Research Article

Numerical Analysis for a Fractional Differential Time-Delay Model of HIV Infection of CD4⁺ T-Cell Proliferation under Antiretroviral Therapy

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We study a fractional differential model of HIV infection of CD4⁺ T-cell, in which the CD4⁺ T-cell proliferation plays an important role in HIV infection under antiretroviral therapy. An appropriate method is given to ensure that both the equilibria are asymptotically stable for $\tau \geq 0$. We calculate the basic reproduction number R_0 , the IFE E_0 , two IPEs E_1^* and E_2^* , and so on, and judge the stability of the equilibrium. In addition, we describe the dynamic behaviors of the fractional HIV model by using the Adams-type predictor-corrector method algorithm. At last, we extend the model to incorporate the term which we consider the loss of virion and a bilinear term during attacking the target cells.

1. Introduction

Human immunodeficiency virus (HIV) is a lentivirus (a member of the retrovirus family) that causes acquired immunodeficiency syndrome (AIDS) [1], a condition in humans in which the immune system begins to fail, leading to life-threatening opportunistic infections. HIV infects primarily vital cells in the human immune system such as helper T-cell (to be specific, CD4⁺ T-cell), macrophages, and dendritic cells. When CD4⁺ T-cell numbers decline below a critical level, cell-mediated immunity is lost and the body becomes progressively more susceptible to opportunistic infections (see [2]).

There are only a few results for dynamics of HIV infection of CD4⁺ T-cell. In 1992, Perelson et al. [3] examined a model for the interaction of HIV with CD4⁺ T-cell who considered four populations: uninfected T-cell, latently infected T-cell, actively infected T cells, and free virus, and they also considered effects of AZT on viral growth and T-cell population dynamics. In 2000, Culshaw and Ruan [4] firstly simplified their model into one consisting of only three components: the healthy CD4⁺ T-cell, infected CD4⁺ T-cell, and free virus

and discussed the existence and stability of the infected steady state, and they studied the effect of the time delay on the stability of the endemically infected equilibrium; criteria were given to ensure that the infected equilibrium is asymptotically stable for all delay.

For backward bifurcations in other disease models, we refer the reader to [5–12]. In [12], this paper analyzed the backward bifurcation sources and application in infectious disease model. HIV/AIDS infection model is a special case of infectious disease model. For recent work on global analysis and persistence of HIV models, we refer the reader to [13–18] and references therein. A discussion on HIV infection and CD4⁺ T-cell depletion is given in the review paper [19].

In 2012, Shu and Wang [12] considered a new model frame that included full logistic growth terms of both healthy and infected CD4⁺ T-cell:

$$\begin{aligned}\frac{d}{dt}T(t) &= s - \mu_1 T(t) + r \frac{T(t) V_I(t)}{C + V_I(t)} - k(1 - n_{rt}) V_I(t) T(t), \\ \frac{d}{dt}I^i(t) &= k(1 - n_{rt}) V_I(t) T(t) - \mu_2 T^i(t),\end{aligned}$$

$$\begin{aligned}\frac{d}{dt}V_I(t) &= (1 - n_p)N\mu_2T^i(t) - \mu_3V_I(t), \\ \frac{d}{dt}V_{NI}(t) &= n_pN\mu_2T^i(t) - \mu_3V_{NI}(t).\end{aligned}\quad (1)$$

Fractional differential equations arise in many engineering and scientific disciplines as the mathematical modeling of systems and processes in various fields, such as physics, mechanics, chemical technology, population dynamics, biotechnology, and economics (see e.g., [20–26]). As one of the important topics in the research differential equations, the boundary value problem has attained a great deal of attention (see [27–41] and the references therein).

Many mathematicians and researchers in the field of application are trying to model fractional order differential equations. In biology, the researchers found that biological membranes with fractional order have the nature of electronic conductivity, so it can be classified as a model of the fractional order. Because of the memory property of fractional calculus, we introduce the fractional calculus into HIV model. Both in mathematics and biology, fractional calculus will correspond with objective reality more than ODE. It is particularly important for us to study fractional HIV model.

Furthermore, delay plays an important role in the process of spreading infectious diseases; it can be used to simulate the incubation period of infectious diseases, the period of patients infected with disease, period of patients immune to disease, and so on. The basic fact reflected by the specific mathematical model with time delay is that the change of trajectory about time t not only depends on the t moment itself but also is affected by some certain conditions before, even the reflection of some certain factors before. This kind of circumstance is abundant in the objective world.

Recently, Yan and Kou [2] have introduced fractional-order derivatives into a model of HIV infection of $CD4^+$ T-cell with time delay:

$$\begin{aligned}D^\alpha T(t) &= s - \mu_T T(t) + rT(t) \left(1 - \frac{T(t) + I(t)}{T_{\max}}\right) \\ &\quad - k_1 T(t) V(t), \\ D^\alpha I(t) &= k_1' T(t - \tau) V(t - \tau) - \mu_I I(t), \\ D^\alpha V(t) &= N\mu_b I(t) - k_1 T(t) V(t) - \mu_v V(t),\end{aligned}\quad (2)$$

with the initial conditions:

$$T(\theta) = T_0, \quad I(0) = 0, \quad V(\theta) = V_0, \quad \theta \in [-\tau, 0]. \quad (3)$$

Motivated by the works mentioned above, we will consider this model where the $CD4^+$ T-cell proliferation does play an important role in HIV infection under antiretroviral therapy; a more appropriate method is given to ensure that both equilibria are asymptotically stable for $\tau \geq 0$. We calculate the basic reproduction number R_0 , the IFE E_0 , two IPEs E_1^* and E_2^* , and so on under certain conditions and judge the stability of the equilibrium. In addition, we describe the dynamic behaviors of the fractional HIV

model by using the Adams-type predictor-corrector method algorithm. At last, we extend the model to incorporate the term which we consider the loss of virion and a bilinear term during attacking the target cells. In this paper, we establish mathematical model as follows:

$$\begin{aligned}D^\alpha T(t) &= s - \mu_1 T(t) + \frac{rT(t)V_I(t)}{C + V_I(t)} \\ &\quad - k(1 - n_{rt})T(t - \tau)V_I(t - \tau), \\ D^\alpha I(t) &= k(1 - n_{rt})T(t - \tau)V_I(t - \tau) - \mu_2 I(t), \\ D^\alpha V(t) &= (1 - n_p)N\mu_2 I(t) - \mu_3 V_I(t),\end{aligned}\quad (4)$$

with the initial conditions:

$$T(\theta) = T_0, \quad I(0) = 0, \quad V(\theta) = V_0, \quad \theta \in [-\tau, 0], \quad (5)$$

where D^α denotes Caputo's fractional derivative of order α with the lower limit zero. $T(t)$, $I(t)$, and $V(t)$ represent the concentration of healthy $CD4^+$ T-cell at time t , infected $CD4^+$ T-cell at time t , and free HIV virus particles in the blood at time t , respectively. The positive constant τ represents the length of the delay in days. A complete list of the parameter values for the model is given in Table 1.

Furthermore, we assume that $T(t) > 0$, $I(t) \geq 0$, and $V(t) \geq 0$ for all $t \geq -\tau$.

This paper is organized in the following way. In the next section, some necessary definitions and lemmas are presented. In Section 3, the stability of the equilibria is given. In Section 4, we calculate some of the data and judge the stability of the equilibrium. In Section 5, we will give the numerical simulation for the fractional HIV model. Finally, the conclusions are given.

2. Preliminaries

In this section, we introduce definitions and lemmas which will be used later.

Definition 1 (see [20, 25]). The fractional (arbitrary) order integral of the function $f : [0, \infty) \rightarrow \mathbb{R}$ of order $p > 0$ is defined by

$$I^p f(x) = \frac{1}{\Gamma(p)} \int_0^x (x-s)^{p-1} f(s) ds. \quad (6)$$

Definition 2 (see [20]). Let $\alpha \geq 0$, $n = [\alpha] + 1$, where $[\alpha]$ denotes the integer part of number α . If $f \in AC^n[a, b]$, the Caputo fractional derivative of order α of f is defined by

$$\begin{aligned}{}^c D^\alpha f(t) &= \frac{1}{\Gamma(n-\alpha)} \int_a^t \frac{f^{(n)}(s)}{(t-s)^{\alpha+1-n}} ds, \\ t &> 0, \quad n-1 < \alpha < n.\end{aligned}\quad (7)$$

Lemma 3 (see [44]). The equilibrium point (x_{eq}, y_{eq}) of the fractional differential system:

$$\begin{aligned}D^\alpha x(t) &= f_1(x, y), \quad D^\alpha y(t) = f_2(x, y), \quad \alpha \in (0, 1], \\ x(0) &= x_0, \quad y(0) = y_0\end{aligned}\quad (8)$$

TABLE 1: Parameters and values of model (4).

Parameter	Description	Value
T	Uninfected $CD4^+$ T-cell population size	1000 mm^{-3}
I	Infected $CD4^+$ T-cell density	0
V_I	Free infectious virus particles	10^{-3} mm^{-3}
V_{NI}	Noninfectious virus particles	10^{-3} mm^{-3}
T_0	$CD4^+$ T-cell population for HIV-negative persons	1000 mm^{-3}
μ_1	Natural death rate of $CD4^+$ T-cell	0.02 day^{-1}
μ_2	Blanket death rate of infected $CD4^+$ T-cell	0.26 day^{-1}
μ_3	Death rate of free virus	2.4 day^{-1}
k	Rate of $CD4^+$ T-cell becoming infected with virus	$2.4 \times 10^{-5} \text{ mm}^3 \text{ day}^{-1}$
k'	Rate of infected cells becoming active	$2 \times 10^{-5} \text{ mm}^3 \text{ day}^{-1}$
s	Source term for uninfected $CD4^+$ T-cell	$10 \text{ day}^{-1} \text{ mm}^{-3}$
N	Number of virions produced by infected $CD4^+$ T-cell	Varies
r	The maximal proliferation rate ($r < \mu_1$)	Varies
C	The half saturation constant of the proliferation process	Varies
n_{rt}	The effectiveness of RTIs ($n_{rt} = 0$ means the therapy is totally ineffective, while $n_{rt} = 1$ indicates the therapy is 100% effective and the cell-to-cell infection is completely stopped)	Varies
n_p	The effectiveness of PIs ($n_p = 1$ meaning the therapy with PIs is 100% effective and no newly infectious virus particles will be produced [42])	Varies
$r \frac{TV_I}{C + V_I}$	The stimulation of T-cell to proliferate in the presence of virus [43]	Varies

is locally asymptotically stable if all the eigenvalues of the Jacobian matrix

$$A = \begin{pmatrix} \frac{\partial f_1}{\partial x} & \frac{\partial f_1}{\partial y} \\ \frac{\partial f_2}{\partial x} & \frac{\partial f_2}{\partial y} \end{pmatrix}, \quad (9)$$

evaluated at the equilibrium point satisfying the following condition:

$$|\arg(\text{eig}(A))| > \frac{\alpha\pi}{2}. \quad (10)$$

The stable and unstable regions for $0 < \alpha \leq 1$ are shown in Figure 1 [45, 46].

Proposition 4 (see [12]). Consider model (4).

(i) Assume that (H1): $r \leq k(1 - n_{rt})C$ is satisfied.

- (a) If $R_0 \leq 1$, then the IFE E_0 is the only equilibrium (Table 3).
- (b) If $R_0 > 1$, then there are two equilibria: the IFE E_0 and a unique IPE E^* .

(ii) Assume that (H2): $r > k(1 - n_{rt})C$ is satisfied. Let $a = (\sqrt{r} - \sqrt{k(1 - n_{rt})C})^2 / \mu_1$; then $0 < a < 1$ ($a < 1$ follows from the assumption that $r < \mu_1$).

- (a) If $R_0 < 1 - a$, then the IFE E_0 is the only equilibrium.

FIGURE 1: Stability region of system (4) with order $0 < \alpha \leq 1$.

- (b) If $1 - a < R_0 < 1$, then there are three equilibria: the IFE E_0 and two IPEs, denoted by $E_1^* = (T_1^*, T_1^i, V_{I1}^*)$ and $E_2^* = (T_2^*, T_2^i, V_{I2}^*)$ such that $V_{I2}^* > V_{I1}^*$.
- (c) If $R_0 = 1 - a$ or $R_0 \geq 1$, then there are two equilibria: the IFE E_0 and a unique IPE.

Remark 5. It is similar to [47, 48] to prove the existence of solution of the fractional delay equations, and the one without no delay time is also parallel to [29, 30].

By the next generation matrix method [11], we easily get

$$\mathcal{F} = \begin{pmatrix} k(1 - n_{rt})V_I T \\ (1 - n_p)N\mu_2 T^i \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} \mu_2 T^i \\ \mu_3 V_I \end{pmatrix}; \quad (11)$$

we obtain the basic reproduction number of model (4)

$$R_0 = \sqrt{\frac{s(1-n_p)(1-n_{rt})kN}{\mu_1\mu_3}}. \quad (12)$$

3. The Stability of the Equilibria

In this section, we investigate the existence of equilibria of system (4).

In order to find the equilibria of system (4), we put

$$\begin{aligned} s - \mu_T T(t) + \frac{rT(t)V_I(t)}{C + V_I(t)} \\ - k(1-n_{rt})T(t-\tau)V_I(t-\tau) &= 0, \\ k(1-n_{rt})T(t-\tau)V_I(t-\tau) - \mu_2 I(t) &= 0, \\ (1-N_p)N\mu_2 I(t) - \mu_3 V_I(t) &= 0. \end{aligned} \quad (13)$$

Following the analysis in [12], we get that system (13) has always the infection free equilibrium (IFE) $E_0 = ((s/\mu_1), 0, 0)$ and the infection persistent equilibrium (IPE) $E^* = (T^*, T^{i*}, V_I^*)$, where

$$\begin{aligned} T^* &= \frac{\mu_3}{(1-n_p)(1-n_{rt})Nk}, \quad I^* = \frac{\mu_3}{(1-n_p)N\mu_2}V_I^*, \\ g(V_I) &= V_I^2 + \left(p - \frac{r}{k(1-n_{rt})} + C\right)V_I + pC, \\ p &= \frac{\mu_1}{(k(1-n_{rt}))(1-R_0)}. \end{aligned} \quad (14)$$

Next, we will discuss the stability for the local asymptotic stability of the viral free equilibrium E_0 and the infected equilibrium E^* .

For the local asymptotic stability of the viral free equilibrium E_0 , we have the following result.

Lemma 6. *If $R_0 < 1$, then E_0 is locally asymptotically stable for $\tau \geq 0$. If $R_0 = 1$, then E_0 is locally stable for $\tau \geq 0$. If $R_0 > 1$, then E_0 is a saddle point with a two-dimensional stable manifold and a one-dimensional unstable manifold.*

Proof. The associated transcendental characteristic equation at $E_0 = (T_0, 0, 0)$ is given by

$$\begin{aligned} (\lambda + \mu_1) \left[\lambda^2 + (\mu^2 + \mu_3)\lambda + \mu_2\mu_3 - k(1-n_{rt}) \right. \\ \left. \times (1-n_p)N\mu_2 T_0 e^{-\lambda\tau} \right] = 0. \end{aligned} \quad (15)$$

Obviously, the above equation has the characteristic root

$$\lambda_1 = -\mu_1 < 0. \quad (16)$$

Next, we consider the transcendental polynomial

$$\begin{aligned} \lambda^2 + (\mu^2 + \mu_3)\lambda + \mu_2\mu_3 \\ - k(1-n_{rt})(1-n_p)N\mu_2 T_0 e^{-\lambda\tau} = 0. \end{aligned} \quad (17)$$

For $\tau = 0$, we get

$$\lambda^2 + (\mu^2 + \mu_3)\lambda + \mu_2\mu_3 - R_0^2\mu_2\mu_3 = 0. \quad (18)$$

We have

$$\lambda_{2,3} = \frac{-(\mu_2 + \mu_3) \pm \sqrt{(\mu_2 + \mu_3)^2 - 4\mu_2\mu_3(1-R_0^2)}}{2}. \quad (19)$$

If $R_0^2 > 1$, the characteristic equation has a positive eigenvalue and two negative eigenvalues. E_0 is thus unstable with a two-dimensional stable manifold and a one-dimensional unstable manifold. If $R_0^2 = 1$, $\lambda_{2,3} = 0$, then E_0 is locally stable. If $R_0^2 < 1$, the other two eigenvalues have negative real parts if and only if $\mu_2\mu_3 - R_0^2\mu_2\mu_3 > 0$, then E_0 is locally asymptotically stable.

For $\tau \neq 0$, we get

$$\begin{aligned} \lambda^2 + (\mu^2 + \mu_3)\lambda + \mu_2\mu_3 \\ - k(1-n_{rt})(1-n_p)N\mu_2 T_0 e^{-\lambda\tau} = 0. \end{aligned} \quad (20)$$

Assume that the above equation has roots $\lambda = \omega(\cos(\beta\pi/2) \pm i \sin(\beta\pi/2))$ for $\omega > 0$ and $\tau > 0$; we get

$$\begin{aligned} \omega^2 \left(\cos \frac{\beta\pi}{2} \pm i \sin \frac{\beta\pi}{2} \right)^2 \\ + \omega (\mu^2 + \mu_3) \left(\cos \frac{\beta\pi}{2} \pm i \sin \frac{\beta\pi}{2} \right) + \mu_2\mu_3 \\ - k(1-n_{rt})(1-n_p)N\mu_2 T_0 e^{-\tau\omega(\cos(\beta\pi/2) \pm i \sin(\beta\pi/2))} = 0. \end{aligned} \quad (21)$$

Separating the real and imaginary parts gives

$$\begin{aligned} \omega^2 \left(\cos^2 \frac{\beta\pi}{2} - \sin^2 \frac{\beta\pi}{2} \right) + \omega \cos \frac{\beta\pi}{2} (\mu_2 + \mu_3) + \mu_2\mu_3 \\ - \frac{k(1-n_{rt})(1-n_p)N\mu_2 T_0 e^{-\tau\omega \cos(\beta\pi/2)}}{\mu_1} \\ \times \cos \left(-\tau\omega \sin \frac{\beta\pi}{2} \right) = 0, \\ 2i\omega^2 \cos \frac{\beta\pi}{2} \sin \frac{\beta\pi}{2} + i\omega \sin \frac{\beta\pi}{2} (\mu_2 + \mu_3) \\ - \frac{k(1-n_{rt})(1-n_p)N\mu_2 T_0 e^{-\tau\omega \cos(\beta\pi/2)}}{\mu_1} \\ \times i \sin \left(-\tau\omega \sin \frac{\beta\pi}{2} \right) = 0. \end{aligned} \quad (22)$$

□

From the second equation of (22), we have

$$\sin \frac{\beta\pi}{2} = 0, \quad (23)$$

that is $(\beta\pi/2) = k\pi, k = 0, 1, 2, \dots$

For $(\beta\pi/2) = k\pi, k = 0, 2, 4, \dots$, substituting into the first equation of (22), we have

$$\omega^2 + \omega(\mu_2 + \mu_3) + \mu_2\mu_3 = \frac{k(1 - n_{rt})(1 - n_p)N\mu_2se^{-\tau\omega}}{\mu_1}. \quad (24)$$

For the parameter values given in Table 1, we take any $R_0^2 < 1$, then the infected equilibrium $E_0 = ((s/\mu_1), 0, 0)$, and we get that the above equation is unequal for $\omega > 0$. Therefore, $\beta \geq 2 > \alpha$.

According to Lemma 3, the uninfected equilibrium E_0 is locally asymptotically stable. The proof is completed.

Next, for the sake of convenience, at $E^* = (T^*, I^*, V^*)$, we give the following symbols:

$$\begin{aligned} A &= \mu_1 + \mu_2 + \mu_3 - \frac{rV_I^*}{C + V_I^*}, \\ B &= k(1 - n_{rt})V_I^*, \\ C &= \mu_1^2 + \mu_1\mu_3 - \frac{rV_I^*(\mu_2 + \mu_3)}{C + V_I^*}, \\ D &= k(1 - n_{rt})V_I^*(\mu_2 + \mu_3), \\ E &= \mu_1\mu_2\mu_3 - \frac{rV_I^*\mu_2\mu_3}{C + V_I^*}, \\ F &= k(1 - n_{rt})V_I^*\mu_2\mu_3 - \frac{V_I^*\mu_2\mu_3rC}{(C + V_I^*)^2} - \mu_1\mu_2\mu_3 + \frac{\mu_2\mu_3rV_I^*}{C + V_I^*}. \end{aligned} \quad (25)$$

Then the characteristic equation of the linear system is

$$\lambda^3 + (A + Be^{-\lambda\tau})\lambda^2 + (C + De^{-\lambda\tau})\lambda + E + Fe^{-\lambda\tau} = 0. \quad (26)$$

Using the results in [49], we get

$$\begin{aligned} D(\lambda) &= \lambda^3 + (A + B)\lambda^2 + (C + D)\lambda + E + F, \\ D'(\lambda) &= 3\lambda^2 + 2(A + B)\lambda + (C + D). \end{aligned} \quad (27)$$

Denote

$$\begin{aligned} D(\lambda) &= - \begin{vmatrix} 1 & A+B & C+D & E+F & 0 \\ 0 & 1 & A+B & C+D & E+F \\ 3 & 2(A+B) & C+D & 0 & 0 \\ 0 & 3 & 2(A+B) & C+D & 0 \\ 0 & 0 & 3 & 2(A+B) & C+D \end{vmatrix} \\ &= 18(A+B)(C+D)(E+F) + (A+B)^2(C+D)^2 \\ &\quad - 4(E+F)(A+B)^3 - 4(C+D)^3 - 27(E+F)^2. \end{aligned} \quad (28)$$

Lemma 7 (see [49]). *The infected equilibrium E^* is asymptotically stable for any time delay $\tau \geq 0$ if either*

- (i) $A + B > 0, E + F > 0, (A + B)(C + D) > E + F$ if $D(\lambda) > 0$;
- (ii) if $D(\lambda) < 0, A + B \geq 0, C + D \geq 0, E + F > 0, \alpha < 2/3$;
- (iii) if $D(\lambda) < 0, A + B < 0, C + D < 0, \alpha > 2/3$, then all roots of $D(\lambda) = 0$ satisfy $|\arg(\lambda)| < \alpha\pi/2$;
- (iv) if $D(\lambda) < 0, A+B > 0, C+D > 0, (A+B)(C+D) = E+F$ for all $\alpha \in [0, 1)$.

4. Comparison with Some of the Data

In this section, we calculate the basic reproduction number R_0 , the IFE E_0 , two IPEs E_1^* and E_2^* , $D(\lambda)$, $A + B$, $C + D$, $E + F$, and $(A + B)(C + D)$. On the basis of these data, we apply all the conditions in Lemma 7 to judge the stability of the equilibrium E_1^* and E_2^* (Table 2).

Remark 8. (1) If $n_{rt} = \{0.1, 0.5, 0.5425\}$, there are two equilibria: the IFE E_0 and a unique IPE E_1^* . In addition, the system at E_1^* satisfies the second condition in Lemma 7, then the IPE E_1^* is locally asymptotically stable (Table 5).

(2) If $n_{rt} = \{0.5426, 0.55, 0.6, 0.7, 0.7252\}$, there are three equilibria: the IFE E_0 and two IPEs. The system at E_1^* doesn't satisfy all the conditions in Lemma 7, then the IPE E_1^* is unstable. The system at E_2^* satisfies the second condition in Lemma 7, then the IPE E_2^* is locally asymptotically stable.

(3) If $n_{rt} = \{0.7253, 0.8, 0.82, 0.8446, 0.85\}$, the IFE E_0 is the only equilibrium.

Remark 9. If $C = \{10, 100, 300, 500, 700, 830, 900\}$, there are two equilibria: the IFE E_0 and a unique IPE E_1^* . In addition, the system at E_1^* satisfies the second condition in Lemma 7, then the IPE E_1^* is locally asymptotically stable.

Remark 10. If $r = \{0, 0.004, 0.008, 0.015, 0.019\}$, there are two equilibria: the IFE E_0 and a unique IPE E_1^* . In addition, the system at E_1^* satisfies the second condition in Lemma 7, then the IPE E_1^* is locally asymptotically stable.

Remark 11. (1) If $N = \{10, 30, 40, 47\}$, the IFE E_0 is the only equilibrium.

(2) If $N = 48$, there are three equilibria: the IFE E_0 and two IPEs. The system at E_1^* and E_2^* does not satisfy all the conditions in Lemma 7, then the IPEs E_1^* and E_2^* are unstable.

(3) If $N = \{60, 88\}$, there are two equilibria: the IFE E_0 and a unique IPE E_1^* . In addition, the system at E_1^* does not satisfy all the conditions in Lemma 7, then the IPE E_1^* is unstable. The system at E_2^* satisfies the second condition in Lemma 7, then the IPE E_2^* is locally asymptotically stable.

(4) If $N = \{89, 100, 200, 600, 1000\}$, there are two equilibria: the IFE E_0 and a unique IPE E_1^* . The system at E_1^* satisfies the second condition in Lemma 7, then the IPE E_2^* is locally asymptotically stable.

(5) If $N = \{10000, 20000\}$, there are two equilibria: the IFE E_0 and a unique IPE E_1^* . The system at E_1^* satisfies the first condition in Lemma 7, then the IPE E_1^* is locally asymptotically stable.

TABLE 2: We take $r = 0.01$, $C = 1$, $N = 100$, $E_0 = (500, 0, 0)$, and $a = (\sqrt{r} - \sqrt{k(1 - n_{tt})C})^2 / \mu_1$, and we get the following.

(a)							
Line	n_{tt}	n_p	$k(1 - n_{tt})C$	$1 - a$	R_0	E_1^*	E_2^*
1	0.1	0.05	0.00002	0.5454	1.6016	(194.9317, 30.9602, 1911.7950)	not exist
2	0.5	0.25	0.00001	0.5340	1.0607	(444.4444, 21.3511, 1040.8668)	not exist
3	0.5425	0.27125	0.00001	0.5326	1.0001	(499.8953, 19.2137, 910.1286)	not exist
4	0.5426	0.2713	0.00001	0.5326	1.0000	(500.0390, 0.000003, 0.0002)	(500.0389, 19.2082, 909.8039)
5	0.55	0.275	0.00001	0.5323	0.9893	(510.8556, 0.0009, 0.0444)	(510.8557, 18.7911, 885.5296)
6	0.6	0.3	0.000009	0.5305	0.9165	(595.2381, 0.0104, 0.4716)	(595.2381, 15.5354, 706.8618)
7	0.7	0.35	0.000007	0.5265	0.7649	(854.7009, 0.1186, 5.0099)	(854.7009, 5.4462, 230.1012)
8	0.7252	0.3626	0.000007	0.5254	0.7249	(951.5244, 0.8287, 34.3354)	(951.5245, 1.0116, 41.9104)
9	0.7253	0.3627	0.000007	0.5253	0.7247	Not exist	Not exist
10	0.8	0.4	0.000005	0.5217	0.6000	Not exist	Not exist
11	0.82	0.41	0.000005	0.5206	0.5644	Not exist	Not exist
12	0.8446	0.4223	0.000004	0.5191	0.5190	Not exist	Not exist
13	0.85	0.425	0.000004	0.5188	0.5087	Not exist	Not exist

(b)							
Line	$D(\lambda)$	$A + B$	$C + D$	$E + F$	$(A + B)(C + D)$	Stability	
1	-1.7882	2.7113	0.1317	0.0258	0.3570	(ii)	
2	-0.5611	2.6825	0.0550	0.0078	0.1477	(ii)	
3	-0.4497	2.6800	0.0484	0.0062	0.1297	(ii)	
4	0.0165, -0.4495	2.6800, 2.6800	0.0484, 0.0484	-0.000001, 0.0062	0.1297, 0.1297	Unsuited (ii)	
5	0.0346, -0.4305	2.6796, 2.6796	0.0473, 0.0473	-0.0003, 0.0060	0.1267, 0.1267	Unsuited (ii)	
6	0.1125, -0.3054	2.6768, 2.6768	0.0399, 0.0399	-0.0014, 0.0042	0.1068, 0.1068	Unsuited (ii)	
7	0.0681, -0.0707	2.6717, 2.6717	0.0263, 0.0263	-0.0008, 0.0010	0.0703, 0.0703	Unsuited (ii)	
8	0.0060, 0.0015	2.6705, 2.6705	0.0232, 0.0232	-0.00003, 0.00003	0.0618, 0.0618	Unsuited (i)	

TABLE 3: We take $r = 0.01$, $n_{tt} = 0.5$, $n_p = 0.25$, $N = 100$, $E_0 = (500, 0, 0)$, and $a = (\sqrt{r} - \sqrt{k(1 - n_{tt})C})^2 / \mu_1$, and we have the following.

(a)							
Line	C	$k(1 - n_{tt})C$	$1 - a$	R_0	E_1^*	E_2^*	
14	10	0.0001	0.6035	1.0607	(444.4444, 21.2037, 1033.6821)	Not exist	
15	100	0.0012	0.7864	1.0607	(444.4444, 19.7599, 963.2939)	Not exist	
16	300	0.0036	0.9200	1.0607	(444.4444, 16.7808, 818.0663)	Not exist	
17	500	0.0060	0.9746	1.0607	(444.4444, 14.1982, 692.1614)	Not exist	
18	700	0.0084	0.9965	1.0607	(444.4444, 12.0858, 589.1841)	Not exist	
19	830	0.0100	1.0000	1.0607	(444.4444, 10.9728, 534.9223)	Not exist	
20	900	0.0108	0.9992	1.0607	(444.4444, 10.4534, 509.6013)	Not exist	

(b)							
Line	$D(\lambda)$	$A + B$	$C + D$	$E + F$	$(A + B)(C + D)$	Stability	
14	-0.5531	2.6825	0.0550	0.0077	0.1477	(ii)	
15	-0.4782	2.6825	0.0551	0.0067	0.1477	(ii)	
16	-0.3449	2.6825	0.0550	0.0049	0.1477	(ii)	
17	-0.2523	2.6825	0.0550	0.0037	0.1477	(ii)	
18	-0.1926	2.6825	0.0551	0.0029	0.1477	(ii)	
19	-0.1668	2.6825	0.0550	0.0025	0.1477	(ii)	
20	-0.1561	2.6825	0.0550	0.0024	0.1477	(ii)

FIGURE 2: In (a)–(d), $\alpha = 0.9$, $N = 100$, and $\tau = 0$.

FIGURE 3: In (a)–(d), $\alpha = 0.9$, $\tau = 2$, $C = 1$, and $r = 0.01$.

FIGURE 4: In (a)–(d), $n_{tt} = 0.5$, $n_p = 0.25$, $\alpha = 0.9$, $\tau = 0$, $C = 10$, and $r = 0.01$.

FIGURE 5: In (a)–(d), $n_{tt} = 0.5$, $n_p = 0.25$, $\alpha = 0.9$, $\tau = 2$, $C = 10$, and $r = 0.01$.

FIGURE 6: In (a)–(d), $n_{rt} = 0.5$, $n_p = 0.25$, $N = 100$, $\tau = 0$, $C = 10$, and $r = 0.01$.

5. Numerical Simulations

In this section, we use the Adams-type predictor-corrector method for the numerical solution of nonlinear system (4) with time delay.

Firstly, we will replace system (4) by the following equivalent fractional integral equations:

$$\begin{aligned} T(t) &= T(0) + I^\alpha \left[s - \mu_T T(t) + \frac{rT(t)V_I(t)}{C + V_I(t)} \right. \\ &\quad \left. - k(1 - n_{rt})T(t - \tau)V_I(t - \tau) \right], \\ I(t) &= I(0) + I^\alpha \\ &\quad \times [k(1 - n_{rt})T(t - \tau)V_I(t - \tau) - \mu_2 I(t)], \\ V(t) &= V(0) + I^\alpha [(1 - n_p)N\mu_2 I(t) - \mu_3 V_I(t)]. \end{aligned} \quad (29)$$

Next, we apply the predict, evaluate, correct, evaluate (PECE) method.

The approximate solution is displayed in (Figures 2(a)–2(d), 3(a)–3(d), 4(a)–4(d), 5(a)–5(d), 6(a)–6(d), and 7(a)–7(d)). When $\alpha = 1$, system (4) is the classical integer-order ODE.

Remark 12. Figures 2 and 3 show that, if $n_{rt} = \{0.7253, 0.8, 0.85\}$ and $\tau = \{0, 2\}$, the system at $E_0 = (500, 0, 0)$ is locally stable.

Remark 13. Figures 4 and 5 show that, if $N = \{10, 30, 40, 47\}$ and $\tau = \{0, 2\}$, the system at $E_0 = (500, 0, 0)$ is locally stable.

Remark 14. Figures 6 and 7 show that, if $\alpha = \{0.9, 0.93, 0.96, 0.99, 1\}$ and $\tau = \{0, 2\}$, the system at $E_0 = (500, 0, 0)$ is locally stable. As α increases, the trajectory of the system approaches the steady state faster and gets close to the integer-order ODE.

6. Extending the Model

In this section, we add the term $-\mu_3 V_I$ in the third equation of model (4) which we consider the loss of virions due to all causes, and we also add the bilinear term $-k(1 - n_{rt})T(t - \tau)V_I(t - \tau)$ which we consider free infectious virions when they enter the target cells. We extend model (4) to the following system of differential equations:

$$\begin{aligned} D^\alpha T(t) &= s - \mu_1 T(t) + \frac{rT(t)V_I(t)}{C + V_I(t)} \\ &\quad - k(1 - n_{rt})T(t - \tau)V_I(t - \tau), \\ D^\alpha I(t) &= k'(1 - n_{rt})T(t - \tau)V_I(t - \tau) - \mu_2 I(t), \end{aligned}$$

FIGURE 7: In (a)–(d), $n_{rt} = 0.5$, $n_p = 0.25$, $N = 100$, $\tau = 2$, $C = 10$, and $r = 0.01$.

$$D^\alpha V(t) = (1 - n_p) N \mu_2 I(t) - \mu_3 V_I(t) - k(1 - n_{rt}) T(t - \tau) V_I(t - \tau). \quad (30)$$

Following the analysis in [12], we get that system (30) has always the uninfected equilibrium $E_0 = ((s/\mu_1), 0, 0)$, and the infected equilibrium $E^{**} = (T^{**}, I^{**}, V_I^{**})$, where

$$\begin{aligned} T^{**} &= \frac{\mu_3}{(1 - n_p) [(1 - n_{rt}) N k' - k]}, \\ I^{**} &= \frac{\mu_3 k'}{\mu_2 [(1 - n_p) N k' - k]} V_I^{**}, \\ g(V_I) &= V_I^2 + V_I \left[\frac{\mu_1}{k(1 - n_{rt})} (1 - R^2) - \frac{r}{(1 - n_{rt}) k} \right. \\ &\quad \left. + \frac{C}{1 - n_{rt}} - \frac{R^2 s}{\mu_3} \right] \\ &\quad + \frac{\mu_1 C}{(1 - n_{rt}) k} - \frac{s C [(1 - n_p) N k' - k]}{k \mu_3} = 0. \end{aligned} \quad (31)$$

By the next generation matrix method [11], we obtain the basic reproduction number of model (30)

$$R^2 = \frac{(1 - n_p) (1 - n_{rt}) N k' s}{\mu_1 \mu_3 + k (1 - n_{rt}) s}. \quad (32)$$

7. Conclusion

In this paper, we modified the ODE model proposed by Shu and Wang [12] and the fractional model proposed by Yan and Kou [2] into a system of fractional order. We study a fractional differential model of HIV infection of $CD4^+$ T cell. We will consider this model where the $CD4^+$ T-cell proliferation does play an important role in HIV infection under antiretroviral therapy. The more appropriate method is given to ensure that both the equilibria are asymptotically stable for $\tau \geq 0$ under some conditions. We calculate the basic reproduction number R_0 , the IFE E_0 , two IPEs E_1^* and E_2^* , and so on, under certain conditions and judge the stability of the equilibrium. According to Tables 1 and 4, we get that, if $n_{rt} = \{0.7253, 0.8, 0.85\}$ and $N = \{10, 30, 40, 47\}$, there is only the IFE E_0 for $\tau \geq 0$. In addition, if $\alpha = \{0.9, 0.93, 0.96, 0.99, 1\}$ under some conditions, there is only the IFE E_0 for $\tau \geq 0$. We describe the dynamic behaviors of the fractional HIV model by using the Adams-type predictor-corrector method algorithm. At last, we extend the model to incorporate the

TABLE 4: We take $C = 10$, $n_{rt} = 0.5$, $n_p = 0.25$, $N = 100$, $E_0 = (500, 0, 0)$, and $a = (\sqrt{r} - \sqrt{k(1 - n_{rt})C})^2 / \mu_1$, we give the following.

(a)						
Line	r	$k(1 - n_{rt})C$	$1 - a$	R_0	E_1^*	E_2^*
21	0	0.0001	0.9940	1.0607	(444.4444, 4.2735, 208.3333)	Not exist
22	0.004	0.0001	0.8632	1.0607	(444.4444, 10.9858, 535.5567)	Not exist
23	0.008	0.0001	0.6920	1.0607	(444.4444, 17.7929, 867.4018)	Not exist
24	0.015	0.0001	0.3782	1.0607	(444.4444, 29.7389, 1449.7703)	Not exist
25	0.019	0.0001	0.1950	1.0607	(444.4444, 36.5710, 1782.8352)	Not exist
(b)						
Line	$D(\lambda)$	$A + B$	$C + D$	$E + F$	$(A + B)(C + D)$	Stability
21	-0.0952	2.6825	0.0550	0.0016	0.1477	(ii)
22	-0.2749	2.6825	0.0551	0.0040	0.1477	(ii)
23	-0.4600	2.6825	0.0550	0.0064	0.1477	(ii)
24	-0.7866	2.6825	0.0550	0.0108	0.1477	(ii)
25	-0.9740	2.6825	0.0550	0.0550	0.1477	(ii)

TABLE 5: We take $C = 10$, $n_{rt} = 0.5$, $n_p = 0.25$, $r = 0.01$, $E_0 = (500, 0, 0)$, and $a = (\sqrt{r} - \sqrt{k(1 - n_{rt})C})^2 / \mu_1$, and we get the following.

(a)						
Line	N	$k(1 - n_{rt})C$	$1 - a$	R_0	E_1^*	E_2^*
26	10	0.00001	0.5340	0.3354	Not exist	Not exist
27	30	0.00001	0.5340	0.5809	Not exist	Not exist
28	40	0.00001	0.5340	0.6708	Not exist	Not exist
29	47	0.00001	0.5340	0.7271	Not exist	Not exist
30	48	0.00001	0.5340	0.7348	(925.9259, 0.6491, 15.1879)	(925.9259, 2.1572, 50.4788)
31	60	0.00001	0.5340	0.8216	(740.7407, 0.0641, 1.8756)	(740.7407, 9.8732, 288.7910)
32	88	0.00001	0.5340	0.9950	(505.0505, 0.0005, 0.0204)	(505.0505, 19.0127, 815.6462)
33	89	0.00001	0.5340	1.0006	(499.3758, 19.2318, 834.4192)	Not exist
34	100	0.00001	0.5340	1.0607	(444.4444, 21.3511, 1040.8668)	Not exist
35	200	0.00001	0.5340	1.5000	(222.2222, 29.9116, 2916.3810)	Not exist
36	600	0.00001	0.5340	2.5981	(74.0741, 35.6123, 10416.5867)	Not exist
37	1000	0.00001	0.5340	3.3541	(44.4444, 36.7520, 17916.6202)	Not exist
38	10000	0.00001	0.5340	10.6066	(4.4444, 38.2906, 186666.6622)	Not exist
39	20000	0.00001	0.5340	15.0000	(2.2222, 38.3761, 374166.6644)	Not exist
(b)						
Line	$D(\lambda)$	$A + B$	$C + D$	$E + F$	$(A + B)(C + D)$	Stability
30	0.1109, 0.0417	2.6742, 2.6723	0.0328, 0.0278	-0.0014, -0.0005	0.0878, 0.0743	Unsuited, unsuited
31	0.0747, -0.1399	2.6784, 2.6738	0.0443, 0.0319	-0.0008, 0.0020	0.1185, 0.0853	Unsuited (i)
32	0.0173, -0.4352	2.6800, 2.6799	0.0483, 0.0482	-0.00001, 0.0060	0.1296, 0.1291	Unsuited (i)
33	-0.4454	2.6801	0.0487	0.0062	0.1307	(ii)
34	-0.5570	2.6826	0.0553	0.0077	0.1483	(ii)
35	-1.5273	2.7050	0.1150	0.0218	0.3110	(ii)
36	-4.7830	2.7950	0.3543	0.0780	0.9903	(ii)
37	-7.1387	2.8850	0.5937	0.1342	1.7129	(ii)
38	30.8901	4.9100	5.9802	1.3978	29.3628	(i)
39	482.3545	7.1600	11.9652	11.9652	85.6708	(i)

term which we consider the loss of virion and a bilinear term during attacking the target cells.

Conflict of Interests

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

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