

Commun. Theor. Phys. **76** (2024) 035001 (14pp) https://doi.org/10.1088/1572-9494/ad2368

Modeling the dynamical behavior of the interaction of T-cells and human immunodeficiency virus with saturated incidence

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Received 7 September 2023, revised 26 January 2024
Accepted for publication 29 January 2024
Published 29 February 2024

Abstract

In the last forty years, the rise of HIV has undoubtedly become a major concern in the field of public health, imposing significant economic burdens on affected regions. Consequently, it becomes imperative to undertake comprehensive investigations into the mechanisms governing the dissemination of HIV within the human body. In this work, we have devised a mathematical model that elucidates the intricate interplay between $CD4^+$ T-cells and viruses of HIV, employing the principles of fractional calculus. The production rate of $CD4^+$ T-cells, like other immune cells depends on certain factors such as age, health status, and the presence of infections or diseases. Therefore, we incorporate a variable source term in the dynamics of HIV infection with a saturated incidence rate to enhance the precision of our findings. We introduce the fundamental concepts of fractional operators as a means of scrutinizing the proposed HIV model. To facilitate a deeper understanding of our system, we present an iterative scheme that elucidates the trajectories of the solution pathways of the system. We show the time series analysis of our model through numerical findings to conceptualize and understand the key factors of the system. In addition to this, we present the phase portrait and the oscillatory behavior of the system with the variation of different input parameters. This information can be utilized to predict the long-term behavior of the system, including whether it will converge to a steady state or exhibit periodic or chaotic oscillations.

Keywords: HIV infection, fractional-calculus, dynamics of HIV, iterative scheme, dynamical behaviour, mathematical model, fractional derivatives

(Some figures may appear in colour only in the online journal)

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1. Introduction

In accordance with available reports, HIV infections inflict damage upon the immune system of afflicted individuals, leading to deleterious effects on vital internal organs such as the kidneys, brain, and heart, ultimately culminating in mortality. Although a definitive cure for this infectious disease remains elusive, there exist efficacious antiretroviral treatments that can substantially ameliorate a patient's health. However, it is worth noting that excessive utilization of these medications may exert adverse effects on the host. Empirical evidence from studies underscores HIV infection as the most pernicious viral threat on a global scale, exerting profound impacts across various sectors. In 2017, an estimated 1.8 million individuals were living with HIV, and tragically, 940,000 of them succumbed to the disease. Nevertheless, there exists a firm belief that the AIDS pandemic can be curtailed through a combination of preventative measures and therapeutic interventions. The utilization of therapy has expanded considerably, with individuals occasionally manifesting symptoms resembling sore throats, rashes, headaches, fevers, and influenza-like manifestations. It is crucial to underscore that when treatment is not administered promptly and effectively, HIV infections progress into severe and life-threatening conditions. The burden of HIV on society encompasses not only the physical and emotional toll on individuals, but also places strains on health-care systems and resources. It fosters a need for comprehensive support structures, education, and public health initiatives to address the multifaceted challenges posed by the virus.

It is acknowledged that mathematical models play a crucial role in exploring the dynamics of infectious diseases and biological processes [1–3]. These models serve as invaluable tools for elucidating the intricacies of complex biological phenomena and furnishing comprehensive insights into the key factors governing biological systems [4–6]. Noteworthy contributions to the field include the establishment of an HIV model and the subsequent identification of statistically optimal control measures in 1999 by researchers, as well as the development of various models in subsequent studies to explore dynamic aspects, including viral mutation, intracellular delays, and other relevant factors [7]. These endeavors collectively contribute to our understanding of HIV and its associated dynamics [8, 9]. The researchers in [10] included two transmission methods, including direct cell-to-cell transfer and infection by free virions, while HIV-positive $CD4^+$ T-cell populations were examined in [11]. In the article [12], Perilson and Nelson describe how they used a mathematical model and determined some significant characteristics of the system. The model originally described by the authors in [12] was further modified by the authors in [13]. Through study and fractional calculus, Samia *et al* [14] examined the phenomenon of the HIV-1 infection. The researchers in [15] used the homotopy analysis methodology to analyze the HIV model, while authors in [16, 17] utilized different concepts and approaches to study the dynamics of HIV. The chaotic dynamics of HIV have been looked at and analyzed in [18]. The incorporation of the drug into the model and its subsequent influence on the overall dynamics has been investigated in the literature [19–21]. In this work, our primary objective is to understand the dynamics of HIV infection by incorporating a saturated incidence rate alongside a variable source term.

Fractional derivatives provide a more versatile way to model and describe the behavior of complex systems, especially those with non-local and memory-dependent characteristics [22–24]. They offer advantages in modeling biological phenomena by providing a more nuanced representation of complex processes, particularly in cases where memory and non-local effects play a crucial role [25–27]. This enhanced modeling capability allows for a more accurate portrayal of disease dynamics, contributing to improved understanding, diagnosis, and treatment strategies [28, 29]. Fractional operators provide a more nuanced and realistic approach to modeling biological systems, offering more accurate predictions and insights for infectious diseases [30, 31]. It is evident that fractional calculus is used in a variety of fields, including chemistry, economics, biology and physics [32, 33]. Many numerical schemes have been developed to visualize the dynamics of a fractional system [34, 35]. Fractional calculus has been used in both older studies and modern research to create more precise information on the dynamics of a system. Specifically, fractional calculus has found application in highlighting the dynamic aspects of infectious diseases. The recently developed Caputo–Fabrizio (CF) operator utilizes an exponential decay law kernel, a choice that yields more precise and accurate outcomes when modeling natural phenomena. The CF operator is a special case of the new generalized Hattaf fractional (GHF) operator introduced in [33]. In this study, our goal is to visualize the dynamical behavior of HIV infection within a fractional framework, aiming to attain results that are more accurate and precise. By incorporating fractional calculus into disease modeling, we aim to better understand the underlying mechanisms, improve prediction accuracy, and potentially enhance the design of control strategies.

The subsequent sections of this paper are organized as follows: in section 2, we delineate the dynamics of HIV, incorporating a saturated incidence rate and a variable source term. To enhance the precision of our findings, we employ the CF fractional operator to illustrate the hypothesized HIV infection system. Section 3 provides an overview of the fundamental principles of fractional theory, essential for the analysis of the recommended HIV infection model. Section 4 introduces a numerical approach to underscore solution pathways, while also presenting the dynamical behavior and phase portrait of the proposed model. Finally, in section 5, we present the concluding remarks summarizing the entirety of our work.

2. Formulation of the model

In this section, we have organized the dynamics of HIV infection in order to elucidate the interactions between the HIV virus and the immune system. A number of researchers developed and tested the reaction of $CD4^+$ T-cells and HIV in

Table 1. Interpretation of the system's state variables and input parameters [46].

Input factors	Descriptions	Values
μ_T	Healthy T-cell mortality rate	0.02 day^{-1}
N	Amount of viruses produced by I	Assumed
$T_{\max}$	Maximum concentration of healthy T-cells	1500 mm^{-3}
k	Healthy T-cell infection rate by free virus	2.4×10^{-5} days^{-1}
I_0	T-cell infection concentration initially	Assumed
μ_V	The pace of death of HIV viruses	2.4 day^{-1}
s	Rate of T supply from precursors	0.1 mm^{-3}
r	Growth rate of the number of healthy T-cells	3 day^{-1}
T_0	Healthy T-cell initial concentration	Assumed
μ_I	The percentage of infected T-cells that die	0.3 day^{-1}
V_0	Amount of HIV-free viruses present	Assumed

the past to explore this intricate phenomena [36–38]. The researchers [39] introduced the HIV transmission phenomena as:

$$\begin{aligned}
\frac{\mathrm{d}T}{\mathrm{dt}} &= \mathrm{s} - \mu_{\mathrm{T}}T + \left(1 - \frac{T+\mathrm{I}}{T_{\max}}\right)rT - kVT,\\
\frac{\mathrm{dI}}{\mathrm{dt}} &= kVT - \mu_{\mathrm{I}}\mathrm{I},\\
\frac{\mathrm{dV}}{\mathrm{d}t} &= \mathrm{N}\mu_{\mathrm{I}}\mathrm{I} - \mu_{\mathrm{V}}\mathrm{V},
\end{aligned} \tag{1}$$

where s represents the rate at which the body produces new T-cells, μ_{T} represents the rate at which T-cells expire, and μ_{V} and μ_{I} indicate the rate at which the particles V and the cells of I expire. The number of cells generated by infected T-cell reproduction is indicated by N while the rate at which healthy T-cells become infected is taken to be k. The HIV model with saturation incidence that Perelson and Nelson [40] is expressed as

$$\begin{aligned}
\frac{\mathrm{d}T}{\mathrm{dt}} &= \left(1 - \frac{T}{T_{\max}}\right)rT - \frac{\beta VT}{1+\alpha\mathrm{V}},\\
\frac{\mathrm{dI}}{\mathrm{dt}} &= \frac{\beta VT}{1+\alpha\mathrm{V}} - \mu_{\mathrm{I}}\mathrm{I},\\
\frac{\mathrm{dV}}{\mathrm{dt}} &= N\mu_{\mathrm{I}}\mathrm{I} - \mu_{\mathrm{V}}\mathrm{V}.
\end{aligned} \tag{2}$$

In the next step, we propose a saturation incidence rate for the propagation of HIV viruses and infected T-cells to healthy $CD4^+$ T-cells. The rate of production of $CD4^+$ T-cells, like other immune cells, can vary depending on factors such as age, health status, and the presence of infections or diseases. Generally, the production of $CD4^+$ T-cells occurs in the bone marrow, where hematopoietic stem cells differentiate into various blood cell types, including T-cells. In healthy individuals, the body continuously produces T-cells to maintain a functional immune system. The exact rate of production can be influenced by factors such as thymic activity, which tends to decline with age. Therefore, we assumed variable source term s(V) given by $\frac{\eta s}{\eta+V}$. Then, we have

$$\begin{aligned}
\frac{\mathrm{d}T}{\mathrm{dt}} &= \frac{\eta\mathrm{s}}{\eta+\mathrm{V}} - \mu_{\mathrm{T}}T + \left(1 - \frac{T}{T_{\max}}\right)rT\\
&\quad - \frac{kVT}{1+\alpha_1\mathrm{V}} - \frac{\alpha IT}{1+\alpha_2\mathrm{I}},\\
\frac{\mathrm{d}L}{\mathrm{dt}} &= \frac{kVT}{1+\alpha_1\mathrm{V}} + \frac{\alpha IT}{1+\alpha_2\mathrm{I}} - \mu_{\mathrm{I}}L - k_2L,\\
\frac{\mathrm{dI}}{\mathrm{dt}} &= k_2L - \mu_{\mathrm{I}}\mathrm{I},\\
\frac{\mathrm{dV}}{\mathrm{dt}} &= \mathrm{N}\mu_{\mathrm{I}}\mathrm{I} - \mu_{\mathrm{V}}\mathrm{V} - \frac{kVT}{1+\alpha_1\mathrm{V}},
\end{aligned} \tag{3}$$

where α indicates the effectiveness of a protease inhibitor and s denotes the rate of cellular infection. In this formulation, the concentration of healthy $CD4^+$ T-cells is denoted by T and the latent stage of T-cells is indicated by L. Furthermore, the concentration of infected T-cells is represented by I while the strength of HIV viruses is symbolized by V in this formulation.

The advantage of fractional order models is that they can capture more complex dynamics and long-range dependencies that may be present in certain biological systems. In the context of diseases like HIV, which exhibit intricate and evolving patterns, these models can provide a more accurate representation of the dynamics. We use fractional calculus to show the aforementioned dynamics in order to provide a more realistic portrayal.

$$\begin{aligned}
{}_0^{\mathrm{CF}}\mathrm{D}_t^{\xi}T &= \frac{\eta s}{\eta+\mathrm{V}} - \mu_T T + \left(1 - \frac{T}{T_{\max}}\right)rT\\
&\quad - \frac{kVT}{1+\alpha_1\mathrm{V}} - \frac{\alpha IT}{1+\alpha_2\mathrm{I}},\\
{}_0^{\mathrm{CF}}\mathrm{D}_t^{\xi}L &= \frac{kVT}{1+\alpha_1\mathrm{V}} + \frac{\alpha IT}{1+\alpha_2\mathrm{I}} - \mu_{\mathrm{I}}L - k_2L,\\
{}_0^{\mathrm{CF}}\mathrm{D}_t^{\xi}\mathrm{I} &= k_2L - \mu_{\mathrm{I}}\mathrm{I},\\
{}_0^{\mathrm{CF}}\mathrm{D}_t^{\xi}\mathrm{V} &= N\mu_{\mathrm{I}}\mathrm{I} - \mu_{\mathrm{V}}\mathrm{V} - \frac{kVT}{1+\alpha_1\mathrm{V}},
\end{aligned} \tag{4}$$

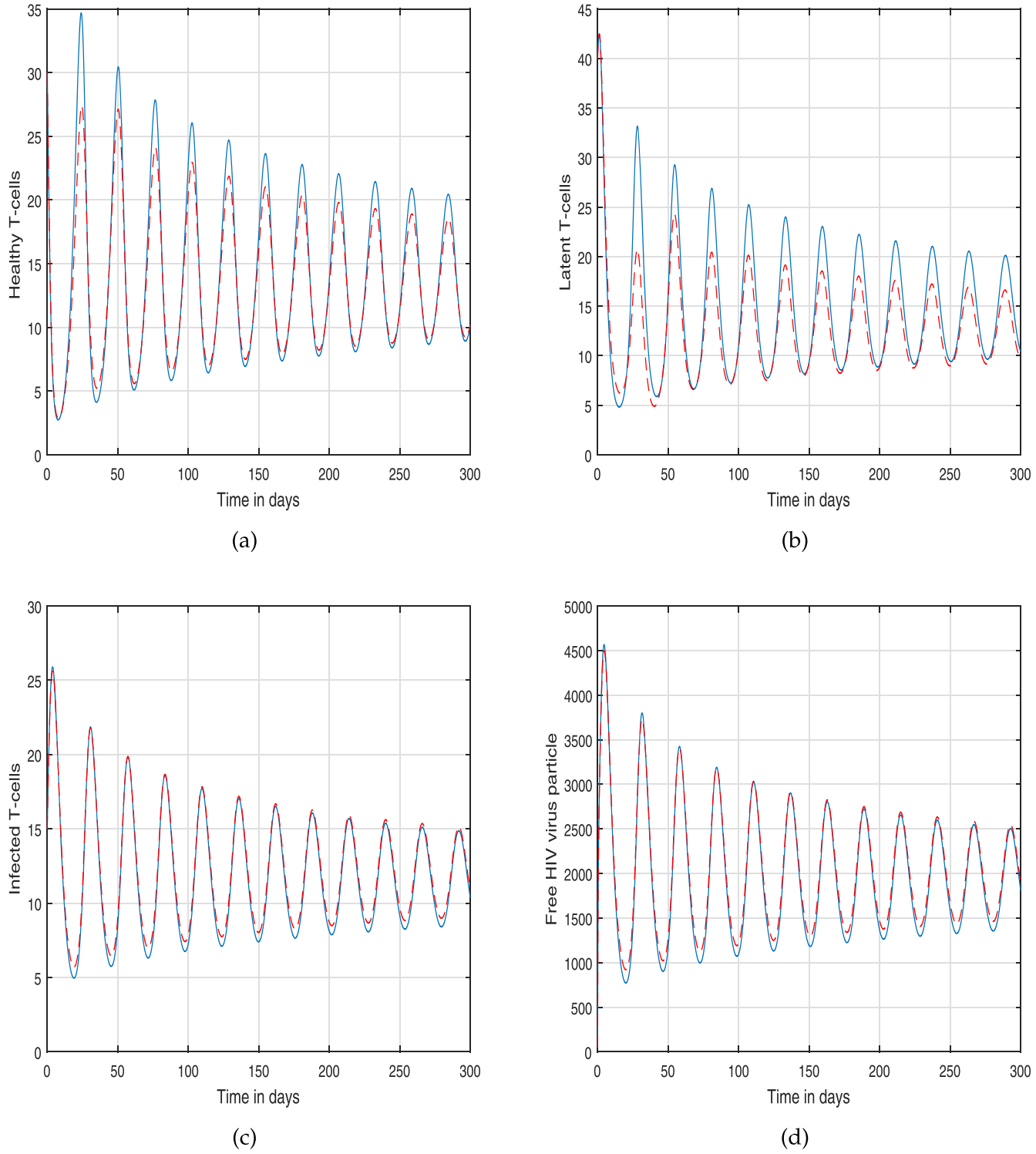

Figure 1. Performing a time series analysis of the prescribed system (4) of HIV infection, with the fractional order $\xi = 0.96$, and values of s and r established as 1.0 and 0.5, respectively.

where ${}_0^{\mathrm{CF}}\mathrm{D}_t^{\xi}$ denotes a fractional derivative of the order ξ by CF. The assessment of our proposed model for HIV infection will rely on a fundamental comprehension of the innovative fractional operator, the CF, which will be elucidated in the subsequent section of the article.

3. Theory of fractional calculus

Here, we present the basic concepts and results of the fractional CF derivative which will be used for further analysis. The following is a list of the basic fundamental ideas:

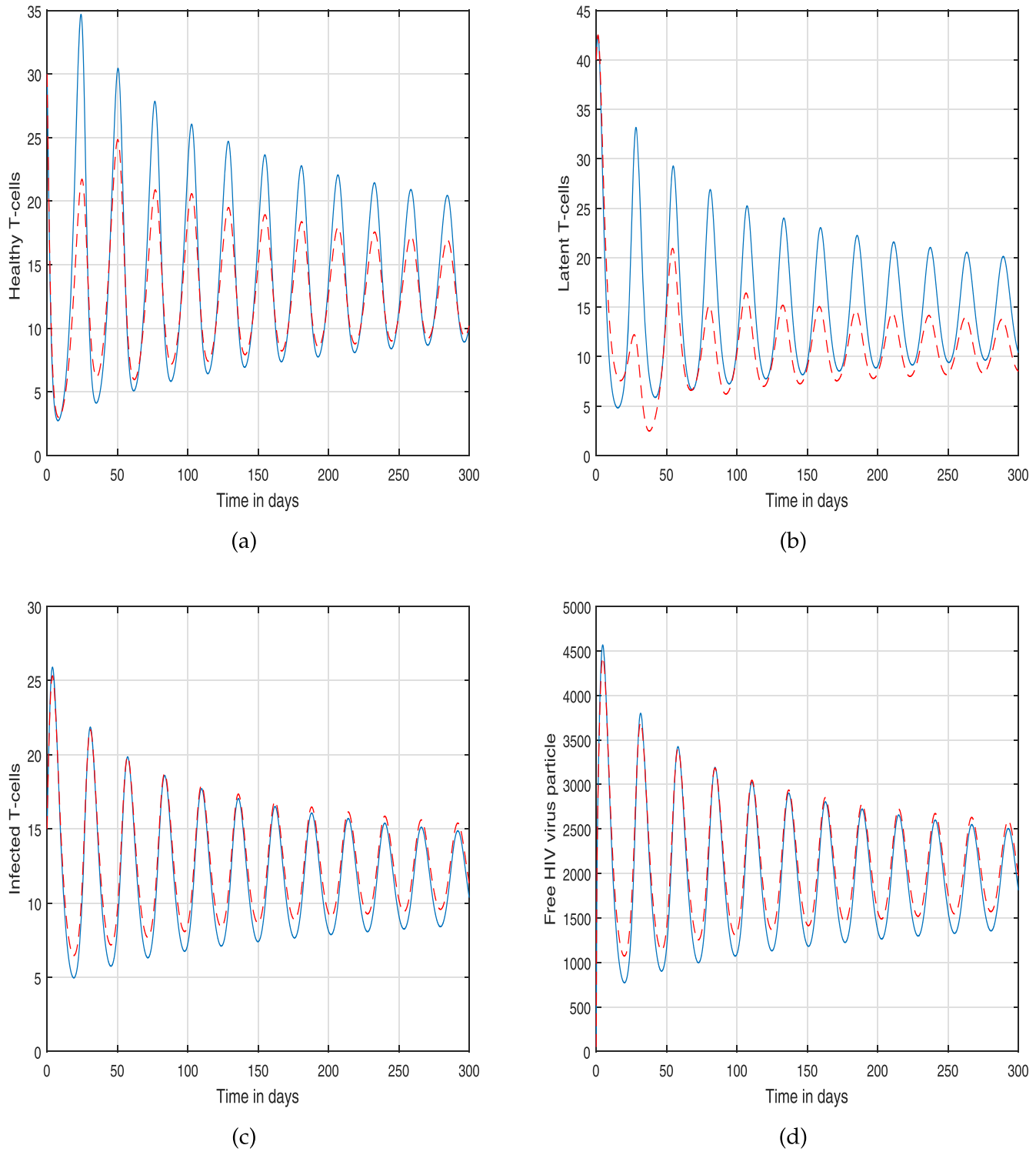

Figure 2. Performing a time series analysis of the model (4) of HIV, employing a fractional order $\xi = 0.92$, a cellular infection rate of $s = 1.0$ and parameter $r = 0.5$.

Definition 1. If f ∈ G^1(a, b) is true and in the case that b exceeds a, then the fractional operator in [41] is given by

$$\mathrm{D}_t^{\xi}(\mathrm{f(t)}) = \frac{\mathcal{U}(\xi)}{1-\xi}\int_{\mathrm{a}}^{\mathrm{t}} f'(\mathrm{y})\mathrm{e}^{\left[-\xi\frac{\mathrm{t-y}}{1-\xi}\right]}\mathrm{dy}, \tag{5}$$

where $\xi \in [0, 1]$ is the order of the operator and $\mathcal{U}(\tau)$ stands for normalcy with $\mathcal{U}(0) = \mathcal{U}(1) = 1$ [41]. Furthermore, the fractional derivative is as follows when $f \notin G^1(a, b)$:

$$\mathrm{D}_t^{\xi}(\mathrm{f(t)}) = \frac{\xi\mathcal{U}(\xi)}{1-\xi}\int_a^t (f(\mathrm{t}) - f(\mathrm{y}))\mathrm{e}^{\left[-\xi\frac{\mathrm{t-y}}{1-\xi}\right]}\mathrm{dy}. \tag{6}$$

Remark 1. Consider the following: $\beta = \frac{1-\xi}{\xi} \in [0, \infty)$ and $\xi = \frac{1}{1+\beta} \in [0, 1]$, equation (6) can thus be expressed as

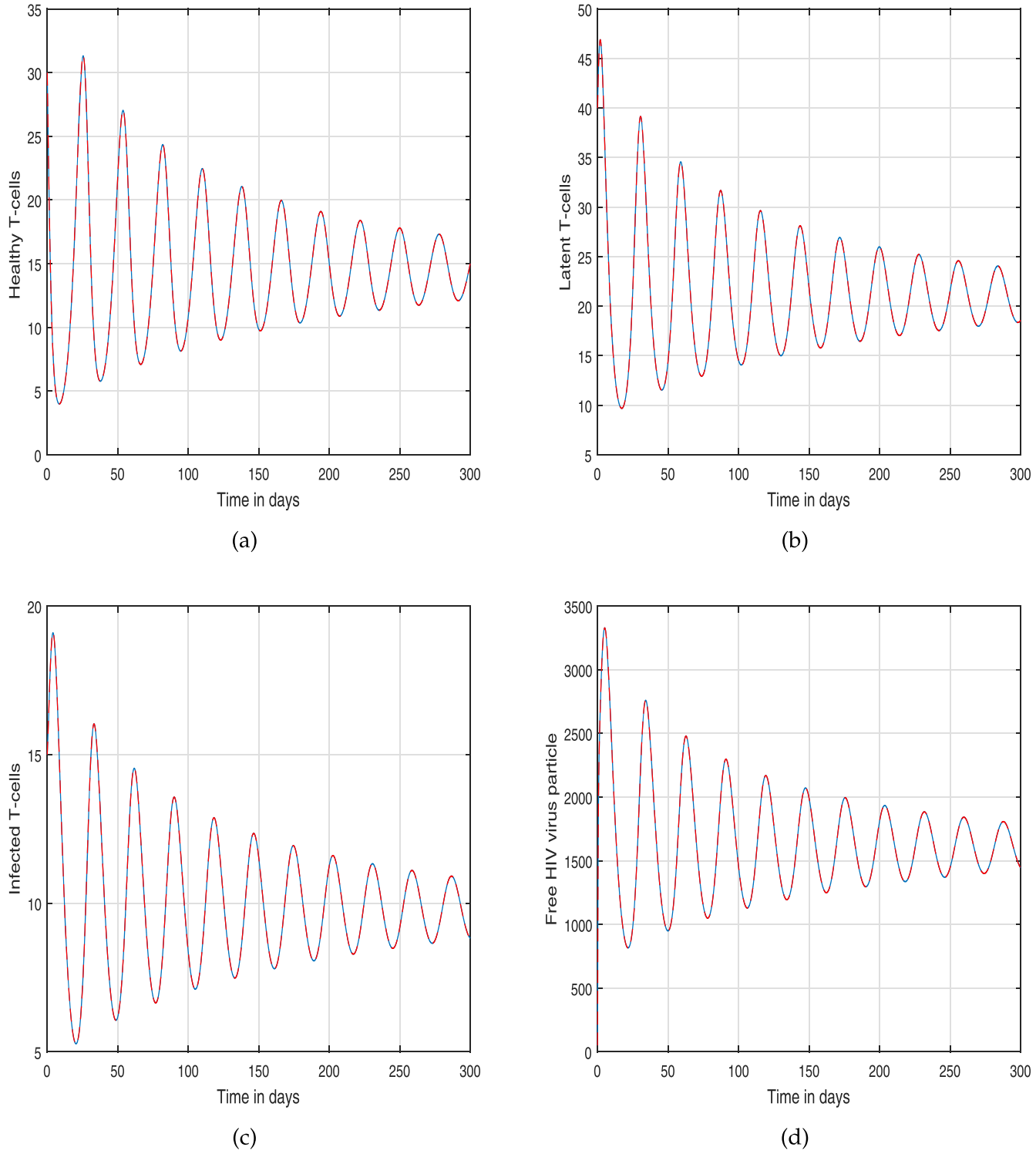

Figure 3. Illustration of the dynamical behavior of the model (4) of HIV by taking the input parameter $k_2 = 1.4 \times 10^{-1}$ instead of $k_2 = 2.4 \times 10^{-1}$ with $\xi = 1.0$.

follows:

$$\begin{aligned} \mathrm{D}_t^{\xi}(\mathrm{f}(\mathrm{t})) &= \frac{N(\beta)}{\beta}\int_{\mathrm{a}}^{\mathrm{t}} f'(\mathrm{y})\mathrm{e}^{\left[-\frac{t-y}{\beta}\right]}\mathrm{dy},\ \mathrm{N}(0) \\ &= N(\infty) = 1, \end{aligned} \tag{7}$$

where $\beta \in [0, \infty]$ and $N(\beta)$ is the normalization of $\mathcal{U}$.

Furthermore, as

$$\lim_{\beta \longrightarrow 0} \frac{1}{\beta}\exp\left[-\frac{\mathrm{t}-\mathrm{y}}{\beta}\right] = \delta(\mathrm{t}-\mathrm{y}). \tag{8}$$

We now proceed to the explanation of the fractional integral, originally presented in [42].

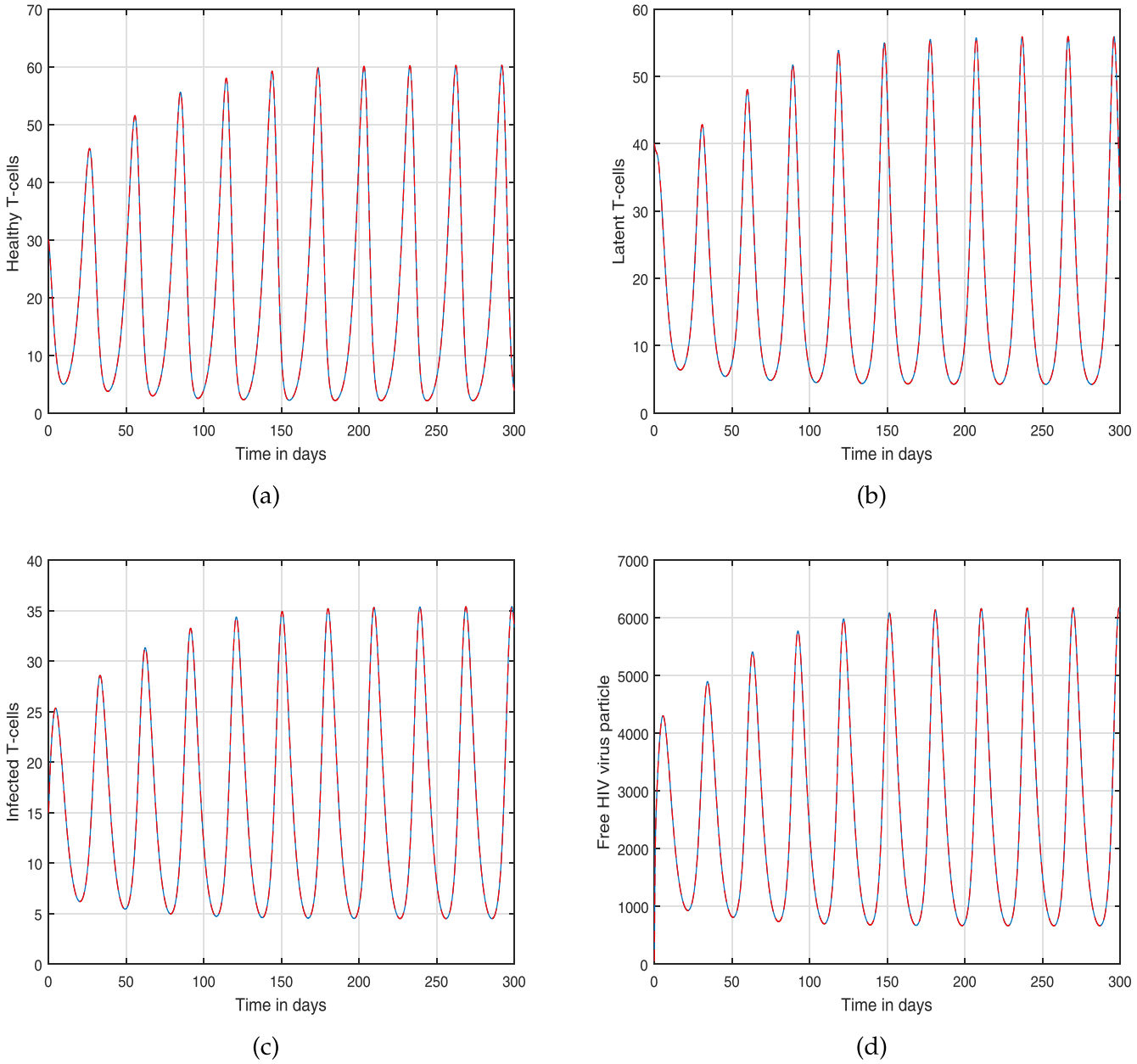

Figure 4. Analyzing the solution trajectories of the system (4) representing HIV infection through a graphical perspective, wherein we consider the input parameter as $\alpha = 0.4 \times 10^{-3}$, instead of the previously used value of $\alpha = 5.4 \times 10^{-3}$ while maintaining $\xi = 1.0$.

Definition 2. The fractional integral of CF for a given function f is given as:

$$
\begin{aligned}
{}^{\mathrm{CF}}I^{\xi}_{\mathrm{tf(t)}} &= \frac{2(1-\xi)}{(2-\xi)\mathcal{U}(\xi)}v(\mathrm{t}) \\
&+\frac{2\xi}{(2-\xi)\mathcal{U}(\xi)}\int_0^t v(\mathrm{u})\mathrm{du}, \quad \mathrm{t} \geqslant 0.
\end{aligned} \tag{9}
$$

where ξ is the order of the integral such that $0 < \xi < 1$ and ${}^{\mathrm{CF}}\mathrm{D}^{\xi}\mathrm{f(t)} = v(\mathrm{t})$.

Remark 2. More examination of the above-mentioned definition 2 reveals that

$$
\frac{2(1-\xi)}{(2-\xi)\mathcal{U}(\xi)} + \frac{2\xi}{(2-\xi)\mathcal{U}(\xi)} = 1, \tag{10}
$$

where $\mathcal{U}(\xi) = \frac{2}{2-\xi}$, $0 \leqslant \xi \leqslant 1$. Nieto and Losada in [42] presented a new Caputo derivative of order ξ utilizing equation (10), which is obtained by

$$
{}^{\mathrm{CF}}\mathrm{D}^{\xi}_{t}(\mathrm{f(t)}) = \frac{1}{1-\xi}\int_0^t \exp\left[\xi\frac{\mathrm{t}-\mathrm{y}}{1-\xi}\right]f'(\mathrm{y})\mathrm{dy}, \quad \mathrm{t} \geqslant 0. \tag{11}
$$

4. Numerical scheme for the model

The key objective of the current section is to numerically illustrate how the recommended fractional model (4) of HIV infection behaves dynamically. In order to demonstrate the

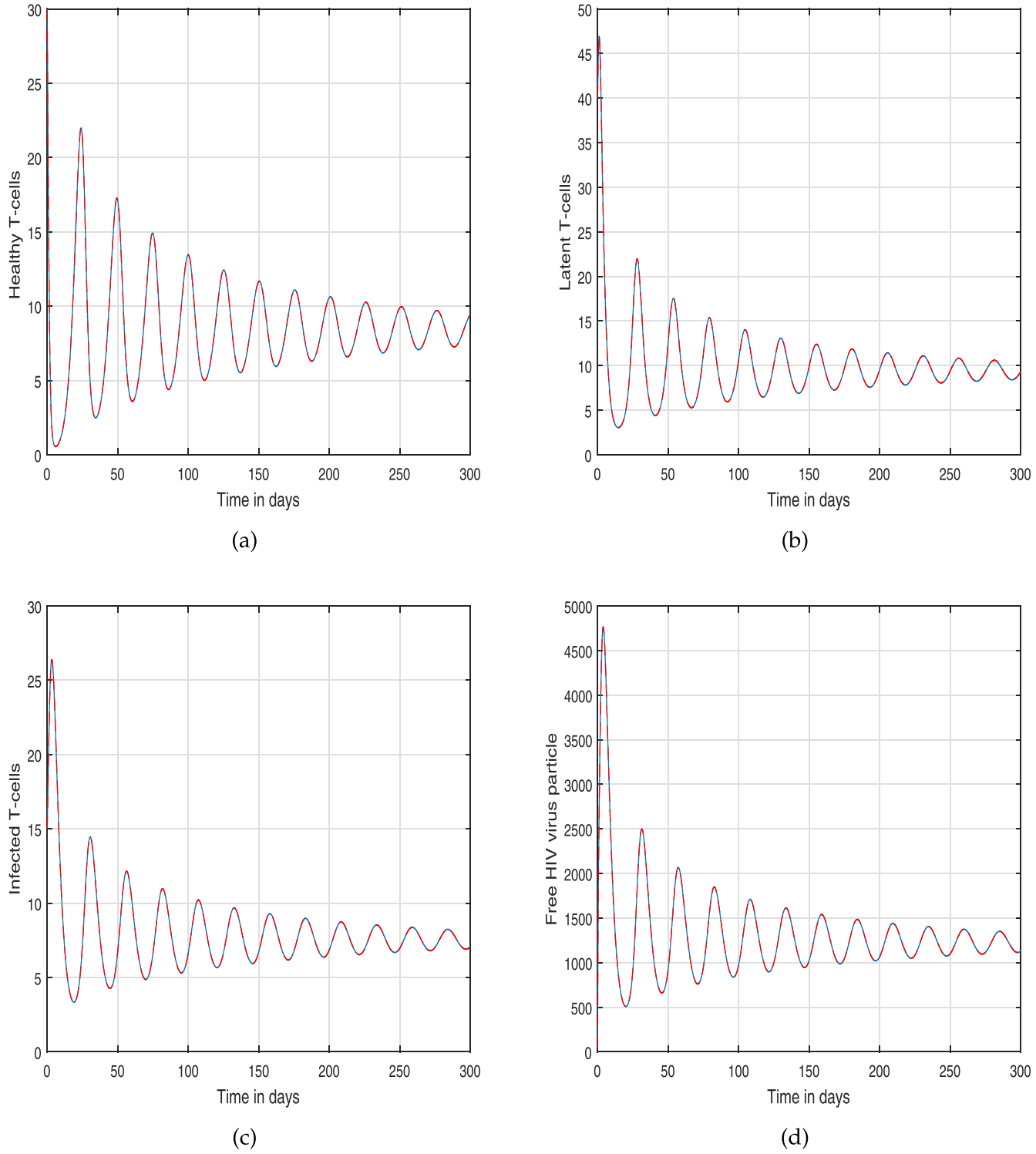

Figure 5. Visualizing the solution pathways of the model (4) describing HIV, where we replace the input parameter k with $k = 4.4 \times 10^{-2}$ in lieu of the previous value of $k = 2.2 \times 10^{-2}$ while maintaining $\xi = 1.0$.

dynamics of the CF fractional systems, the literature [43–45] has offered multiple numerical methods. To illustrate the dynamics of our fractional system (4), which is more reliable, practical, and stable, we shall employ the [45] technique. Our suggested fractional model of HIV infection is first expressed using the Volterra type, and it is then further simplified using the fundamental theorem of integration. Our proposed model of HIV infection's first equation states that in order to obtain the numerical scheme,

$$w_1(\mathrm{t}) - \mathrm{w}_1(0) = \frac{1-\xi}{\mathcal{U}(\xi)}\mathcal{K}_1(\mathrm{t}, \mathrm{w}_1) + \frac{\xi}{\mathcal{U}(\xi)}\int_0^{\mathrm{t}} \mathcal{K}_1(\omega, \mathrm{w}_1)\mathrm{d}\omega. \tag{12}$$

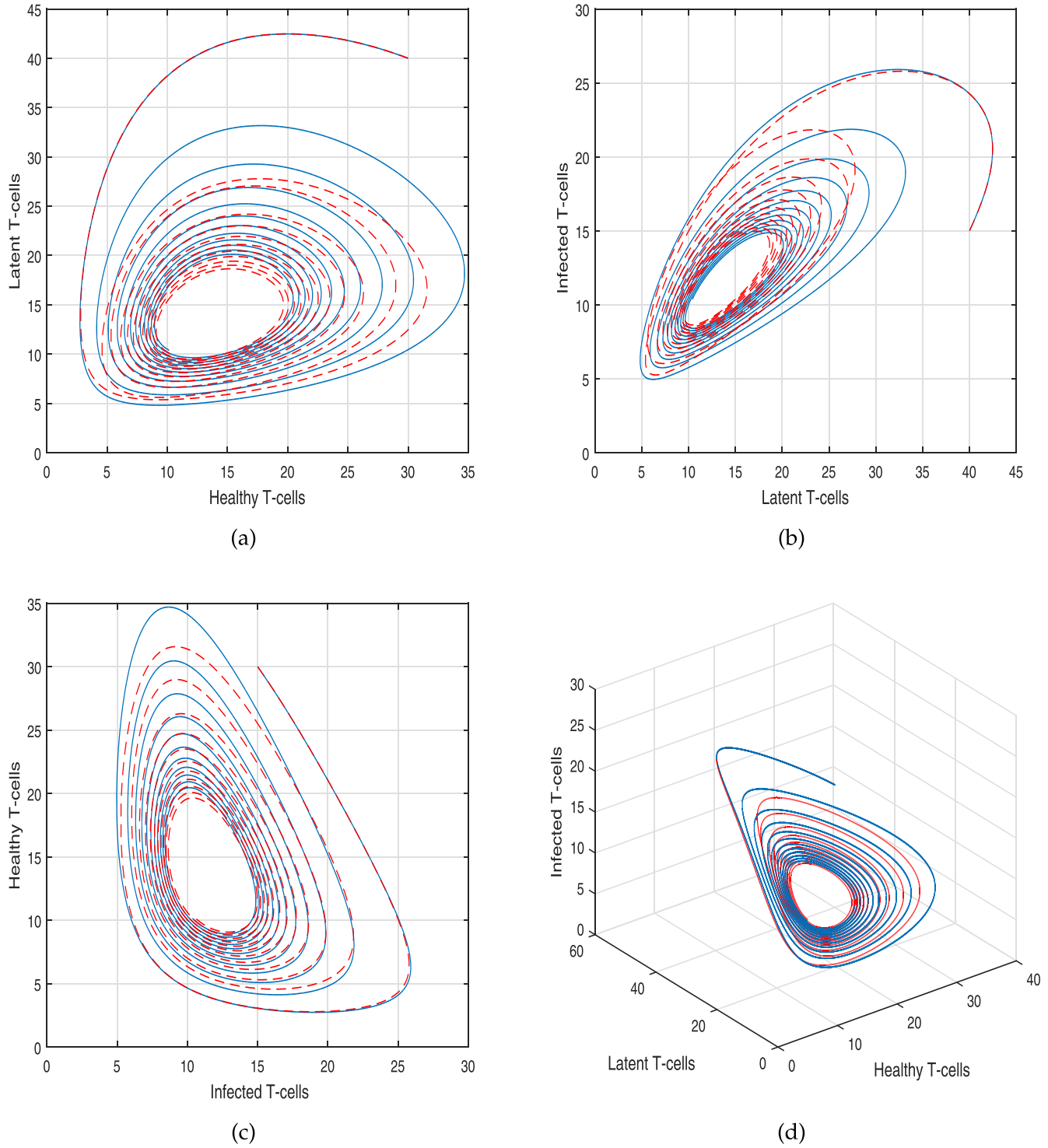

Figure 6. Analyzing the phase portrait of the recommended system (4) of HIV with the input parameter $s = 1.0$, $r = 0.5$ and $\xi = 0.98$.

Then, using the time $t = t_{v+1}$, $v = 0, 1,...,$; Consequently, the below mentioned results are obtained

$$w_1(t_{v+1}) - w_1(0) = \frac{1-\xi}{\mathcal{U}(\xi)}\mathcal{K}_1(t_v, \mathrm{w}_1(t_v)) + \frac{\xi}{\mathcal{U}(\xi)}\int_0^{t_{v+1}} \mathcal{K}_1(t, \mathrm{w}_1)\mathrm{d}t. \quad (13)$$

and

$$w_1(t_v) - w_1(0) = \frac{1-\xi}{\mathcal{U}(\xi)}\mathcal{K}_1(t_{v-1}, w_1(t_{v-1})) + \frac{\xi}{\mathcal{U}(\xi)}\int_0^{t_v} \mathcal{K}_1(t, w_1)\mathrm{d}t. \quad (14)$$

The following formula is used to determine how the system's

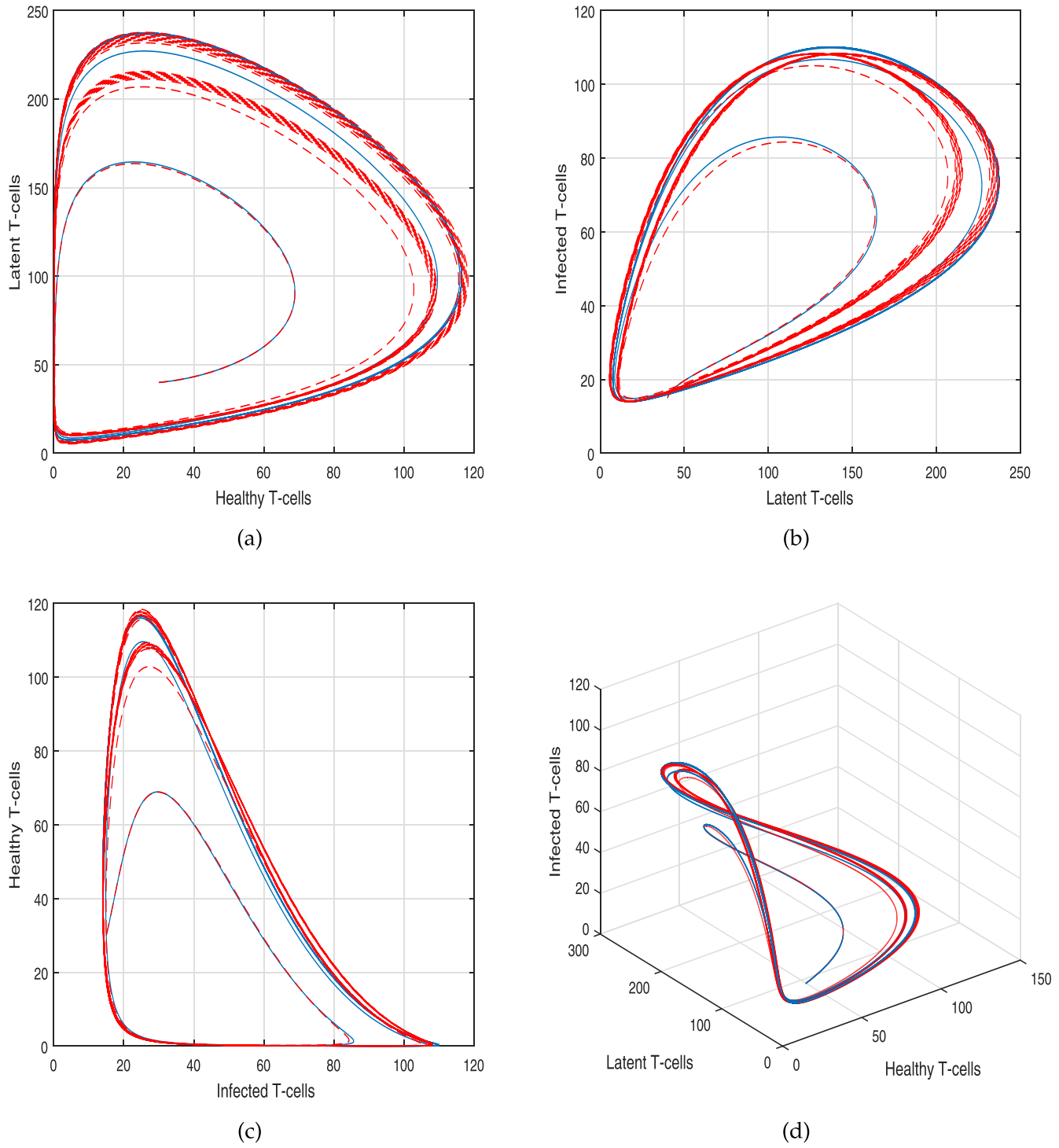

Figure 7. Plotting the phase portrait of the recommended system (4) of HIV infection with the input parameter $s = 0.50$, $r = 1.5$ and $\xi = 0.98$.

consecutive terms differ:

$$w_{1_{v+1}} - w_{1_v} = \frac{1-\xi}{\mathcal{U}(\xi)}(\mathcal{K}_1(t_v, w_{1_m}) - \mathcal{K}_1(t_{v-1}, w_{1_{v-1}})) + \frac{\xi}{\mathcal{U}(\xi)}\int_v^{t_{v+1}} \mathcal{K}_1(t, w_1)\mathrm{d}t. \quad (15)$$

We also use an interpolation polynomial to the above-mentioned approximation function $\mathcal{K}_1(t, w_1)$ in the time interval $[t_r, t_{r+1}]$ and obtain

$$\mathcal{H}_{\mathrm{r(t)}} \cong \frac{\mathcal{K}_1(t_r, w_r)}{g}(\mathrm{t} - \mathrm{t}_{r-1}) - \frac{\mathcal{K}_1(\mathrm{t}_{r-1}, \mathrm{w}_{r-1})}{\mathrm{g}}(\mathrm{t} - \mathrm{t}_r), \quad (16)$$

The equation $\mathcal{H}_{\mathrm{r(t)}}$ is applied in order to calculate the value of the subsequent integral, where g is the time spent and

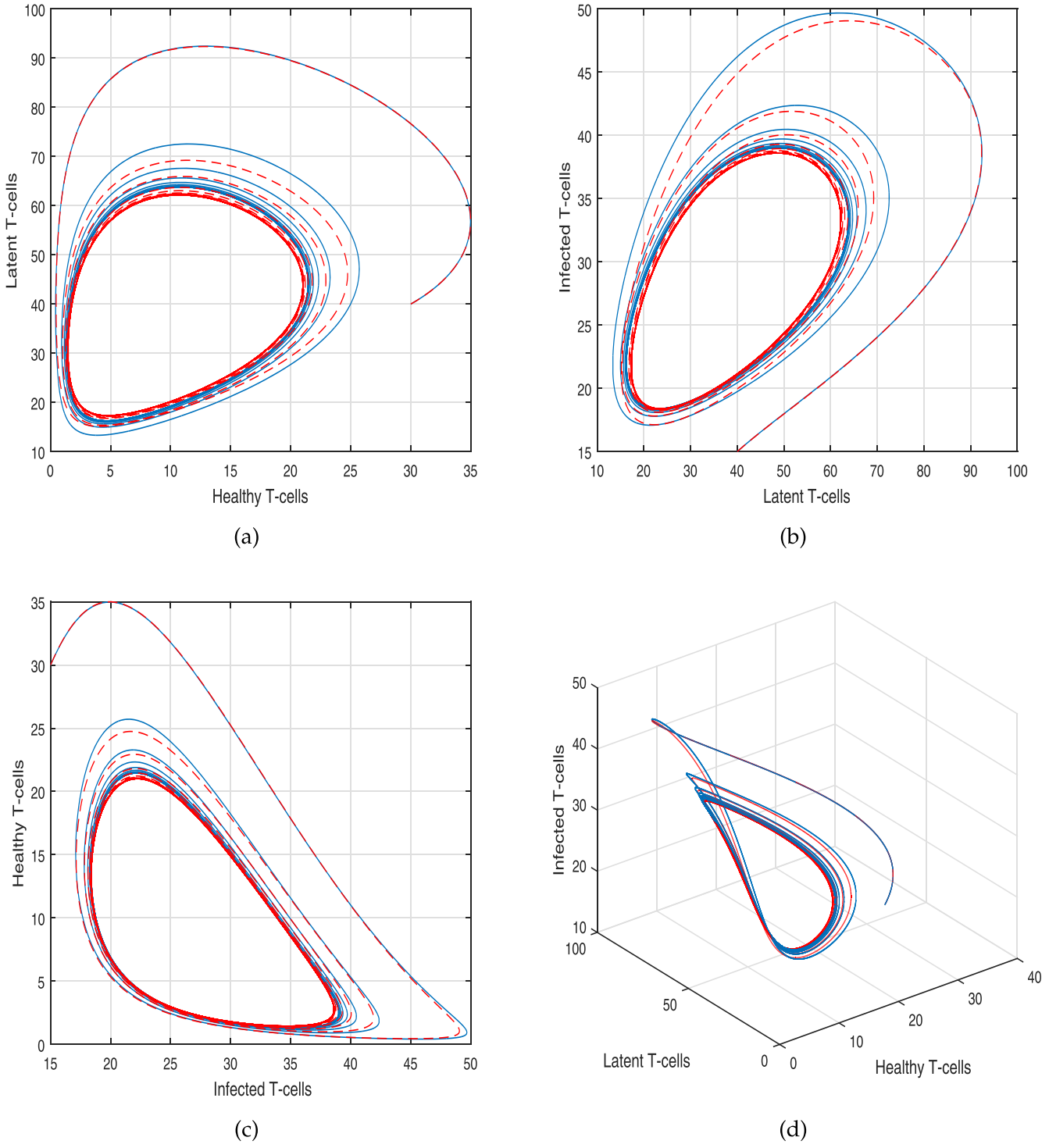

Figure 8. Illustration of the phase portrait of the recommended system (4) of HIV infection with the input parameter $s = 5.0$, $r = 0.5$ and $\xi = 0.98$.

$g = t_v - t_{v-1}$.

$$\int_v^{t_{v+1}} \mathcal{K}_1(t, w_l)dt = \int_v^{t_{v+1}} \left(\frac{\mathcal{K}_1(t_v, w_{l_v})}{g}(t - t_{v-1}) - \frac{\mathcal{K}_1(t_{v-1}, w_{l_{v-1}})}{g}(t - t_v) \right) dt,$$
$$= \frac{3g}{2}\mathcal{K}_1(t_v, w_{l_v}) - \frac{g}{2}\mathcal{K}_1(t_{v-1}, w_{l_{v-1}}). \quad (17)$$

By changing the value of (17) in equation (15), we obtain the following.

$$w_{l_{v+1}} = w_{l_v} + \left(\frac{1-\xi}{\mathcal{U}(\xi)} + \frac{3\xi g}{2\mathcal{U}(\xi)} \right) \mathcal{K}_1(t_v, w_{l_v}) - \left(\frac{1-\xi}{\mathcal{U}(\xi)} + \frac{\xi g}{2\mathcal{U}(\xi)} \right) \mathcal{K}_1(t_{v-1}, w_{l_{v-1}}), \quad (18)$$

The above is the approximation of the first equation of our fractional system (4) of HIV infection. For the 2nd and 3rd equations of system 4, we obtained the following using the same technique:

$$w_{2_{v+1}} = w_{2_v} + \left(\frac{1-\xi}{\mathcal{U}(\xi)} + \frac{3\xi g}{2\mathcal{U}(\xi)}\right)\mathcal{K}_2(\mathrm{t}_v, \mathrm{w}_{2_v}) - \left(\frac{1-\xi}{\mathcal{U}(\xi)} + \frac{\xi g}{2\mathcal{U}(\xi)}\right)\mathcal{K}_2(t_{v-1}, w_{2_{v-1}}), \tag{19}$$

and

$$w_{3_{v+1}} = w_{3_v} + \left(\frac{1-\xi}{\mathcal{U}(\xi)} + \frac{3\xi g}{2\mathcal{U}(\xi)}\right)\mathcal{K}_3(\mathrm{t}_v, \mathrm{w}_{3_v}) - \left(\frac{1-\xi}{\mathcal{U}(\xi)} + \frac{\xi g}{2\mathcal{U}(\xi)}\right)\mathcal{K}_3(\mathrm{t}_{v-1}, w_{3_{v-1}}). \tag{20}$$

For the CF operator, this method uses a two-step Adams–Bashforth technique that takes into consideration both the exponential decay rule and the nonlinearity of the kernel. In [45], the stability and convergence of the method have been discussed. In this work, we mainly focus on the dynamical behavior of the system to conceptualize the impact of different parameters on the system. The analytic aspects of the recommended system will be investigated in our future work. Also, we will perform comparative analysis of the numerical method with existing methods [34, 35].

5. Numerical results

HIV/AIDS still has a serious detrimental impact on affected families despite major global efforts to control it. Reduced income from employment, greater health-care costs, and a loss of capital required to close the gap between income and expenses all add to this burden. In order to prevent these losses, it is imperative to do research into the fundamental causes of HIV infection. The main goal of this research is to visualize the dynamical behavior of the system in order to understand how various factors affect it. We examine the effects of the input variables on the dynamics of HIV using a variety of numerical scenarios. For numerical purposes, we take the values of input parameters from table 1 while the values of state-variables are assumed to be $T(0) = 30$, $L(0) = 40$, $I(0) = 15$ and $V(0) = 50$.

We run many simulations to see how input elements affect the system and to illustrate how these parameters affect the system, how HIV spreads and how it is controlled inside the body of the host. Figures 1–2 show the system's oscillatory behavior selecting fractional-order values. The system's solution pathways have been discovered to be significantly impacted by the fractional parameter. It has been highlighted that the parameter ξ exerts a favorable influence on the dynamics of HIV and could potentially serve as a preventive measure. In figures 3–5, we have presented the alterations in the input parameters k_2, α, and k, demonstrating their respective impacts on the solution trajectories of the system. Additionally, in figures 6–8, we have depicted the phase portraits of the recommended HIV infection model under varying values of r and s, providing insights into the long-term behavior of the system. In figure 7, we assumed the values of $\mathrm{s} = 0.50$, $\mathrm{r} = 1.5$ and $\xi = 98$ to comprehend the dynamical behavior of the HIV model which provides a deeper understanding of the system for effective control strategies. The presence of chaos and oscillations in the recommended model can be attributed to the system's inherent nonlinearity. Notably, our observations underscore the substantial influence wielded by the input parameters, with a reduction in ξ holding the potential to mitigate the incidence of HIV infections. Consequently, it is advisable for policymakers to consider the manipulation of these input factors as a potential strategy.

Our numerical findings investigated the oscillatory behavior and phase portrait of the system with different input values of the parameters. This information can be used to predict the long-term behavior of the system, including whether it will converge to a steady state or exhibit periodic or chaotic oscillations. These phenomena are highly sensitive to the input parameters of the system. The presence of chaos in the system is reflected in its phase portrait, with the trajectories exhibiting a complex and irregular pattern. Also, we predict that these phenomena are due to the strong nonlinearity of the system and are dependable on each other. These issues are important because of the essential knowledge they provide about the HIV infection process.

Delays are important in biological modeling because they reflect the temporal intricacies of biological phenomena, allowing for a more accurate representation of the dynamic and time-dependent nature of living systems [47, 48]. Incorporating delays in mathematical models enhances their realism and predictive power in understanding and simulating complex biological dynamics [49]. Delays in the dynamics of HIV are motivated by the biological processes inherent to the infection, including the time it takes for infection and replication, immune response activation, treatment initiation, viral load dynamics, latency in reservoirs, development of immune memory, and the evolution of drug resistance. In future work, we will incorporate a delay in our model to enhance their accuracy and provide a more realistic representation of HIV dynamics in the human body.

6. Conclusion

HIV is unequivocally acknowledged as a pathogenic agent that preferentially targets the immune system, precipitating a diminution in T-cell populations and concomitantly compromising the host organism's immune competence, thereby impeding its ability to mount effective defenses against additional pathogenic agents. Presently, the global public health community confronts a substantial challenge posed by the intricate interplay between HIV and T-cells. In our pursuit to comprehend this complex phenomenon, we have devised a mathematical model. Our model intricately captures the interactions involving HIV viruses, infected T-cells, and healthy T-cells. To characterize this HIV system, we have

harnessed the fractional operator of Caputo–Fabrizio. By employing numerical techniques, we have unveiled the dynamic behavior of the HIV system under various scenarios. Our investigations have demonstrated the profound influence of fractional-order on the solution pathways of HIV infection, as we systematically varied crucial input parameters associated with infection management and prevention. Additionally, by scrutinizing the phase portrait, we have gleaned pivotal insights into system behavior, revealing the presence of periodic orbits, limit cycles, and various types of attractors. In future work, we intend to investigate our model of HIV infection to assess the impact of medical advancements on the virus' progression and explore innovative treatment modalities. Moreover, we will improve our model to incorporate the effects of vaccinations and medications, enabling a comprehensive analysis of their influence on the system.

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