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A Nonstandard Finite Difference Scheme for Solving Fractional–Order Model of HIV–1 Infection of CD4$^+$ T–Cells

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ABSTRACT In this paper, we introduce fractional-order into a model of HIV–1 infection of CD4$^+$ T–cells. We study the effect of the changing the average number of viral particles N with different sets of initial conditions on the dynamics of the presented model. The nonstandard finite difference (NSFD) scheme is implemented to examine the dynamic behaviors in the fractional-order HIV-1 infection model. Numerical results show that the NSFD approach is simple and accurate for solving fractional-order HIV-1 infection model.

KEYWORDS HIV–1 model •n onstandard finite difference scheme • fractional differential equations • Grunwald-Letnikov derivative • stability

1. INTRODUCTION

Human immune deficiency virus (HIV) is a lenti virus (a member of the retro virus family) that causes acquired immuno deficiency syndrome (AIDS), 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.

The HIV epidemic is widely acknowledged to be the most severe health crisis of modern times. HIV continues to spreadat alarming rates through many parts of the world, and there have been few victories in the efforts to contain it. This istrue despite remarkable

advances in our understanding of the molecular biology of the virus and its effects on the body advances that have led to major therapeutic discoveries in the second decade of the epidemic. For those who are able toobtain treatment with antiretroviral drugs, HIV infection has been transformed from a fatal illness into a chronic condition. This has led to dramatic reductions in mortality and morbidity from the illness. However, despite these advances on the biomedical front, the epidemic continues to spread and treatment remains unavailable to the overwhelming majority of those who require it.

It causes destruction of millions of people and expenditure of enormous amount of money in research and healthcare. Though, on the medical frontier there have been many advances, but still there is no effective cure or vaccine available for HIV. It is, therefore, essential that sufficient attention must be paid to study the dynamics of this fatal disease to subsequently control its spread. Mathematical models have been proven valuable in understanding the dynamics of HIV infection [9,14,17]. Many researchers discussed on this models.

In [12] Perelson was developed a simple model for the primary infection with HIV. This model has been important in the field of mathematical modeling of HIV infection, and many other models have been proposed, which take this model as their inspiration. Perelson et al. extended the model in [13] and discussed some behavior of the model. They defined the model by considering four categories: uninfected $CD4^+$T-cells, latently infected $CD4^+$ T-cells, productively infected $CD4^+$ T-cells and virus population.

Rong et al. [16] modified the model further by incorporating anti-retroviral effects to study the evolution of drug resistance. They considered three classes of $CD4^+$T-cells: uninfected cells, infected cells in eclipse phase and productively infected cells. The model depends on the observation that for a virus, when it enters a resting $CD4^+$ T-cell, viral RNA may not be completely reverse transcribed into DNA. In [5] Haiping modified the system of ordinary differential equations (ODEs) model proposed by Culshaw and Ruan [3] into a system of fractional-order. They showed that the model established in this paper possesses non-negative solutions, as desired in any population dynamics. They obtained a restriction on the number of viral particles released per infectious cell, in order for infection to be sustained. Following Rong et al., we assume here that a fraction of infected $CD4^+$ T-cells return to the uninfected class. In view of this, the following model is proposed:

$$\begin{aligned} T' &= s - kVT - dT + bT^*, \\ T^{*\prime} &= kVT - (b+\delta)T^*, \\ V' &= N\delta T^* - cV, \end{aligned} \tag{1}$$

with initial conditions:

$$T(0) = T_0, \qquad T^*(0) = T_0^*, \qquad V(0) = V_0.$$

In this model, T, T^* and V denote the concentration of uninfected $CD4^+$ cells, infected $CD4^+$ T-cells and free HIV virus particles in the blood, respectively. The parameter*s* stand for the inflow rate of $CD4^+$ T-cells and d its natural death rate. The parameter k represents the rate of infection of T-cells, δ represents death rate of infected T-cells and includes the possibility of death by bursting of infected T-cells, hence $\delta \geq d$. The factor b is the rate at which infected cells return to uninfected class. In addition, c presents the death rate of virus and N is the average number of viral particles produced by an infected cell.

This paper is organized as follows: In the next section,we elaborate some basic definitions and properties of the Grunwald-Letnikov (GL) approximation and provides a brief overview of the important features of the procedures for constructing NSFD schemes for systems of ODEs. In Section 3, we introduce fractional-order into the model that describes HIV-1 infection of $CD4^+$ T-cells and also stability theorem and Routh-Hurwitz stability conditions are given for the local asymptotic stability of the fractional systems. In Section 4, we will discuss the stability analysis of fractional system. In Section 5, we present the idea of NSFD scheme for solving the fractional-order HIV-1 infection of $CD4^+$ T-cells model. Finally in the last section, numerical results demonstrate that the NSFD approach is easy to be implemented and accurate when applied to the fractional-order HIV−1 infection model.

2. PRELIMINARIES AND NOTATIONS

In this section, some basic definitions and properties of the fractional calculus theory and nonstandard discretization are discussed.

2.1 Fundamentals of Fractional-Order

Fractional differential equations (FDEs) have gained considerable importance due to their applications in various sciences, such as physics, mechanics, chemistry and engineering [15]. In the recent years, the dynamic behaviors of fractional-order differential systems have received increasing attention. Although the concept of the fractional calculus was discussed in the same time interval of integer-order calculus, the complexity and the lack of applications postponed its progress till a few decades ago. Recently, most of the dynamical systems based on the integer-order calculus have been modified into the fractional-order domain due to the extra degrees of freedom and the flexibility which can be used to precisely fit the experimental data much better than the integer-order modeling.

2.2 GL Approximation

The GL method of approximation for the one-dimensional fractional derivative is as follows [15]:

$$D^{\alpha}x(t) = f\big(t, x(t)\big), \qquad x(0) = x_0, \qquad t \in [0, t_f], \tag{2}$$

$$D^{\alpha}x(t) = \lim_{h\to 0} h^{-\alpha} \sum_{j=0}^{[\frac{t_f}{h}]} (-1)^j \binom{\alpha}{j} x(t - jh),$$

where $0 < \alpha < 1$, D^{α} denotes the fractional derivative, h is the step size and $\left[\frac{t_f}{h}\right]$ represents the integer part of $\frac{t_f}{h}$. Therefore, Eq. (2) is discretized in the next form,

$$\sum_{j=0}^{n} c_j^{\alpha} x_{n-j} = f(t_n, x_n), \qquad n = 1, 2, 3, \ldots$$

where $t_n = n\ h$ and c_j^{α} are the GL coefficients defined as:

$$c_j^{\alpha} = \left(1 - \frac{1+\alpha}{j}\right) c_{j-1}^{\alpha}, \qquad c_0^{\alpha} = h^{-\alpha}, \qquad j = 1, 2, 3, \ldots$$

2.3 NSFD Discretization

The initial foundation of NSFD schemes came from the exact finite difference schemes. These schemes are well developed by Mickens [10, 11] in the past decades. These schemes are developed for compensating the weaknesses such as numerical instabilities that may be caused by standard finite difference methods. Regarding the positivity, boundedness and monotonicity of solutions, NSFD schemes have a better performance over the standard finite difference schemes, due to its flexibility to construct a NSFD scheme that can preserve certain properties and structures, which are obeyed by the original equations.

The advantages of NSFD schemes have been shown in many numerical applications. González-Parra et al. [4] and Arenas et al. [2] developed NSFD schemes to solve population and biological models. Jordan [6] constructed NSFD schemes for heat transfer problems. We now give an outline of the critical points which will allow the construction of NSFD discretizations for ODEs. Consider the autonomous ODE given by

$$x' = f(x), \qquad x(0) = x_0, \qquad t \in [0, t_f],$$

where $f(x)$ is, in general, a nonlinear function of x. For a discrete-time grid with step size, $\Delta t = h$, we replace the independent variable t by

$$t \approx t_n = nh, \qquad n = 0, 1, 2, \ldots, N$$

where $h = \frac{t_f}{N}$. The dependent variable $x(t)$ is replaced by

$$x(t) \approx x_n,$$

where x_n is the approximation of $x(t_n)$.

The first NSFD requirement is that the dependent functions should be modeled nonlocally on the discrete–time computational grid. Particular examples of this include the following functions [10, 11].

$$\begin{cases} xy \approx 2x_{n+1}y_n - x_{n+1}y_{n+1}, \\ x^2 \approx x_{n+1}x_n, \\ x^3 \approx \left(\dfrac{x_{n+1} + x_{n-1}}{2}\right) x_n^2. \end{cases}$$

A standard way for representing a discrete first-derivative is given by

$$x' \cong \frac{x_{n+1} - x_n}{h}.$$

However, the NSFD scheme requires that x' has the more general representation

$$x' \cong \frac{x_{n+1} - x_n}{\emptyset(h)},$$

where the denominator function, i.e. $\emptyset$ has the properties:

$(i)\ \emptyset(h) = h + O(h^2),$
$(ii)\ \emptyset(h)\ \ is\ an\ increasing\ function\ of\ h,$
$(iii)\ \emptyset(h)\ may\ depend\ on\ the\ parameters\ appearing\ in\ the\ differential\ equations.$

The paper of Mickens [11] gives a general procedure for determining $\emptyset(h)$ for systems of ODEs. An example of the NSFD discretization process is its application to the decay equation

$$x' = -\lambda x$$

where λ is a constant. The discretization scheme [11] is

$$\frac{x_{n+1} - x_n}{\emptyset} = -\lambda x_n, \qquad \emptyset(h, \lambda) = \frac{1 - e^{-\lambda h}}{\lambda}.$$

Another example is given by

$$x' = \lambda_1 x - \lambda_2 x^2,$$

where the NSFD scheme is

$$\frac{x_{n+1} - x_n}{\emptyset} = \lambda_1 x_n - \lambda_2 x_{n+1} x_n, \qquad \emptyset(h, \lambda_1) = \frac{e^{\lambda_1 h} - 1}{\lambda_1}.$$

It should be noted that the NSFD schemes for both ODEs are exact in the sense that $x_n = x(t_n)$ for all applicable values of $h > 0$. In general, for an ODE with polynomial terms,

$$x' = ax + (NL), \qquad NL \equiv nonlinear\ terms$$

the NSFD discretization for the linear expressions is derived by Mickens [11]

$$\frac{x_{n+1} - x_n}{\emptyset} = ax_n + (NL)_n,$$

where the denominator function is

$$\emptyset(h, a) = \frac{e^{ah} - 1}{a}.$$

It follows that if x' is a function of x which does not have a linear term, then the denominator function would be just h, i.e. $\emptyset(h) = h$. By applyingthis technique and using the GL discretizationmethod, it yields the following relations:

$$x_{n+1} = \frac{-\sum_{j=1}^{n+1} c_j^{\alpha} x_{n+1-j} + f(t_{n+1}, x_{n+1})}{c_0^{\alpha}}, \qquad n = 0, 1, 2, \ldots$$

where $c_0^{\alpha} = \emptyset(h)^{-\alpha}$.

3. FRACTIONAL-ORDER HIV–1 INFECTION EPIDEMIC MODEL

In this section, we introduce fractional-order into the model (1) of HIV-1 infection of the $CD4^+$ T-cells. The new system is described by the following set of fractional ODEs of order $\alpha_1, \alpha_2, \alpha_3$:

$$\begin{aligned} &D^{\alpha_1} T = s - kVT - dT + bT^*, \\ &D^{\alpha_1} T^* = kVT - (b + \delta)T^*, \\ &D^{\alpha_1} V = N\delta T^* - cV, \\ &0 < \alpha_i \leq 1, \qquad\qquad i = 1, 2, 3 \end{aligned} \tag{3}$$

with initial conditions

$$T(0) = T_0, \qquad T^*(0) = T_0^*, \qquad V(0) = V_0.$$

In order to analyze the stability of the model, stability theorem on fractional-order systems and fractional Routh-Hurwitz stability conditions for fractional-order differential equations are introduced. The first stability theorem has been given for fractional-order systems.

Theorem 1. [8] Consider the following fractional-order system:

$$D^{\alpha} x = f(x), \quad x(0) = x_0, \tag{4}$$

where $0 < \alpha \leq 1$ and $x \in R^n$. The equilibrium points of system Eqs. (4) are calculated by solving the equation $f(x) = 0$. These points are locally asymptotically stable if all eigenvalues λ of the Jacobian matrix $J = \frac{\partial f}{\partial x}$ evaluated at the equilibrium point satisfy:

$$|\arg(\lambda)| > \frac{\alpha\pi}{2}.$$

The Jacobian matrix J system Eqs. (3) of the equilibrium point $E = (T, T^*, V)$ is computed as

$$J(E) = \begin{pmatrix} -kV - d & b & -kT \\ kV & -b - \delta & kT \\ 0 & N\delta & -c \end{pmatrix}, \tag{5}$$

the existence and local stability conditions of this equilibrium point E is as follows. Suppose that $D(P)$ denotes the discriminant of a polynomial P

$$P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{6}$$

and

$$D(P) = 18a_1a_2a_3 + (a_1a_2)^2 - 4a_3(a_1)^3 - 4(a_2)^3 - 27(a_3)^2,$$

using the results of [1] we have the following Routh-Hurwitz stability conditions for FDEs:
(i) If $D(P) > 0$, then the necessary and sufficient condition for the equilibrium point E, to be locally asymptotically stable, is $a_1 > 0,\ a_3 > 0,\ a_1a_2 - a_3 > 0$.
(ii) If $D(P) < 0,\ a_1 \geq 0,\ a_2 \geq 0,\ a_3 > 0$, then the equilibrium point E is locally asymptotically stable for $\alpha < \frac{2}{3}$. Also, if

$$D(P) < 0, \quad a_1 < 0, \quad a_2 < 0, \quad \alpha > \frac{2}{3},$$

then all roots of Eq. (6) satisfy the condition $|\arg(\lambda)| < \frac{\alpha\pi}{2}$.
(iii) If $D(P) < 0,\ a_1 > 0,\ a_2 > 0,\ a_1a_2 - a_3 = 0,$ then the equilibrium point E is locally asymptotically stable for all $\alpha \in [0,1)$.
(iv) The necessary condition for the equilibrium point E, to be locally asymptotically stable, is $a_3 > 0$.

In the next section, we discuss the asymptotic stability of the equilibrium point E of the system Eqs. (3).

4. STABILITY ANALYSIS OF THE MODEL

To evaluate the equilibrium points of system Eqs. (3), let

$$\begin{aligned} s - kVT - dT + bT^* &= 0, \\ kVT - (b+\delta)T^* &= 0\,, \\ N\delta T^* - cV &= 0. \end{aligned}$$

Then the equilibrium points are $E_0 = \left(\frac{s}{d}, 0,0\right)$ and $E_1(\tilde{T}, \widetilde{T^*}, \tilde{V})$ for $R_0 > 1$, where

$$\tilde{T} = \frac{(b+\delta)c}{N\delta k}, \qquad \widetilde{T^*} = \frac{s}{\delta}\left(\frac{R_0 - 1}{R_0}\right), \qquad \tilde{V} = \frac{N\delta T^*}{c},$$

and the basic reproduction number of system Eqs. (3) is

$$R_0 = \frac{N\delta ks}{cd(b+\delta)}.$$

The local stability conditions of these equilibrium points are as follows.

(i) The Jacobian matrix (5) at the equilibrium point $E_0 = (\frac{s}{d}, 0\ ,0)$ is

$$J\left(\frac{s}{d}, 0\ ,0\right) = \begin{pmatrix} -d & b & -\frac{ks}{d} \\ 0 & -b-\delta & \frac{ks}{d} \\ 0 & N\delta & -c \end{pmatrix},$$

with the characteristic equation

$$P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where

$$a_1 = c + b + \delta + d, \qquad a_2 = -\frac{skN\delta - cdb - cd\delta - cd^2 - d^2b - d^2\delta}{d},$$
$$a_3 = -skN\delta + cdb + cd\delta.$$

Theorem 2. Assume that $\frac{1}{2} < R_0 < 1$. Then the disease free equilibrium point E_0 of system Eqs. (3) is asymptotically stable for all α.

Proof. Since $R_0 < 1$, hence $a_3 > 0$. Moreover

$$a_1a_2 - a_3 = -\frac{(b+\delta+c)(-d^2b - cdb - cd\delta + skN\delta - d^2\delta - d^3 - cd^2)}{d} > 0.$$

Now if $\frac{1}{2} < R_0 < 1$ then

$$D(P) = \frac{(4skN\delta - 2cd\delta - 2cdb + d\delta^2 + 2bd\delta + c^2d + db^2)\mu^2}{d^3} > 0,$$

where

$$\mu = skN\delta - cdb - cd\delta + d^2\delta - d^3 + cd^2 + d^2b.$$

Since $a_1 > 0$, hence all inequalities part (i) of the fractional Routh-Hurwitz conditions are satisfied. Therefore, if $\frac{1}{2} < R_0 < 1$ then the disease free equilibrium point E_0 is locally asymptotically stable for all α. □

(ii) The Jacobian matrix (5) at the equilibrium point $E_1(\tilde{T}, \widetilde{T^*}, \tilde{V})$ is

$$J(\tilde{T}, \widetilde{T^*}, \tilde{V}) = \begin{pmatrix} -\frac{skNR_0 - skN + dcR_0}{cR_0} & b & -\frac{c(b+\delta)}{N\delta} \\ \frac{kNs(R_0-1)}{cR_0} & -b-\delta & \frac{c(b+\delta)}{N\delta} \\ 0 & N\delta & -c \end{pmatrix},$$

with the characteristic equation

$$P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where

$$a_1 = \frac{skN(R_0-1) + dcR_0 + R_0\delta c + c^2R_0 + cR_0b}{cR_0},$$

$$a_2 = \frac{skN\delta(R_0-1) + skN(R_0-1) + dcR_0b + dcR_0\delta + dc^2R_0}{cR_0},$$

$$a_3 = \frac{skN\delta(R_0-1)}{R_0}.$$

Observe that if $R_0 > 1$ then $a_1 > 0$, $a_2 > 0$ and $a_3 > 0$.

5. NSFD Discretization for Fractional–Order HIV–1 Infection Model

By system Eqs. (3) and applying Mickens scheme by replacing the step size h by a function $\emptyset(h)$ and using the GL discretization method, it can be seen that:

$$\sum_{j=0}^{n+1} c_j^{\alpha_1} T_{n+1-j} = s - kT_{n+1}V_n - dT_{n+1} + bT_n^*, \tag{7}$$

$$\sum_{j=0}^{n+1} c_j^{\alpha_2} T_{n+1-j}^* = kT_{n+1}V_n - (b+\delta)T_{n+1}^*, \tag{8}$$

$$\sum_{j=0}^{n+1} c_j^{\alpha_3} V_{n+1-j} = N\delta T_{n+1}^* - cV_{n+1}. \tag{9}$$

Comparing Eqs. (7)–(9) with system Eqs. (3), we note the following statements:

1. The linear and nonlinear terms on the right-hand side of first equation in system (3) are in the form
$$-TV \approx -T_{n+1}V_n, \qquad -T \approx -T_{n+1}, \qquad T^* \approx T_n^*.$$
2. The linear and nonlinear terms on the right-hand side of second equation in (3) are:
$$TV \approx T_{n+1}V_n, \qquad -T^* \approx -T_{n+1}^*.$$
3. The linear terms on the right-hand side of third equation system (3) are:
$$T^* \approx T_{n+1}^*, \qquad -V \approx -V_{n+1}.$$

Invoking some algebraic manipulations on Eqs. (7)–(9), the following relations are obtained:

$$T_{n+1} = \frac{-\sum_{j=1}^{n+1} c_j^{\alpha_1} T_{n+1-j} + s + bT_n^*}{c_0^{\alpha_1} + d + kV_n},$$

$$T_{n+1}^* = \frac{-\sum_{j=0}^{n+1} c_j^{\alpha_2} T_{n+1-j}^* + kT_{n+1}V_n}{c_0^{\alpha_2} + b + \delta},$$

$$V_{n+1} = \frac{-\sum_{j=1}^{n+1} c_j^{\alpha_3} V_{n+1-j} + N\delta T_{n+1}^*}{c_0^{\alpha_3} + c},$$

where
$$c_0^{\alpha_1} = \emptyset_1(h)^{-\alpha_1}, \qquad c_0^{\alpha_2} = \emptyset_2(h)^{-\alpha_2}, \qquad c_0^{\alpha_3} = \emptyset_3(h)^{-\alpha_3},$$

by [18]
$$\emptyset_1(h) = \frac{e^{dh} - 1}{d}, \qquad \emptyset_2(h) = \frac{e^{(\delta+b)h} - 1}{(\delta + b)}, \qquad \emptyset_3(h) = \frac{e^{ch} - 1}{c}.$$

6. NUMERICAL RESULTS

Analytical studies always remain incomplete without numerical verification of the results. In this section, we present numerical simulation to illustrate the results obtained in previous sections. Now we solve the fractional-order HIV-1 infection epidemic model in two cases. The approximate solutions are displayed in Figures 1–4, for different $0 < \alpha_i \leq 1$ and $i = 1, 2, 3$.

Case 1. We exploited the following data set: $s = 10$, $b = 0.2$, $k = 0.000024$, $d = 0.01$, $\delta = 0.16$, $c = 3.4$ and $N = 1000$. For this set of data $R_0 = 3.137 > 1$, $D(P) = -4.868$ and

$$a_1 = 3.818, \qquad a_2 = 0.208, \qquad a_3 = 0.026,$$

with $a_1 a_2 - a_3 = 0.771$. Thus, the disease free equilibrium point E_1 is locally asymptotically stable for $\alpha < \frac{2}{3}$. It can be verified that the system goes to infected steady state $(318.75, 42.57, 2003.9)$. The results are depicted in Figures 1 and 2 for the initial conditions in the first case study are $T(0) = 1000$, $T^*(0) = 0$, $V(0) = 0.001$ with simulation time $5000s$ and step size $h = 1.1$.

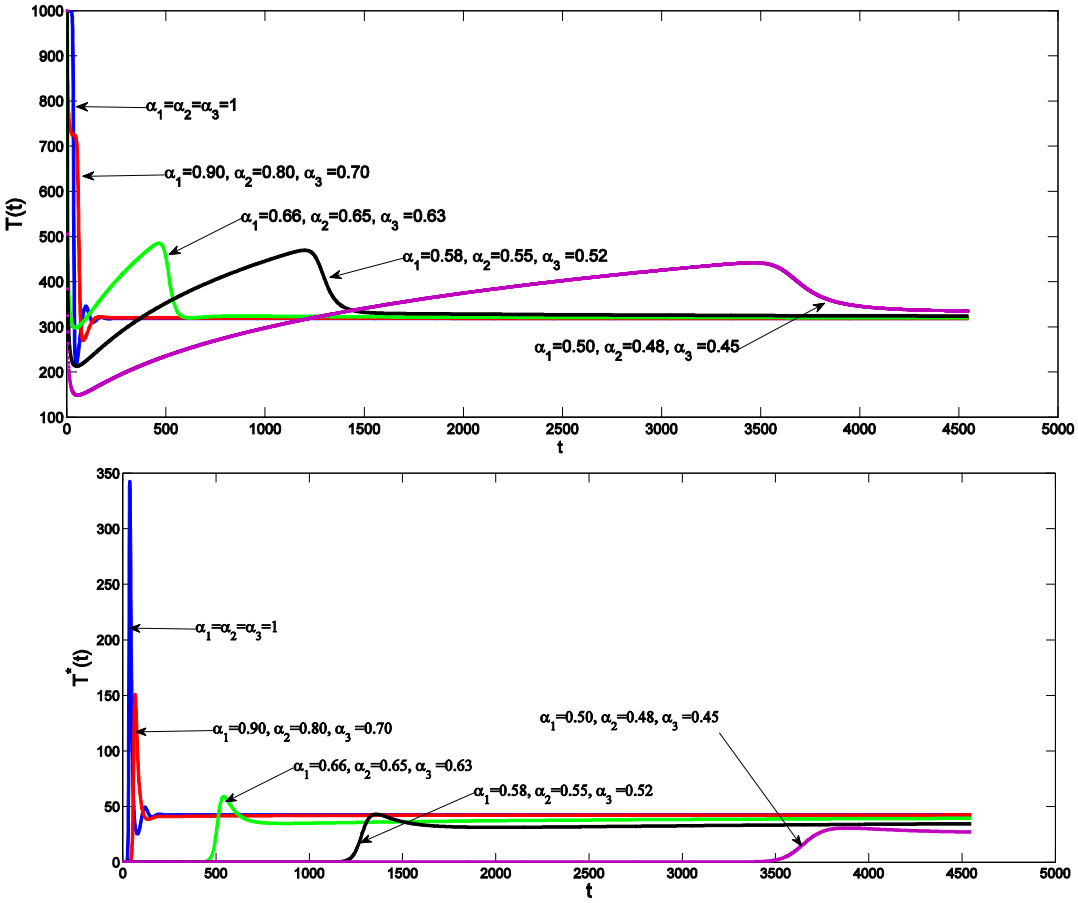

Figure 1. The concentration of the uninfected $CD4^+$ T-cells at $N = 1000$ with step size $h = 1.1$.

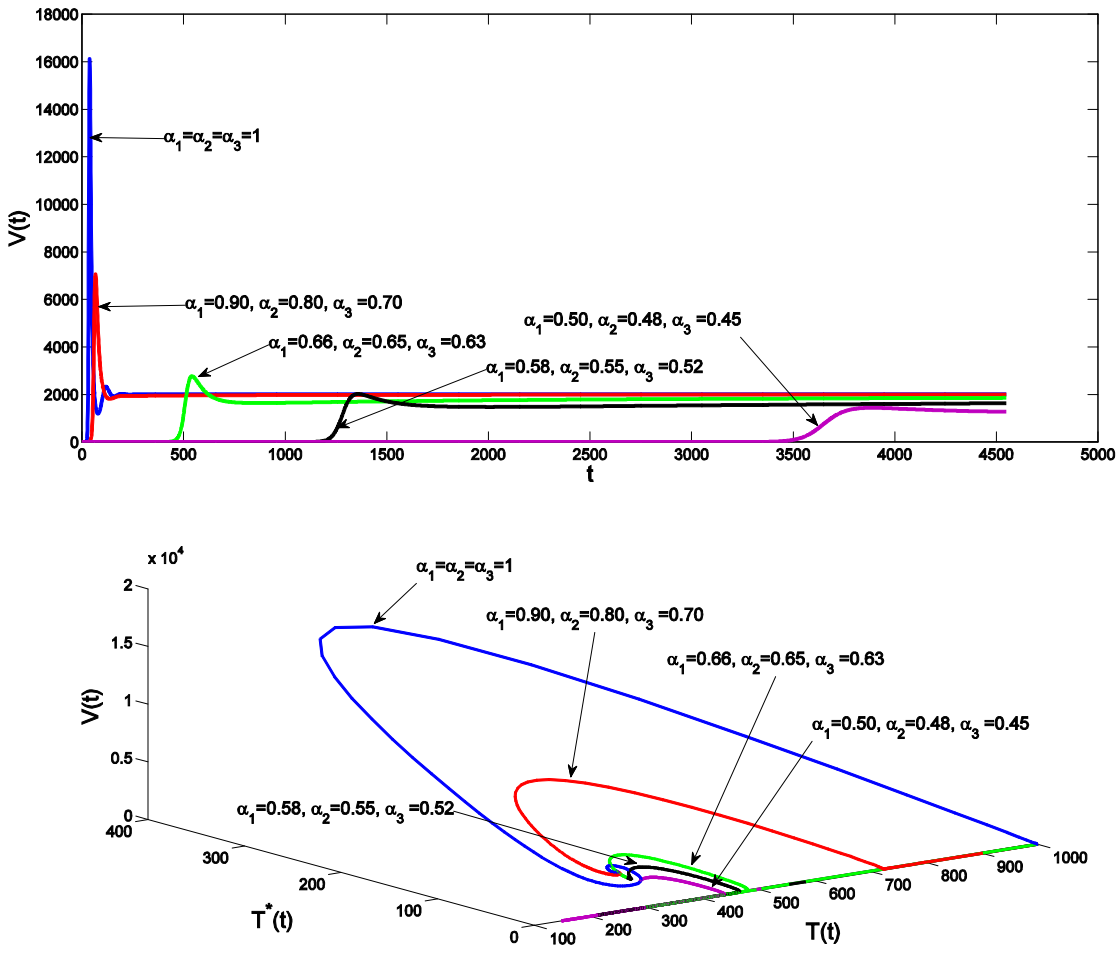

Figure 2. The concentration of the free HIV virus particles at $N = 1000$ with step size $h = 1.1$.

Case 2. We exploited the following data set: $s = 10$, $b = 0.2$, $k = 0.000024$, $d = 0.01$, $\delta = 0.16$, $c = 3.4$ and $N = 1600$. For this set of data $R_0 = 5.019 > 1$, $D(P) = -8.417$ and

$$a_1 = 3.860, \qquad a_2 = 0.359, \qquad a_3 = 0.049,$$

with $a_1 a_2 - a_3 = 1.338$. Thus, the disease free equilibrium point E_1 is locally asymptotically stable for $\alpha < \frac{2}{3}$. It can be verified that the system goes to infected steady state $(199.218, 3768.382, 50.048)$. The results are depicted in Figures 3 and 4 for the initial conditions in the second case study are $T(0) = 1000$, $T^*(0) = 10, V(0) = 10$ with simulation time $3000s$ and step size $h = 1.5$.

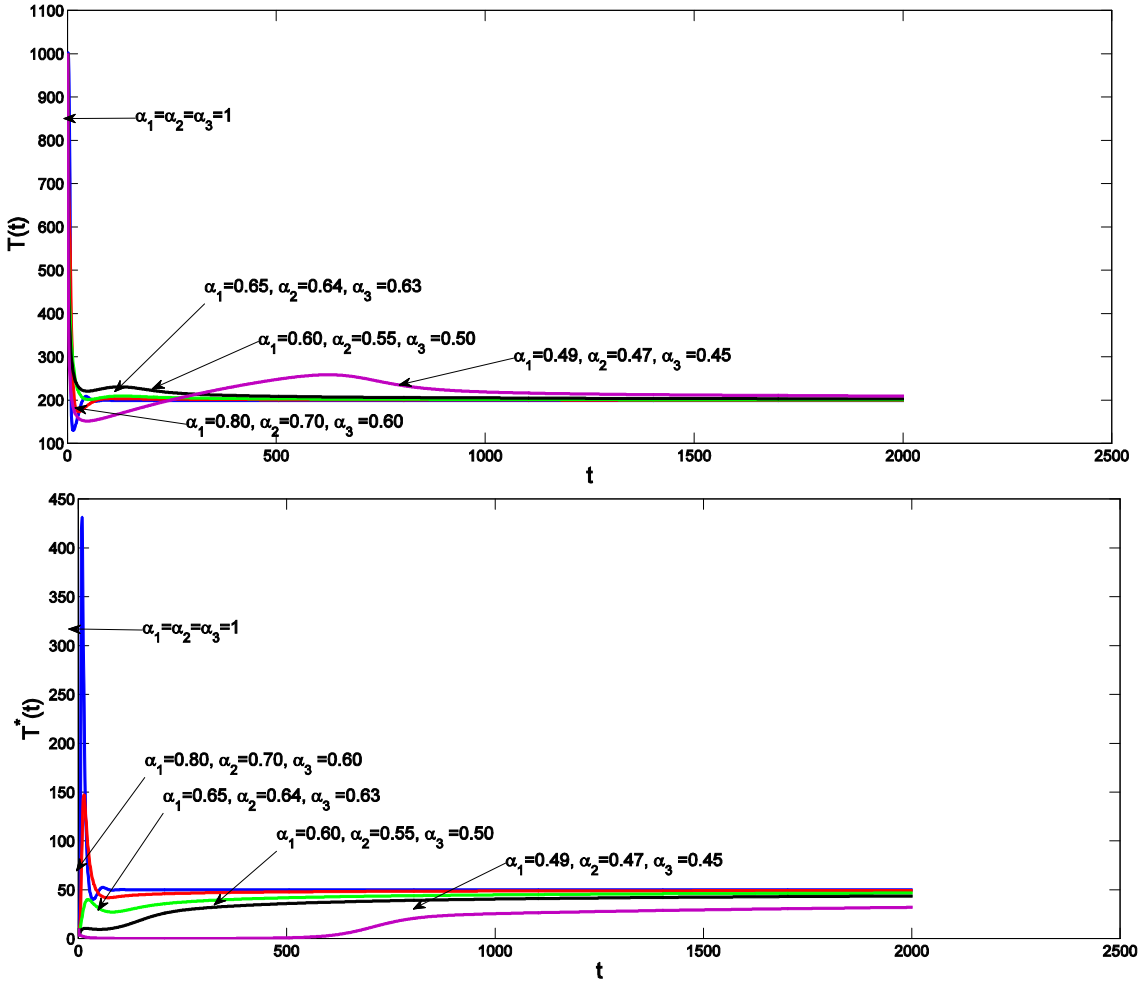

Figure 3. The concentration of the uninfected $CD4^+$ T-cells at $N = 1600$ with step size $h = 1.5$.

7. CONCLUSION

In this paper we studied the fractional-order HIV-1 infection model. From the obtained results in the presented figures, it turns outthat in the primary stage of the infection with the (HIV) virus, a dramatically decrease in the level of the $CD4^+$T-cells occurs because of the death of such infectedcells. On the other hand, the number of the free HIV virus particles and the number of susceptible $CD4^+$ T-cells increase. This assumes that the growth of healthy T-cells slows down during the course of HIV infection.We have to give an attention to the parameter b whichis called the reverting rate of infected cells to uninfected

class due to non-completion of reverse transcription.Further, since only small fraction of infected cells will revert back due to incompletion of reverse transcription, we expect the reverting rate b to be small. The basic reproduction number of the presented model (3) is given in as:

$$R_0 = \frac{N\delta ks}{cd(b+\delta)}.$$

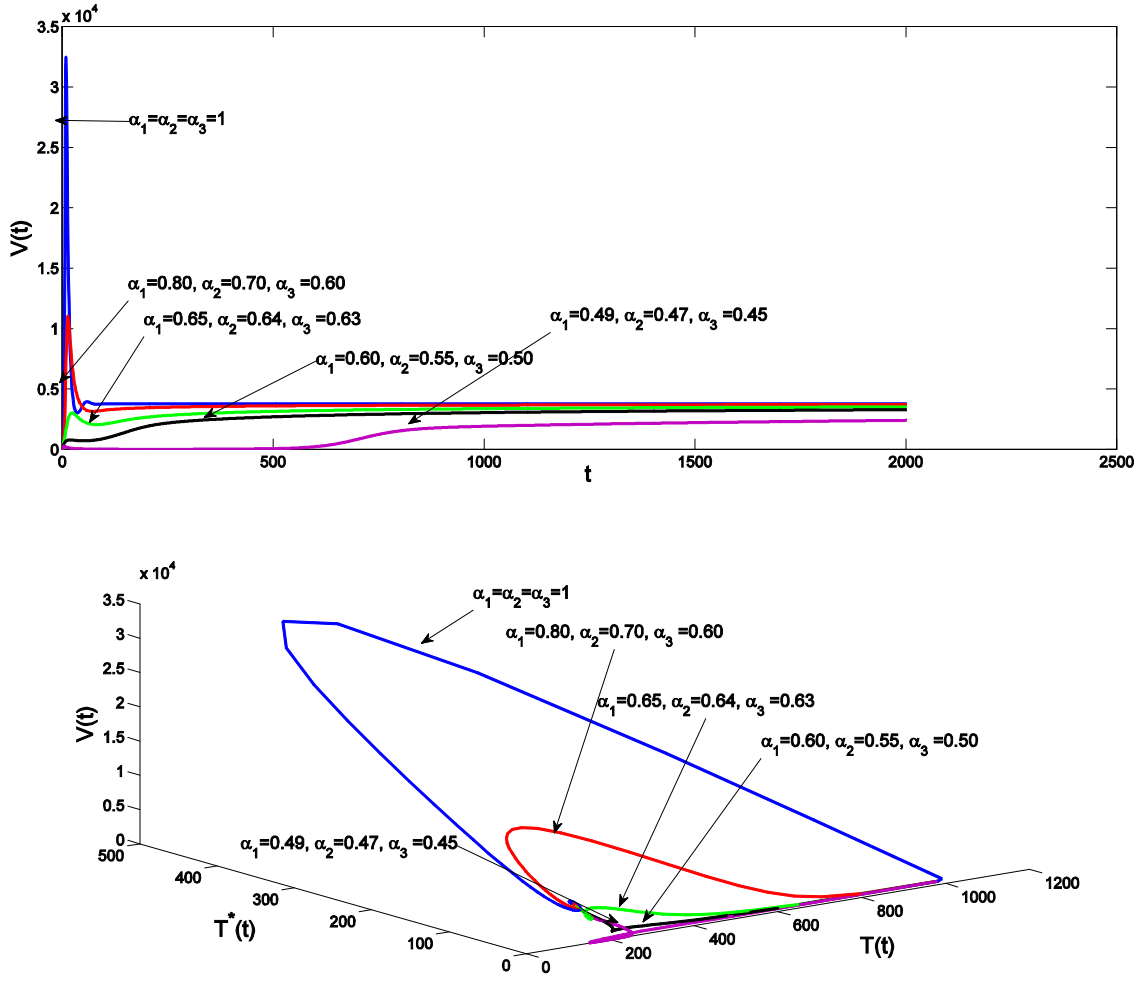

Figure4. The concentration of the free HIV virus particles at $N = 1600$ with step size $h = 1.5$.

It represents the average number of secondary infection caused by a single infected T-cell in an entirely susceptible T-cell population, through out its infectious period. For system (3), if the basic reproduction number $R_0 \leq 1$, the virus is cleared and no HIV infection persists. If $R_0 > 1$, the HIV infection persists in the T-cell population. In the two presented cases, $R_0 = 3.137$ when $N = 1000$, (see figures1-2) and $R_0 = 5.019$ when $N = 1600$

(see figures 3-4), the system goes to infected steady state. From the definition of R_0, it can be seen that R_0 decreases as there verting rate, b of infected cells increases. Hence R_0 can be low for a high parametric value of b. Increasing the value of N will decrease the numbers of uninfected $CD4^+$ T-cells and increases the number of free viruss ubstantially, but does not change the stability of the steady state. The concentration of susceptible $CD4^+$ T-cells $T(t)$, infected $CD4^+$ T-cells $I(t)$, and free HIV virus particles $V(t)$ in the blood have been obtained, therefore when $\alpha \to 1$ the solution of the fractionalmodel (3), $T_\alpha(t)$, $T_\alpha^*(t)$, $V_\alpha(t)$, reduces to the standard solution $T(t)$, $T^*(t)$, $V(t)$ Finally, the recent appearance of fractional differential equations as models in some fields of applied mathematics makes it necessary to investigate methods of solution for such equations (analytical and numerical) and we hope that this work is a step in this direction.

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