

Research Article

Dynamical Behaviour and Chaotic Phenomena of HIV Infection through Fractional Calculus

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The infection of human immunodeficiency virus (HIV) is a serious and potentially incurable infection. There is no cure for HIV and is a public health issue around the world. That is why, it is valuable to investigate the intricate phenomena of HIV infection and provide some control interventions to lessen its economic burden. In this research work, the dynamics of HIV via fractional calculus to conceptualize the intricate phenomena of this viral infection has been formulated and conceptualized. We have shown the rudimentary concept of fractional calculus in Atangana–Baleanu framework. A novel numerical technique is presented for the chaotic and dynamic behaviour of the proposed model. The oscillatory and chaotic phenomena of the system have been shown with the fluctuation of different input factors of the system. Furthermore, we have shown the affect of fractional order on the proposed system of HIV infection. Most critical input parameters are highlighted through numerical simulations and suggested control intervention to the policy makers. Finally, we have shown the stability result and the convergence condition for the proposed numerical scheme.

1. Introduction

Mathematics and Biology are extricable linked in different research areas. Genetics, environmental science, population dynamics, medical science, and other fields all benefit from mathematical biology. Mathematics is used to conceptualize, understand, and visualize intricate biological phenomena [1, 2]. It is well known that the development of theoretical principles for biology is referred to theoretical biology while investigation of biological phenomena or processes through mathematical tools is known to be mathematical biology. This implies that mathematics plays an important role to interrogate a biological system. When traditional lab tests are either unfeasible or too difficult to answer a research topic, biologists turn to mathematics to create models that highlight the key factors of transmission process of an infection. Scientists can use these approaches to forecast the likelihood of certain outcomes while simultaneously fine-tuning their research subjects. Different biological events

and processes can be described mathematically in terms of delay, impulsive, stochastic [3], fractional and ordinary differential equations, and so on [4]. In formulating these mathematical models, various assumptions, laws, and axioms that govern these processes are used to demonstrate the complex dynamics of biological events. HIV infection is a serious public health concern worldwide, having claimed about 33 million lives to date, and a slew of mathematical models for the human immune system has been created to depict the complete spectrum of infection. The interaction of human immunodeficiency (HIV) and immune system has been described. HIV has been reported to be an effective agent in achieving immunodeficiency syndrome (AIDS) which impairs the capability of the body to fend against various illnesses. HIV infection is an incurable fatal disease that has killed the lives of about 44,200,000 people. It is reported in 2020 that 37.6 million people are infected by HIV around the world and 1.5 million people are newly infected. However, therapy that works, caring, assessment, as well as

protection of HIV has led to people living longer and healthier lives with HIV. When the HIV virus penetrates a healthy individual's body, it propagates rapidly and causes CD4⁺ T-cells destruction that act on the immune system.

The symptoms and signs of HIV infection in its initial phases include the flu, nighttime cravings, coughing, losing weight, a headache, diarrhoea, sunburn, body aches and joint pain, tonsillitis, as well as a dry mouth. The process is still in its initial phases, the virus carries more weight into the bloodstream, and the HIV infection spreads more easily through the body than at other stages. In addition, HIV-infected viruses are spread through body fluids (blood, tears, urine, saliva, and so on) and infect the uninfected person. It is really obvious that CD4⁺ T-cells have the fighting ability against infections; additionally, those same CD4⁺ T-cells play a significant role as in immune system's modification, so their precession would have a wide range of consequences which can entirely disrupt its immune system's functioning. Because the retention time of such lymphocytes is utilized. Therefore, we define the phase of HIV infection, determining their importance using a mathematical formulation becomes essential. For effective illustration of the interaction of CD4⁺ T-cells and HIV-infected viruses, a number of mathematical models were developed.

Several scholars investigated the kinetics of HIV infection using various assumptions. The authors in [5] studied the interaction of CD4⁺ T-cells and HIV virus through a mathematical model. Perelson and Nelson [6] also developed a novel model of HIV incorporating the following classes: late infection, constantly infected and non-infected, and the HIV viral particle community. Several recognized characteristics of AIDS were proven their research clinically [6]. Following that, Culshaw and Raun [7] formulated the dynamics of HIV infection and studied HIV dynamics. Bushnaq et al. [8] performed research in which they explored the stability and persistence of HIV/AIDS model, as well as the role of recall throughout the biomechanics with HIV infection, using formalized paraphrasing and fractional differentiation. To highlight the dynamic monitoring behaviours of HIV infection, the authors used a range of methodologies to investigate HIV dynamics [9, 10] while the researchers in [11, 12] computationally interrogated the dynamics of HIV. The main objective of this research work is to formulate the dynamics of HIV infection using a variable term from source rather than a fixed quantity for fresh CD4⁺ T-cells. In addition to this, our objective is to visualize the role of input parameters on the output of the system and to investigate the most critical factors of the system for the control of this viral infection.

Non-integer models are famous due to their more valuable, reliable, deeper, and precise knowledge in different areas of science and technology [13, 14]. Due to its inherited characteristics and memory definition, fractional systems [15] perform more accurately. It is also well known that nonlocal behaviour of the system can easily be represented through fractional-calculus [16]. The fraction calculus offered very precise information for such

phenomena, especially for the dynamics of infectious diseases and engineering systems. In fraction calculus, Caputo, Riemann–Liouville, Hilfer, and a few more operations have core laws and have limitations in modeling natural phenomena. Atangana and Baleanu developed a new derivative in 2016 that extended the Mittag–Leffler function to nonlocal and nonsingular cases [16]. This newly developed has been successfully used in different areas of science and engineering [17, 18]. Therefore, the mathematical biologists are interested to investigate the transmission process of different infections through this novel operator to provide accurate results and to conceptualize the contribution of memory in the dynamical behaviour of different diseases. The authors in [19] represented the transmission phenomena of rubella disease through Atangana–Baleanu operators. The transmission phenomena of COVID-19 have been investigated through AB operator in some African countries [20]. This novel operator more accurately represents natural phenomena rather than the previous operators. Thus, we opt to investigate the dynamics of healthy CD4⁺ T-cells, infected CD4⁺ T-cells, and free viruses of HIV infection through fractional-calculus via Atangana–Baleanu operator.

The research work is structured as follows. The fractional formulation of the HIV infection of CD4⁺ T-cells is presented in Section 2 of this article. We introduced a new numerical technique for the analysis of the proposed fractional model in Section 3. In section four, we highlighted the chaotic and oscillatory concepts of the model with fluctuation of different input parameters. Furthermore, the most critical scenario is visualized through these numerical analyses. The proposed numerical scheme's convergence and stability findings have been demonstrated. The last portion of the article contains the entire work's concluding remarks.

2. Structure of HIV Dynamics

The most critical necessity for understanding HIV/AIDS infections is to understand the interaction of HIV and CD4⁺ T-cells. It is reported that these cells are created throughout the bone marrow and moved to the medulla and then went through special differentiation for maturation into uninfected CD4⁺ T-cells. In the human body, the maximal weight is achieved by thymus. The thymus in humans achieves its maximal weight at maturation stage and then gradually grows more complicated. The effect of thymic drainage from adults is small even though the adult thymus is active and its few lymphocytes function as recruits for T-cells and uninfected T-cells. The provided model focuses on CD4⁺ T-cells. The number of CD4⁺ T-cells that tell us more about the early symptoms can be used to assess the persistence of HIV infection.

The current work is interested to interrogate the oscillatory and path tracking behaviour of the HIV dynamics. These analyses detect the most critical factor and also help the policy makers to identify input factor for the prevention of infection. The assumptions in [11] give the following mathematical descriptions:

$$\begin{aligned} \frac{dT}{dt} &= s - \mu_T T - kVT + rT \left(1 - \frac{T+I}{T_{\max}}\right), \\ \frac{dI}{dt} &= kVT - \mu_I I, \\ \frac{dV}{dt} &= N\mu_I I - \mu_V V. \end{aligned} \tag{1}$$

Here, the state variables $T(t)$ and $I(t)$ indicate the concentration of healthy and infected while $V(t)$ indicates HIV virus freely available in the blood, respectively. In Table 1, we have shown the initial conditions and all the parameters with description. In the next section, we will extend the model with a fractional framework.

2.1. HIV Infection’s Fractional Dynamics. Here, a constant source term s introduced in [11] is replaced by the variable $s(V) = s \exp(-\kappa V)$ in the proposed model. The new source term included the model indicating the amount of healthy T-cells generated by thymus as a function of viral load concentration. Because the greater viral load lowers the generation of healthy T lymphocytes, the source term is seen as a variable rather than a constant. Further explanation and detail are given in the research [21–23]. Then, the above system of differential equation (1) in fractional framework with our new assumptions is given by

$$\begin{aligned} {}_0^{ABC}D_t^\ell T &= s \exp(-\kappa V) - \mu_T T + rT \left(1 - \frac{T+I}{T_{\max}}\right) - kVT, \\ {}_0^{ABC}D_t^\ell I &= kVT - \mu_I I, \\ {}_0^{ABC}D_t^\ell V &= N\mu_I I - \mu_V V - kVT, \end{aligned} \tag{2}$$

where ${}_0^{ABC}D_t^\ell$ indicates the derivative of Atangana–Baleanu in the Caputo sense of order ℓ . The following portion of the study will go through the rudimentary knowledge of ABC derivative, which will be used to analyze our HIV infection model. This fractional derivative has been recently introduced which is successfully utilized in different research fields.

3. Results of Fractional Calculus

Fractional calculus theory is rich in applications and has been applied to many problems in engineering, physics, economics, biology, and many other areas of technology and science. Recent research has shown that they provide more accurate, precise, and reliable results [24, 25]. Here, we introduce the basic idea of fractions for analyzing our system of HIV. In the following, some basic results and concepts of ABC fractional derivative are presented for analysis.

Definition 1. Let us take f such that $g \in H^1(p, q)$, $p < q$, then ABC derivative with order ζ is given as follows:

$${}_p^{ABC}D_t^\zeta g(t) = \frac{B(\zeta)}{1-\zeta} \int_p^t g'(\zeta) E_\zeta \left[-\zeta \frac{(t-\xi)^\zeta}{1-\zeta} \right] d\xi, \tag{3}$$

where ζ belongs to the closed interval $[0, 1]$.

Definition 2. Assume $f(t)$ be any given function, then the integral of the abovementioned operator is indicated by ${}_p^{ABC}I_t^\zeta g(t)$ and is given by

$${}_p^{ABC}I_t^\zeta g(t) = \frac{1-\zeta}{B(\zeta)} g(t) + \frac{\zeta}{B(\zeta)\Gamma(\zeta)} \int_p^t g(\zeta)(t-\xi)^{\zeta-1} d\xi. \tag{4}$$

Here, as the fractional-order ζ approaches to 0, we obtained the initial function.

Theorem 1 (see [16]). *Let us take f such that $f \in C[p, q]$ where f is continuous, then the following holds true:*

$$\|{}_p^{ABC}D_t^\zeta(g(t))\| < \frac{B(\zeta)}{1-\zeta} \|f(t)\|, \text{ with } \|f(t)\| = \max_{p \leq t \leq q} |g(t)|. \tag{5}$$

Furthermore, it fulfills the following:

$$\|{}_p^{ABC}D_t^\zeta g_1(t) - {}_p^{ABC}D_t^\zeta g_2(t)\| < \varrho_1 \|g_1(t) - g_2(t)\|, \tag{6}$$

which is called Lipschitz condition.

Theorem 2 (see [16]). *Let us take a fractional system of the form as follows:*

$${}_p^{ABC}D_t^\zeta g(t) = u(t), \tag{7}$$

the above system has the following unique solution:

$$g(t) = \frac{1-\zeta}{B(\zeta)} u(t) + \frac{\zeta}{B(\zeta)\Gamma(\zeta)} \int_p^t u(\zeta)(t-\xi)^{\zeta-1} d\xi. \tag{8}$$

$${}_0^{ABC}D_t^\ell y(t) = \mathcal{K}(t, y(t)). \tag{9}$$

4. Numerical Approach for Fractional Derivative

Here is a numerical method that emphasises the fractional model of HIV infection’s oscillatory behaviour and chaos. Numerous numerical techniques have been developed and described to visualize fractional order models. For the fractional dynamics of HIV, we will use a new scheme presented in [26] to describe the solution pathway in (2). To derive the numerical schemes needed for our system (2), we first adopt the following fractional system.

Then, by the theory of fractional calculus, we get

$$y(t) - y(0) = \frac{1-\ell}{ABC(\ell)} \mathcal{K}(t, y(t)) + \frac{\ell}{ABC(\ell)\Gamma(\ell)} \int_0^t (t-\tau)^{\ell-1} \mathcal{K}(\tau, y(\tau)) d\tau. \tag{10}$$

TABLE 1: Description of state-variables and input parameters with corresponding values.

Symbols	Parameter and state-variable interpretation	Values
V_0	HIV virus concentration	Assumed
T_{\max}	Maximum number of healthy T-cells	1500 mm^{-3}
N	The quantity of virus generated by infected T-cells	Assumed
μ_V	Death rate of HIV virus	2.4 day^{-1}
μ_I	Death rate of infected T-cells	0.3 day^{-1}
μ_T	Death rate of healthy T-cells	0.02 day^{-1}
r	Growth rate of healthy T-cells	3 day^{-1}
s	Healthy T-cells supply rate from precursors	0.1 mm^{-3}
I_0	T-cells population with infection	Assumed
T_0	T-cells population in healthy form	Assumed
k	Infection rate of T-cells by free virus	$2.4 \times 10^{-5} \text{ days}^{-1}$

Here, we take the time $t = t_n$, then the above implies that

$$y(t_n) - y(0) = \frac{1 - \ell}{\text{ABC}(\ell)} \mathcal{K}(t_{n-1}, y(t_{n-1})) + \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)} \int_0^{t_n} (t_n - \tau)^{\ell-1} \mathcal{K}(\tau, y(\tau)) d\tau, \quad (11)$$

and for t_{n+1} , we get

$$y(t_{n+1}) - y(0) = \frac{1 - \ell}{\text{ABC}(\ell)} \mathcal{K}(t_{n+1}, y(t_{n+1})) + \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\ell-1} \mathcal{K}(\tau, y(\tau)) d\tau. \quad (12)$$

From the above, we can find the difference as follows:

$$y(t_{n+1}) - y(t_n) = \frac{1 - \ell}{\text{ABC}(\ell)} [\mathcal{K}(t_n, y(t_n)) - \mathcal{K}(t_{n-1}, y(t_{n-1}))] + \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\ell-1} \mathcal{K}(\tau, y(\tau)) d\tau - \int_0^{t_n} (t_n - \tau)^{\ell-1} \mathcal{K}(\tau, y(\tau)) d\tau. \quad (13)$$

This further implies that

$$y(t_{n+1}) - y(t_n) = \frac{1 - \ell}{\text{ABC}(\ell)} [\mathcal{K}(t_n, y(t_n)) - \mathcal{K}(t_{n-1}, y(t_{n-1}))] + B_{\ell,1} - B_{\ell,2}, \quad (14)$$

in which

$$B_{\ell,1} = \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\ell-1} \mathcal{K}(\tau, y(\tau)) d\tau. \quad (15)$$

The next step is to get it using an approximation as follows:

$$P(t) \cong \frac{\mathcal{K}(t_n, y_n)}{h} (t - t_{n-1}) - \frac{\mathcal{K}(t_{n-1}, y_{n-1})}{h} (t - t_n). \quad (16)$$

We take $h = t_m - t_{m-1}$ and obtain the following:

$$\begin{aligned}
 B_{\ell,1} &= \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\ell-1} \left[\frac{\mathcal{K}(t_n, y_n)}{h} (t - t_{n-1}) - \frac{\mathcal{K}(t_{n-1}, y_{n-1})}{h} (t - t_n) \right] d\tau \\
 &= \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)} \int_0^{t_{n+1}} (t_{n+1} - \tau)^{\ell-1} \left[\frac{\mathcal{K}(t_n, y_n)}{h} (t - t_{n-1}) - \frac{\mathcal{K}(t_{n-1}, y_{n-1})}{h} (t - t_n) \right] d\tau,
 \end{aligned}
 \tag{17}$$

which implies that

$$B_{\ell,1} = \frac{\ell \mathcal{K}(t_n, y_n)}{\text{ABC}(\ell)\Gamma(\ell)h} \left[\frac{2ht_{n+1}^\ell}{\ell} - \frac{t_{n+1}^{\ell+1}}{\ell+1} \right] - \frac{\ell \mathcal{K}(t_{n-1}, y_{n-1})}{\text{ABC}(\ell)\Gamma(\ell)h} \left[\frac{ht_{n+1}^\ell}{\ell} - \frac{t_{n+1}^{\ell+1}}{\ell+1} \right].
 \tag{18}$$

In the same way, we can find

$$B_{\ell,2} = \frac{\ell \mathcal{K}(t_n, y_n)}{\text{ABC}(\ell)\Gamma(\ell)h} \left[\frac{ht_n^\ell}{\ell} - \frac{t_n^{\ell+1}}{\ell+1} \right] - \frac{\mathcal{K}(t_{n-1}, y_{n-1})}{\text{ABC}(\ell)\Gamma(\ell)h}.
 \tag{19}$$

Next, we have the following:

$$\begin{aligned}
 y(t_{n+1}) - y(t_n) &= \frac{1-\ell}{\text{ABC}(\ell)} [\mathcal{K}(t_n, y(t_n)) - \mathcal{K}(t_{n-1}, y(t_{n-1}))] + \frac{\ell \mathcal{K}(t_n, y_n)}{\text{ABC}(\ell)\Gamma(\ell)h} \left[\frac{2ht_{n+1}^\ell}{\ell} - \frac{t_{n+1}^{\ell+1}}{\ell+1} \right] \\
 &\quad - \frac{\ell \mathcal{K}(t_{n-1}, y_{n-1})}{\text{ABC}(\ell)\Gamma(\ell)h} \left[\frac{ht_{n+1}^\ell}{\ell} - \frac{t_{n+1}^{\ell+1}}{\ell+1} \right] - \frac{\ell \mathcal{K}(t_n, y_n)}{\text{ABC}(\ell)\Gamma(\ell)h} \left[\frac{ht_n^\ell}{\ell} - \frac{t_n^{\ell+1}}{\ell+1} \right] \\
 &\quad + \frac{\mathcal{K}(t_{n-1}, y_{n-1})}{\text{ABC}(\ell)\Gamma(\ell)h}.
 \end{aligned}
 \tag{20}$$

The above gives us

$$\begin{aligned}
 y(t_{n+1}) &= y(t_n) + \mathcal{K}(t_n, y_n) \left[\frac{1-\ell}{\text{ABC}(\ell)} + \frac{\ell}{\text{ABC}(\ell)h} \left\{ \frac{2ht_{n+1}^\ell}{\ell} - \frac{t_{n+1}^{\ell+1}}{\ell+1} \right\} \right. \\
 &\quad \left. - \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)h} \left\{ \frac{ht_n^\ell}{\ell} - \frac{t_n^{\ell+1}}{\ell+1} \right\} \right] + \mathcal{K}(t_{n-1}, y_{n-1}) \times, \\
 &\quad \left[\frac{\ell-1}{\text{ABC}(\ell)} - \frac{\ell}{\text{ABC}(\ell)\Gamma(\ell)h} \left\{ \frac{ht_{n+1}^\ell}{\ell} - \frac{t_{n+1}^{\ell+1}}{\ell+1} + \frac{t_n^{\ell+1}}{h\text{ABC}(\ell)\Gamma(\ell)} \right\} \right].
 \end{aligned}
 \tag{21}$$

The above approach is a two-step Adams–Bashforth method for the ABC fractional derivative; this takes into consideration the kernels nonlinearity, as well as the Atangana–Baleanu operator’s exponential decay law. Furthermore, we will discuss the convergence and stability of the suggested numerical approach in the upcoming part. We

conducted numerous simulations for the better conceptualization of the complicated phenomena of HIV infection. For numerical simulation, the model parameter values and state-variable initial values are shown in Table 1 which is utilized for numerical calculations. Figures 1–4 depict the time series analysis of all the three compartment of the

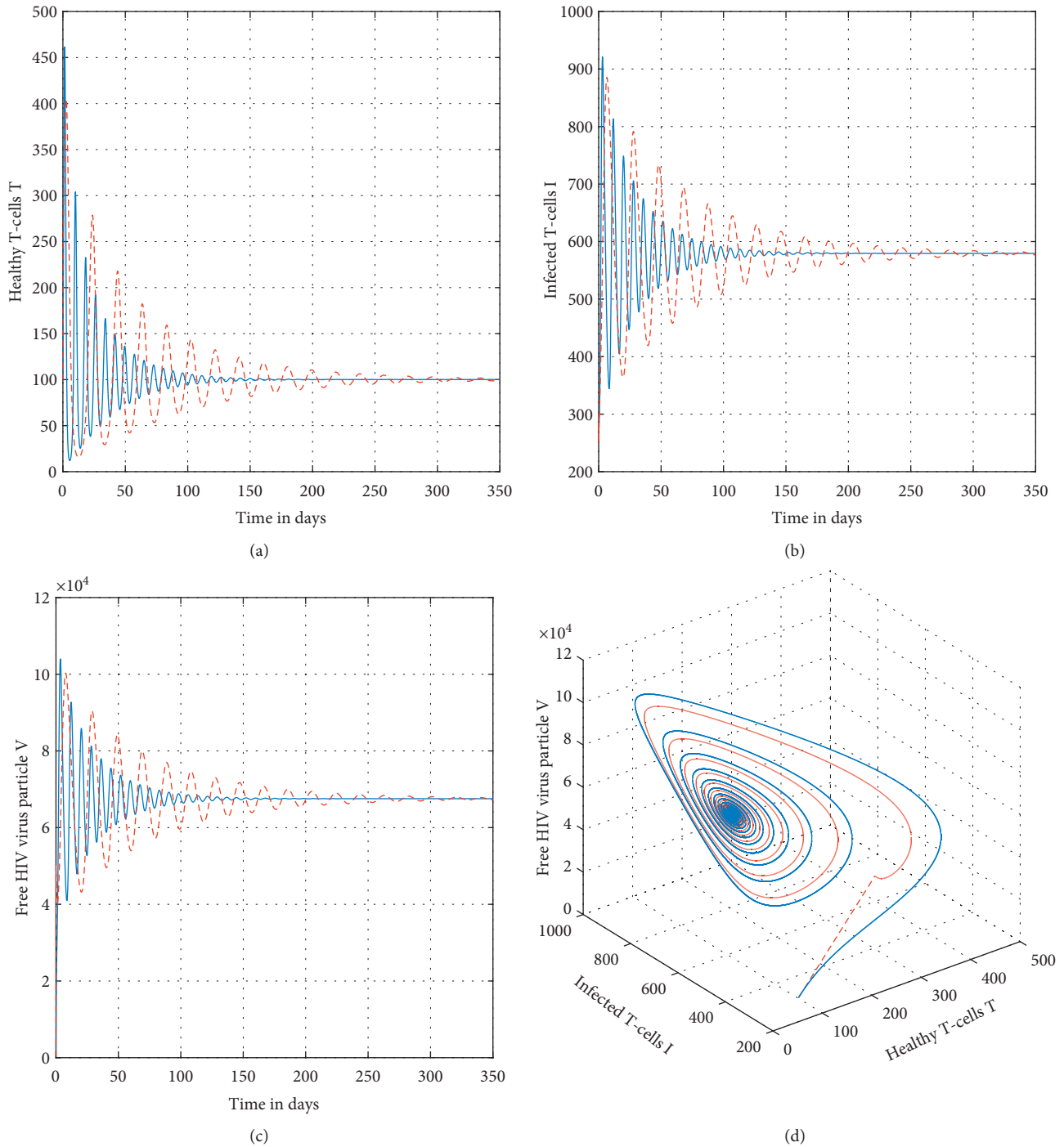


FIGURE 1: Graphical view analysis of the fractional model (2) of HIV by taking the index of memory $\vartheta = 0.4$.

proposed system with the variation of the index of memory ℓ , i.e., $\ell = 0.4, 0.6, 0.8, 1.0$ to show the dynamical behaviour of HIV infection. It has been observed through numerical outcomes that the parameter ℓ can be used as preventive parameter. Figures 5–8 depict the chaotic behaviour of our system (2) with various values of index of memory ℓ . We noticed that the index of memory ℓ can be also be utilized as chaotic control parameter. Many scientific and engineering applications rely heavily on the chaotic behaviour of the

system. It is well known that there is indeed a strong inclination to conceive and depict chaotic system behaviour. The chaotic modeling validates the feasibility and scalability of the suggested mathematical model, which can then be applied towards the novel chaos systems. We showed that perhaps ℓ had a considerable contribution and may be utilized as an effective parameter for preventative actions. Furthermore, we have shown the impact of several input factors on the dynamics of the system in Figures 9–11.

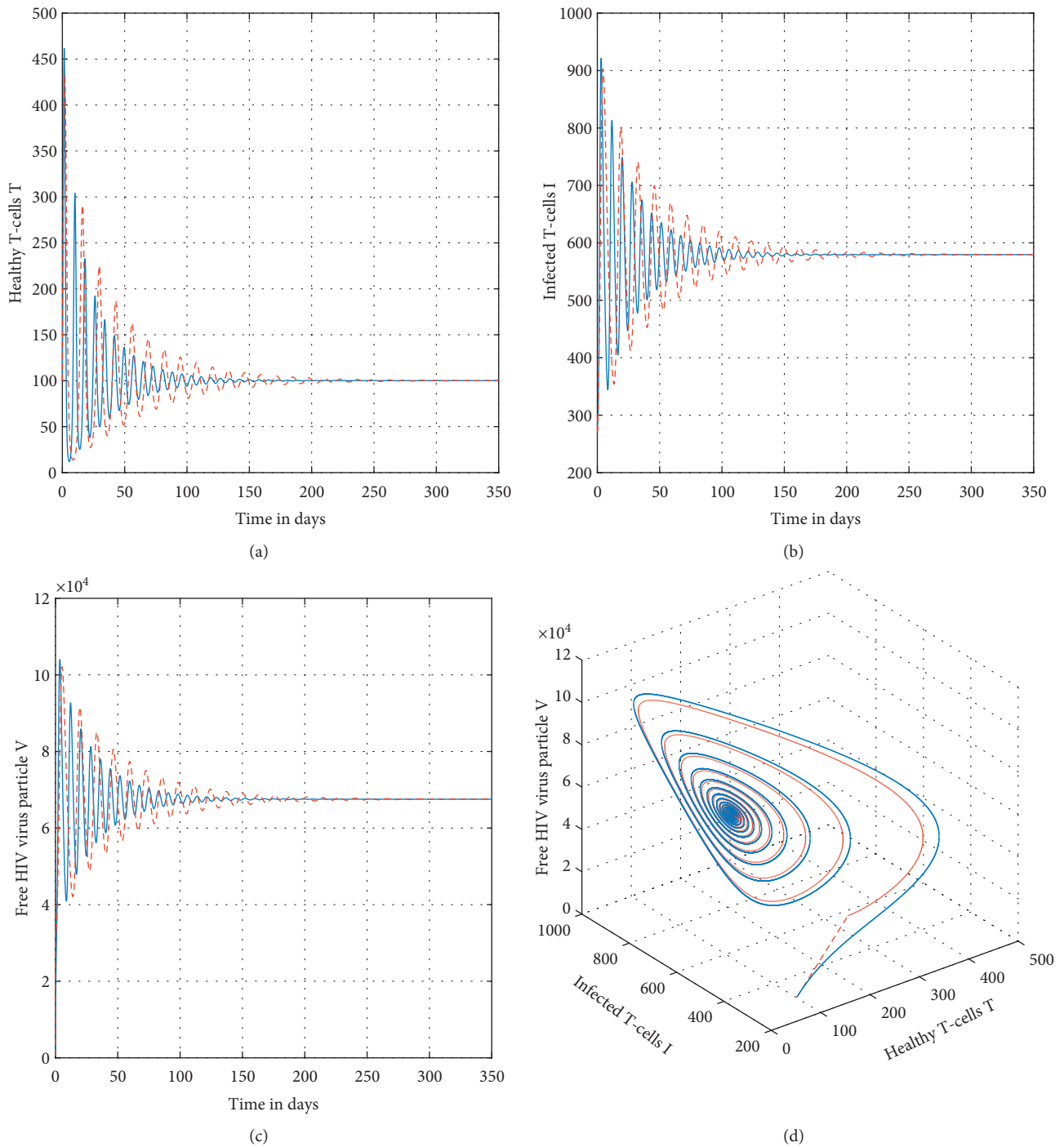


FIGURE 2: Graphical view analysis of the fractional model (2) of HIV by taking the index of memory $\vartheta = 0.6$.

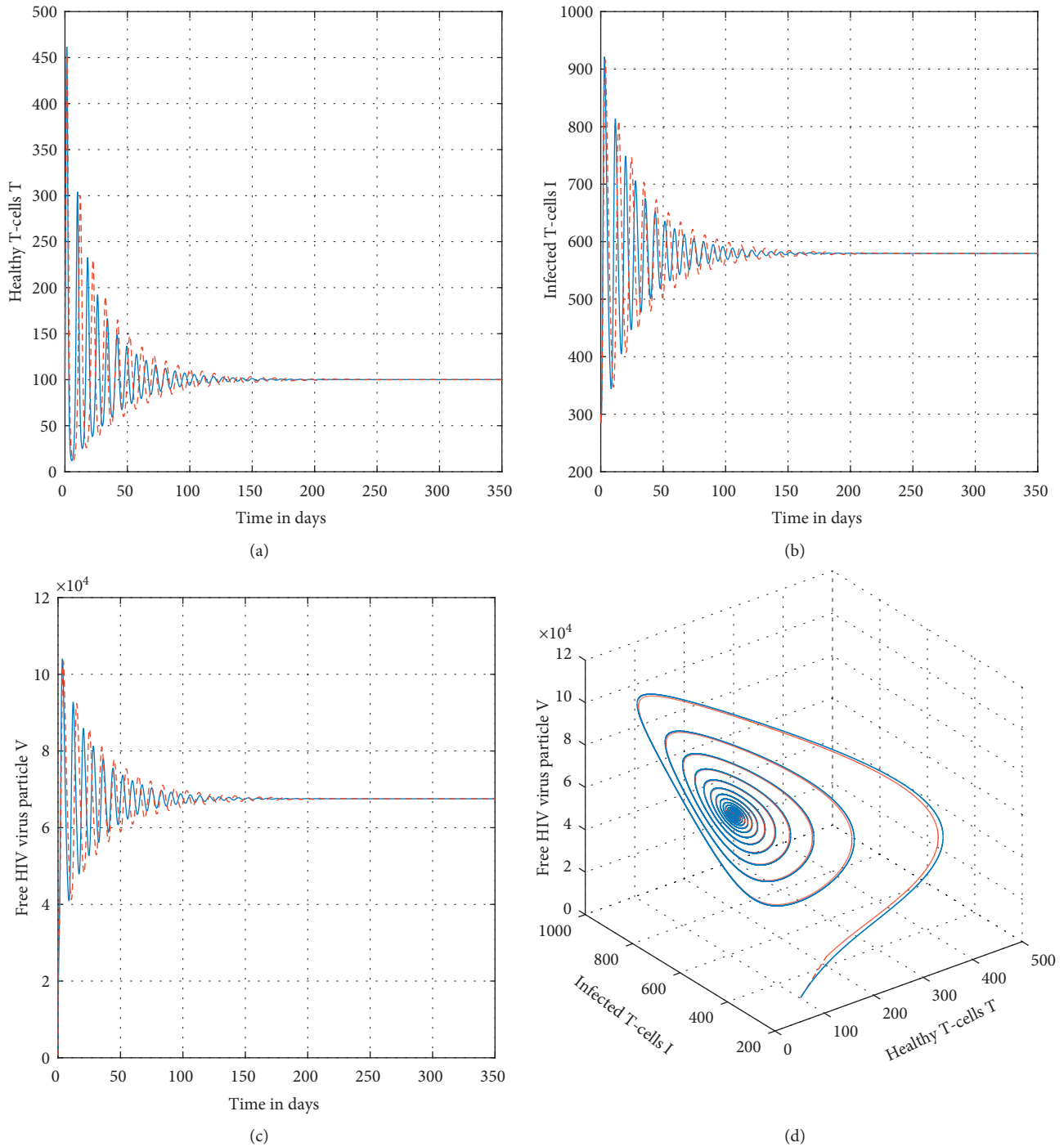


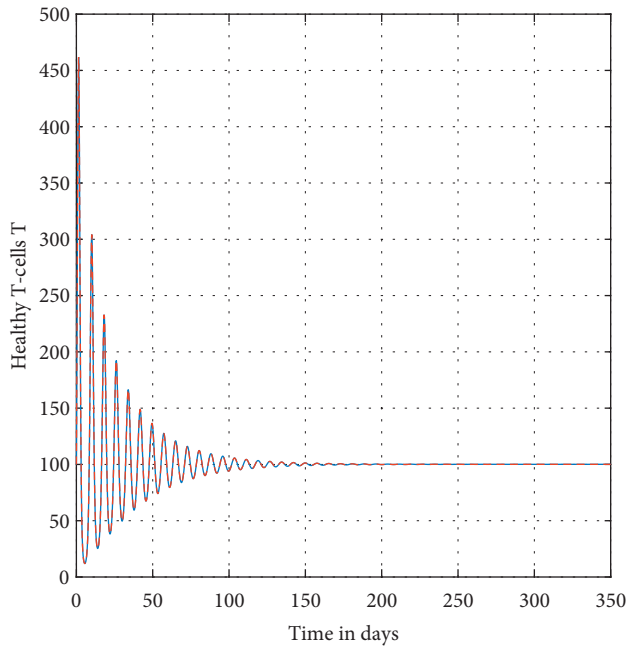
FIGURE 3: Graphical view analysis of the fractional model (2) of HIV by taking the index of memory $\vartheta = 0.8$.

Simulations reveal that the suggested numerical scheme is simple to implement and quick to execute. However, more study will be required to investigate the effectiveness of this technique in terms of consistency, accuracy, and computing cost. In the next step, we will discuss convergence and stability result of the above numerical method. The convergence result of the above method has been given as follows.

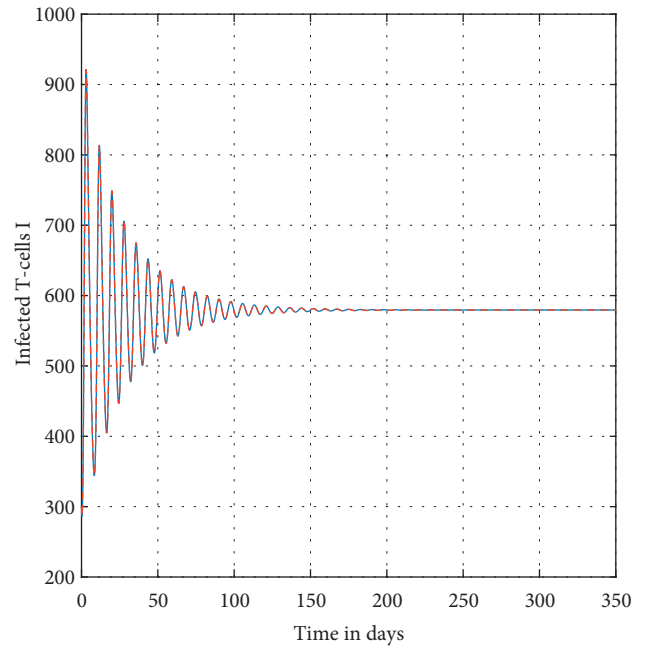
Theorem 3. Assume that g be a continuous and bounded function and $x(\tau)$ be the solution of the fractional system as follows:

$${}_{0}^{ABC}D_{\tau}^{\vartheta}x(\tau) = g(\tau, x(\tau)), \quad (22)$$

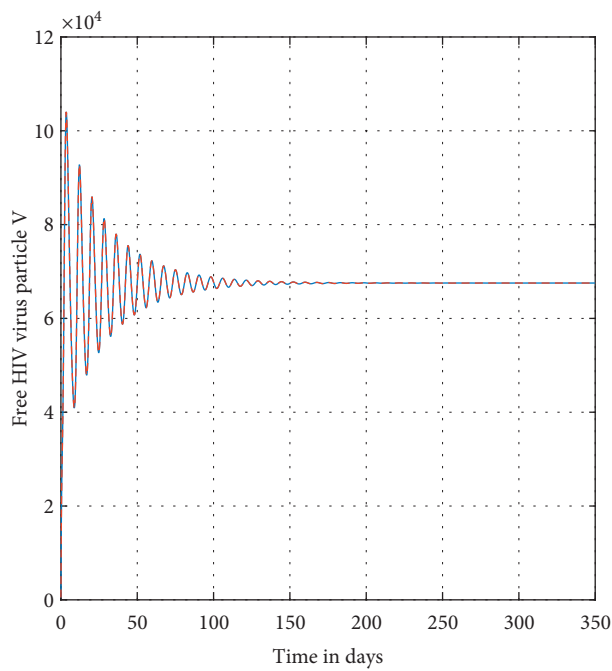
then the solution of $x(\tau)$ is as follows:



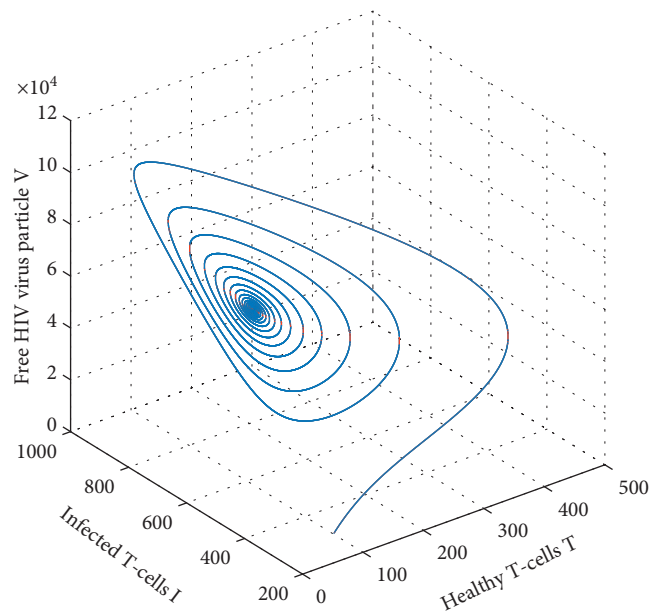
(a)



(b)



(c)



(d)

FIGURE 4: Graphical view analysis of the fractional model (2) of HIV by taking the index of memory $\vartheta = 1.0$.

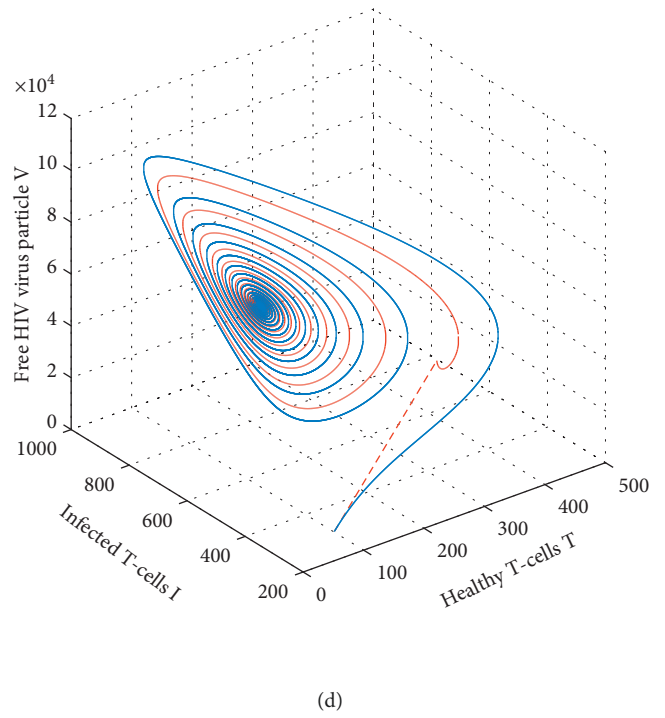
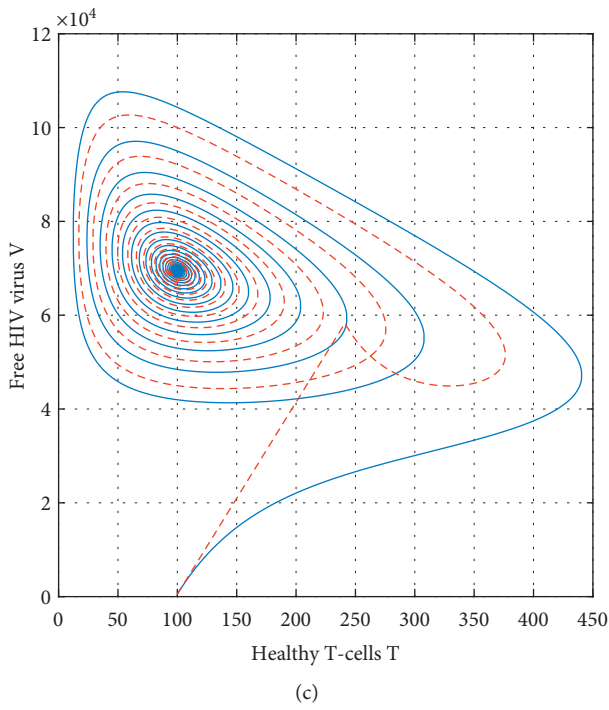
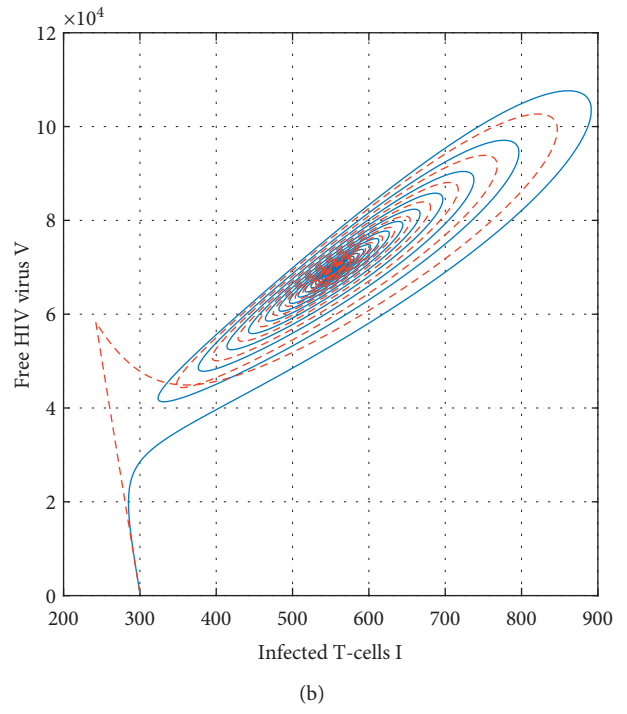
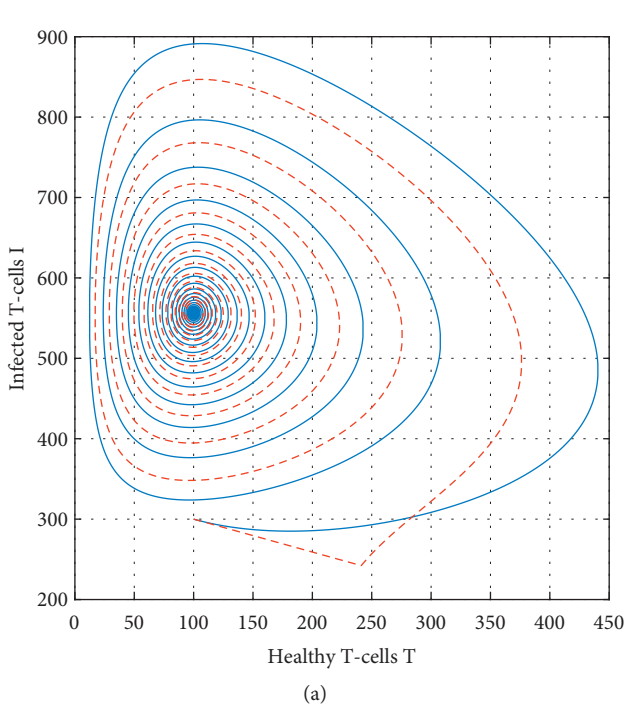


FIGURE 5: Graphical view analysis of the dynamical behaviour of the fractional model (2) to represent its chaotic plot with the index of memory $\vartheta = 0.35$.

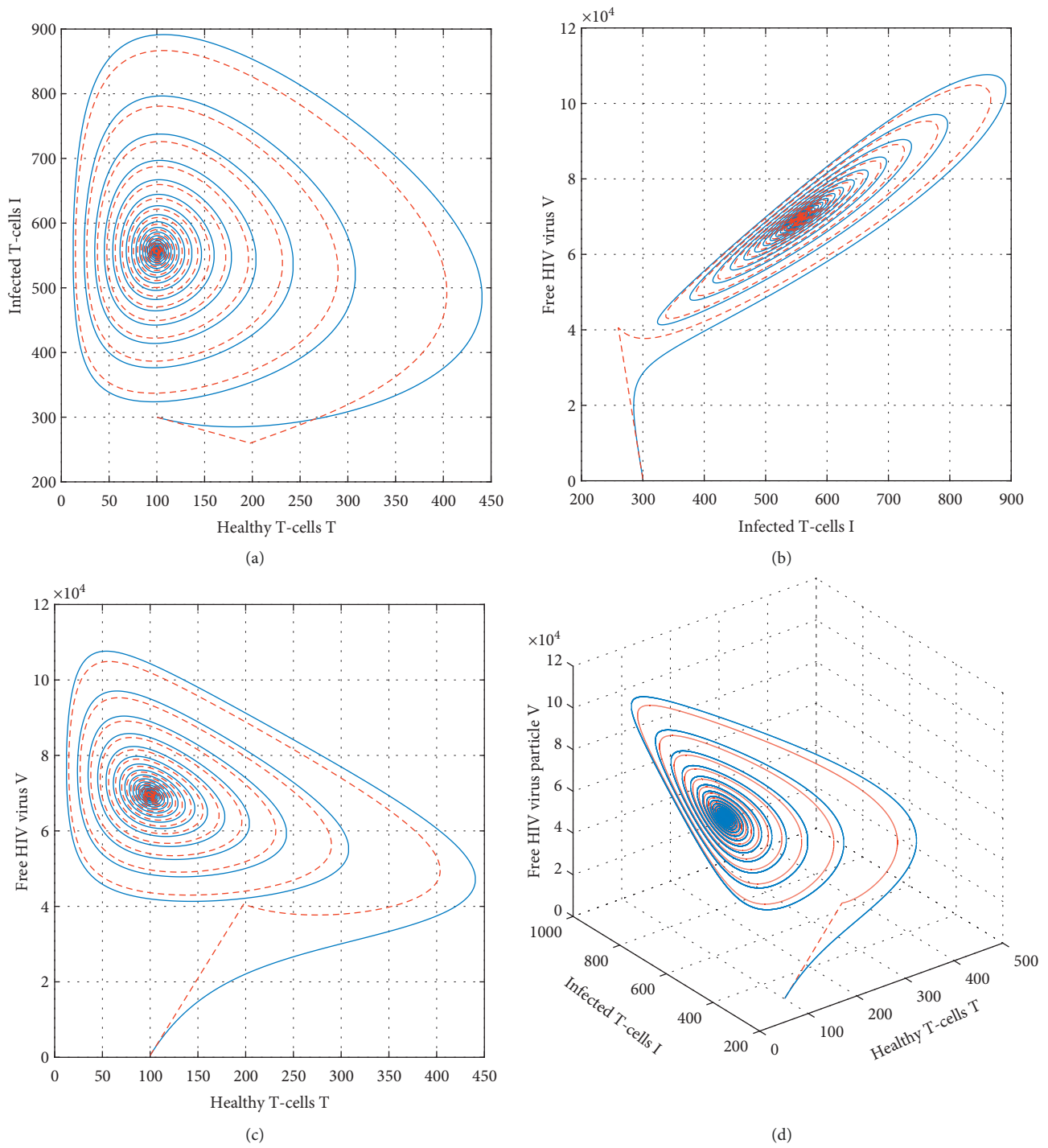


FIGURE 6: Graphical view analysis of the chaotic phenomena of the suggested fractional model (2) of HIV with the index of memory $\vartheta = 0.55$.

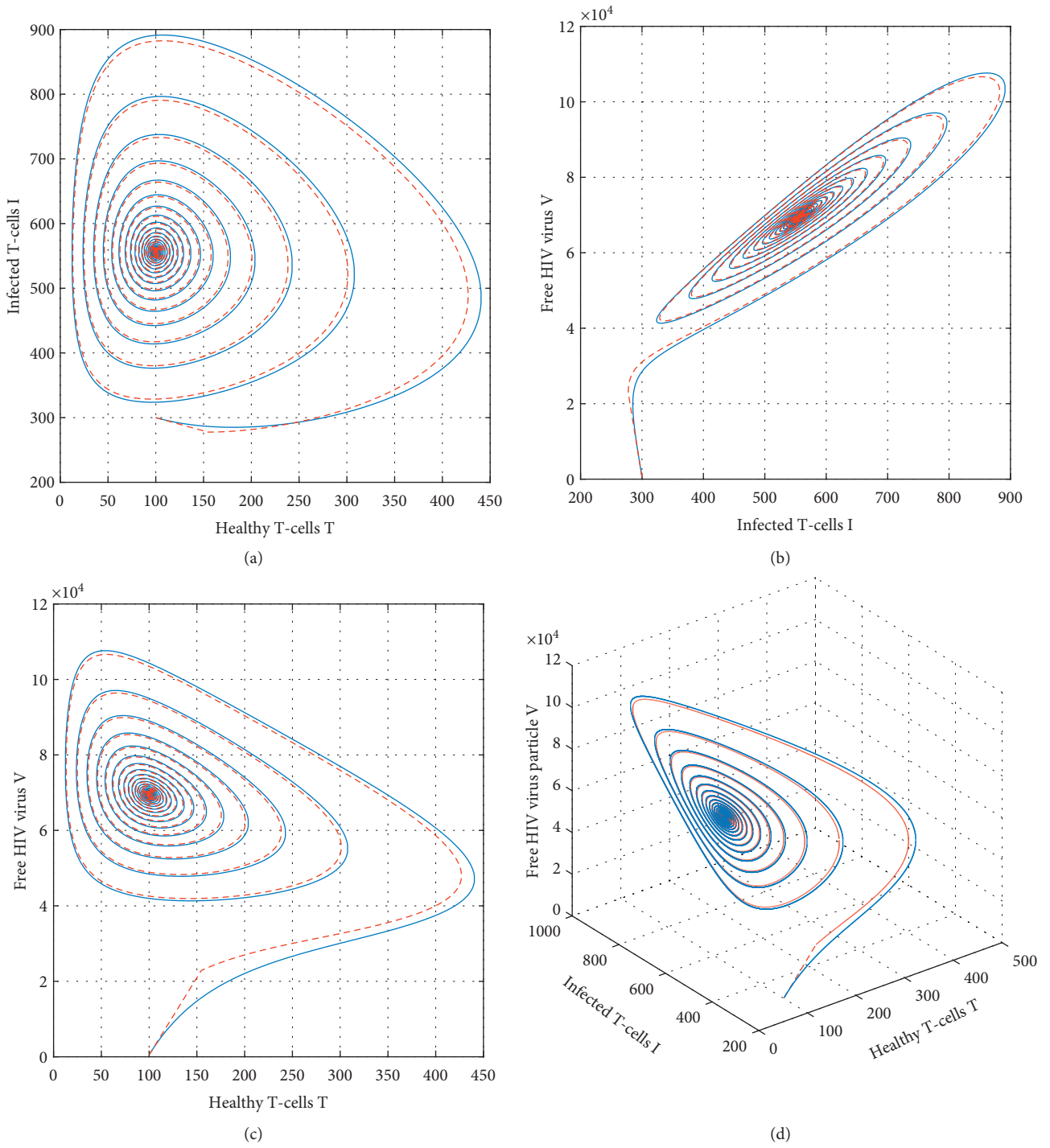


FIGURE 7: Graphical view analysis of the suggested fractional model (2) of HIV to represent its chaotic plot with the index of memory $\rho = 0.75$.

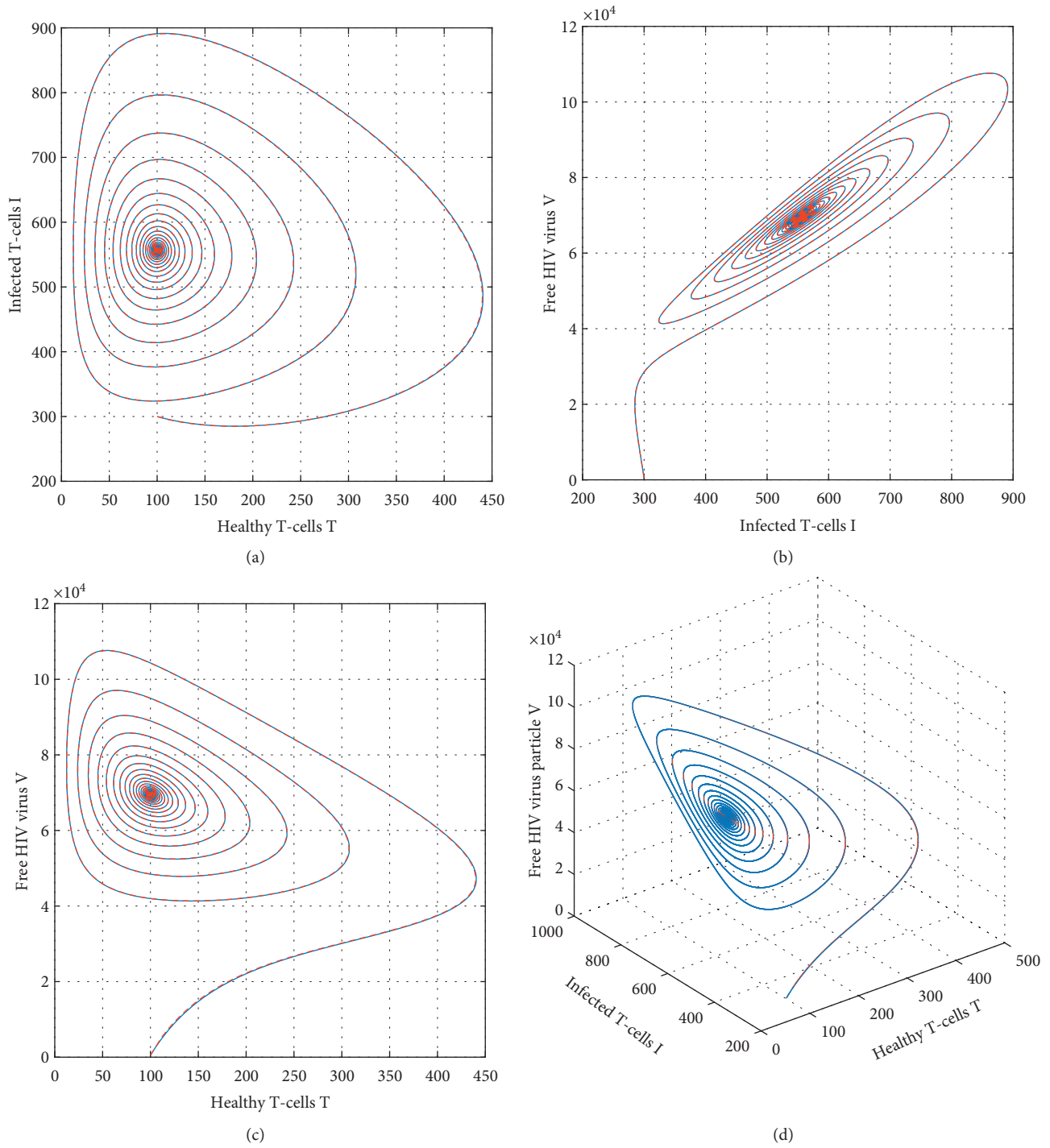


FIGURE 8: Graphical view analysis of the time series of the suggested fractional model (2) of HIV to represent its chaotic plot with the index of memory $\vartheta = 0.95$.

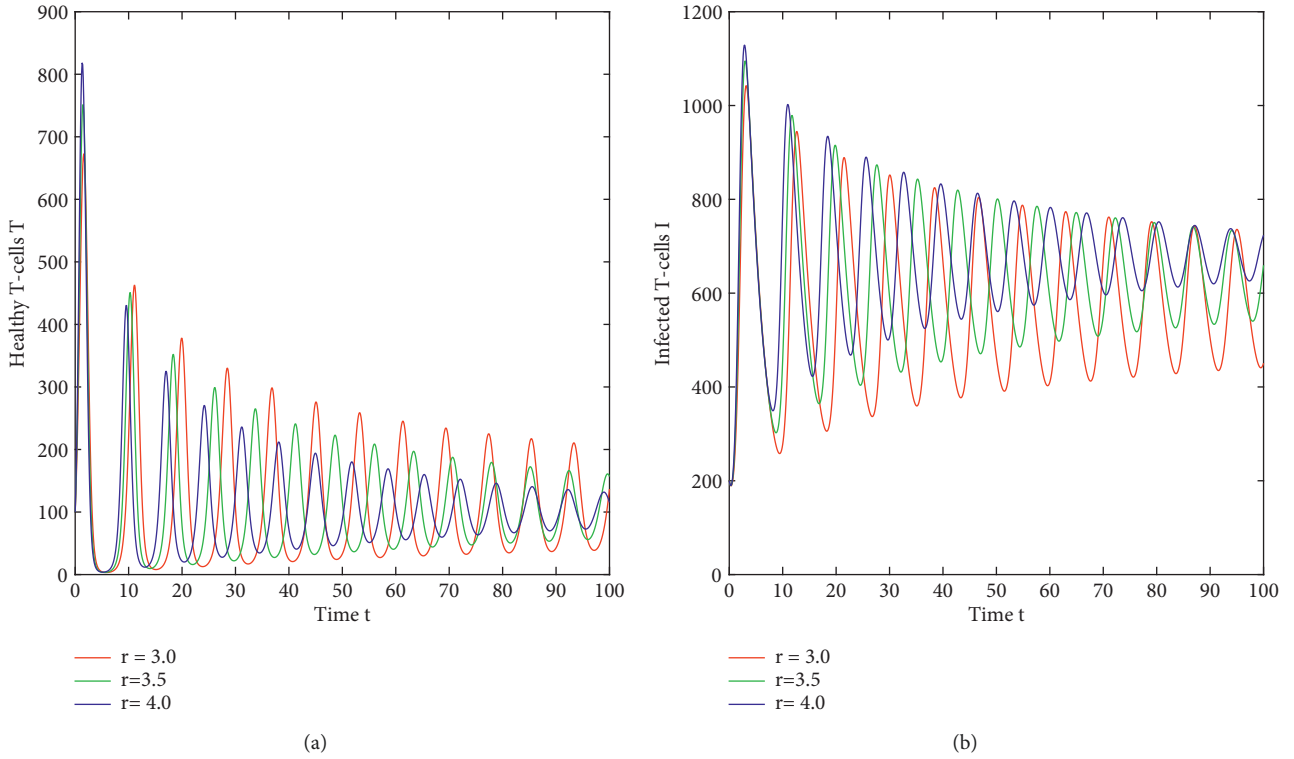


FIGURE 9: Graphical view analysis of the suggested fractional model (2) of HIV with the variation of r , i.e., $r = 3.0, 3.5, 4.0$.

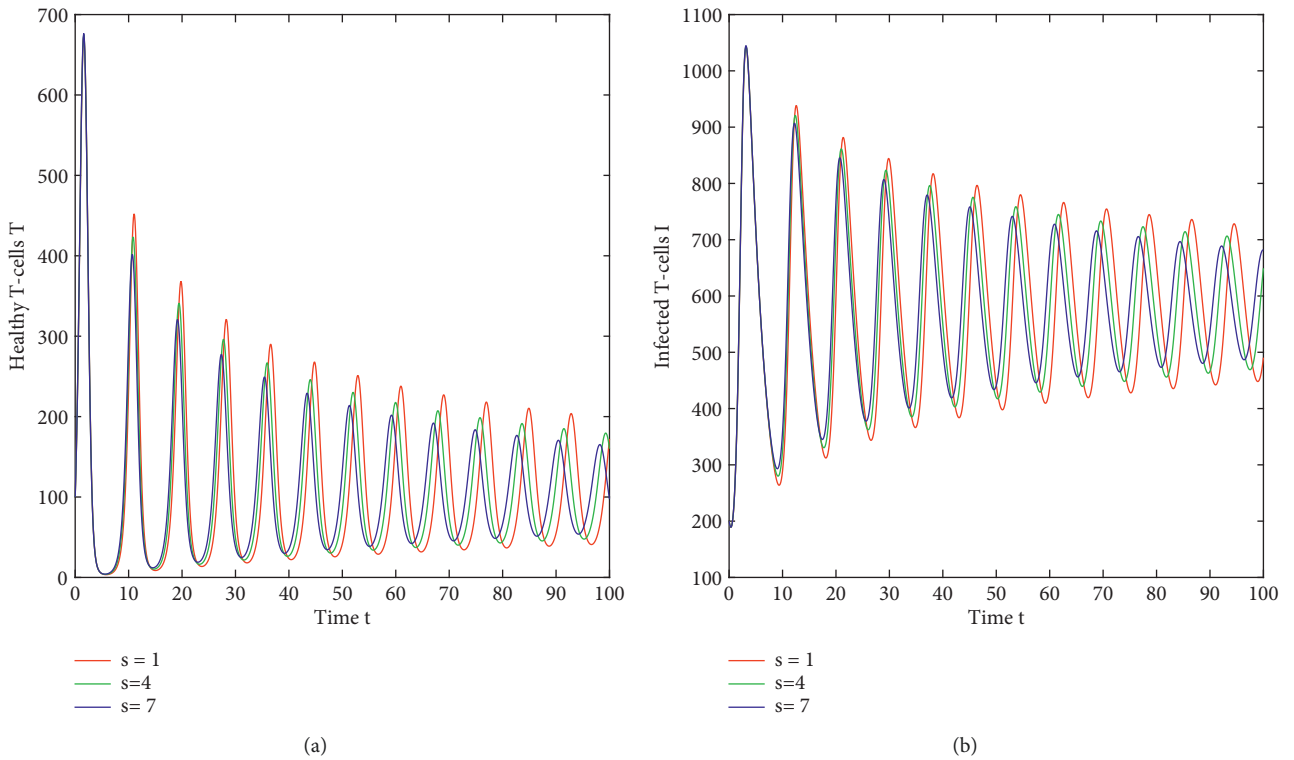


FIGURE 10: Graphical view analysis of the time series of the suggested fractional model (2) of HIV with the variation of s , i.e., $s = 1.0, 4.0, 7.0$.

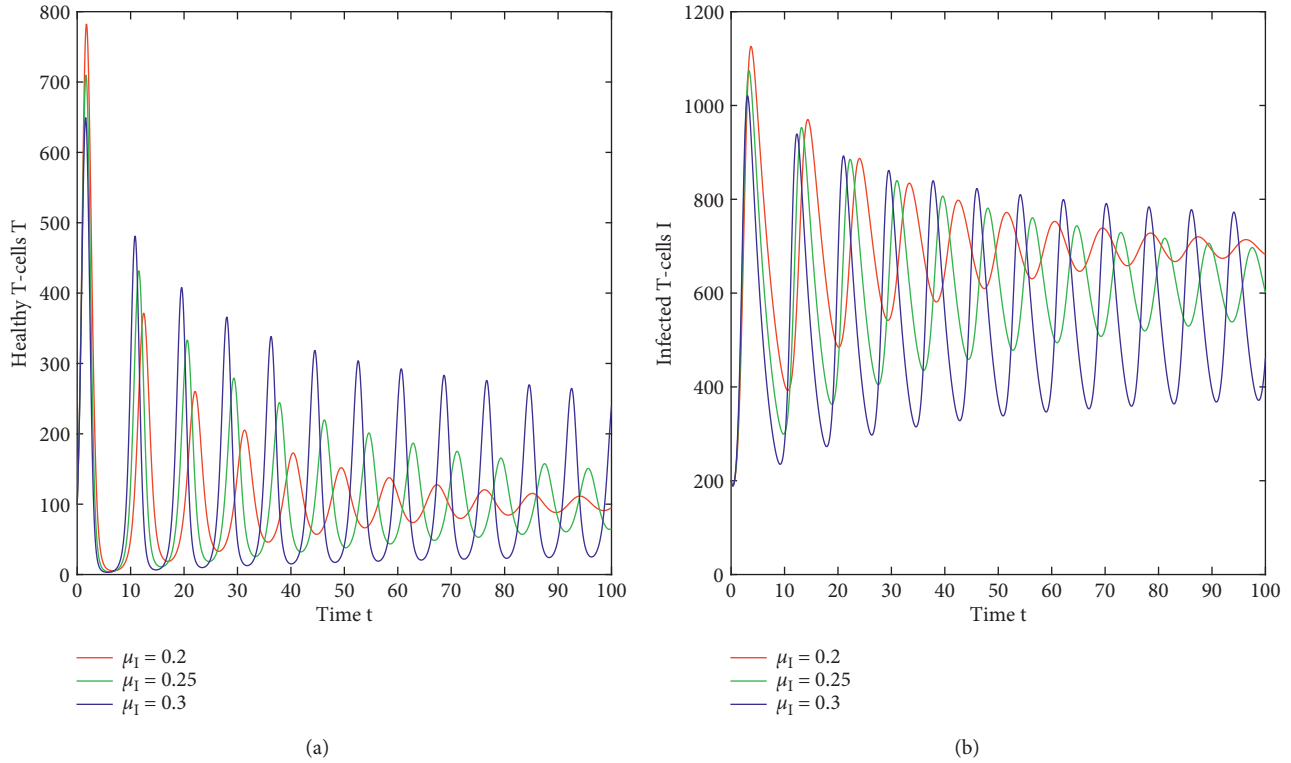


FIGURE 11: Illustration of the time series of the suggested fractional model (2) of HIV with the variation of μ_I , i.e., $\mu_I = 0.2, 0.25, 0.30$.

$$\begin{aligned}
 x_{n+1} &= x_n + g(\tau_n, x_n) \\
 &\left[\frac{1 - \vartheta}{ABC(\vartheta)} + \frac{\vartheta}{ABC(\vartheta)h} \left\{ \frac{2ht_{n+1}^\vartheta}{\vartheta} - \frac{t_{n+1}^{\vartheta+1}}{\vartheta + 1} \right\} - \frac{\vartheta}{h\Gamma(\vartheta)ABC(\vartheta)} \left\{ \frac{ht_n^\vartheta}{\vartheta} - \frac{t_n^{\vartheta+1}}{\vartheta + 1} \right\} \right] \\
 &+ g(\tau_{n-1}, x_{n-1}) \times \left[\frac{\vartheta - 1}{ABC(\vartheta)} - \frac{\vartheta}{ABC(\vartheta)\Gamma(\vartheta)h} \left[\frac{2h\tau_{n+1}^\vartheta}{\vartheta} - \frac{\tau_{n+1}^{\vartheta+1}}{\vartheta + 1} + \frac{\tau^{\vartheta+1}}{ABC(\vartheta)h\Gamma(\vartheta)} \right] \right] + H_\vartheta,
 \end{aligned} \tag{23}$$

in which $\|H_\vartheta\|_\infty < N$.

Proof. To prove the required result, we proceed in the following manner:

$$\begin{aligned}
 x_{n+1} - x_n &= \frac{1 - \vartheta}{ABC(\vartheta)} (g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})) + \frac{\vartheta}{\Gamma(\vartheta)ABC(\vartheta)} \\
 &\times \left[\int_0^{\tau_{n+1}} (\tau_{n+1} - \tau)^{\vartheta-1} g(\tau, x(\tau)) d\tau - \int_0^{\tau_n} (\tau_n - \tau)^{\vartheta-1} g(\tau, x(\tau)) d\tau \right] \\
 &= \frac{1 - \vartheta}{ABC(\vartheta)} (g_n - g_{n-1}) + \frac{\vartheta}{\Gamma(\vartheta)ABC(\vartheta)} \\
 &\left\{ \int_0^{\tau_{n+1}} \frac{\tau - \tau_{n-1}}{\tau_n - \tau_{n-1}} g(\tau_n, x_n) + \frac{\tau - \tau_n}{\tau_{n-1} - \tau_n} g(\tau_{n-1}, x_{n-1}) + \frac{g^{n+1}(\tau)}{(n+1)!} \prod_{i=0}^n (\tau - \tau_i) \right\} (\tau_{n+1} - \tau)^{\vartheta-1} d\tau
 \end{aligned}$$

$$\begin{aligned}
& - \int_0^{\tau_n} \left\{ \frac{\tau - \tau_{n-1}}{\tau_n - \tau_{n-1}} g(\tau_n, x_n) + \frac{\tau - \tau_n}{\tau_{n-1} - \tau_n} f(\tau_{n-1}, y_{n-1}) + \frac{g^n(\tau)}{(n)!} \prod_{i=0}^{n-1} (\tau - \tau_i) \right\} \times (\tau_n - \tau)^{\vartheta-1} d\tau \\
& = \int_0^{\tau_{n+1}} \frac{g^{(n+1)}(\tau)}{(n+1)!} \prod_{i=0}^n (\tau_{n+1} - \tau)^{\vartheta-1} (\tau - \tau_i) d\tau + J(\tau, \vartheta, n) \\
& - \int_0^{\tau_n} \frac{g^{(n)}(\tau)}{(n)!} \prod_{i=0}^n (\tau_n - \tau)^{\vartheta-1} (\tau - \tau_i) d\tau.
\end{aligned} \tag{24}$$

This further gives

$$x_{n+1} - x_n = J(\tau, \vartheta, n) + H_\vartheta(\tau), \tag{25}$$

in which we have

$$\begin{aligned}
J(\tau, \vartheta, n) & = g(\tau_n, x_n) \\
& \left\{ \frac{1 - \vartheta}{ABC(\vartheta)} + \frac{\vartheta}{AB(\vartheta)h} \left[\frac{2h\tau_{n+1}^\vartheta}{\vartheta} - \frac{\tau_{n+1}^{\vartheta+1}}{\vartheta+1} \right] - \frac{\vartheta}{ABC(\vartheta)h\Gamma(\vartheta)} \left[\frac{h\tau_n^\vartheta}{\vartheta} - \frac{\tau_n^{\vartheta+1}}{\vartheta+1} \right] \right\} \\
& + g(\tau_{n-1}, x_{n-1}) \left\{ \frac{\vartheta}{ABC(\vartheta)} - \frac{\vartheta}{hABC(\vartheta)\Gamma(\vartheta)} \left(\frac{h\tau_{n+1}^\vartheta}{\vartheta} - \frac{\tau_{n+1}^{\vartheta+1}}{\vartheta+1} + \frac{\tau_n^{\vartheta+1}}{h\Gamma(\vartheta)ABC(\vartheta)} \right) \right\},
\end{aligned} \tag{26}$$

$$H_\vartheta(t) = \int_0^{\tau_{n+1}} \frac{g^{(n+1)}(\tau)}{(n+1)!} \prod_{i=0}^n (\tau_{n+1} - \tau)^{\vartheta-1} (\tau - \tau_i) d\tau - \int_0^{\tau_n} \frac{g^n(\tau)}{(n)!} \prod_{i=0}^{n-1} (\tau - \tau_i) (\tau_n - \tau)^{\vartheta-1} d\tau. \tag{27}$$

Next, we need to prove the following:

$$\begin{aligned}
\|H_\vartheta(\tau)\|_\infty & = \left\| \int_0^{\tau_{n+1}} \frac{g^{(n+1)}(\tau)}{(n+1)!} \prod_{i=0}^n (\tau - \tau_i) (\tau_{n+1} - \tau)^{\vartheta-1} d\tau - \int_0^{\tau_n} \frac{g^n(\tau)}{(n)!} \prod_{i=0}^{n-1} (\tau - \tau_i) (\tau_n - \tau)^{\vartheta-1} d\tau \right\|_\infty \\
& < \left\| \int_0^{\tau_{n+1}} \frac{g^{(n+1)}(\tau)}{(n+1)!} \prod_{i=0}^n (\tau - \tau_i) (\tau_{n+1} - \tau)^{\vartheta-1} d\tau \right\|_\infty + \left\| \int_0^{\tau_n} \frac{g^n(\tau)}{(n)!} \prod_{i=0}^{n-1} (\tau_n - \tau)^{\vartheta-1} (\tau - \tau_i) d\tau \right\|_\infty \\
& < \max_{\tau \in [0, \tau_{n+1}]} \frac{|g^{(n+1)}(\tau)|}{(n+1)!} \left\| \prod_{i=0}^n (\tau - \tau_i) \right\|_\infty \frac{t_{n+1}^\vartheta}{\vartheta} + \max_{\tau \in [0, \tau_{n+1}]} \frac{|g^n(\tau)|}{(n)!} \left\| \prod_{i=0}^{n-1} (\tau - \tau_i) \right\|_\infty \frac{\tau_n^\vartheta}{\vartheta} \\
& < \sup_{\tau \in [0, \tau_{n+1}]} \left\{ \max_{\tau \in [0, \tau_{n+1}]} \frac{|g^{(n+1)}(\tau)|}{(n+1)!}, \max_{\tau \in [0, \tau_{n+1}]} \frac{|g^n(\tau)|}{(n)!} \right\} \left(n! \frac{h^{n+1}}{4\vartheta} \tau^{\vartheta_{n+1}} + (n-1)! \frac{h^n}{4\vartheta} \tau^{\vartheta_n} \right),
\end{aligned} \tag{28}$$

which is the required result. \square

Atangana–Baleanu fraction framework in Caputo sense is given by

Theorem 4. Assume that g fulfills Lipschitz condition, then the stability condition for the above numerical scheme in

$$\|g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})\|_\infty \longrightarrow 0, \tag{29}$$

as $n \rightarrow \infty$.

Proof. To prove the required result, we first take

$$x_{n-1} - x_n = \frac{1 - \vartheta}{ABC(\vartheta)} [g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})] + \frac{\vartheta}{ABC(\vartheta)\Gamma(\vartheta)} \times \left[\int_0^{\tau_{n+1}} g(\tau, x(\tau)) (\tau_{n+1} - \tau)^{\vartheta-1} d\tau - \int_0^{\tau_n} g(\tau, x(\tau)) (\tau_n - \tau)^{\vartheta-1} d\tau \right]. \tag{30}$$

The following is obtained by taking the norm on both sides:

$$\begin{aligned} \|x_{n-1} - x_n\|_\infty &< \left\| \frac{1 - \vartheta}{ABC(\vartheta)} [g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})] \right\|_\infty + \frac{\vartheta}{\Gamma(\vartheta)ABC(\vartheta)} \\ &\times \left\| \left[\int_0^{\tau_{n+1}} (\tau_{n+1} - \tau)^{\vartheta-1} g(\tau, x(\tau)) d\tau - \int_0^{\tau_n} (\tau_n - \tau)^{\vartheta-1} g(\tau, x(\tau)) d\tau \right] \right\|_\infty \\ &< \frac{1 - \vartheta}{ABC(\vartheta)} \|g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})\|_\infty \\ &+ \frac{\vartheta}{ABC(\vartheta)\Gamma(\vartheta)} \left\| \int_0^{\tau_{n+1}} g(\tau, x(\tau)) (\tau_{n+1} - \tau)^{\vartheta-1} d\tau \right\|_\infty \\ &+ \frac{\vartheta}{ABC(\vartheta)\Gamma(\vartheta)} \left\| \int_0^{\tau_n} g(\tau, x(\tau)) (\tau_n - \tau)^{\vartheta-1} d\tau \right\|_\infty. \end{aligned} \tag{31}$$

This implies that

$$\begin{aligned} \|x_{n+1} - x_n\|_\infty &< \frac{1 - \vartheta}{ABC(\vartheta)} \|g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})\|_\infty \\ &+ \frac{\vartheta}{ABC(\vartheta)\Gamma(\vartheta)} \left\| \int_0^{\tau_{n+1}} (\tau_{n+1} - \tau)^{\vartheta-1} \sum_{i=0}^n \prod_{0 \leq i \leq n} \frac{(\tau - \tau_i)}{(-1)^i h} g(\tau_i, x_i) d\tau \right\|_\infty \\ &+ \frac{\vartheta}{ABC(\vartheta)\Gamma(\vartheta)} \left\| \int_0^{\tau_n} (\tau_n - \tau)^{\vartheta-1} \sum_{i=0}^{n-1} \prod_{0 \leq i \leq n-1} g(\tau_i, x_i) d\tau \right\|_\infty \\ &< \frac{1 - \vartheta}{ABC(\vartheta)} \|g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})\|_\infty + \|P_n^\vartheta(\tau)\|_\infty + \|H_n^\vartheta(\tau)\|_\infty, \end{aligned} \tag{32}$$

in which

$$\begin{aligned} \|P_n^\vartheta(\tau)\|_\infty &= \left\| \int_0^{\tau_{n+1}} (\tau_{n+1} - \tau)^{\vartheta-1} \sum_{i=0}^n \frac{(\tau - \tau_i)}{(-1)^i h} g(\tau_i, x_i) d\tau \right\|_\infty \\ &\leq \sum_{i=0}^n \frac{\|g(\tau_i, x_i)\|_\infty}{h} \frac{\tau_{n+1}^\vartheta}{\vartheta} \prod_{i=0}^n |\tau - \tau_i| \end{aligned} \quad (33)$$

$$\begin{aligned} &\leq \sum_{i=0}^n \frac{\|g(\tau_i, x_i)\|_\infty}{h} \frac{\tau_{n+1}^\vartheta}{\vartheta} \frac{n! h^n}{4}, \\ \|H_n^\vartheta(\tau)\|_\infty &\leq \sum_{i=0}^n \frac{\|g(\tau_i, x_i)\|_\infty}{h} \frac{\tau_n^\vartheta}{\vartheta} (n-1)! \frac{h^{n-1}}{4}. \end{aligned} \quad (34)$$

As a result, we obtain

$$\begin{aligned} \|x_{n-1} - x_n\|_\infty &< \frac{1 - \vartheta}{\text{ABC}(\vartheta)} \|g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})\|_\infty \\ &+ \sum_{i=0}^n \frac{\|g(\tau_i, x_i)\|_\infty}{4\vartheta} \tau_{n+1}^\vartheta h^{n-1} n! + \sum_{i=0}^{n-1} \frac{\|g(\tau_{i-1}, x_{i-1})\|_\infty}{4\vartheta} \tau_n^\vartheta h^{n-3} (n-1)! \\ &< \frac{Mn!h^n}{4\vartheta} \left\{ \frac{\tau_{n+1}^\vartheta (n+1)}{h} + \frac{\tau_n^\vartheta}{h^2} \right\} + \frac{1 - \vartheta}{\text{ABC}(\vartheta)} \|g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})\|_\infty, \end{aligned} \quad (35)$$

which implies that $|g(\tau_n, x_n) - g(\tau_{n-1}, x_{n-1})|$ goes to zero as n goes to ∞ and as h tends to zero, then $Mn!h^n/4\vartheta$ tends to zero, where $M = \max_{\tau \in [0, \tau_{n+1}]} |g(\tau, x(\tau))|$. \square

5. Concluding Remarks

HIV/AIDS has a significant impact on economic growth by limiting the availability of human capital. AIDS is killing a high number of people in underdeveloped nations due to a lack of effective prevention, treatment, health care, and nutrition. Therefore, it is significant to interrogate the transmission pathway of HIV to identify the role of different input factors on the output of infection. In this work, we structured the dynamics of CD4^+ T-cells in HIV infection through fractional calculus. We presented the proposed model through Atangana–Baleanu derivative in the Caputo sense. The rudimentary properties of fractional calculus have been introduced for the examination of the system. We provided a new numerical scheme for addressing the Atangana–Baleanu fractional derivative to conceptualize the dynamics of HIV. The oscillatory and chaotic plots have been presented with the variation of different input parameters. It has been shown that fractional order has an influence on the chaotic behaviour of the suggested model. The memory index ℓ is expected to improve the system and may have been used as a control parameter. We illustrated the impact of input parameters r , s , and μ_I on the concentration level of healthy and infected CD4^+ T-cells. On the basis of our results, the most critical factors of the system are highlighted. We highlighted the influence of different input

parameter on the dynamics of HIV infection. Furthermore, the convergence and stability result of the system have been shown. In future research work, we opt to highlight the influence of time delay on the infection of HIV infection to highlight the importance of time delay for the control and to validate our results through experimental data.

Data Availability

No data were used to support the study.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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