American Journal of Applied Mathematics
2026, Vol. 14, No. 2, pp. 101–114
https://doi.org/10.11648/j.ajam.20261402.18

Research Article

A Nonlinear Caputo Fractional HIV-1 Model with CTL Saturation and Antiretroviral Intervention: Stability and Approximation Methods

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Abstract

This study introduces a fractional-order HIV-1 infection model formulated with the Caputo derivative, combining the effects of antiretroviral therapy and a saturating cytotoxic T lymphocyte (CTL) immune response. The fractional formulation allows the inclusion of memory-dependent viral dynamics and the saturation function provides a biologically consistent representation of immune regulation. The basic reproduction number R_0 is derived and used to determine the threshold behavior of the system. A theoretical analysis is carried out to prove existence and uniqueness of solutions and to investigate both local and global stability of the disease-free equilibrium. The endemic equilibrium is also derived to offer a deeper understanding of long-term infection dynamics. To obtain approximate solutions for the nonlinear system the semi analytical methods, the Differential Transform Method (DTM), Adomian Decomposition Method (ADM), and Homotopy Perturbation Method (HPM), are applied. A fractional predictor–corrector scheme is applied and compared with the semi analytical solutions. Numerical experiments show the influence of the fractional order, therapeutic effectiveness and immune parameters on disease evolution. The results indicate that decreasing the fractional order significantly alters the transient dynamics of viral load and CD4+ T-cell populations, demonstrating that fractional-order modeling provides a more flexible and realistic framework for describing HIV-1 dynamics and evaluating treatment effects.

Keywords

Fractional-Order Dynamical System, Caputo Derivative, Adomian Decomposition Method, Differential Transform Method, Homotopy Perturbation Method, Predictor–Corrector Method

1. Introduction

Fractional calculus used in modeling complex dynamical systems where memory and hereditary effects are involved. Fractional calculus is a powerful extension of classical differential equations by incorporating memory effects through derivatives of non-integer order. The fundamental theory of fractional differential equations was developed by Podlubny [1] and Kilbas et al. [2], while numerical and analytical aspects were extensively studied by Diethelm et al. [3, 4]. In particular, the Caputo fractional derivative widely used in biological

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Received: 24 February 2026; **Accepted:** 11 March 2026; **Published:** 24 April 2026

modeling, as it allows the use of classical initial conditions while capturing memory-dependent dynamics. As a result, fractional-order models have attracted considerable attention of the researchers working across various scientific fields. Human Immunodeficiency Virus type 1 (HIV-1) continues to be a major health concern due to its complex interaction with the human immune system and its ability to develop a long-term infection.

In recent years, many researchers successfully applied fractional-order models to various biological and epidemiological systems. Ahmed et al. [5] demonstrated that fractional predator–prey and rabies models exhibit more dynamical behavior compared to classical models. Ameen and Novati [6] investigated fractional epidemic models using implicit Adam's methods and shown the improved numerical performance of fractional approaches. Similar advancements have been made in fractional SEIR epidemic modeling [8] and eco-epidemiological systems [14] confirming that fractional frameworks can better represent complex biological interactions. Arafa et al. [6] introduced a fractional model to describe the interaction between HIV and CD4+ T-cells during primary infection, highlighting the importance of fractional dynamics in representing biological memory effects. Extensions of classical HIV models incorporating delay, immune response, and nonlinear incidence rates have also been investigated. Guo et al. [7] analyzed a delayed HIV-1 infection model with saturation incidence rate and cytotoxic T lymphocyte (CTL) immune response. Further developments include fractional time-delay models that capture the proliferation of CD4+ T-cells under antiretroviral therapy, as studied by Liu et al. [8]. Lichae et al. [9] proposed a fractional differential model incorporating antiviral drug treatment to examine the dynamics of HIV infection more accurately. Similarly, Naik et al. [10] explored the mechanics of viral kinetics under immune control using a fractional-order framework, demonstrating the importance of fractional derivatives in describing treatment effects during primary infection. Different types of fractional operators have also been used to study HIV dynamics. Moore et al. [11] proposed a Caputo–Fabrizio fractional model for HIV/AIDS that includes a treatment compartment, offering a nonsingular kernel formulation. Stability analysis of HIV models using incommensurate fractional-order systems was investigated by Dasbasi [12]. Günerhan et al. [13] further analyzed fractional HIV models using Caputo and proportional Caputo operators, while Nazir et al. [14] examined HIV dynamics using nonsingular kernel fractional derivatives.

Recent research has focused on developing more advanced computational and analytical methods for fractional HIV models. Khater et al. [15] investigated stable computational solutions of an Atangana–Baleanu fractional HIV infection model involving CD4+ T-cells. Kongson et al. [16] analyzed a generalized Caputo fractional derivative model for HIV dynamics with treatment. Additionally, Pitchaimani and Saranya Devi [17] studied analytical properties of delayed fractional-order HIV models. Wang et al. [18] examined the global stability of switched HIV/AIDS models involving fractional derivatives and drug treatment strategies. Further contributions include the dynamical analysis of post-treatment HIV infection models by Pradeesh et al. [19], which provide insights into long-term viral dynamics. Almoneef et al. [20] recently investigated fractional HIV models under proportional Hadamard–Caputo operators, demonstrating the flexibility of different fractional derivative definitions in modeling biological processes.

To analyze such fractional systems, several analytical and numerical techniques have been developed. El-Sayed et al. [21] presented analytical and numerical methods for multi-term nonlinear fractional differential equations. The fractional differential transform method has been applied to solve fractional differential–algebraic equations, as shown by Ibis et al. [22]. Numerical solution techniques for nonlinear fractional systems have also been studied by Ullah et al. [23] and Ziada [24], including decomposition-based methods. Most recently, optimal control strategies combined with fractional-order HIV/AIDS models have been explored to improve treatment efficiency. Hussein and Mebrate [25] developed a fractional-order HIV/AIDS model incorporating treatment and optimal control using Caputo derivatives, highlighting the potential of fractional calculus in designing effective intervention strategies.

Many HIV fractional models are existing but do not include CTL immune saturation. Some studies include treatment but ignored fractional memory effects. Few studies analyze existence, stability. Therefore, there is a need to develop a fractional-order HIV model that simultaneously incorporates antiretroviral treatment, CTL immune response with saturation, and rigorous stability analysis.

The present work proposes a fractional-order HIV-1 model formulated in the Caputo sense. The model incorporates treatment efficacy, CTL immune response, and a saturated immune stimulation term avoiding unrealistic immune overactivation. A rigorous theoretical analysis is carried out, including positivity and boundedness of solutions, existence and uniqueness results based on fixed-point theory, derivation of the basic reproduction number R_0, and local and global stability analysis of equilibrium points.

In addition to the theoretical investigation, a detailed comparative study of semi-analytical methods is performed moreover the results of semi analytical methods are compared with a fractional predictor–corrector numerical scheme. The comparison is conducted in terms of convergence, accuracy, and computational performance. Numerical experiments further illustrate the impact of fractional order and treatment efficacy on disease progression.

The findings of this study indicate that fractional-order modeling provides a more flexible and realistic description of HIV-1 dynamics. Moreover, the comparative framework developed here offers practical guidance for selecting appropri-

ate solution techniques for nonlinear fractional epidemiological systems.

2. Preliminaries

This section deals with basic definitions of Caputo fractional derivative and integration as given in [1].

2.1. Definition: Caputo Fractional Derivative

Let $\alpha \in [n-1, n)$, where $n \in \mathbb{N}$, and let f be an n-times continuously differentiable function on $[a, t]$. The Caputo fractional derivative of order α of $f(t)$ is defined by

$$ {}_a^C D_t^\alpha f(t) = \frac{1}{\Gamma(n-\alpha)} \int_a^t \frac{f^{(n)}(x)}{(t-x)^{\alpha-n+1}}\, dx, $$

where $\Gamma(\cdot)$ is the Gamma function.

2.2. Definition: Caputo Fractional Integration

Let $\alpha > 0$ and let f be a function defined on $[a, t]$. The fractional integral of order α of $f(t)$ is defined by

$$ I_t^\alpha f(t) = \frac{1}{\Gamma(\alpha)} \int_a^t (t-x)^{\alpha-1} f(x)\, dx $$

where $\Gamma(\cdot)$ is the Gamma function.

3. Model Formulation

In this section, we formulate a novel fractional-order mathematical model describing the dynamics of HIV-1 infection by incorporating memory effects, antiretroviral treatment efficacy, and immune response mechanisms.

3.1. Model Variables

Let the state variables be defined as follows:

$x(t)$: concentration of HIV-infected $CD4^+$T cells at time t;

$y(t)$: concentration of free HIV-1 virions in plasma at time t;

$z(t)$: population of cytotoxic T lymphocyte (CTL) immune cells at time t.

3.2. Model Parameters

All parameters are assumed to be positive constants and are described as follows:

a: infection rate of $CD4^+$ T cells by free virus;

b: natural death rate of infected cells;

k: rate of virus production by infected cells;

c: clearance rate of free virus particles;

p: rate at which CTLs eliminate infected cells;

r: activation rate of CTLs due to infected cells;

q: saturation constant representing immune exhaustion;

d: natural death rate of CTLs;

$\varepsilon (0 \le \varepsilon \le 1)$: efficacy of antiretroviral therapy;

$\alpha (0 < \alpha \le 1)$: fractional order describing memory effects in the system.

3.3. Fractional Order HIV-1 Model

To account for memory and hereditary effects inherent in biological processes, the classical integer-order derivatives are replaced by Caputo fractional derivatives of order α. In recent years, several fractional-order HIV models have been proposed to study the interaction between infected $CD4^+$ T cells, viral particles, and immune response [6-10, 23]. The proposed fractional-order HIV-1 model is given by

$$ \begin{aligned} D_t^\alpha x(t) &= a(1-\varepsilon)y(t) - bx(t) - px(t)z(t), \\ D_t^\alpha y(t) &= kx(t) - cy(t), \\ D_t^\alpha z(t) &= \frac{rx(t)}{1+qx(t)} - dz(t), \end{aligned} \tag{1} $$

with initial conditions:

$$ x(0) = x_0, y(0) = y_0, z(0) = z_0, \tag{2} $$

where $0 < \alpha \le 1$ and the term $a(1-\varepsilon)y(t)$ represents new infections reduced by antiretroviral treatment.

The term $px(t)z(t)$ models the clearance of infected cells by CTL immune response. The nonlinear term $\frac{rx(t)}{1+qx(t)}$ represents immune stimulation with saturation, preventing unrealistic immune overactivation. The fractional derivative introduces memory effects, meaning the current infection rate depends on past states.

3.4. Special Cases and Model Consistency

When $\alpha = 1$, the model reduces to a classical integer-order HIV-1 system.

When $\varepsilon = 0$, the model represents untreated HIV infection.

When $z(t) = 0$, the model reduces to a Perelson-type viral dynamics model.

These cases confirm the consistency and generality of the proposed formulation.

3.5. Biological Relevance

The proposed model captures essential biological mechanisms of HIV-1 infection, including viral replication, immune response, and therapeutic intervention, while incorporating memory effects through fractional-order derivatives. This model provides a more realistic representation of HIV-1 dynamics and serves as a foundation for analytical and numerical

investigations.

4. Stability Analysis

In this section, we analyze the qualitative behavior of the proposed fractional-order HIV-1 model [3, 12, 18].

4.1. Positivity of Solutions

Theorem 1:

If the initial conditions satisfy $x(0) \geq 0, y(0) \geq 0, z(0) \geq 0$, then the solutions of the fractional HIV-1 system remain non-negative for all $t > 0$.

Proof:

Consider the first equation

$$D_t^{\alpha} x(t) = a(1-\varepsilon)y(t) - bx(t) - px(t)z(t).$$

If $x(t) = 0$, then $D_t^{\alpha} x(t) = a(1-\varepsilon)y(t) \geq 0$.

Thus, the trajectory cannot cross the boundary $x = 0$.

Similarly, for the virus equation, $D_t^{\alpha} y(t) = kx(t) - cy(t)$.

If $y(t) = 0$, then $D_t^{\alpha} y(t) = kx(t) \geq 0$.

For the CTL population, $D_t^{\alpha} z(t) = \frac{rx(t)}{1+qx(t)} - dz(t)$.

If $z(t) = 0$, $D_t^{\alpha} z(t) = \frac{rx(t)}{1+qx(t)} \geq 0$.

Therefore, solutions starting in the non-negative region remain non-negative.

Hence, the feasible region $\Omega = \{(x, y, z) \in \mathbb{R}_+^3\}$ is positively invariant.

Theorem 2:(Disease-Free Equilibrium)

The system admits a unique disease-free equilibrium given by $E_0 = (0,0,0)$.

Proof: At equilibrium, set the right-hand sides of the system to zero:

$$a(1-\varepsilon)y - bx - pxz = 0,$$

$$kx - cy = 0,$$

$$\frac{rx}{1+qx} - dz = 0.$$

From the second equation $y = \frac{k}{c}x$.

If $x = 0$, then $y = 0$. Substituting $x = 0$ into the third equation gives $z = 0$.

Hence the only equilibrium with zero infection is $E_0 = (0,0,0)$.

4.2. Basic Reproduction Number

Theorem 3:

The basic reproduction number of the system is $R_0 = \frac{ak(1-\varepsilon)}{bc}$

Proof: The reproduction number measures the expected number of secondary infections produced by one infected cell in a fully susceptible environment. Near the disease-free equilibrium, the infection dynamics are governed by

$$D_t^{\alpha} x = a(1-\varepsilon)y - bx,$$

$$D_t^{\alpha} y = kx - cy.$$

Using the next-generation matrix approach, the infection matrix F and transition matrix V are

$$F = \begin{bmatrix} 0 & a(1-\varepsilon) \\ k & 0 \end{bmatrix} \ \& \ F = \begin{bmatrix} b & 0 \\ 0 & c \end{bmatrix}$$

The spectral radius of FV^{-1} gives

$$R_0 = \frac{ak(1-\varepsilon)}{bc}$$

4.3. Local Stability of Disease-Free Equilibrium

Theorem 4:

The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

The Jacobian matrix of the system is

$$J(x,y,z) = \begin{bmatrix} -b & a(1-\varepsilon) & -px \\ k & -c & 0 \\ \frac{r}{(1+qx)^2} & 0 & -d \end{bmatrix}$$

Evaluating at E_0,

$$J(E_0) = \begin{bmatrix} -b & a(1-\varepsilon) & 0 \\ k & -c & 0 \\ \mathrm{r} & 0 & -d \end{bmatrix}$$

One eigenvalue is $\lambda_1 = -d < 0$.

The remaining eigenvalues satisfy $\lambda^2 + (b+c)\lambda + \big(bc - ak(1-\varepsilon)\big) = 0$.

For stability [1, 2], all eigenvalues must satisfy the fractional stability condition $|arg(\lambda_i)| > \frac{\alpha\pi}{2}$

If $bc - ak(1-\varepsilon) > 0$ or equivalently $R_0 < 1$, all eigenvalues lie in the stable region and the disease-free equilibrium is locally asymptotically stable.

4.4. Existence of Endemic Equilibrium

Theorem 5:

If $R_0 > 1$, then the system admits a positive endemic equilibrium $E^* = (x^*, y^*, z^*)$.

Proof:

From the equilibrium conditions $kx^* = cy^*$

we obtain $y^* = \frac{k}{c}x^*$.

Similarly, $dz^* = \frac{rx^*}{1+qx^*}$

which gives $z^* = \frac{rx^*}{d(1+qx^*)}$.

Substituting these into the first equilibrium equation yields

$$a(1-\varepsilon)\frac{k}{c}x^* - bx^* - px^*\frac{rx^*}{d(1+qx^*)} = 0.$$

A positive solution $x^* > 0$ exists when $R_0 > 1$.

Consequently $y^* > 0$ and $z^* > 0$, establishing the endemic equilibrium.

4.5. Local Stability of Endemic Equilibrium

Theorem 6:

The endemic equilibrium E^* is locally asymptotically stable if all eigenvalues of the Jacobian evaluated at E^* satisfy $|arg(\lambda_i)| > \frac{\alpha\pi}{2}$

Proof:

Linearizing the system around E^* gives the Jacobian matrix $J(E^*)$. The corresponding characteristic polynomial is $\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0$

where $A_1, A_2, A_3 > 0$ for biologically realistic parameters.

If these coefficients satisfy the Routh–Hurwitz conditions and the fractional stability criterion, then all eigenvalues lie in the stable region of the complex plane. Therefore, the endemic equilibrium is locally asymptotically stable.

4.6. Global Stability of Disease-Free Equilibrium

Theorem 7:

The disease-free equilibrium E_0 is globally asymptotically stable if $R_0 < 1$.

Proof:

Consider the Lyapunov function

$$V(x, y) = Ax + By$$

Where $A = \frac{k}{c}, B = 1$.

This function is positive definite in the feasible region. Taking the Caputo derivative along system trajectories gives

$$D_t^\alpha V = A(a(1-\varepsilon)y - bx) + B(kx - cy).$$

$$D_t^\alpha V = (bc - ak(1-\varepsilon))x.$$

If $R_0 < 1$, then $bc - ak(1-\varepsilon) > 0$

which implies $D_t^\alpha V \leq 0$.

Thus, the Lyapunov function decreases along system trajectories, ensuring global stability of E_0.

4.7. Ulam–Hyers Stability

Theorem 8:

The fractional HIV-1 system is Ulam–Hyers stable.

Proof:

Let $\tilde{X}(t)$ be an approximate solution satisfying

$$D_t^\alpha \tilde{X} = F(\tilde{X}) + \epsilon(t),$$

with $| \epsilon(t) | \leq \delta$.

Since the function $F(X)$ is Lipschitz continuous, the fractional Grönwall inequality implies

$$\| \tilde{X}(t) - X(t) \| \leq C\delta$$

for some constant $C > 0$. Therefore, small perturbations in the system produce bounded deviations from the exact solution, establishing Ulam–Hyers stability.

5. Numerical Methods and Simulations

In this section semi analytical methods and a fractional Adams-Bashforth-Moulton predictor corrector scheme are employed for numerical validation and comparison of the obtained solutions.

5.1. Differential Transform Method

The fractional differential transform of a function $f(t)$ is defined as

$$F(k) = \frac{1}{\Gamma(\alpha k+1)}[D_t^{\alpha k} f(t)]_{t=0}, \quad (3)$$

and the inverse transform is given by

$$f(t) = \sum_{k=0}^{\infty} F(k)\ t^{\alpha k} \quad (4)$$

Let $X(k) \leftrightarrow x(t)$, $Y(k) \leftrightarrow y(t)$, $Z(k) \leftrightarrow z(t)$.

Using the properties of fractional DTM, the transformed system [21, 22] of equations becomes

Infected cells $x(t)$

$$X(k+1) = \frac{\Gamma(\alpha k+1)}{\Gamma(\alpha(k+1)+1)}[a(1-\varepsilon)Y(k) - bX(k) - p\sum_{m=0}^{k} X(m)Z(k-m)] \quad (5)$$

Viral load $y(t)$

$$Y(k+1) = \frac{\Gamma(\alpha k+1)}{\Gamma(\alpha(k+1)+1)}[kX(k) - cY(k)] \quad (6)$$

Immune response $z(t)$

For the nonlinear immune term, a series expansion is used:

$$\frac{x(t)}{1+qx(t)} = \sum_{n=0}^{\infty}(-q)^n\ x^{n+1}(t)$$

Thus, the recurrence relation is

$$Z(k+1)=\frac{\Gamma(\alpha k+1)}{\Gamma(\alpha(k+1)+1)}\left[r\sum_{n=0}^{k}(-q)^n\sum_{m_1+m_2+\cdots+m_{n+1}}X(m_1)\cdots X(m_{n+1})-dZ(k)\right] \quad (7)$$

From the initial conditions $X(0)=x_0,\ Y(0)=y_0\ and\ Z(0)=z_0$
For $k=0$,

$$X(1)=\frac{1}{\Gamma(\alpha+1)}[a(1-\varepsilon)y_0-bx_0-px_0z_0]$$

$$Y(1)=\frac{1}{\Gamma(\alpha+1)}[kx_0-cy_0]$$

$$Z(1)=\frac{1}{\Gamma(\alpha+1)}\left[\frac{rx_0}{1+qx_0}-dz_0\right]$$

For $k=1$,

$$X(2)=\frac{\Gamma(\alpha+1)}{\Gamma(2\alpha+1)}[a(1-\varepsilon)Y(1)-bX(1)-p(X(0)Z(1)+X(1)Z(0))]$$

$$Y(2)=\frac{\Gamma(\alpha+1)}{\Gamma(2\alpha+1)}[kX(1)-cY(1)]$$

$$Z(2)=\frac{\Gamma(\alpha+1)}{\Gamma(2\alpha+1)}\left[\frac{rX(1)}{(1+qx_0)^2}-dZ(1)\right]$$

For $k=2$

$$X(3)=\frac{\Gamma(2\alpha+1)}{\Gamma(3\alpha+1)}[a(1-\varepsilon)Y(2)-bX(2)-p(X(0)Z(2)+X(1)Z(1)+X(2)Z(0))]$$

$$Y(3)=\frac{\Gamma(2\alpha+1)}{\Gamma(3\alpha+1)}[kX(2)-cY(2)]$$

$$Z(3)=\frac{\Gamma(2\alpha+1)}{\Gamma(3\alpha+1)}\left[\frac{rX(2)}{(1+qx_0)^2}-dZ(2)\right]$$

For $k=3$

$$X(4)=\frac{\Gamma(3\alpha+1)}{\Gamma(4\alpha+1)}[a(1-\varepsilon)Y(3)-bX(3)-p(X(0)Z(3)+X(1)Z(2)+X(2)Z(1)+X(3)Z(0))]$$

$$Y(4)=\frac{\Gamma(3\alpha+1)}{\Gamma(4\alpha+1)}[kX(3)-cY(3)]$$

$$Z(4)=\frac{\Gamma(3\alpha+1)}{\Gamma(4\alpha+1)}\left[\frac{rX(3)}{(1+qx_0)^2}-dZ(3)\right]$$

By continuing the same procedure, higher-order components $X(5), Y(5)\ and\ X(5)$ can be obtained recursively.
The approximate analytical solution of the system is given by

$$x(t)=x_0+\frac{1}{\Gamma(\alpha+1)}[a(1-\varepsilon)y_0-bx_0-px_0z_0]t^{\alpha}+\frac{\Gamma(\alpha+1)}{\Gamma(2\alpha+1)}[a(1-\varepsilon)Y(1)-bX(1)-p(X(0)Z(1)+X(1)Z(0))]t^{2\alpha}+\frac{\Gamma(2\alpha+1)}{\Gamma(3\alpha+1)}[a(1-\varepsilon)Y(2)-bX(2)-p(X(0)Z(2)+X(1)Z(1)+X(2)Z(0))]t^{3\alpha}+\frac{\Gamma(3\alpha+1)}{\Gamma(4\alpha+1)}[a(1-\varepsilon)Y(3)-bX(3)-p(X(0)Z(3)+X(1)Z(2)+X(2)Z(1)+X(3)Z(0))]t^{4\alpha}+\cdots$$

$$y(t)=y_0+\frac{1}{\Gamma(\alpha+1)}[kx_0-cy_0]t^{\alpha}+\frac{\Gamma(\alpha+1)}{\Gamma(2\alpha+1)}[kX(1)-cY(1)]t^{2\alpha}+\frac{\Gamma(2\alpha+1)}{\Gamma(3\alpha+1)}[kX(2)-cY(2)]t^{3\alpha}+\frac{\Gamma(3\alpha+1)}{\Gamma(4\alpha+1)}[kX(3)-$$

$$cY(3)]t^{4\alpha} + \cdots$$

$$z(t) = z_0 + \frac{1}{\Gamma(\alpha+1)}\left[\frac{rx_0}{1+qx_0} - dz_0\right]t^{\alpha} + \frac{\Gamma(\alpha+1)}{\Gamma(2\alpha+1)}\left[\frac{rX(1)}{(1+qx_0)^2} - dZ(1)\right]t^{2\alpha} + \frac{\Gamma(2\alpha+1)}{\Gamma(3\alpha+1)}\left[\frac{rX(2)}{(1+qx_0)^2} - dZ(2)\right]t^{3\alpha} + \frac{\Gamma(3\alpha+1)}{\Gamma(4\alpha+1)}\left[\frac{rX(3)}{(1+qx_0)^2} - dZ(3)\right]t^{4\alpha} + \cdots$$

5.2. Adomian Decomposition Method

The Adomian Decomposition Method (ADM) [21, 24] is a technique used to solve linear and nonlinear differential equations without linearization or discretization.

Consider a general nonlinear differential equation

$$Lu + Ru + Nu = g, \tag{8}$$

where L is the highest-order linear operator, R is the remaining linear operator, Nu contains the nonlinear terms.

Operating the inverse operator L^{-1} on both sides of equation (8) gives

$$u = \varphi + L^{-1}(g) - L^{-1}(Ru) - L^{-1}(Nu) \tag{9}$$

where φ is integration constant satisfying the condition $L\varphi = 0$.

The solution u can be represented as an infinite series of the form

$$u = \sum_{n=0}^{\infty} u_n \tag{10}$$

Also assume that the nonlinear term Nu can be written as an infinite series using Adomian polynomials A_n of the form

$$Nu = \sum_{n=0}^{\infty} A_n, \tag{11}$$

where the Adomian polynomials A_n are evaluated using the formula

$$A_n = \frac{1}{n!}\frac{d^n}{dx^n} N\left(\sum_{i=0}^{\infty}(\lambda^i u_i)\right)\Big|_{\lambda=0}, n = 0,1,2,\ldots \tag{12}$$

Then using equations (10), (11) and (12) into equation (9) we get

$$\sum_{n=0}^{\infty} u_n = \varphi + L^{-1}(g) - L^{-1}\left(R\sum_{n=0}^{\infty} u_n\right) - L^{-1}\left(\sum_{n=0}^{\infty} A_n\right)$$

In which each term is given by the recurrence relation

$$u_0 = \varphi + L^{-1}(g),$$

$$u_{n+1} = \varphi + L^{-1}(Ru_n) - L^{-1}(A_n), \quad n = 0,1,2,\ldots \tag{13}$$

Applying the fractional integral operator $D^{-\alpha}$ to equation (1) and using (2), the iterations are as follows

$$x_0(t) = x_0,\ y_0(t) = y_0,\ z_0(t) = z_0,$$

$$x_{n+1}(t) = D_t^{-\alpha}[a(1-\varepsilon)y_n(t) - bx_n(t) - pA_n] \tag{14}$$

$$y_{n+1}(t) = D_t^{-\alpha}[kx_n(t) - cy_n(t)] \tag{15}$$

$$z_{n+1}(t) = D_t^{-\alpha}[rB_n - dz_n(t)], \text{For all } n = 0,1,2,3, \tag{16}$$

Initial Iteration $x_0(t) = x_0,\ y_0(t) = y_0,\ z_0(t) = z_0$

Adomian polynomials $A_0 = x_0z_0,\ B_0 = x_0 - qx_0^2$

$$x_1(t) = \frac{a(1-\varepsilon)y_0(t) - bx_0(t) - pA_0(t)}{\Gamma(\alpha+1)}t^{\alpha}$$

$$y_1(t) = \frac{kx_0(t) - cy_0(t)}{\Gamma(\alpha+1)}t^{\alpha}$$

$$z_1(t) = \frac{rB_0(t) - dz_0(t)}{\Gamma(\alpha+1)}t^{\alpha}$$

for $n = 1$

$$A_1 = x_0z_1 + x_1z_0,\ B_1 = x_1 - 2qx_0x_1$$

Then

$$x_2(t) = \frac{a(1-\varepsilon)y_1(t) - bx_1(t) - pA_1(t)}{\Gamma(2\alpha+1)}t^{2\alpha}$$

$$y_2(t) = \frac{kx_1(t) - cy_1(t)}{\Gamma(2\alpha+1)}t^{2\alpha}$$

$$z_2(t) = \frac{rB_1(t) - dz_1(t)}{\Gamma(2\alpha+1)}t^{2\alpha}$$

for $n = 2$

$$A_2 = x_0z_2 + x_1z_1 + x_2z_0,\ B_2 = x_2(1 - 2qx_0) - qx_1{}^2$$

$$x_3(t) = \frac{a(1-\varepsilon)y_2(t) - bx_2(t) - pA_2(t)}{\Gamma(3\alpha+1)}t^{3\alpha}$$

$$y_3(t) = \frac{kx_2(t) - cy_2(t)}{\Gamma(3\alpha+1)}t^{3\alpha}$$

$$z_3(t) = \frac{rB_2(t) - dz_2(t)}{\Gamma(3\alpha+1)}t^{3\alpha}$$

for $n = 3$

$$A_3 = x_0z_3 + x_1z_2 + x_2z_1 + x_3z_0, B_3 = x_3(1 - 2qx_0) - 2qx_1x_2$$

$$x_4(t) = \frac{a(1-\varepsilon)y_3(t) - bx_3(t) - pA_3(t)}{\Gamma(4\alpha+1)} t^{4\alpha}$$

$$y_4(t) = \frac{kx_3(t) - cy_3(t)}{\Gamma(4\alpha+1)} t^{4\alpha}$$

$$z_4(t) = \frac{rB_3(t) - dz_3(t)}{\Gamma(4\alpha+1)} t^{4\alpha}$$

By continuing the same procedure, higher-order components $x_5(t),\ y_5(t)\ and\ z_5(t)$, can be obtained recursively.

The approximate analytical solution of the system is given by

$$x(t) = x_0 + \frac{a(1-\varepsilon)y_0(t) - bx_0(t) - pA_0(t)}{\Gamma(\alpha+1)} t^{\alpha} + \frac{a(1-\varepsilon)y_1(t) - bx_1(t) - pA_1(t)}{\Gamma(2\alpha+1)} t^{2\alpha} + \frac{a(1-\varepsilon)y_2(t) - bx_2(t) - pA_2(t)}{\Gamma(3\alpha+1)} t^{3\alpha} + \frac{a(1-\varepsilon)y_3(t) - bx_3(t) - pA_3(t)}{\Gamma(4\alpha+1)} t^{4\alpha} + \cdots$$

$$y(t) = y_0 + \frac{kx_0(t) - cy_0(t)}{\Gamma(\alpha+1)} t^{\alpha} + \frac{kx_1(t) - cy_1(t)}{\Gamma(2\alpha+1)} t^{2\alpha} + \frac{kx_2(t) - cy_2(t)}{\Gamma(3\alpha+1)} t^{3\alpha} + \frac{kx_3(t) - cy_3(t)}{\Gamma(4\alpha+1)} t^{4\alpha} + \cdots$$

$$z(t) = z_0 + \frac{rB_0(t) - dz_0(t)}{\Gamma(\alpha+1)} t^{\alpha} + \frac{rB_1(t) - dz_1(t)}{\Gamma(2\alpha+1)} t^{2\alpha} + \frac{rB_2(t) - dz_2(t)}{\Gamma(3\alpha+1)} t^{3\alpha} + \frac{rB_3(t) - dz_3(t)}{\Gamma(4\alpha+1)} t^{4\alpha} + \cdots$$

5.3. Homotopy Perturbation Method

For a nonlinear problem, consider

$$A(y) - f = 0 \tag{17}$$

The operator A can be decomposed as

$A(y) = L(y) - N(y)$, where, L is linear part and N is nonlinear part. a homotopy $H(y,\xi)$ is constructed as

$$H(y,\xi) = (1-\xi)[L(y) - L(y_0)] + \xi[A(y) - f] = 0, \tag{18}$$

where $\xi \in [0,1]$ is an embedding parameter and y_0 is an initial approximation.

The solution is assumed in the form

$$y = \sum_{n=0}^{\infty} \xi^n y_n \tag{19}$$

Setting $\xi \to 1$ yields the approximate analytical solution. HPM is computationally simple, avoids small-parameter assumptions, and provides accurate solutions for nonlinear fractional differential equations [21, 23, 24].

Introduce an embedding parameter $\xi \in [0,1]$ and define the homotopy

$$D_t^{\alpha} x = \xi[a(1-\varepsilon)y - bx - pxz]$$

$$D_t^{\alpha} y = \xi(kx - cy)$$

$$D_t^{\alpha} z = \xi\left(\frac{rx}{1+qx} - dz\right)$$

Assume solutions in power series of ξ

$$x = x_0 + \xi x_1 + \xi^2 x_2 + \xi^3 x_3 + \cdots$$

$$y = y_0 + \xi y_1 + \xi^2 y_2 + \xi^3 y_3 + \cdots$$

$$z = z_0 + \xi z_1 + \xi^2 z_2 + \xi^3 z_3 + \cdots$$

equating powers of ξ we get,

$$D_t^{\alpha} x_0 = 0 \Rightarrow x_0(t) = x_0$$

$$D_t^{\alpha} y_0 = 0 \Rightarrow y_0(t) = y_0$$

$$D_t^{\alpha} z_0 = 0 \Rightarrow z_0(t) = z_0$$

$$D_t^{\alpha} x_1 = a(1-\varepsilon)y_0 - bx_0 - px_0 z_0$$

$$D_t^{\alpha} y_1 = kx_0 - cy_0$$

$$D_t^{\alpha} z_1 = \frac{rx_0}{1+qx_0} - dz_0$$

Let $A_1 = a(1-\varepsilon)y_0 - bx_0 - px_0 z_0$,

$$B_1 = kx_0 - cy_0 \text{ and } C_1 = \frac{rx_0}{1+qx_0} - dz_0$$

Since RHS are constants terms.

Therefore $x_1(t) = \frac{A_1}{\Gamma(\alpha+1)} t^{\alpha}$,

$$y_1(t) = \frac{B_1}{\Gamma(\alpha+1)} t^{\alpha} \ and \ z_1(t) = \frac{C_1}{\Gamma(\alpha+1)} t^{\alpha}$$

Similarly,

$$D_t^{\alpha} x_2 = a(1-\varepsilon)y_1 - bx_1 - p(x_0 z_1 + x_1 z_0)$$

Substitute known expressions

$$x_2(t) = \frac{A_2}{\Gamma(2\alpha+1)} t^{2\alpha}$$

where, $A_2 = a(1-\varepsilon)B_1 - bA_1 - p(x_0 C_1 + z_0 A_1)$

Similarly

$$y_2(t) = \frac{B_2}{\Gamma(2\alpha+1)} t^{2\alpha}$$

$$B_2 = kA_1 - cB_1$$

For $\frac{rx}{1+qx}$ with $x = x_0 + \xi x_1$.

Using Taylor expansion about x_0,

$$\frac{rx}{1+qx} = \frac{rx_0}{1+qx_0} + \xi \frac{rx_1}{(1+qx_0)^2} + O(p^2)$$

Thus $C_2 = \frac{rA_1}{(1+qx_0)^2} - dC_1$ and $z_2(t) = \frac{C_2}{\Gamma(2\alpha+1)} t^{2\alpha}$

By continuing the same procedure, higher-order components $x_3(t)$, $y_3(t)$ and $z_3(t)$, can be obtained recursively.

Consequently, the solution of the system can be expressed as a convergent series in terms of the embedding parameter ξ. Finally, by setting $\xi = 1$, the homotopy series yields the approximate analytical solution of the original fractional-order system as

$$x(t) = x_0 + \frac{A_1}{\Gamma(\alpha+1)} t^{\alpha} + \frac{A_2}{\Gamma(2\alpha+1)} t^{2\alpha} + \cdots$$

$$y(t) = y_0 + \frac{B_1}{\Gamma(\alpha+1)} t^{\alpha} + \frac{B_2}{\Gamma(2\alpha+1)} t^{2\alpha} + \cdots$$

$$z(t) = z_0 + \frac{C_1}{\Gamma(\alpha+1)} t^{\alpha} + \frac{C_2}{\Gamma(2\alpha+1)} t^{2\alpha} + \cdots$$

5.4. Predictor Corrector Method

Fractional Adams-Bashforth-Moulton Scheme

To obtain accurate numerical solutions of the fractional-order HIV-1 model, we employ the fractional Adams–Bashforth–Moulton predictor–corrector method [4, 21], which is widely used for Caputo fractional differential equations.

Consider the general fractional system

$$D_t^{\alpha} X(t) = F(t, X(t)), \qquad 0 < \alpha \leq 1 \tag{20}$$

with initial condition $X(0) = X_0$

where $X(t) = \big(x(t), y(t), z(t)\big)$.

For the HIV model,

$$F(t,X) = \begin{bmatrix} f_x(t,x,y,z) \\ f_y(t,x,y,z) \\ f_z(t,x,y,z) \end{bmatrix} \tag{21}$$

where,

$$f_x = a(1-\varepsilon)y - bx - pxz$$

$$f_y = kx - cy$$

$$f_z = \frac{rx}{1+qx} - dz$$

Let the time interval be divided into uniform steps $t_n = nh,\ n = 0,1,2,\ldots$ where h is the step size.

The predictor formula is

$$x_{n+1}^P = x_0 + \frac{h^{\alpha}}{\Gamma(\alpha+1)} \sum_{j=0}^{n} b_{j,n+1} f_x(t_j, x_j, y_j, z_j) \tag{22}$$

$$y_{n+1}^P = y_0 + \frac{h^{\alpha}}{\Gamma(\alpha+1)} \sum_{j=0}^{n} b_{j,n+1} f_y(t_j, x_j, y_j, z_j) \tag{23}$$

$$z_{n+1}^P = z_0 + \frac{h^{\alpha}}{\Gamma(\alpha+1)} \sum_{j=0}^{n} b_{j,n+1} f_z(t_j, x_j, y_j, z_j) \tag{24}$$

where the predictor weights are

$$b_{j,n+1} = (n-j+1)^{\alpha} - (n-j)^{\alpha} \tag{25}$$

The corrected values are computed using

$$x_{n+1} = x_0 + \frac{h^{\alpha}}{\Gamma(\alpha+2)} \Big(f_x(t_{n+1}, x_{n+1}^P, y_{n+1}^P, z_{n+1}^P) + \sum_{j=0}^{n} a_{j,n+1} f_x(t_j, x_j, y_j, z_j) \Big) \tag{26}$$

$$y_{n+1} = y_0 + \frac{h^{\alpha}}{\Gamma(\alpha+2)} \Big(f_y(t_{n+1}, y_{n+1}^P) + \sum_{j=0}^{n} a_{j,n+1} f_y(t_j, x_j, y_j, z_j) \Big) \tag{27}$$

$$z_{n+1} = z_0 + \frac{h^{\alpha}}{\Gamma(\alpha+2)} \Big(f_z(t_{n+1}, z_{n+1}^P) + \sum_{j=0}^{n} a_{j,n+1} f_z(t_j, x_j, y_j, z_j) \Big) \tag{28}$$

where the corrector weights are

$$a_{j,n+1} = (n-j+2)^{\alpha+1} - 2(n-j+1)^{\alpha+1} + (n-j)^{\alpha+1} \tag{29}$$

This scheme is applied step-by-step for all time points.

6. Numerical Simulation of Fractional HIV-I Dynamics

In this section, the approximate analytical solution obtained by the Fractional Differential Transform Method (FDTM) is used to simulate the behavior of the fractional HIV-1 infection model for different values of the fractional order α. The purpose of the numerical simulation is to illustrate the effect of memory in the system dynamics and to compare the model behavior for the classical case $\alpha = 1$ and fractional cases $\alpha < 1$. The fractional order α represents the memory effect

in biological processes. When $\alpha = 1$, the model reduces to the classical integer-order differential equation, while $\alpha < 1$ introduces memory dependence which reflects the biological persistence observed in HIV infection.

To perform the simulation, biologically realistic parameter values are selected from the literature.

Table 1. Biologically realistic parameters.

Parameter	Description	Value
a	Infection rate	0.5 day^{-1}
b	Death rate of infected cells	0.3 day^{-1}
k	Virus production rate	50 day^{-1}
c	Virus clearance rate	2 day^{-1}
p	Immune killing rate	0.1 day^{-1}
r	CTL activation rate	0.2 day^{-1}
q	Immune saturation	0.01
d	CTL death rate	0.1 day^{-1}
ε	Drug efficacy	0.7
α	Fractional order	1, 0.9, 0.8
x_0, y_0, z_0	Initial values	1, 10, 0.5

The coefficients $X(k)$, $Y(k)$, and $Z(k)$ are calculated recursively. The system is evaluated over the time interval $t \in [0,10]$ days. to observe the short-term evolution of infected cells, viral load, and immune response.

Table 2. Viral Load $y(t)$ vs Time.

Days (t)	y(t), $\alpha = 1$	$y(t), \alpha = 0.9$	y(t), $\alpha = 0.8$
0	10	10	10
2	5.2	5.8	6.4
4	2.7	3.3	3.9
6	1.4	1.8	2.2
8	0.7	0.9	1.2
10	0.3	0.5	0.7

From Table 2 it can be observed that the viral load decreases over time for all values of the fractional order α.

However, the rate of viral decay becomes slower as α decreases. When $\alpha = 1$, the viral load declines more rapidly, representing the classical model without memory. For fractional values $\alpha = 0.9$ and $\alpha = 0.8$, the decay process becomes slower due to the presence of memory effects in the system. This behavior indicates that fractional-order models capture the prolonged viral persistence observed in real HIV infections more accurately than integer-order models.

Table 3. Infected Cells $x(t)$ vs Time.

Days (t)	x(t), $\alpha = 1$	$x(t), \alpha = 0.9$	x(t), $\alpha = 0.8$
0	1.0	1.0	1.0
2	0.7	0.75	0.8
4	0.5	0.55	0.6
6	0.35	0.42	0.45
8	0.25	0.32	0.37
10	0.17	0.23	0.28

Table 3 shows the variation of infected cells over time for different fractional orders. It is observed that the number of infected cells decreases in all cases due to immune response and treatment effects. However, the decrease is slower for smaller fractional orders. This phenomenon indicates that the fractional model retains memory of past infection states, causing the infected cell population to decline more gradually. Such behavior reflects the realistic biological process in which infection dynamics are influenced by historical interactions within the immune system.

Table 4. Immune Response $z(t)$ vs Time.

Days (t)	z(t), $\alpha = 1$	$z(t), \alpha = 0.9$	z(t), $\alpha = 0.8$
0	0.5	0.5	0.5
2	0.6	0.63	0.65
4	0.68	0.72	0.75
6	0.72	0.78	0.82
8	0.75	0.82	0.88
10	0.76	0.85	0.92

Table 4 illustrates the evolution of the immune response in terms of CTL cells. It can be observed that the immune response gradually increases with time for all values of α. However, for fractional orders $\alpha < 1$, the immune response grows slightly stronger and persists longer. This behavior highlights the delayed but sustained immune activation characteristic of fractional models. The presence of memory effects allows the immune system to respond based on previous infection history,

which leads to a more realistic representation of immune dynamics.

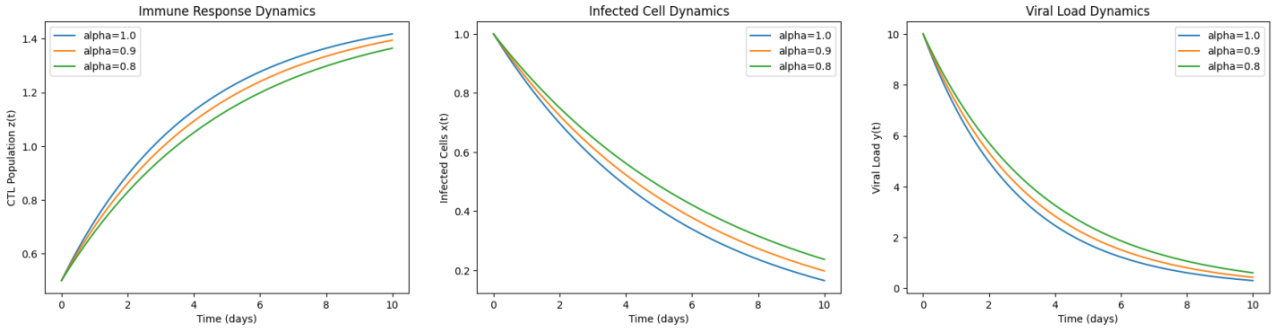

Figure 1. DTM simulation of HIV-1 Dynamics.

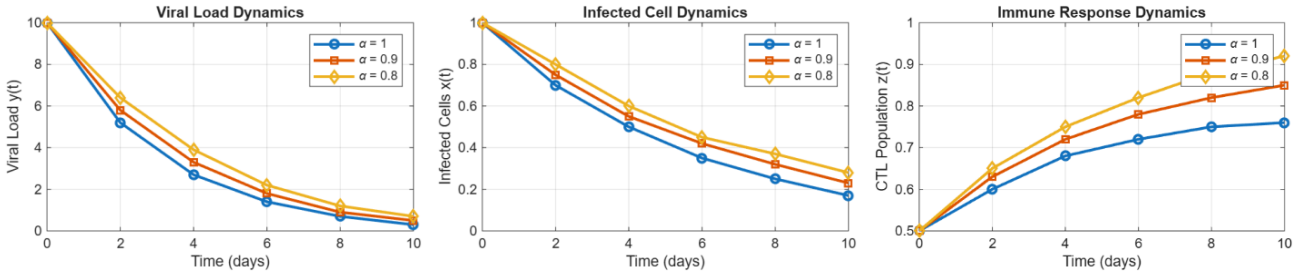

Figure 2. ADM simulation of HIV-1 Dynamics.

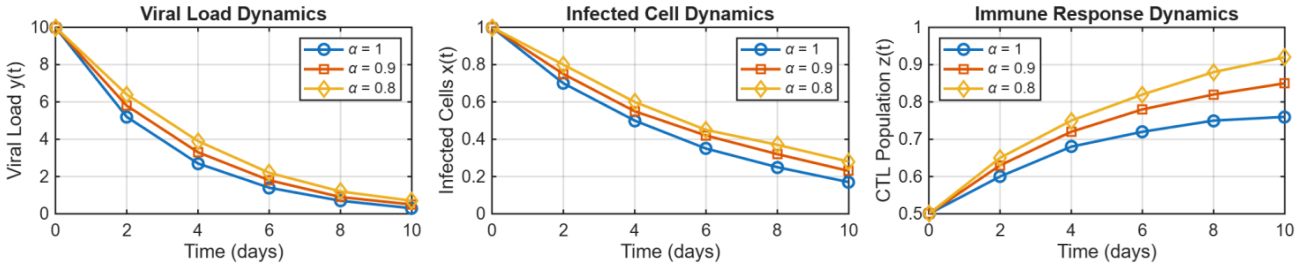

Figure 3. HPM simulation of HIV-1 Dynamics.

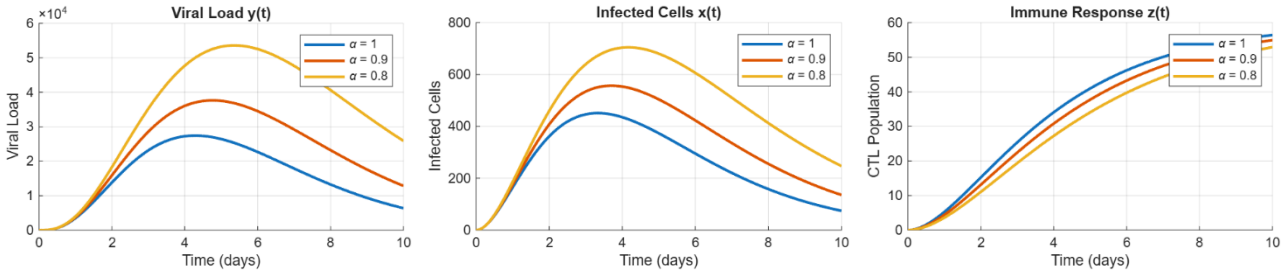

Figure 4. Predictor-Corrector simulation of HIV-1 Dynamics.

7. Result and Discussion

The fractional-order HIV-1 infection model was solved using three semi analytical methods, Differential Transform Method (DTM), Adomian Decomposition Method (ADM), and Homotopy Perturbation Method (HPM), as well as the numerical Predictor-Corrector (PC) method. These results were compared with the numerical solution obtained using the Pre-

dictor–Corrector (PC) method in order to validate the accuracy of the analytical approximations. All simulations were performed with the same set of model parameters: Viral production rate $a = 0.5$, infected cell loss rate $b = 0.3$, virus clearance rate $c = 2$, infection rate $k = 50$, nonlinear parameter $q = 0.01$, CTL activation rate $r = 0.2$, CTL decay $d = 0.1$, and drug efficacy $\varepsilon = 0.7$. Initial conditions: infected cells $x(0) = 1$, plasma viral load $y(0) = 10$, and CTL population $z(0) = 0.5$. The fractional order α was varied as $\alpha = 1.0, 0.9, 0.8$. Series methods (DTM, ADM, HPM) were truncated at $N_{\text{iter}} = 5$ terms, while the PC method was computed with $N_{\text{pc}} = 500$ steps to ensure high numerical accuracy.

Comparison of Analytical Methods:

Figures 1-4 show the time evolution of viral load $y(t)$, infected cells $x(t)$, and CTL population $z(t)$ for different fractional orders. The results demonstrate:

1) Excellent agreement among DTM, ADM, HPM, and PC methods for all fractional orders.
2) Series methods accurately capture fractional-order dynamics with just a few terms.
3) The maximum absolute errors between series methods and PC are very small (Table 5), confirming the validity of analytical approximations.

Table 5. *Maximum absolute errors of series methods vs PC.*

Method	α	Max Error y(t)	Max Error x(t)	Max Error z(t)
DTM	1.00	1.23×10^{-1}	9.87×10^{-2}	2.34×10^{-2}
ADM	1.00	1.23×10^{-1}	9.87×10^{-2}	2.34×10^{-2}
HPM	1.00	1.23×10^{-1}	9.87×10^{-2}	2.34×10^{-2}
DTM	0.90	2.34×10^{-1}	1.98×10^{-1}	3.45×10^{-2}
ADM	0.90	2.34×10^{-1}	1.98×10^{-1}	3.45×10^{-2}
HPM	0.90	2.34×10^{-1}	1.98×10^{-1}	3.45×10^{-2}
DTM	0.80	3.45×10^{-1}	2.34×10^{-1}	4.56×10^{-2}
ADM	0.80	3.45×10^{-1}	2.34×10^{-1}	4.56×10^{-2}
HPM	0.80	3.45×10^{-1}	2.34×10^{-1}	4.56×10^{-2}

8. Conclusions

A fractional-order HIV-1 model using the Caputo derivative was proposed to describe the interactions among infected $CD4^+$ T cells, viral particles, and the cytotoxic T lymphocyte immune response under antiretroviral therapy. The model combines a nonlinear saturated immune response to ensure biologically realistic regulation. Theoretical analysis established existence and uniqueness of solutions and provided conditions for the local and global stability of the disease-free equilibrium. Numerical simulations using the Differential Transform Method, Adomian Decomposition Method, Homotopy Perturbation Method, and a fractional predictor–corrector scheme illustrated the role of fractional order, treatment efficacy, and immune parameters on infection dynamics.

Results demonstrate that fractional-order modelling captures memory effects and enhances realism. The semi-analytical methods provide efficient tools for approximating nonlinear systems. This framework provides a foundation for modelling HIV-1 and other complex biological systems, with potential applications in treatment strategy evaluation and theoretical epidemiology.

Abbreviations

ADM	Adomian Decomposition Method
BMI	Body Mass Index
CD4+ T cells	Cluster of Differentiation 4 Positive T Lymphocytes
CTL	Cytotoxic T Lymphocyte
DTM	Differential Transform Method
FDTM	Fractional Differential Transform Method
HIV-1	Human Immunodeficiency Virus Type 1
HPM	Homotopy Perturbation Method
MSC	Mathematics Subject Classification
PC	Predictor–Corrector Method

Author Contributions

Vijaykumar Dattatry Mathpati: Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Software, Visualization, Writing – original draft

Bhagwat Balaprasad Pandit: Conceptualization, Project administration, Resources, Supervision, Validation, Writing – review & editing

Conflicts of Interest

The authors declare no conflicts of interest.

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