\phi(j+1)h} = \mathcal{G}_w^m e^{i\phi j h}. \end{aligned}$$

The term $\mathcal{G}_w^m e^{i\phi j h}$ is employed into the above equation for division, one gets:

$$\begin{aligned} & -\nu_3 \mathcal{G}_w e^{-i\phi h} + (1 + 2\nu_3 + \Delta tb) \mathcal{G}_w - \nu_3 \mathcal{G}_w e^{i\phi h} = 1, \\ & -\nu_3 \mathcal{G}_w \left(e^{-i\phi h} + e^{i\phi h} \right) + (1 + 2\nu_3 + \Delta tb) \mathcal{G}_w = 1, \\ & -2\nu_3 \mathcal{G}_w \cos(\phi h) + (1 + 2\nu_3 + \Delta tb) \mathcal{G}_w = 1, \\ & \left(-2\nu_3 + 4\nu_3 \sin^2 \left(\frac{\phi h}{2} \right) + 1 + 2\nu_3 + \Delta tb \right) \mathcal{G}_w = 1. \end{aligned}$$

$$|\mathcal{G}_w| = \left| \frac{1}{4\nu_3 \sin^2 \left(\frac{\phi h}{2} \right) + 1 + \Delta tb} \right| < 1 \quad (3.6)$$

3.3 Positivity of numerical scheme

The positivity property of numerical scheme was introduced in [2] by conducting M -matrix theory [20]. The positivity of numerical scheme has important role in mathematical epidemiology to provide their behavior. In this paper, the dynamical system consists of three sub populations namely susceptible (U), infected (V), and recovered (W) sub populations. Then for each time $t > 0$, $U(x, t) > 0$, $V(x, t) > 0$, and $W(x, t) > 0$.

Theorem 8. *For all $k = 1, 2, 3, \dots$ indicating the time period of dynamical system, then the systems (2.1)-(2.3) provide the positivity, i.e., $U^k > 0$, $V^k > 0$, and $W^k > 0$.*

Proof. We firstly rewrite the dynamical system (2.1)-(2.3) into the following matrices

$$\begin{aligned} \mathcal{A}U^{k+1} &= U^k, \\ \mathcal{B}V^{k+1} &= V^k, \\ \mathcal{C}W^{k+1} &= W^k. \end{aligned} \quad (3.7)$$

The square matrices of \mathcal{A} , \mathcal{B} , and \mathcal{C} are:

$$\mathcal{A} = \begin{pmatrix} a_3 & a_1 & 0 & \cdots & \cdots & \cdots & \cdots & 0 \\ a_4 & a_3 & a_2 & \ddots & & & & \vdots \\ 0 & a_4 & a_3 & a_2 & \ddots & & & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & & \ddots & a_4 & a_3 & a_2 & 0 \\ \vdots & & & \ddots & a_4 & a_3 & a_2 \\ 0 & \cdots & \cdots & \cdots & 0 & a_5 & a_3 \end{pmatrix}, \quad (3.8)$$

$$\mathcal{B} = \begin{pmatrix} b_3 & b_1 & 0 & \cdots & \cdots & \cdots & \cdots & 0 \\ b_4 & b_3 & b_2 & \ddots & & & & \vdots \\ 0 & b_4 & b_3 & b_2 & \ddots & & & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & & \vdots \\ \vdots & & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & & & \ddots & b_4 & b_3 & b_2 & 0 \\ \vdots & & & & \ddots & b_4 & b_3 & b_2 \\ 0 & \cdots & \cdots & \cdots & \cdots & 0 & b_5 & b_3 \end{pmatrix}, \quad (3.9)$$

$$\mathcal{C} = \begin{pmatrix} c_3 & c_1 & 0 & \cdots & \cdots & \cdots & \cdots & 0 \\ c_4 & c_3 & c_2 & \ddots & & & & \vdots \\ 0 & c_4 & c_3 & c_2 & \ddots & & & \vdots \\ \vdots & \ddots & \ddots & \ddots & \ddots & \ddots & & \vdots \\ \vdots & & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots \\ \vdots & & & \ddots & c_4 & c_3 & c_2 & 0 \\ \vdots & & & & \ddots & c_4 & c_3 & c_2 \\ 0 & \cdots & \cdots & \cdots & \cdots & 0 & c_5 & c_3 \end{pmatrix}, \quad (3.10)$$

where

$$\begin{aligned} a_1 = a_5 &= -2\nu_1, \quad a_2 = a_4 = -\nu_1, \quad a_3 = 1 + 2\nu_1 + \Delta t d + \Delta t(1 - u_1)\beta V_j^m - \Delta t u_2, \\ b_1 = b_5 &= -2\nu_2, \quad b_2 = b_4 = -\nu_2, \quad b_3 = 1 + 2\nu_2 + \Delta t(s + a) - \Delta t(1 - u_1)\beta U_j^m, \\ c_1 = c_5 &= -2\nu_3, \quad c_2 = c_4 = -\nu_3, \quad c_3 = 1 + 2\nu_3 + \Delta t b. \end{aligned}$$

Moreover, the column matrices of U^k, V^k , and W^k are:

$$\begin{aligned} U^k &= U_j^m + \Delta t \lambda, \\ V^k &= V_j^m, \\ W^k &= W_j^m + \Delta t s V_j^m + \Delta t u_2 U_j^m. \end{aligned}$$

Here, M -matrix consists of the matrices \mathcal{A}, \mathcal{B} , and \mathcal{C} . Therefore, the above equations become

$$\begin{aligned} U^{k+1} &= \mathcal{A}^{-1} U^k, \\ V^{k+1} &= \mathcal{B}^{-1} V^k, \\ W^{k+1} &= \mathcal{C}^{-1} W^k. \end{aligned} \quad (3.11)$$

We firstly suppose that U^k, V^k and W^k are positive. Further step, by conducting the M -matrix criterion, the positivity of U^{k+1}, V^{k+1} and W^{k+1} are satisfied. In view of mathematical induction principle, the proof of theorem is achieved. \square

3.4 Consistency of numerical scheme

The consistency of the non-standard finite difference indicates how close the numerical scheme and the system of differential equations are, where the system of the numerical scheme is firstly approximated by Taylor's series as in [15, 39, 30].

Theorem 9. Given a system of differential equations $\mathcal{P}g = f$ and finite difference scheme $\mathcal{P}_{\Delta x, \Delta t}h = f$. Then, the finite difference scheme is consistent with the system of differential equations if for any smooth function $\phi(x, t)$ satisfies

$$\mathcal{P}_{\Delta x, \Delta t}\phi - \mathcal{P}\phi \rightarrow 0 \text{ as } (\Delta x, \Delta t) \rightarrow 0.$$

Proof. It follows from (2.2), the operator \mathcal{P} can be written as follows

$$\mathcal{P}\phi(x, t) = \frac{\partial}{\partial t} - D_i \frac{\partial^2}{\partial x^2}, \quad \text{where } i = 1, 2, 3, \text{ and } \phi(x, t) = (U(x, t), V(x, t), W(x, t)). \quad (3.12)$$

Moreover, we provide that

$$\mathcal{P}_{\Delta x, \Delta t}\phi(x, t) = \frac{\phi_j^{m+1} - \phi_j^m}{\Delta t} - D_i \frac{\phi_{j-1}^{m+1} - 2\phi_j^{m+1} + \phi_{j+1}^{m+1}}{\Delta x^2}, \quad \text{where } i = 1, 2, 3, \text{ and } \phi(x, t) = (U(x_j, t_m), V(x_j, t_m), W(x_j, t_m)). \quad (3.13)$$

By conducting (3.12)-(3.13), let us define that

$$\begin{aligned} U_j^{m+1} &= U(x_j, t_m) + \frac{\Delta t}{1!} U_t(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{tt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{ttt}(x_j, t_m) + \dots \\ U_{j-1}^{m+1} &= U(x_j, t_{m+1}) - \frac{\Delta x}{1!} U_x(x_j, t_{m+1}) + \frac{(\Delta x)^2}{2!} U_{xx}(x_j, t_{m+1}) - \frac{(\Delta x)^3}{3!} U_{xxx}(x_j, t_{m+1}) \\ &\quad + \frac{(\Delta x)^4}{4!} U_{xxxx}(x_j, t_{m+1}) - \dots \\ &= \left(U(x_j, t_m) + \frac{\Delta t}{1!} U_t(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{tt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{ttt}(x_j, t_m) + \dots \right) \\ &\quad - \frac{\Delta x}{1!} \left(U_x(x_j, t_m) + \frac{\Delta t}{1!} U_{xt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{xttt}(x_j, t_m) + \dots \right) \\ &\quad + \frac{(\Delta x)^2}{2!} \left(U_{xx}(x_j, t_m) + \frac{\Delta t}{1!} U_{xxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xxtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{xxttt}(x_j, t_m) + \dots \right) \\ &\quad - \dots \end{aligned}$$

$$\begin{aligned}
U_{j+1}^{m+1} &= U(x_j, t_{m+1}) + \frac{\Delta x}{1!} U_x(x_j, t_{m+1}) + \frac{(\Delta x)^2}{2!} U_{xx}(x_j, t_{m+1}) + \frac{(\Delta x)^3}{3!} U_{xxx}(x_j, t_{m+1}) \\
&\quad + \frac{(\Delta x)^4}{4!} U_{xxxx}(x_j, t_{m+1}) + \dots \\
&= \left(U(x_j, t_m) + \frac{\Delta t}{1!} U_t(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{tt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{ttt}(x_j, t_m) + \dots \right) \\
&\quad + \frac{\Delta x}{1!} \left(U_x(x_j, t_m) + \frac{\Delta t}{1!} U_{xt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{xttt}(x_j, t_m) + \dots \right) \\
&\quad + \frac{(\Delta x)^2}{2!} \left(U_{xx}(x_j, t_m) + \frac{\Delta t}{1!} U_{xxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xxtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{xxttt}(x_j, t_m) + \dots \right) \\
&\quad + \dots
\end{aligned}$$

By substituting the above Taylor series into the following discretization of U

$$\frac{U_j^{m+1} - U_j^m}{\Delta t} - D_1 \frac{U_{j-1}^{m+1} - 2U_j^{m+1} + U_{j+1}^{m+1}}{(\Delta x)^2} - \lambda + dU_j^{m+1} + (1 - u_1)\beta U_j^{m+1} V_j^m + u_2 U_j^{m+1} = 0,$$

one has:

$$\begin{aligned}
&\left(U_t(x_j, t_m) + \frac{\Delta t}{2!} U_{tt}(x_j, t_m) + \frac{(\Delta t)^2}{3!} U_{ttt}(x_j, t_m) + \frac{(\Delta t)^3}{4!} U_{tttt}(x_j, t_m) + \dots \right) \\
&- D_1 \left(U_{xx}(x_j, t_m) + \frac{\Delta t}{1!} U_{xxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xxtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{xxttt}(x_j, t_m) + \dots \right) \\
&- \frac{D_1 (\Delta x)^2}{12} \left(U_{xxxx}(x_j, t_m) + \frac{\Delta t}{1!} U_{xxxxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xxxxtt}(x_j, t_m) + \dots \right) - \dots \\
&- \lambda + (d + (1 - u_1)\beta V(x_j, t_m) + u_2) \left(U(x_j, t_m) + \frac{\Delta t}{1!} U_t(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{tt}(x_j, t_m) + \dots \right) = 0.
\end{aligned}$$

By employing the limit $(\Delta x, \Delta t) \rightarrow 0$. Then, the following terms become

$$\left[\begin{array}{l} \left(\frac{\Delta t}{2!} U_{tt}(x_j, t_m) + \frac{(\Delta t)^2}{3!} U_{ttt}(x_j, t_m) + \frac{(\Delta t)^3}{4!} U_{tttt}(x_j, t_m) + \dots \right) \\ - D_1 \left(\frac{\Delta t}{1!} U_{xxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xxtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} U_{xxttt}(x_j, t_m) + \dots \right) \\ - \frac{D_1 (\Delta x)^2}{12} \left(U_{xxxx}(x_j, t_m) + \frac{\Delta t}{1!} U_{xxxxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{xxxxtt}(x_j, t_m) + \dots \right) \\ + (d + (1 - u_1)\beta V(x_j, t_m) + u_2) \left(\frac{\Delta t}{1!} U_t(x_j, t_m) + \frac{(\Delta t)^2}{2!} U_{tt}(x_j, t_m) + \dots \right) \end{array} \right] \rightarrow 0.$$

Since

$$\frac{\partial U}{\partial t} = U_t(x_j, t_m), \quad \frac{\partial U}{\partial x} = U_x(x_j, t_m), \quad \frac{\partial^2 U}{\partial x^2} = U_{xx}(x_j, t_m), \quad \frac{\partial^2 U}{\partial x \partial t} = U_{xt}(x_j, t_m), \dots$$

one can derive

$$\begin{aligned}
&\frac{U_j^{m+1} - U_j^m}{\Delta t} - D_1 \frac{U_{j-1}^{m+1} - 2U_j^{m+1} + U_{j+1}^{m+1}}{(\Delta x)^2} - \lambda + dU_j^{m+1} + (1 - u_1)\beta U_j^{m+1} V_j^m + u_2 U_j^{m+1} \\
&- \left(\frac{\partial U}{\partial t} - D_1 \frac{\partial^2 U}{\partial x^2} - \lambda + (d + (1 - u_1)\beta V + u_2) U \right) \rightarrow 0 \text{ as } (\Delta x, \Delta t) \rightarrow 0.
\end{aligned} \tag{3.14}$$

Thus, the scheme of compartment U is consistent. Moreover, the consistency is also achieved for the schemes of compartments V and W by using the similar ways. Then one has

$$\left[\begin{array}{l} \left(\frac{\Delta t}{2!} V_{tt}(x_j, t_m) + \frac{(\Delta t)^2}{3!} V_{ttt}(x_j, t_m) + \frac{(\Delta t)^3}{4!} V_{tttt}(x_j, t_m) + \dots \right) \\ - D_2 \left(\frac{\Delta t}{1!} V_{xxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} V_{xxtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} V_{xxttt}(x_j, t_m) + \dots \right) \\ - \frac{D_2(\Delta x)^2}{12} \left(V_{xxxx}(x_j, t_m) + \frac{\Delta t}{1!} V_{xxxxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} V_{xxxxtt}(x_j, t_m) + \dots \right) \\ + ((s+a) - (1-u_1)\beta U(x_j, t_m)) \left(\frac{\Delta t}{1!} V_t(x_j, t_m) + \frac{(\Delta t)^2}{2!} V_{tt}(x_j, t_m) + \dots \right) \end{array} \right] \rightarrow 0,$$

and

$$\left[\begin{array}{l} \left(\frac{\Delta t}{2!} W_{tt}(x_j, t_m) + \frac{(\Delta t)^2}{3!} W_{ttt}(x_j, t_m) + \frac{(\Delta t)^3}{4!} W_{tttt}(x_j, t_m) + \dots \right) \\ - D_3 \left(\frac{\Delta t}{1!} W_{xxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} W_{xxtt}(x_j, t_m) + \frac{(\Delta t)^3}{3!} W_{xxttt}(x_j, t_m) + \dots \right) \\ - \frac{D_3(\Delta x)^2}{12} \left(W_{xxxx}(x_j, t_m) + \frac{\Delta t}{1!} W_{xxxxt}(x_j, t_m) + \frac{(\Delta t)^2}{2!} W_{xxxxtt}(x_j, t_m) + \dots \right) \\ + b \left(\frac{\Delta t}{1!} W_t(x_j, t_m) + \frac{(\Delta t)^2}{2!} W_{tt}(x_j, t_m) + \dots \right) \end{array} \right] \rightarrow 0,$$

implying that

$$\begin{aligned} & \frac{V_j^{m+1} - V_j^m}{\Delta t} - D_2 \frac{V_{j-1}^{m+1} - 2V_j^{m+1} + V_{j+1}^{m+1}}{(\Delta x)^2} + (s+a)V_j^{m+1} - (1-u_1)\beta U_j^m V_j^{m+1} \\ & - \left(\frac{\partial V}{\partial t} - D_2 \frac{\partial^2 V}{\partial x^2} + ((s+a) - (1-u_1)\beta U)V \right) \rightarrow 0 \quad \text{as } (\Delta x, \Delta t) \rightarrow 0. \end{aligned} \quad (3.15)$$

and

$$\begin{aligned} & \frac{W_j^{m+1} - W_j^m}{\Delta t} - D_3 \frac{W_{j-1}^{m+1} - 2W_j^{m+1} + W_{j+1}^{m+1}}{(\Delta x)^2} + bW_j^{m+1} - sV_j^m - u_2 U_j^m \\ & - \left(\frac{\partial W}{\partial t} - D_3 \frac{\partial^2 W}{\partial x^2} + bW - sV - u_2 U \right) \rightarrow 0 \quad \text{as } (\Delta x, \Delta t) \rightarrow 0. \end{aligned} \quad (3.16)$$

Based on the results in Eqs. (3.14), (3.15), (3.16), we can conclude that $(\mathcal{P}_{\Delta x, \Delta t} - \mathcal{P})\phi(x, t) \rightarrow 0$ as $(\Delta x, \Delta t) \rightarrow 0$ for any smooth function $\phi(x, t)$. 54 □

3.5 Example of numerical scheme

In this section, we perform the example of simulation for the discretization results of HID-AIDS model. The following parameters are all assumed

$$\begin{aligned} & \lambda = 10, \beta = 0.002, d = 0.02, a = 0.24, s = 0.2, b = 0.02, \\ & D_1 = 0.01, D_2 = 0.01, D_3 = 0.01. \end{aligned}$$

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Moreover, the initial conditions in (2.2) and the combinations of two treatments (HAART, Immunotherapy) are given below

$$\begin{aligned} & \text{28} \quad U(x, 0) = 20, V(x, 0) = 20, W(x, 0) = 2, \\ & \text{33} \quad (u_1 = 0; u_2 = 0), (u_1 = 0.5; u_2 = 0), (u_1 = 0; u_2 = 0.001), (u_1 = 0.5; u_2 = 0.001). \end{aligned}$$

Table 1: Basic reproduction number for varying values of u_1 and u_2

u_2 (fixed $u_1 = 0.001$)	\mathcal{R}_0	u_1 (fixed $u_2 = 0.001$)	\mathcal{R}_0
0.001	3.9643	0.001	3.9643
0.01	2.7750	0.01	3.9286
0.1	0.6937	0.1	3.5714
0.5	0.1601	0.5	1.9841
0.7	0.1156	0.7	1.1905

All basic reproduction numbers (\mathcal{R}_0) in Figures 1, 2, and 3 are in the endemic stage with the values of reproduction numbers respectively $\mathcal{R}_0 = 4.1667; 2.0833; 1.9841$. Based on those three basic reproduction numbers, we can see that the smallest basic reproduction number is $\mathcal{R}_0 = 1.9841$ with two treatments at once (HAART and Immunotherapy). Then, followed by the second small basic reproduction number $\mathcal{R}_0 = 2.0833$ with only one treatment of HAART, and the biggest basic reproduction number is achieved with $\mathcal{R}_0 = 4.1667$ without any treatments. From these all experiments, we can conclude that the two combinations of treatment are the most effective which can be represented in Figure 4.

The susceptible, infected, and recovered sub populations are respectively represented in blue, green, and red in Figure 4. Moreover, Figure 4a represents the comparisons between sub population without HAART and Immunotherapy (solid line) and sub population only with HAART. The comparisons between sub population without HAART and Immunotherapy (solid line) and sub population with HAART and Immunotherapy at once (dotted line) are shown in Figure 4b. Figure 4b provides the comparisons between sub population only with HAART (dashed line) and sub population with HAART and Immunotherapy at once (dotted line). Moreover, the only one treatment of HAART is less efficient in decreasing the spread of infected sub population. The only one treatment of Immunotherapy provides the basic reproduction number $\mathcal{R}_0 = 3.9683$ which is bigger than $\mathcal{R}_0 = 2.0833$ (treatment of only HAART). If we compare between HAART and Immunotherapy based on those two basic reproduction numbers, we can conclude that the treatment of HAART is more effective than the treatment of Immunotherapy.

The disease-free stage can be obtained by giving the value of HAART ($u_1 = 0.5$) and Immunotherapy ($u_2 = 0.1$) with the basic reproduction number $\mathcal{R}_0 = 0.3472 < 1$. The result of disease-free stage can be seen in Figure 5. Based on the basic reproduction number stated in Theorem 1, the treatments of HAART (u_1) and immunotherapy (u_2) have significant role to make the value of \mathcal{R}_0 smaller, i.e., the higher the values of u_1, u_2 are, the smaller the value of \mathcal{R}_0 is. The varying values of $u_1 = u_2 = 0.001; 0.01; 0.1; 0.5; 0.7$ are listed in Table 1 and represented in Figures (6a)-(6b). If we correlate Table 1 and Figures (6a)-(6b), we can conclude that the increasing number of treatments with Immunotherapy causes the CD4⁺T cell infection rate to decrease (this is indicated by sloping V profile). The more sloping V profile indicates the smaller the basic reproduction number as well. Moreover, based on the results in Table 1 and Figures (6a)-(6b), by conducting the same varying values of u_1 and u_2 , then the immunotherapy (u_2) is more effective than HAART (u_1) in reducing the infected CD4⁺T cell, indicated by the decrease of basic reproduction number (by immunotherapy) is faster than the decrease of basic reproduction number (by HAART). This case is in line with the formula of basic reproduction number, where the parameter of immunotherapy (u_2) is as a divider in that formula.

Moreover, we validate our dynamical system with the real data by conducting the classical

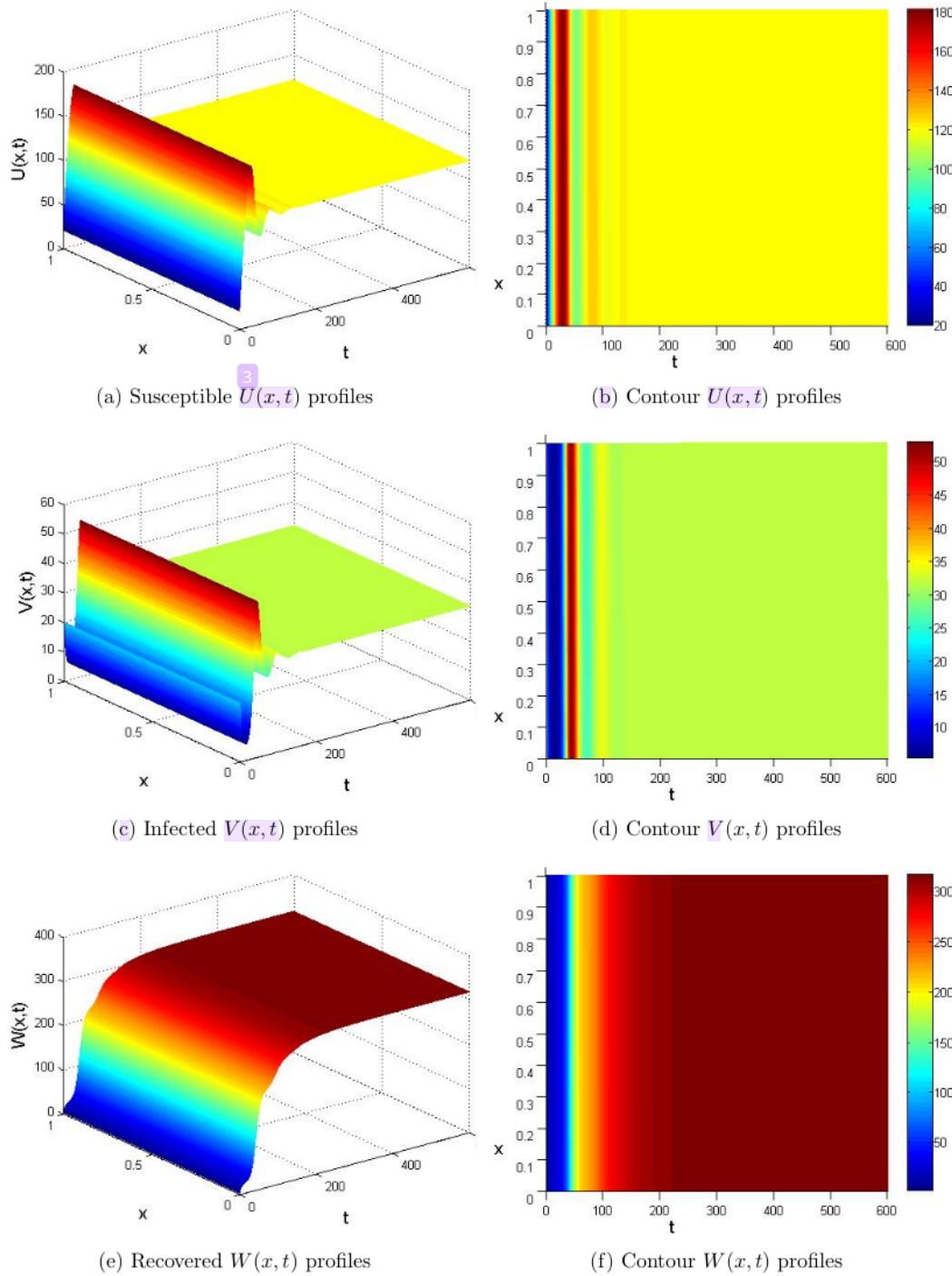


Figure 1: Profiles of $(U, V, W)(x, t)$ for $u_1 = 0, u_2 = 0$

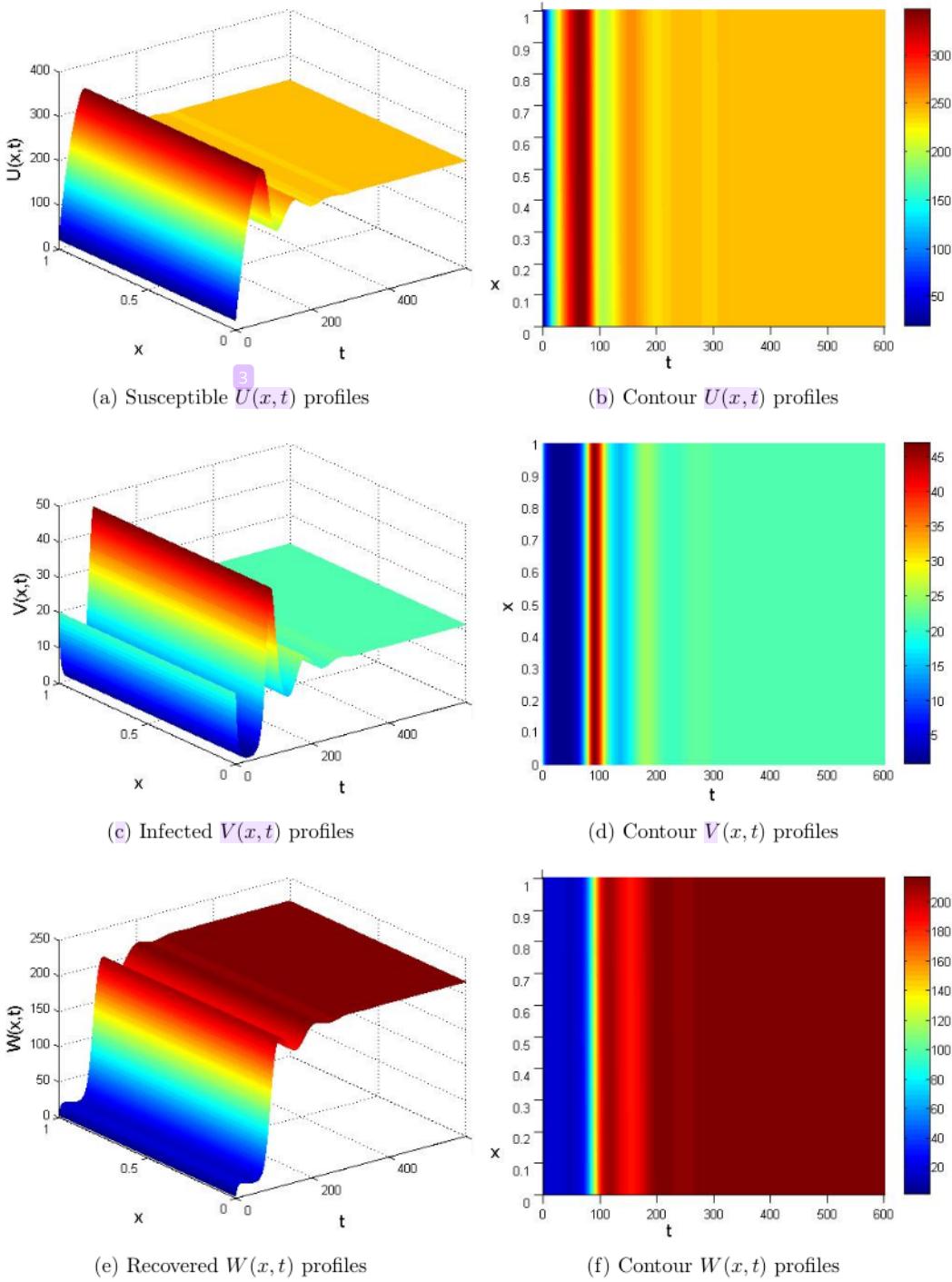


Figure 2: Profiles of $(U, V, W)(x, t)$ for $u_1 = 0.5, u_2 = 0$

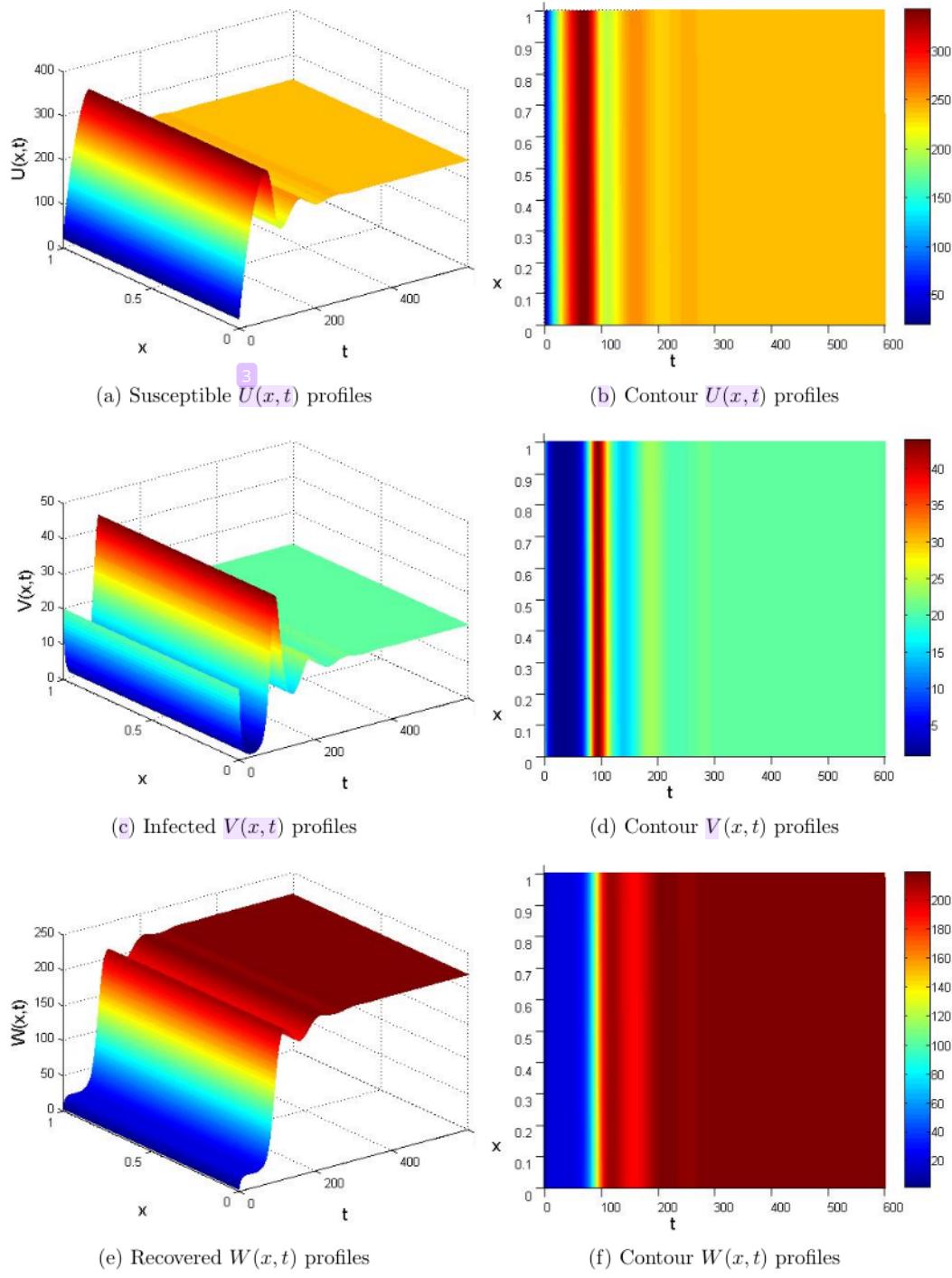
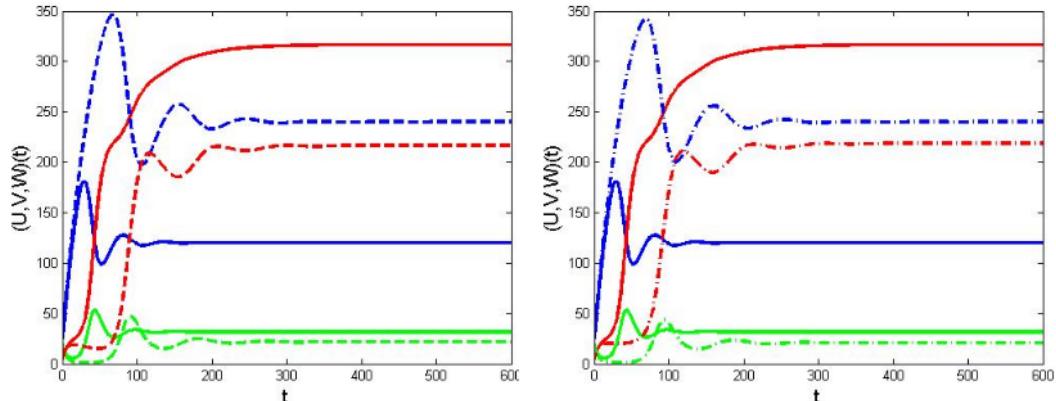
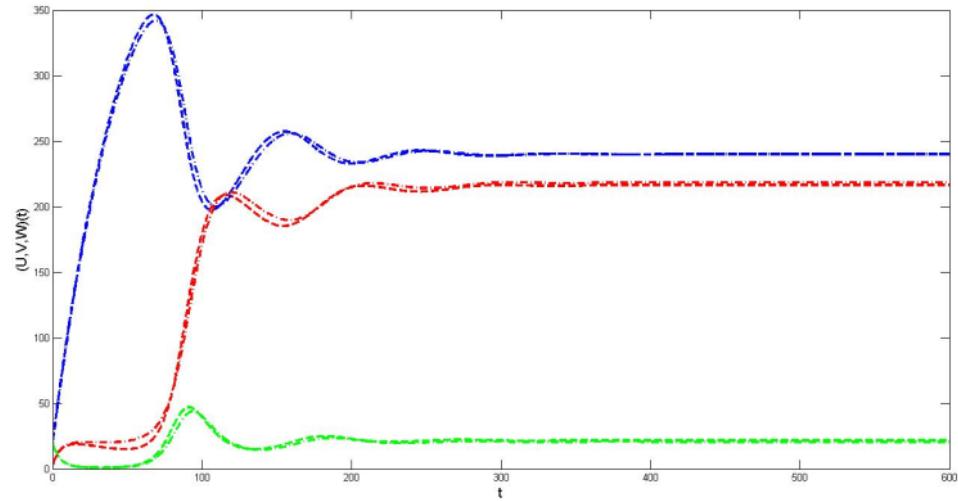


Figure 3: Profiles of $(U, V, W)(x, t)$ for $u_1 = 0.5, u_2 = 0.001$



(a) $u_1 = 0, u_2 = 0$ (solid line) versus $u_1 = 0.5, u_2 = 0$ (b) $u_1 = 0, u_2 = 0$ (solid line) versus $u_1 = 0.5, u_2 = 0.001$ (dashed line)



(c) $u_1 = 0.5, u_2 = 0$ (dashed line) versus $u_1 = 0.5, u_2 = 0.001$ (dotted line)

Figure 4: Profiles of $(U, V, W)(t)$ for varying values u_1 and u_2 (susceptible: blue, infected: green, recovered: red)

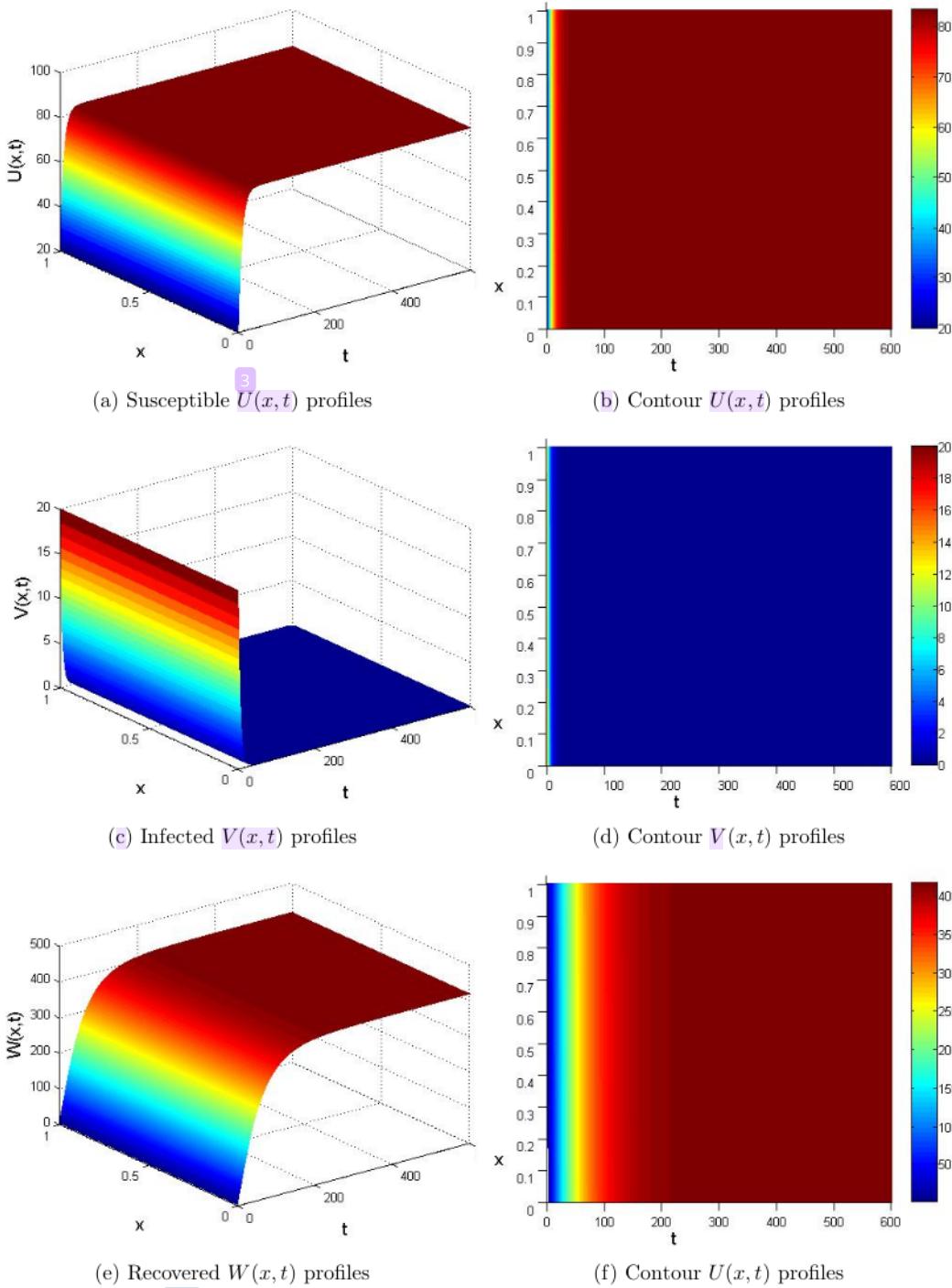
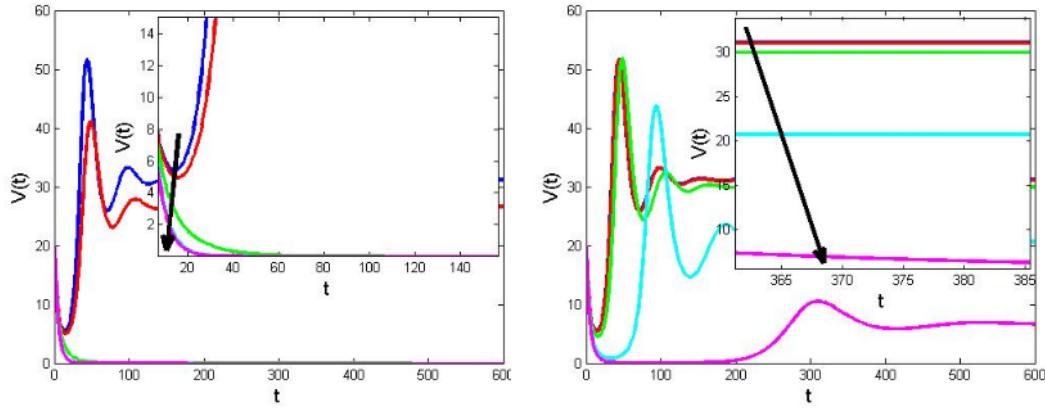


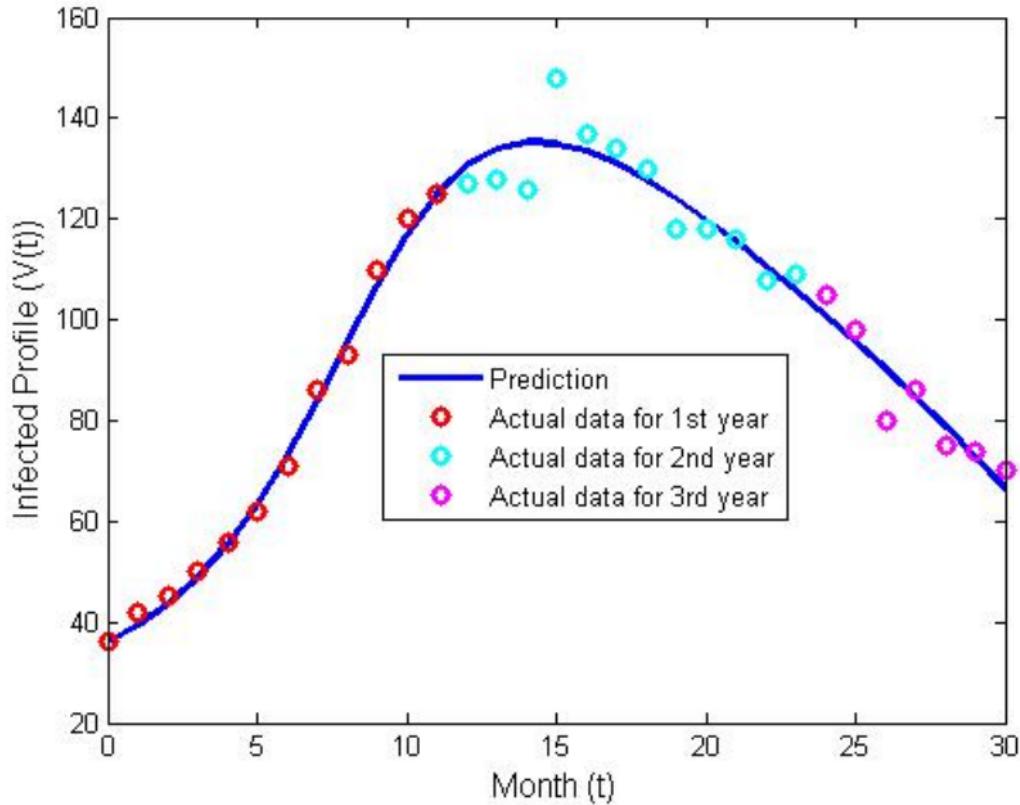
Figure 5: Profiles of $(U, V, W)(x, t)$ for $u_1 = \bar{0.5}$ and $u_2 = 0.1$

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(a) varying u_2 and fixed $u_1 = 0.001$

(b) varying u_1 and fixed $u_2 = 0.001$



(c) Infected profile (prediction versus actual data)

Figure 6: (6a)-(6b) Infected CD4⁺T cell for varying immunotherapy and HAART, (6c) predicted result and actual data of HIV-AIDS for 2 years 7 months

formula, least square technique written as

$$RMSE(\mathcal{N}) = \sum_{k=1}^M (\mathcal{Y}_{pred}(k) - \mathcal{Y}_{data}(k)), \quad (3.17)$$

where M is the number of real data, \mathcal{Y}_{pred} is the numerical result of our dynamical system, \mathcal{Y}_{data} is the real data of HIV-AIDS taken for 2 years 7 months, and \mathcal{N} is the unknown parameters of our dynamical system in (2.2). Our dynamical system in (2.2) is represented as follows

$$\frac{\partial \mathcal{Y}(x, t)}{\partial t} = \frac{\partial \mathcal{Y}(x, t)}{\partial x^2} + \mathcal{F}(x, t, \mathcal{Y}, \mathcal{N}), \quad (3.18)$$

where Eq. (3.18) is approximated by the non-standard finite difference. Our goal is to minimize the objective function

$$\min_{\mathcal{N}} RMSE(\mathcal{N}), \quad (3.19)$$

subject to Eq. (3.18).

The more detailed algorithm of parameter estimation can be addressed in [38] and the algorithm of optimization is in [24]. We divide the real data into three regions as represented in Figure 6c, where the largest infected profile is achieved in the region of 2nd year. The trend of infected profile for 2 years 7 months, it is initially increased in the region of 1st year and finally decreased in the region of 3rd year. The simulation results with the finite difference scheme (blue) are closed enough to the real data (red, cyan, magenta). This indicates that our dynamical system model is reliable enough to be used in predicting the spread of HIV-AIDS.

4 Conclusions

In this paper, we study the spatio-temporal HIV-AIDS model, where the change of system not only in time but also in space. The equilibrium points for disease-free and endemic are obtained by considering the diffusion parameters of D_1, D_2 and D_3 equal to zero. This condition also applies in providing the basic reproduction number (\mathcal{R}_0). The local stability of disease-free and endemic equilibrium points are established by definition of Fourier series, where the aim of using this Fourier series is because of the diffusion term. Moreover, we employ the non-standard finite difference (NSFD) to approximate our model into the numerical steps. The stability criterion of NSFD in our proposed model is established based on the standard way of using Von-Neumann stability. We can see that the stability criterion for NSFD of our model is less than 1, i.e., $|\mathcal{G}_u, \mathcal{G}_v, \mathcal{G}_w| < 1$. Moreover, the M -matrix theory is employed to provide the positivity of numerical scheme of our model. This technique only makes sure that there is no any negative population, i.e., susceptible, infected, and recovered sub populations. Meanwhile, knowing how close the numerical scheme of NSFD and the system of differential equations are, then consistency are employed. The experiment section is provided to know the effectiveness between HAART and Immunotherapy in reducing the infected CD4⁺T cells. Based on the results, we can conclude that the treatment of HAART and Immunotherapy at once is the most efficient in decreasing the infected CD4⁺T cells with the basic reproduction number ($\mathcal{R}_0 = 1.9841$). Moreover, based on the formula of basic reproduction number, the treatments of HAART and immunotherapy have significant role in reducing the infected CD4⁺T cells, i.e., the higher the values of u_1 and u_2

are, the smaller the value of \mathcal{R}_0 is. By conducting the least square technique to our dynamical system, then our dynamical system is eligible to predict the spread of HIV-AIDS based on the validation results with the actual data.

75 Data availability

Data will be made available on request.

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