



Analytic study for a fractional model of HIV infection of CD4⁺T lymphocyte cells



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Abstract

In this article, we implemented homotopy transform methods, namely, homotopy analysis transform method and homotopy perturbation Sumudu transform method to examine the fractional model for HIV infection of CD4⁺T lymphocyte cells. Proliferation of CD4⁺T lymphocyte cells is driven by a combination of the homeostatic response to cells depletion (CD4⁺T cells counts) and viral load (HIV levels). The attraction of both the methods is that an approach of HPSTM is used and on other hand by HATM a large admissible convergence range of series solution is described for standard as well as fractional order nonlinear problems.

Keywords: Homotopy perturbation Sumudu transform method, Laplace transform method, Homotopy analysis transform method, fractional model for HIV infection of CD4⁺T lymphocyte cells, \mathfrak{h} -curves.

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1. Introduction

The model of fractional differential equations have been showed beyond doubt to be an excellent tool to reveal the hidden aspect in various field of science and engineering associated with non-locality [1, 3, 4, 9, 32]. These successes lead up with proposed techniques to system of time-fractional differential equations occurring in HIV infection of CD4⁺T lymphocyte cells. The CD4⁺T lymphocyte cells are the white blood cells that play a crucial role in protecting human body from infection by improving the immune response [10]. They activate body's immune response against "Intruders" like bacteria and viruses. The CD4⁺T lymphocyte cells got killed by the HIV, once virus entered in the body and replicate itself. A healthy individual has CD4⁺T lymphocyte cell count normal as 800 to 1200/ mm³. A fall in CD4⁺T lymphocyte cells count may be due to a thymus failure or a defect in bone. One of the methods

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to decide that the person is living with HIV and is susceptible to move towards various opportunistic infections is detected with very low count of $CD4^+T$ lymphocyte cells (less than $200/mm^3$) [28].

A considerable and remarkable work is done earlier in the field of mathematical dynamical model for HIV infection of $CD4^+T$ cells are: a combination of Adomian decomposition method and Laplace transform [25], multistep Laplace decomposition scheme [8], differential transform technique [34], Bessel collocation method [38], homotopy decomposition method [2] all mentioned above methods have comparatively local point effects and low convergence speed. In proposed techniques we provide rapid convergence solution series in efficient and effective way for strongly nonlinear fractional differential equations, uniformly valid for large/small parameters, appear these restrictive assumptions in traditional perturbation techniques.

The power of homotopy perturbation Sumudu transform method (HPSTM) [20, 30, 31] is well coupling of Sumudu transform algorithm and homotopy perturbation technique by using He's polynomials. The Sumudu transform was firstly proposed and applied by Watugala [36]. The pioneering work in connection with the development of important and basic results of the Sumudu transform were conducted by many authors notably Belgacem et al. [6], Belgacem and Al-Shemas [5] and Khalaf and Belgacem [16]. Homotopy perturbation method (HPM) is propounded by Ji-Huan He and used it in varies fields of research [12–14]. On the other hand homotopy analysis transform method (HATM) [11, 17, 19, 27, 33] demonstrate that how the Laplace transform can be employed to obtain the solutions of time depended fractional dynamical model for HIV infection of lymphocyte cells by manipulating the homotopy analysis method (HAM). The HAM was offered by Liao [21] by making use of the theory of the homotopy [15] an important part of topology [29]. A book entitled "Beyond Perturbation: Introduce to the Homotopy Analysis Method" [22] which demonstrates the basic plan of the HAM and its relationship with other analytic schemes and some of its uses in scientific fields [23, 24, 37]. Unlike HAM, HATM is not required the assumption of auxiliary linear operator, uses the differentiation property of Laplace transform for Caputo fractional derivative [7, 18].

2. Basic idea of HPSTM

We demonstrate the basic plan of HPSTM, by using a time-fractional nonlinear differential equation possessing the initial condition of the form

$$D_t^\alpha y(x, t) = R y(x, t) + N y(x, t) + g(x, t), \quad n-1 < \alpha \leq n, \quad \alpha > 0, \quad (2.1)$$

subject to the initial condition $D_0^m y(x, 0) = f_m(x)$, where $m = 0, \dots, n-1$, $D_0^n y(x, 0) = 0$, and $n = [\alpha]$, where is $D_t^\alpha y(x, t)$ the Caputo fractional derivative of the function $y(x, t)$, R is the linear differential operator, N represents the general nonlinear differential and $g(x, t)$ designates the source term.

On using the Sumudu transform operator on Eq. (2.1), it yields

$$S[y] = u^\alpha \sum_{k=0}^{n-1} u^{-\alpha+k} y^{(k)}(x, 0) + u^\alpha S[R y] + u^\alpha S[N y] + u^\alpha S[g(x, t)]. \quad (2.2)$$

Next, on applying the inversion of Sumudu transform operator on Eq. (2.2), it gives

$$y = F(x, t) + S^{-1} [u^\alpha S[R y] + u^\alpha S[N y]], \quad (2.3)$$

where $F(x, t)$ indicates the term occurring from the known function $g(x, t)$ and the initial conditions.

Now we use the HPM

$$y(x, t) = \sum_{n=0}^{\infty} P^n y_n(x, t). \quad (2.4)$$

We deform the nonlinear term as

$$N y(x, t) = \sum_{n=0}^{\infty} P^n H_n(y). \quad (2.5)$$

Using the He’s polynomials $H_n(y)$ [26] given as:

$$H_n(y_0, \dots, y_n) = \frac{1}{\Gamma(n+1)} \frac{\partial^n}{\partial P^n} \left[N \left(\sum_{r=0}^{\infty} P^r y_r(x, t) \right) \right], n = 0, 1, 2, \dots$$

Substituting (2.4) and (2.5) in (2.3), we have

$$\sum_{n=0}^{\infty} P^n y_n = F(x, t) + P \left[S^{-1} \left[u^\alpha S \left[R \left(\sum_{n=0}^{\infty} P^n y_n \right) \right] + u^\alpha S \left[N \left(\sum_{n=0}^{\infty} P^n H_n \right) \right] \right] \right].$$

This is the mixture of the Sumudu transform and the HPM with the aid of He’s polynomials. Comparing the coefficients of like power of P, it yields

$$\begin{aligned} P^0 : y_0 &= F(x, t), \\ P^1 : y_1 &= S^{-1} [u^\alpha S [R(y_0) + H_0(y)]], \\ P^2 : y_2 &= S^{-1} [u^\alpha S [R(y_1) + H_1(y)]], \\ &\vdots \\ P^n : y_n &= S^{-1} [u^\alpha S [R(y_{n-1}) + H_{n-1}(y)]], \\ &\vdots \end{aligned}$$

The approximate the analytical solution $y(x, t)$ of Eq. (2.1) is presented as

$$y(x, t) = y_0(x, t) + \sum_{k=1}^{\infty} y_k(x, t).$$

3. Basic idea of HATM

To demonstrate the basic plan of this algorithm, we take a general fractional nonlinear non-homogeneous partial differential equation expressed as

$$D_t^\alpha u(x, t) + Ru(x, t) + Nu(x, t) = g(x, t), \quad n - 1 < \alpha \leq n, \tag{3.1}$$

where $D_t^\alpha u(x, t)$ indicates the Caputo fractional derivative of the function $u(x, t)$, R stands for the linear differential operator, N denotes the general nonlinear differential operator and $g(x, t)$ is the source term.

By applying the Laplace transform on both sides of equation (3.1), we get

$$s^\alpha L[u] - \sum_{k=0}^{n-1} s^{\alpha-k-1} u^{(k)}(x, 0) + L[Ru] + L[Nu] = L[g(x, t)].$$

On simplifying

$$L[u] - \frac{1}{s^\alpha} \sum_{k=0}^{n-1} s^{\alpha-k-1} u^{(k)}(x, 0) + \frac{1}{s^\alpha} [L[Ru] + L[Nu] - L[g(x, t)]] = 0.$$

We define the nonlinear operator

$$\begin{aligned} N[\phi(x, t; q)] &= L[\phi(x, t; q)] - \frac{1}{s^\alpha} \sum_{k=0}^{n-1} s^{\alpha-k-1} \phi^{(k)}(x, t; q)(0^+) \\ &\quad + \frac{1}{s^\alpha} [L[R\phi(x, t; q)] + L[N\phi(x, t; q)] - L[g(x, t)]], \end{aligned}$$

where $q \in [0, 1]$ and $\phi(x, t; q)$ is a real function of x, t and q . Now using the same procedure as used in a series of papers [11, 17, 19, 27, 33], we get the following m th-order deformation equation:

$$L [u_m(x, t) - \chi_m u_{m-1}(x, t)] = \hbar \mathfrak{R}_m(\vec{u}_{m-1}).$$

Applying the inverse Laplace transform, we have

$$u_m(x, t) = \chi_m u_{m-1}(x, t) + \hbar L^{-1}[\mathfrak{R}_m(\vec{u}_{m-1})],$$

where

$$\mathfrak{R}_m(\vec{u}_{m-1}) = \frac{1}{(m-1)!} \frac{\partial^{m-1} N[\phi(x, t; q)]}{\partial q^{m-1}} \Big|_{q=0},$$

and

$$\chi_m = \begin{cases} 0, & m \leq 1, \\ 1, & m > 1. \end{cases}$$

4. Fractional dynamical model for HIV infection of CD4⁺T lymphocyte cells

We examine the dynamic model for HIV infection of CD4⁺T lymphocyte cells in fractional form. This mathematical model is characterized by a system of the linear and nonlinear differential equations of fractional order as follows

$$\begin{cases} \frac{d^\mu T}{dt^\mu} = \delta - \alpha T + rT(1 - \frac{T+I}{T_{\max}}) - kVT, \\ \frac{d^\eta I}{dt^\eta} = kVT - \beta I, \\ \frac{d^\omega V}{dt^\omega} = N\beta I - \gamma V, \end{cases} \quad 0 < \mu, \eta, \omega \leq 1, \tag{4.1}$$

having the initial conditions

$$T(0) = T_0, \quad I(0) = I_0, \quad V(0) = V_0. \tag{4.2}$$

This model is expressed the basic three components $T(t)$, $I(t)$ and $V(t)$ which denote concentration of susceptible CD4⁺T cells, CD4⁺T cells infected by the HIV viruses and virus population of CD4⁺T cells by HIV in the blood, respectively. $rT(1 - \frac{T+I}{T_{\max}})$ is logistic growth of the healthy CD4⁺T cells, and proliferation of infected CD4⁺T cells is neglected. r is the rate at which T cells multiply through mitosis when T cells are stimulated by antigen or mitogen. T_{\max} is the maximum level of CD4⁺T cells in the human body [35]. Each infected CD4⁺T cell is assumed to produce 1 virus particles during its lifetime, including any of its daughter cells. The body is believed to produce CD4⁺T cells from precursors in the bone marrow and thymus at a constant rate δ . The term kVT describes the incidence of HIV infection of healthy CD4⁺T cells, where $k > 0$ is the infection of T -cells. $N\beta$ is the rate of production of virions by infected cells, where N is the average number of virus particles produced by an infected T -cell and γ is the death rate of virus particles [35]. In a normal human body, the level of CD4⁺T cells is 800/1200mm³. CD4⁺T cells are also named as T helper cells or leukocytes. These CD4⁺T cells with order cells in human immunity systems fight against disease. We set the following initial conditions and parameters for numerical approximate solutions [2]

$$T(0) = 0.1, \quad I(0) = 0, \quad V(0) = 0.1, \quad \alpha = 0.02, \quad \beta = 0.3, \\ \gamma = 2.4, \quad \delta = 0.1, \quad k = 0.0027, \quad r = 3, \quad T_{\max} = 1,500, \quad N = 10.$$

5. Implementation of HPSTM

We apply the HPSTM for solving system of time-fractional differential equations arising in model for HIV infection of CD4⁺T lymphocyte cells (4.1), and compared with well existing results.

Operating the Sumudu transform on Eq. (4.1), we get

$$\begin{cases} S\left(\frac{dT}{dt^\mu}\right) = S\left(\delta - \alpha T + rT\left(1 - \frac{T+I}{T_{\max}}\right) - kVT\right), \\ S\left(\frac{d^n I}{dt^\eta}\right) = S(kVT - \beta I), \\ S\left(\frac{d^\omega V}{dt^\omega}\right) = S(N\beta I - \gamma V), \end{cases}$$

$$\begin{cases} S(T) = 0.1 + u^\mu S\left(\delta - \alpha T + rT\left(1 - \frac{T+I}{T_{\max}}\right) - kVT\right), \\ S(I) = u^\eta S(kVT - \beta I), \\ S(V) = 0.1 + u^\omega S(N\beta I - \gamma V). \end{cases} \tag{5.1}$$

Operating with the inverse of Sumudu transform on Eq. (5.1), we have

$$\begin{cases} T = 0.1 + S^{-1}\left(u^\mu S\left(\delta - \alpha T + rT\left(1 - \frac{T+I}{T_{\max}}\right) - kVT\right)\right), \\ I = S^{-1}\left(u^\eta S(kVT - \beta I)\right), \\ V = 0.1 + S^{-1}\left(u^\omega S(N\beta I - \gamma V)\right). \end{cases} \tag{5.2}$$

The nonlinear terms noticed as, $A = T^2, B = T \cdot I, C = V \cdot T$ at right side of system (5.2) will be represented by an infinite series of He’s polynomials

$$\begin{cases} F_m(T_0, \dots, T_m) = \frac{1}{\Gamma(m+1)} \frac{\partial^m}{\partial P^m} [A(\sum_{r=0}^\infty P^r T_r(t))], m = 0, 1, 2, \dots \\ G_m(T_0, \dots, T_m, I_0, \dots, I_m) = \frac{1}{\Gamma(m+1)} \frac{\partial^m}{\partial P^m} [B(\sum_{r=0}^\infty P^r T_r(t) I_r(t))], m = 0, 1, \dots \\ H_m(V_0, \dots, V_m, T_0, \dots, T_m) = \frac{1}{\Gamma(m+1)} \frac{\partial^m}{\partial P^m} [C(\sum_{r=0}^\infty P^r V_r(t) T_r(t))], m = 0, 1, \dots \end{cases}$$

Now using the HPM, we get

$$\begin{cases} \sum_{m=0}^\infty P^m T_m = 0.1 + PS^{-1}\left(u^\mu \left(S\left(\delta + (-\alpha + r) \sum_{m=0}^\infty P^m T_m - \frac{r}{T_{\max}} \left(\sum_{m=0}^\infty P^m F_m + \sum_{m=0}^\infty P^m G_m\right) - k \sum_{m=0}^\infty P^m H_m\right)\right)\right), \\ \sum_{m=0}^\infty P^m I_m = PS^{-1}\left(u^\eta \left(S(k \sum_{m=0}^\infty P^m H_m - \beta \sum_{m=0}^\infty P^m I_m)\right)\right), \\ \sum_{m=0}^\infty P^m V_m = 0.1 + PS^{-1}\left(u^\omega \left(S(N\beta \sum_{m=0}^\infty P^m I_m - \gamma \sum_{m=0}^\infty P^m V_m)\right)\right). \end{cases}$$

Equating the coefficient of like powers of P, we have

$$\begin{cases} P^0 : T_0 = 0.1, & P^0 : I_0 = 0, & P^0 : V_0 = 0.1, \\ P^1 : T_1 = S^{-1}\left(u^\mu \left(S\left(\delta + (-\alpha + r)T_0 - \frac{r}{T_{\max}}(F_0 + G_0) - kH_0\right)\right)\right), \\ & = S^{-1}\left(u^\mu \left(S\left(\delta + (-\alpha + r)T_0 - \frac{r}{T_{\max}}(T_0^2 + T_0 I_0) - kV_0 T_0\right)\right)\right), \\ & = 0.397953 \frac{t^\mu}{\Gamma(\mu+1)}, \\ P^1 : I_1 = S^{-1}\left(u^\eta \left(S(kH_0 - \beta I_0)\right)\right), \\ & = S^{-1}\left(u^\eta \left(S(kV_0 T_0 - \beta I_0)\right)\right) = 0.000027 \frac{t^\eta}{\Gamma(\eta+1)}, \\ P^1 : V_1 = S^{-1}\left(u^\omega \left(S(N\beta I_0 - \gamma V_0)\right)\right) = -0.24 \frac{t^\omega}{\Gamma(\omega+1)}, \\ P^2 : T_2 = S^{-1}\left(u^\mu \left(S\left((- \alpha + r)T_1 - \frac{r}{T_{\max}}(F_1 + G_1) - kH_1\right)\right)\right), \\ & = S^{-1}\left(u^\mu \left(S\left((- \alpha + r)T_1 - \frac{r}{T_{\max}}(2T_0 T_1 + T_0 I_1 + T_1 I_0) - k(V_0 T_1 + V_1 T_0)\right)\right)\right), \\ & = -5.4 \times (10)^{-9} \frac{t^{\mu+\eta}}{\Gamma(\mu+\eta+1)} + 6.4 \times (10)^{-5} \frac{t^{\mu+\omega}}{\Gamma(\mu+\omega+1)}, \\ P^2 : I_2 = S^{-1}\left(u^\eta \left(S(k(V_0 T_1 + V_1 T_0) - \beta I_1)\right)\right), \\ & = S^{-1}\left(u^\eta \left(S(kV_0 T_0 - \beta I_0)\right)\right) \\ & = -8.1 \times (10)^{-6} \frac{t^{2\eta}}{\Gamma(2\eta+1)} + 0.000107447 \frac{t^{\mu+\eta}}{\Gamma(\mu+\eta+1)} - 0.0000648 \frac{t^{\eta+\omega}}{\Gamma(\eta+\omega+1)}, \\ P^2 : V_2 = S^{-1}\left(u^\omega \left(S(N\beta I_1 - \gamma V_1)\right)\right) = 8.1 \times (10)^{-5} \frac{t^{\eta+\omega}}{\Gamma(\eta+\omega+1)} + 0.567 \frac{t^{2\omega}}{\Gamma(2\omega+1)}, \\ \vdots \end{cases}$$

and so on, in this way the remaining components can be obtained and the solution of the system of Eq. (4.1) is presented by infinite series as

$$\begin{cases} T(t) = \sum_{l=0}^{\infty} T_l(t) = T_0 + T_1 + T_2 + T_3 + \dots, \\ I(t) = \sum_{l=0}^{\infty} I_l(t) = I_0 + I_1 + I_2 + I_3 + \dots, \\ V(t) = \sum_{l=0}^{\infty} V_l(t) = V_0 + V_1 + V_2 + V_3 + \dots. \end{cases}$$

It's clear to see that the HPSTM solution is same as obtained by HDM [2].

6. Implementation of the HATM

In this section, we apply HATM in a realistic and efficient way to handle nonlinear fractional model for HIV infection of lymphocyte cells (4.1) leads three coupled equations, found more general solution with large convergence domain, compared to LADM [8], DTM [34], BCM [38], HDM [2], are a particular case of the HATM solution series when $\hbar = -1$.

System (4.1) and Eq. (4.2) suggest that we express the nonlinear operators as

$$\begin{cases} N^1[\psi_1(t; q), \psi_2(t; q), \psi_3(t; q)] \\ \quad = L[\psi_1(t; q)] - (1 - \chi_m) \frac{1}{s} T_0 - \frac{1}{s^\mu} L[\delta - \alpha\psi_1(t; q) + r\psi_1(t; q) \\ \quad \quad - \frac{r}{T_{\max}}(\psi_1(t; q) - \psi_1^2(t; q) - \psi_2(t; q)\psi_1(t; q)) - k\psi_3(t; q)\psi_1(t; q)], \\ N^2[\psi_1(t; q), \psi_2(t; q), \psi_3(t; q)] \\ \quad = L[\psi_2(t; q)] - (1 - \chi_m) \frac{1}{s} I_0 - \frac{1}{s^\eta} L[k\psi_3(t; q)\psi_1(t; q) - \beta\psi_2(t; q)], \\ N^3[\psi_1(t; q), \psi_2(t; q), \psi_3(t; q)] \\ \quad = L[\psi_3(t; q)] - (1 - \chi_m) \frac{1}{s} V_0 - \frac{1}{s^\omega} L[N\beta\psi_2(t; q) - \gamma\psi_3(t; q)], \end{cases}$$

and the Laplace operator as

$$\begin{cases} L[T_m - \chi_m T_{m-1}] = \hbar R_{1,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}], \\ L[I_m - \chi_m I_{m-1}] = \hbar R_{2,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}], \\ L[V_m - \chi_m V_{m-1}] = \hbar R_{3,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}], \end{cases} \tag{6.1}$$

where

$$\begin{cases} R_{1,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}] = L\{T_{m-1}\} - (1 - \chi_m) \frac{1}{s} T_0 - \frac{1}{s^\mu} L\{(1 - \chi_m) \delta - \alpha T_{m-1} + r T_{m-1} \\ \quad - \frac{r}{T_{\max}}(\sum_{i=0}^{m-1} T_i T_{m-1-i} + \sum_{i=0}^{m-1} I_i T_{m-1-i}) - k \sum_{i=0}^{m-1} V_i T_{m-1-i}\}, \\ R_{2,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}] = L\{I_{m-1}\} - (1 - \chi_m) \frac{1}{s} I_0 - \frac{1}{s^\eta} L\{k \sum_{i=0}^{m-1} V_i T_{m-1-i} - \beta I_{m-1}\}. \\ R_{3,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}] = L\{V_{m-1}\} - (1 - \chi_m) \frac{1}{s} V_0 - \frac{1}{s^\omega} L\{N\beta I_{m-1} - \gamma V_{m-1}\}. \end{cases} \tag{6.2}$$

Applying the inverse Laplace transforms in (6.1) and using (6.2), we have

$$\begin{cases} T_m = \chi_m T_{m-1} + \hbar L^{-1} \left\{ R_{1,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}] \right\}, \\ I_m = \chi_m I_{m-1} + \hbar L^{-1} \left\{ R_{2,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}] \right\}, \\ V_m = \chi_m V_{m-1} + \hbar L^{-1} \left\{ R_{3,m}[\vec{T}_{m-1}, \vec{I}_{m-1}, \vec{V}_{m-1}] \right\}. \end{cases} \tag{6.3}$$

We solve the system of (6.3) for $m = 1, 2, 3, \dots$, and using initial conditions and parameters, we get the following results

$$\left\{ \begin{array}{l} T_0(t) = T_0 = 0.1, \quad I_0(t) = I_0 = 0, \quad V_0(t) = V_0 = 0.1, \\ T_1(t) = -0.397953\hbar \frac{t^\mu}{\Gamma(\mu+1)}, \quad I_1(t) = -0.000027\hbar \frac{t^\eta}{\Gamma(\eta+1)}, \quad V_1(t) = 0.24\hbar \frac{t^\omega}{\Gamma(\omega+1)} \\ T_2(t) = -0.397953\hbar(\hbar+1) \frac{t^\mu}{\Gamma(\mu+1)} + 1.18563\hbar^2 \frac{t^{2\mu}}{\Gamma(2\mu+1)} - 5.4 \times (10)^{-9}\hbar^2 \frac{t^{\mu+\eta}}{\Gamma(\mu+\eta+1)} \\ \quad + 6.4 \times (10)^{-5}\hbar^2 \frac{t^{\mu+\omega}}{\Gamma(\mu+\omega+1)}, \\ I_2(t) = -0.000027\hbar(\hbar+1) \frac{t^\eta}{\Gamma(\eta+1)} - 8.1 \times (10)^{-6}\hbar^2 \frac{t^{2\eta}}{\Gamma(2\eta+1)} \\ \quad + 0.000107447\hbar^2 \frac{t^{\mu+\eta}}{\Gamma(\mu+\eta+1)} - 0.0000648\hbar^2 \frac{t^{\eta+\omega}}{\Gamma(\eta+\omega+1)}, \\ V_2(t) = 0.24\hbar(\hbar+1) \frac{t^\omega}{\Gamma(\omega+1)} + 8.1 \times (10)^{-5}\hbar^2 \frac{t^{\eta+\omega}}{\Gamma(\eta+\omega+1)} + 0.567\hbar^2 \frac{t^{2\omega}}{\Gamma(2\omega+1)}, \\ \vdots \end{array} \right.$$

and so on, in this way the remaining components can be computed by using the software Maple. The approximate series solutions can be obtained by

$$\left\{ \begin{array}{l} T(t) = \sum_{l=0}^{\infty} T_l(t) = T_0 + T_1 + T_2 + T_3 + \dots, \\ I(t) = \sum_{l=0}^{\infty} I_l(t) = I_0 + I_1 + I_2 + I_3 + \dots, \\ V(t) = \sum_{l=0}^{\infty} V_l(t) = V_0 + V_1 + V_2 + V_3 + \dots \end{array} \right. \quad (6.4)$$

It’s clear to see that when we set $\hbar = -1$ in HATM series solution, it converge to HPSTM and HDM [2] series solution. Figures 1-3 represents the rate of change of three variables $T(t)$, $I(t)$ and $V(t)$ for standard motion $\mu = \eta = \omega = 1$ and Brownian motions $\mu = \eta = \omega = 0.95, 0.85, 0.75$ of 3rd order HPSTM and HATM solution series. Figures 4-6 display the \hbar –curves. It’s obvious that the middle point of \hbar –curves interval i.e., $\hbar = -1$ is a proper choice, at this point the numerical solution converges to series solution for standard motion as well as Brownian motions. The horizontal line segment indicates the valid region of \hbar which insures the convergence of the series solution.

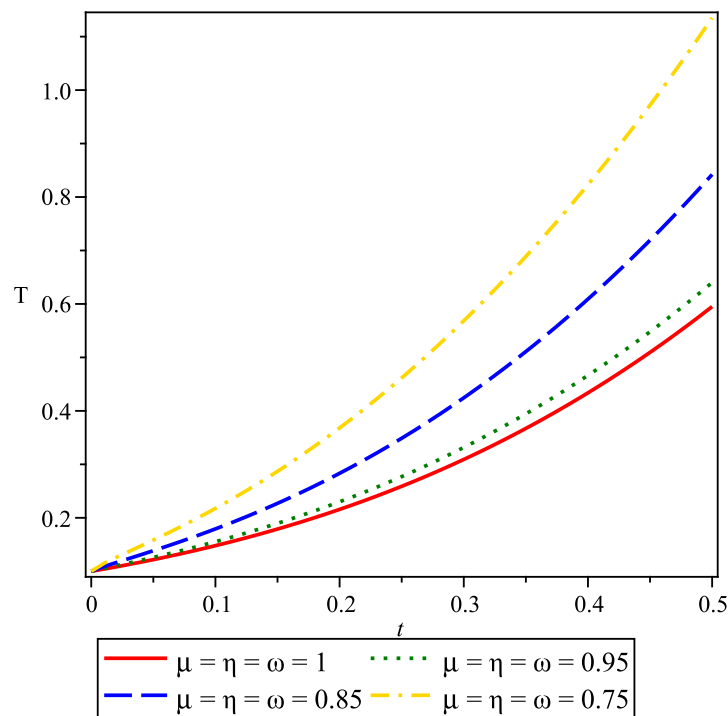


Figure 1: Plot of $T(t)$ vs. time t for standard motion $\mu = \eta = \omega = 1$ and Brownian motions $\mu = \eta = \omega = 0.95, 0.85, 0.75$ of 3rd order HPSTM and HATM solution series.

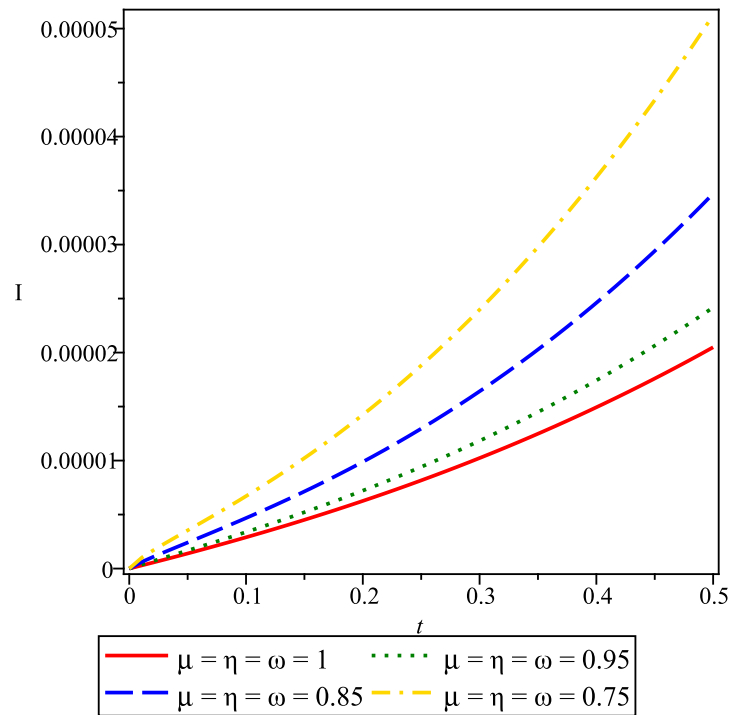


Figure 2: Plot of $I(t)$ vs. time t for standard motion $\mu = \eta = \omega = 1$ and Brownian motions $\mu = \eta = \omega = 0.95, 0.85, 0.75$ of 3rd order HPSTM and HATM solution series.

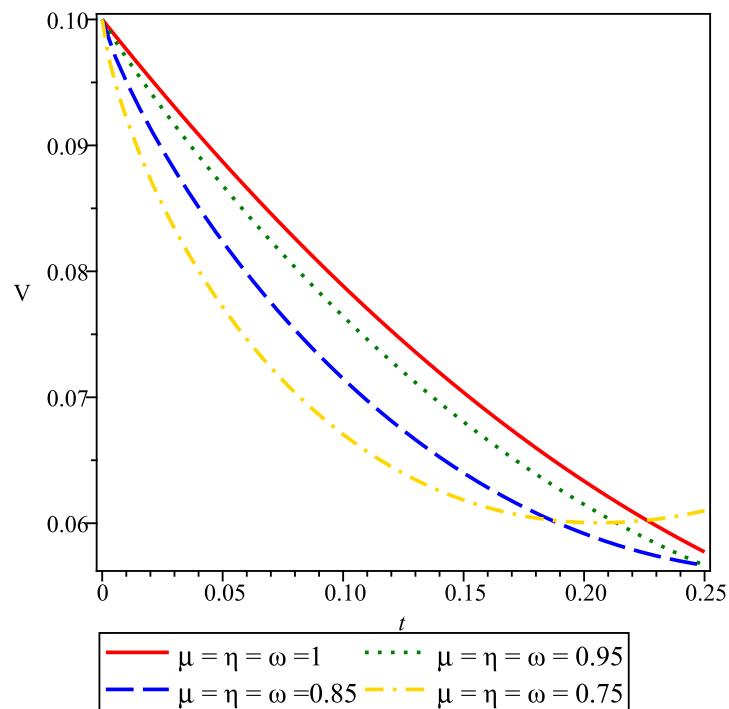


Figure 3: Plot of $V(t)$ vs. time t for standard motion $\mu = \eta = \omega = 1$ and Brownian motions $\mu = \eta = \omega = 0.95, 0.85, 0.75$ of 3rd order HPSTM and HATM solution series.

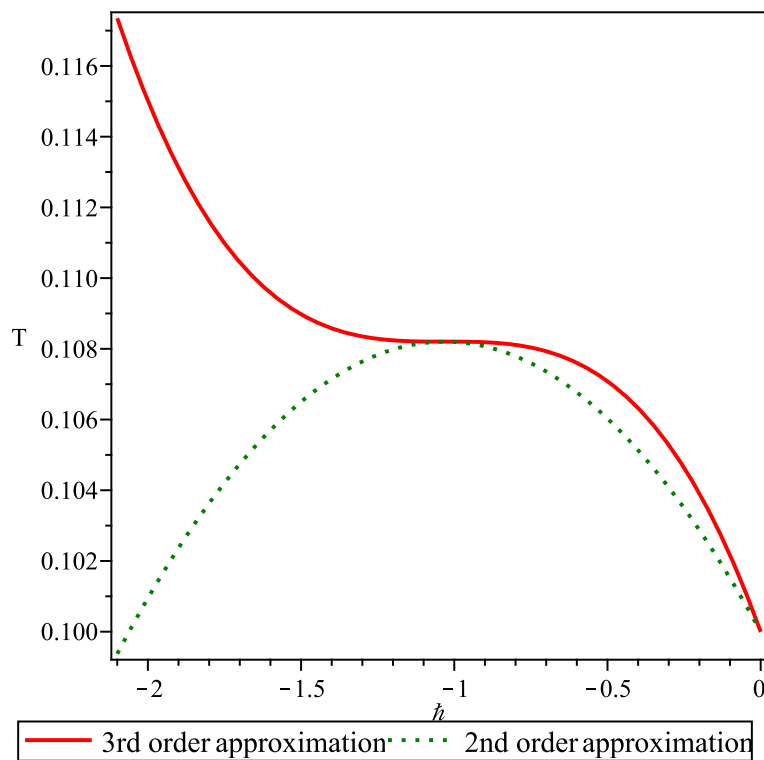


Figure 4: h -curve for $T(t)$ when $\mu = \eta = \omega = 1$.

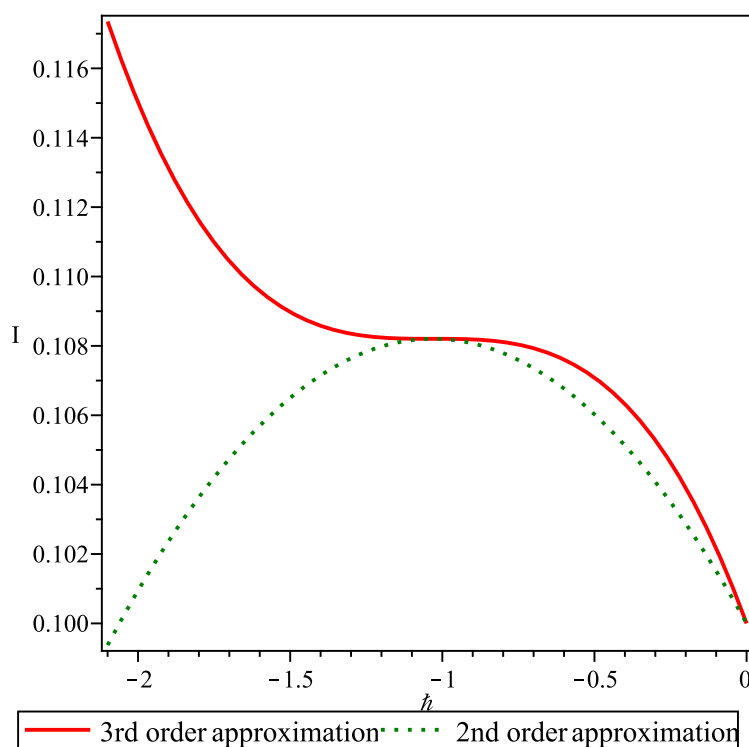


Figure 5: h -curve for $I(t)$ when $\mu = \eta = \omega = 1$.

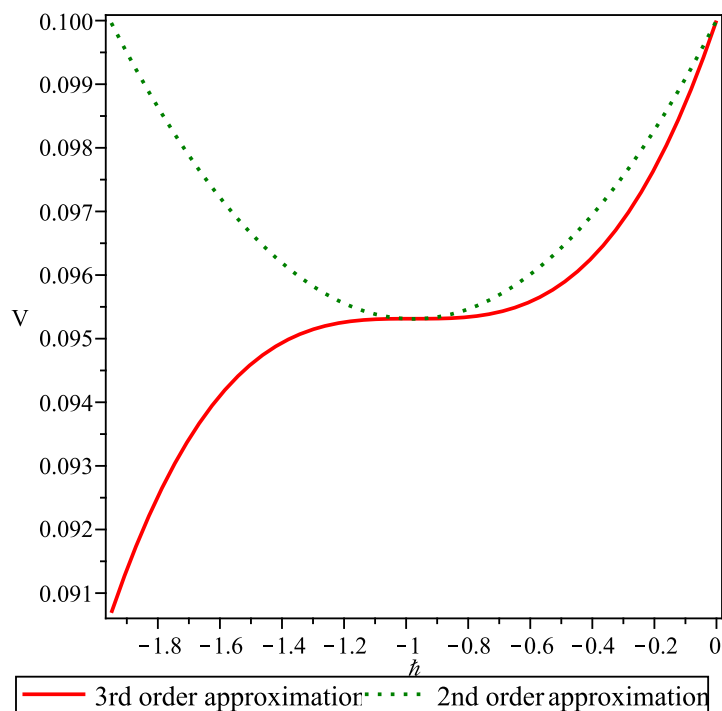


Figure 6: h – curve for $V(t)$ when $\mu = \eta = \omega = 1$.

7. Conclusions

In this paper, two powerful coupled methods, HPSTM and HATM are successfully applied to derive the solution of the fractional model for HIV infection of $CD4^+T$ lymphocyte cells, as compared with existing results. It can be concluded that the proposed techniques are more convenient, straight forward and power full than the standard methods STM, HPM and LTM, HAM. The HATM is capable to provide rapid and considerably large convergence region by choosing the appropriate values of convergence-control parameter h . By selecting $h = -1$ the solution series converge to the solution series of HPSTM.

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