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Solutions of Fractional model of human T-cell lymphotropic virus I (HTLV-I) infection of $CD4^+$ T-cells using HAM

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Abstract

In this paper, we introduce fractional-order model of (HTLV-I) infection of $CD4^+$ T-cells. Homotopy analysis method (HAM) is implemented to give approximate and analytical solutions of the presented problem. The fractional derivatives are described in the Caputo sense. The method introduces a promising tool for solving many linear and nonlinear fractional differential equations. In these schemes, the solution takes the form of a convergent series with easily computable components. Numerical results show that the approach is easy to implement and accurate when applied to ordinary differential equations of fractional order.

Keywords: *Homotopy analysis method, Fractional order ordinary differential equations, Models of infectious diseases.*

1 Introduction

Human T-cell lymphotropic virus type I (HTLV-I) infection is associated with a variety of human diseases. Human T-cell lymphotropic virus (HTLV) is a member of the exogeneous human retroviruses that have a tropism for T lymphocytes. HTLV-I belongs to the delta-type retroviruses, which also include bovine

leukemia virus; human T-cell leukemia virus type II (HTLV-II), and simian T-cell leukemia virus [15]. Infection with HTLV-I is now a global epidemic, affecting 10 million to 20 million people. This virus has been linked to life-threatening, incurable diseases:

a) Adult T-cell leukemia (ATL).
b) HTLV-I-associated myelopathy/tropical spastic paraparesis (HAM/TSP).

These syndromes are important causes of mortality and morbidity in the areas where HTLV-I is endemic, mainly in the tropics and subtropics [17]. There are large endemic areas in southern Japan, the Caribbean, Central and West Africa, the Middle East, Melanesia, and equatorial regions of Africa. In Europe and North America, the virus is found chiefly in immigrants from the endemic areas and in some communities of intravenous drug users. There is neither a vaccine against the virus, nor a satisfactory treatment for the malignancy or the in- flammatory syndromes HTLV-I is transmitted via three major routes:

(i) Transmission from mother to child by breast feeding.
(ii) Transmission from male to female (more frequent than from female to male) by sexual contact.
(iii) Transmission by infected blood, either by blood transfusion or by the contaminated needles among drug abusers.

Like HIV, HTLV-I targets $CD4^+$ T-cells, the most abundant white cells in the immune system, decreasing the body's ability to fight infection. Primary infection leads to chronic infection, the proviral load of which can be extremely high, approximately 30–50% [18]. Unlike in the case of HIV infection [4], however, only a small percentage of infected individuals develop the disease and 2–5% percent of HTLV-I carriers develop symptoms of ATL [15]. Also, there is very little cell-free virus in the plasma. Almost all viral genetic material resides, in DNA form, integrated within the host genome of infected cells. HTLV-I infection is achieved primarily through cell-to-cell contact [17]. The HTLV model is:

$$\begin{aligned}
\frac{dT}{dt} &= \lambda - \mu_T T - \kappa VT, \\
\frac{dI}{dt} &= \kappa_1 VT - (\mu_L + \alpha)I, \\
\frac{dV}{dt} &= \alpha I - (\mu_A + \rho)V, \\
\frac{dL}{dt} &= \rho V + \beta L\left(1 - \frac{L}{L_{max}}\right) - \mu_M L.
\end{aligned} \tag{1}$$

The activity of which produces a DNA copy of the viral genome that is integrated into the DNA of the host genome. After this takes place, the latency period can persist for a long period of time. Latently infected cells contain the virus, but do not produce DNA and are incapable of contagion. When such cells are stimulated by antigen, they can become active and infect healthy cells. Taking these factors

into consideration, Stilianakis and Seydel [17] proposed a model that formulates a system of nonlinear differential equations that divides $CD4^+$ T-cells into four compartments: uninfected $CD4^+$ T-cells, latently infected cells, actively infected cells, and leukemia cells. Patricia Katri et al modified the classic model [15] for the system of non-linear differential equations to distinguish, in terms of parameters, between contact and infectivity rates. The resulting ODE model is (1).

2 Fractional Calculus

Fractional calculus (FC) has been extensively applied in many fields [1]. Many mathematicians and applied researchers have tried to model real processes using the fractional calculus. Petrovic et al developed a fractional-order mathematical model of a human root dentin [16]. In biology, it has been deduced that the membranes of cells of biological organism have fractional-order electrical conductance [3] and then are classified in groups of non-integer order models. Fractional derivatives embody essential features of the behavior of the pattern formation in bacterial colonies [5]. Also, it has been shown that modelling the behavior of brainstem vestibule-oculumotor neurons by fractional ordinary differential equations (FODE) has more advantages than classical integer-order modelling [2]. FODE are naturally related to systems with memory which exists in most biological systems. Also, they are closely related to fractals, which are abundant in biological systems [1,9]. However, the convergence region of the corresponding results is rather small, as shown later in this paper. Recently, Liao [10,11,12], proposed a powerful analytic method, namely the homotopy analysis method (HAM), for solving linear and nonlinear differential and integral equations. Different from perturbation techniques, the homotopy analysis method does not depend upon any small or large parameters. Besides, it logically contains other non-perturbation techniques, such as Adomian decomposition method, homotopy perturbation method.

3 Homotopy Analysis Method (HAM)

Several methods have been suggested to solve fractional differential equations. These methods include the homotopy perturbation method, Adomian decomposition method and variation iteration method. However, the convergence region of the corresponding results is rather small, as shown later in this paper. Recently, Liao [12], proposed a powerful analytic method, namely the homotopy analysis method (HAM), for solving linear and nonlinear differential and integral equations. Different from perturbation techniques, the homotopy analysis method does not depend upon any small or large parameters. Besides, it logically contains other non-perturbation techniques, such as Adomian's decomposition method, homotopy perturbation method. In this paper, (HAM) is applied to solve nonlinear

fractional initial-value problem of the non-fatal epidemic model to obtain symbolic approximate solutions for linear and nonlinear differential equations of fractional order [8]. (HAM) is different from all analytical methods; it provides us with a simple way to adjust and control the convergence region of the series solution by introducing the auxiliary parameter h, and the auxiliary function H(t) [6,7]. In fact, it is the auxiliary parameter h that provides us, for the first time, a simple way to ensure the convergence of the series solution. Due to this reason, it seems reasonable to rename h the convergence-control parameter. It should be emphasized that, without the use of the convergence-parameter h, one had to assume that the homotopy series is convergent. However, with the use of the convergence-parameter h, such an assumption is unnecessary; because it seems that one can always choose a proper value of h to obtain convergent homotopy-series solution. So, the use of the convergence-parameter h in the zeroth-order deformation equation greatly modifies the early homotopy analysis method. Since then, the homotopy analysis method has been developing greatly, and more generalized zeroth-order deformation equations are suggested by Liao [10]. Besides, the so-called ‘‘homotopy perturbation method” (proposed in 1998) is exactly the same as the early homotopy analysis method (proposed in 1992) and is a special case of the late homotopy analysis method in case of $h = -1$.

Consider the following system of (FDE):

$$D^{\alpha_i}\left(u_i(t)\right) = f_i(t, u_1, \dots, u_n), \quad i = 1,2,3, \dots, n, \quad 0 \le \alpha_i \le 1 \tag{2}$$

Subject to the initial conditions:

$$u_i(0) = a_i, \quad i = 1,2, \dots, n \tag{3}$$

Liao [10] constructed the so-called zeroth-order deformation equation:

$$(1 - q)\mathcal{L}_i[\phi_i(t, q) - u_{i0}(t)] = q h_i H_i(t) N_i[\phi_i(t, q)], \quad i = 1,2,3, \dots . n, \tag{4}$$

subject to the initial conditions:

$$\phi_i(0, q) = a_i \tag{5}$$

where $q \in [0,1]$ is an embedding parameter, N_i are nonlinear operators, $\mathcal{L}_i$ are auxiliary linear operators satisfy $\mathcal{L}_i(0) = 0$, $u_{i0}(t)$ are initial guesses satisfy the initial conditions (3), $h_i \neq 0$ are auxiliary parameters, $H_i(t) \neq 0$ are auxiliary functions, $\phi_i(t, q)$are unknown functions. It should be emphasized that one has great freedom to choose, the auxiliary linear operators $\mathcal{L}_i$, the auxiliary parameters h_i and the auxiliary functions H_i. Obviously, when $q \neq 0$, since $u_{i0}(t)$ satisfy the initial conditions (3) and $\mathcal{L}_i(0) = 0$, we have

$$\phi_i(t, 0) = u_{i0}(t), \quad i = 1,2,3, \dots, n, \tag{6}$$

when q = 1, since $h_i \neq 0$ and $H_i(t) \neq 0$, the zeroth-order deformation equation (4) and (5) are equivalent to (2) and (3), hence

$$\phi_i(t,1) = u_{i0}(t), \quad i = 1,2,3,\dots,n. \tag{7}$$

Thus, as q increasing from 0 to 1, the solutions $\phi_i(t,q)$ various from $u_{i0}(t)$ to $u_i(t)$. Expanding $\phi_i(t,q)$ in Taylor series with respect to the embedding parameter q, one has

$$\phi_i(t,q) = u_{i0}(t) + \sum_{m=1}^{\infty} u_{im}(t)q^m \ , \ i = 1,2,3,\dots,n, \tag{8}$$

Where

$$u_{im}(t) = \frac{1}{m!}\frac{\partial^m \phi_i(t,q)}{\partial q^m}\Big|_{q=0} \ , i = 1,2,3,\dots,n. \tag{9}$$

Assume that the auxiliary parametersh$_i$, the auxiliary functions $H_i(t)$, the initial approximations $u_{i0}(t)$ and the auxiliary linear operators $\mathcal{L}_i$ are properly chosen so that the series (8) converges at q = 1. Then at q = 1, and by (7) the series (8) becomes

$$u_i(t) = u_{i0}(t) + \sum_{m=1}^{\infty} u_{im}(t) \ , \ i = 1,2,3,\dots,n. \tag{10}$$

and now define the vector

$$\vec{u}_i = \{u_{i0}, u_{i1,} u_{i2}, \dots, u_{ij}\}, \qquad i = 1,2,3,\dots j. \tag{11}$$

Differentiating equations (4) m times with respect to the embedding parameter q, then setting q = 0 and dividing them by m!, finally using (9), we have the so-called mth-order deformation equations:

$$\mathcal{L}_i[u_{im} - \mathcal{X}_m u_{i(m-1)}(t)] = h_i H_i(t) \Re_{im}\left(\vec{u}_{i(m-1)}(t)\right), \quad i = 1,2,\dots,n, \tag{12}$$

Subject to the conditions:

$$u_{im}(0) = 0, \quad i = 1,2,\dots,n, \ m = 1,2,3,\dots n, \tag{13}$$

Where

$$\Re_{im}\left(\vec{u}_{i(m-1)}(t)\right) = \frac{1}{(m-1)!}\frac{\partial^{m-1} N_i(\phi_i(t,q))}{\partial q^{m-1}}\Big|_{q=0} \tag{14}$$

and

$$\mathcal{X}_m = \begin{cases} 0, & m \leq 1 \\ 1, & m > 1 \end{cases} \tag{15}$$

If we choose the linear operator $\mathcal{L}_i = D^{\alpha_i}$ then according to (12), we have

$$J^{\alpha_i}D^{\alpha_i}\left[u_{im} - \mathcal{X}_m u_{i(m-1)}(t)\right] = h_i J^{\alpha_i}\left[H_i(t)\Re_{im}\left(\vec{u}_{i(m-1)}(t)\right)\right] \qquad (16)$$

Finally it seems that, as long as a nonlinear fractional order differential equation has at least one solution, then one can always construct a kind of zeroth-order deformation equation to get convergent homotopy-series solution as

$$u_{im} = \mathcal{X}_m u_{i(m-1)}(t) + h_i J^{\alpha_i}\left[H_i(t)\Re_{im}\left(\vec{u}_{i(m-1)}(t)\right)\right] \qquad (17)$$

and $u_i = u_{i0} + u_{i1} + u_{i2} + u_{i3+\cdots}$

4 Model Derivation

We first give the definition of fractional-order integration and fractional-order differentiation [13,14]. For the concept of fractional derivative, we will adopt Caputo's definition, which is a modification of the Riemann-Liouville definition and has the advantage of dealing properly with initial value problems.

Definition 4.1 *The fractional integral of order $\alpha > 0$ of a function $f: R^+ \to R$ is given by*

$$J^{\alpha}f(x) = \frac{1}{\Gamma(\alpha)}\int_0^x (x-t)^{\alpha-1}f(t)\,dt, \qquad \alpha > 0, x > 0,$$

$$J^0 f(x) = f(x).$$

Hence we have

$$J^{\alpha}t^{\gamma} = \frac{\Gamma(\gamma+1)}{\Gamma(\alpha+\gamma+1)}t^{\alpha+\gamma}, \qquad \alpha > 0, \gamma > -1, t > 0$$

Definition 4.2 *Riemann–Liouville and Caputo fractional derivatives of order αof a continuous function $f: R^+ \to R$ is given respectively by*

$$D_*^{\alpha}f(x) = D^m\left(J^{m-\alpha}f(x)\right),$$

$$D^{\alpha}f(x) = J^{m-\alpha}\left(D^m f(x)\right),$$

Where $m - 1 < \alpha \le m, m \in N$.

Now we introduce fractional-order into the model of HIV infection of $CD4^+$ T - cells. The new system is described by the following set of FODEs of order $\alpha_1, \alpha_1, \alpha_3, \alpha_3 > 0$:

$$
\begin{aligned}
&D^{\alpha_1}(T) = \lambda - \mu_T T - \kappa V T,\\
&D^{\alpha_2}(I) = \kappa_1 V T - (\mu_L + \alpha) I,\\
&D^{\alpha_3}(V) = \alpha I - (\mu_A + \rho) V,\\
&D^{\alpha_4}(L) = \rho V + \beta L\left(1 - \frac{L}{L_{max}}\right) - \mu_M L.
\end{aligned} \tag{18}
$$

With initial conditions: $T(0) = 1000, I(0) = 250, V(0) = 1.5, L(0) = 0$
All the parameters are assumed to be positive as in table (1) as follows

Table 1: Parameters values

Parameters	Values
μ_T	$0.6\ mm^3/day$
μ_L	$0.006\ mm^3/day$
μ_A	$0.05\ mm^3/day$
μ_M	$0.0005\ mm^3/day$
κ	$0.1\ mm^3/day$
κ_1	$0.1\ mm^3/day$
β	$0.0003\ mm^3/day$
α	$0.0004\ mm^3/day$
ρ	$0.00004\ mm^3/day$
L_{max}	$2200\ mm^3/day$
λ	$6\ mm^3/day$

In view of the homotopy analysis method presented above, if we select the auxiliary functions $H_1 = H_2 = H_3 = H_4 = 1$, we can construct the homotopy:

$$\Re_{1m}\left(\vec{T}_{(m-1)}(t)\right) = D^{\alpha_1}(T_{m-1}) - \lambda + \mu_T T + \kappa\left(\sum_{i=0}^{m-1} V_i T_{m-1-i}\right),$$

$$\Re_{2m}\left(\vec{I}_{(m-1)}(t)\right) = D^{\alpha_2}(I_{m-1}) - \kappa_1\left(\sum_{i=0}^{m-1} V_i T_{m-1-i}\right) + (\mu_L + \alpha) I_{m-1}$$

$$\Re_{3m}\left(\vec{V}_{(m-1)}(t)\right) = D^{\alpha_3}(V_{m-1}) - \alpha I_{m-1} + (\mu_A + \rho) V_{m-1},$$

$$\Re_{4m}\left(\vec{L}_{(m-1)}(t)\right) = D^{\alpha_4}(L_{m-1}) - \rho V_{m-1} - \beta L_{m-1} + \frac{\beta}{L_{max}}\left(\sum_{i=0}^{m-1} L_i L_{m-1-i}\right) + \mu_M L$$

Consequently, we have

$$T_m = \chi_m T_{m-1} + h_1 J^{\alpha_1}\left[\Re_{1m}\left(\vec{T}_{(m-1)}(t)\right)\right],$$

$$I_m = \chi_m I_{m-1} + h_1 J^{\alpha_2}\left[\Re_{2m}\left(\vec{I}_{(m-1)}(t)\right)\right],$$

$$V_m = \chi_m V_{m-1} + h_1 J^{\alpha_3}\left[\Re_{3m}\left(\vec{V}_{(m-1)}(t)\right)\right],$$

$$L_m = \chi_m V_{m-1} + h_1 J^{\alpha_4}\left[\Re_{3m}\left(\vec{L}_{(m-1)}(t)\right)\right].$$

5 Numerical Results and Discussion

One can choose the values of $h_1, h_2, h_3,$ and h_4 from the so called h-curves. Fig.1. represents some of the h-curves while Fig.2. represents the HAM 3rd order series solution of system (18) in case of $h_1 = h_2 = h_3 = h_4 = h$ and $\alpha_1 = \alpha_2 = \alpha_3 = \alpha$.

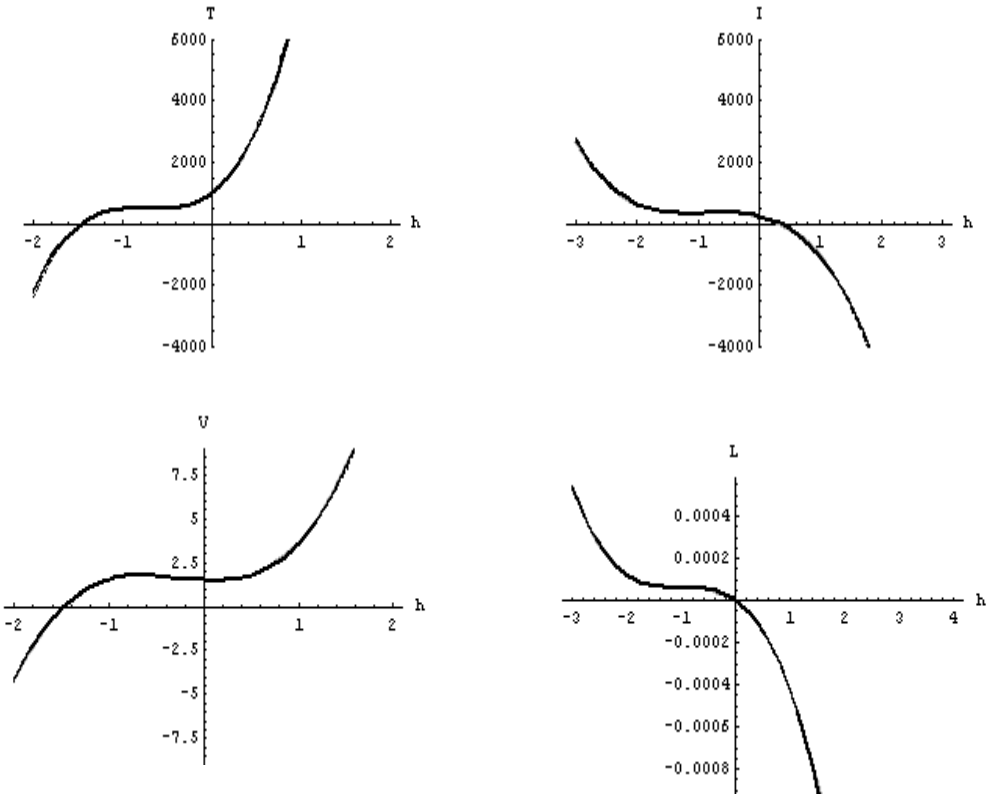

Figure 1: The h-curves of the system (18) at different values of α: Gray solid line (α = 1), Dashed line (α = 0.99), Dotted line (α = 0.95).

The first few components of the homotopy analysis solution $x(t)$, $y(t)$ and $z(t)$, are calculated. We computed the first three terms of the HAM series solution for the system (18). We present two of them as follows:

$$T_1 = \frac{t^{\alpha_1} h_1(-\lambda + T_0\mu_t + kT_0V_0)}{\Gamma[1+\alpha_1]}$$

$$I_1 = \frac{t^{\alpha_2} h_2(-k_1 T_0 V_0 + (\alpha + \mu_l) I_0)}{\Gamma[1+\alpha_2]}$$

$$V_1 = \frac{t^{\alpha_3} h_3((\rho + \mu_a) V_0 - \alpha I_0)}{\Gamma[1+\alpha_3]}$$

$$L_1 = \frac{t^{\alpha_4} h_4(-\rho V_0 - \beta L_0 + \mu_m L_0 + \frac{\beta L_0^2}{L_{max}})}{\Gamma[1+\alpha_4]}$$

$$T_2 = \frac{t^{\alpha_1} h_1(-\lambda + k T_0 V_0 + T_0 \mu_t)}{\Gamma[1+\alpha_1]} + \frac{t^{\alpha_1} {h_1}^2(-\lambda + k T_0 V_0 + T_0 \mu_t)}{\Gamma[1+\alpha_1]} +$$

$$+ \frac{t^{2\alpha_1} {h_1}^2 \mu_t(-\lambda + k T_0 V_0 + T_0 \mu_t)}{\Gamma[1+2\alpha_1]} + \frac{t^{\alpha_1+\alpha_3} h_1 k h_3 T_0\big(-\alpha i_0 + V_0(\rho + \mu_a)\big)}{\Gamma[1+\alpha_1+\alpha_3]} +$$

$$\frac{t^{2\alpha_1} k {h_1}^2 V_0(-\lambda + k T_0 V_0 + T_0 \mu_t)}{\Gamma[1+2\alpha_1]}$$

$$I_2 = \frac{t^{\alpha_2} h_2(-k_1 T_0 V_0 + I_0(\alpha + \mu_l))}{\Gamma[1+\alpha_2]} + h_2\Big(\frac{t^{\alpha_2} h_2\big(-k_1 T_0 V_0 + I_0(\alpha + \mu_l)\big)}{\Gamma[1+\alpha_2]} +$$

$$\frac{t^{2\alpha_2} h_2(\alpha + \mu_l)(-k_1 T_0 V_0 + I_0(\alpha + \mu_l))}{\Gamma[1+2\alpha_2]} - k_1\Big(\frac{t^{\alpha_2+\alpha_3} h_3 T_0\big(-\alpha I_0 + V_0(\rho + \mu_a)\big)}{\Gamma[1+\alpha_2+\alpha_3]}$$

$$+ \frac{t^{\alpha_1+\alpha_2} h_1 V_0(-\lambda + k T_0 V_0 + T_0 \mu_t)}{\Gamma[1+\alpha_1+\alpha_2]}\Big)\Big)$$

$$V_2 = \frac{t^{\alpha_3} h_3((\rho + \mu_a) V_0 - \alpha I_0)}{\Gamma[1+\alpha_3]} + h_3\Big(\frac{t^{\alpha_3} h_3\big((\rho + \mu_a) V_0 - \alpha I_0\big)}{\Gamma[1+\alpha_3]} +$$

$$\frac{t^{2\alpha_3} h_3(\rho + \mu_a)((\rho + \mu_a) V_0 - \alpha I_0)}{\Gamma[1+2\alpha_3]} - \frac{t^{\alpha_2+\alpha_3} \alpha h_2(-k_1 T_0 V_0 + (\alpha + \mu_l) I_0)}{\Gamma[1+\alpha_2+\alpha_3]}\Big)$$

$$L_2 = \frac{t^{\alpha_4} h_4(-\beta L_0 + \frac{\beta L_0^2}{L_{\max}} - \rho V_0 + L_0 \mu_m)}{\Gamma[1+\alpha_4]}$$

$$+ h_4\Big(-\frac{t^{\alpha_3+\alpha_4} \rho h_3\big(-\alpha i_0 + V_0(\rho + \mu_a)\big)}{\Gamma[1+\alpha_3+\alpha_4]} +$$

$$\frac{t^{\alpha_4}h_4(-\beta L_0+\frac{\beta L_0^2}{L_{\max}}-\rho V_0+L_0\mu_m)}{\Gamma[1+\alpha_4]}-\frac{t^{2\alpha_4}\beta h_4(-\beta L_0+\frac{\beta L_0^2}{L_{\max}}-\rho V_0+L_0\mu_m)}{\Gamma[1+2\alpha_4]}+$$

$$\frac{2t^{2\alpha_4}\beta h_4 L_0(-\beta L_0+\frac{\beta L_0^2}{L_{\max}}-\rho V_0+L_0\mu_m)}{\Gamma[1+2\alpha_4]L_{\max}}$$

$$+\frac{t^{2\alpha_4}h_4\mu_m(-\beta L_0+\frac{\beta L_0^2}{L_{\max}}-\rho V_0+L_0\mu_m)}{\Gamma[1+2\alpha_4]})$$

Thus, the HAM series solution of the given system (18) can be given by:

$T=T_0+T_1+T_2+T_3+\cdots$
$I=I_0+I_1+I_2+I_3+\cdots$
$V=V_0+V_1+V_2+V_3+\cdots$
$L=L_0+L_1+L_2+L_3+\cdots$

One can choose the values of $h_1, h_2, h_3, and\ h_4$from the so called *h*-curves. fig.1. represents some of the h-curves while fig.2. represents the HAM 3rd order series solution of system (18) in case of $h_1=h_2=h_3=h_4=h$ and $\alpha_1=\alpha_2=\alpha_3=\alpha_4=\alpha$.

The definition of fractional derivative involves an integration which is non local operator (as it is defined on an interval) so fractional derivative is a non local operator. In other word, calculating time-fractional derivative of a function $\mathbf{f(t)}$ at some time $\mathbf{t=t_1}$ requires all the previous history, i.e. all $\mathbf{f(t)}$ from $\mathbf{t=0\ to\ t=t_1}$. The reason of using fractional order differential equations (FOD) is that FOD are naturally related to systems with memory which exists in most biological systems. Also they are closely related to fractals which are abundant in biological systems. The results derived of the fractional system (18) are of a more general nature. We would like to put your attention that time fractional derivatives change also the solutions we usually get in standard system (1). The concept of fractional or non-integer order derivation and integration can be traced back to the genesis of integer order calculus itself. Most of the mathematical theory applicable to the study of non-integer order calculus was developed through the end of 19th century. However it is in the past hundred years that the most intriguing leaps in engineering and scientific application have been found. The calculation technique has in some cases had to change to meet the requirement of physical reality. The derivatives are understood in the Caputo sense. The general response expression contains a parameter describing the order of the fractional derivative that can be varied to obtain various responses.

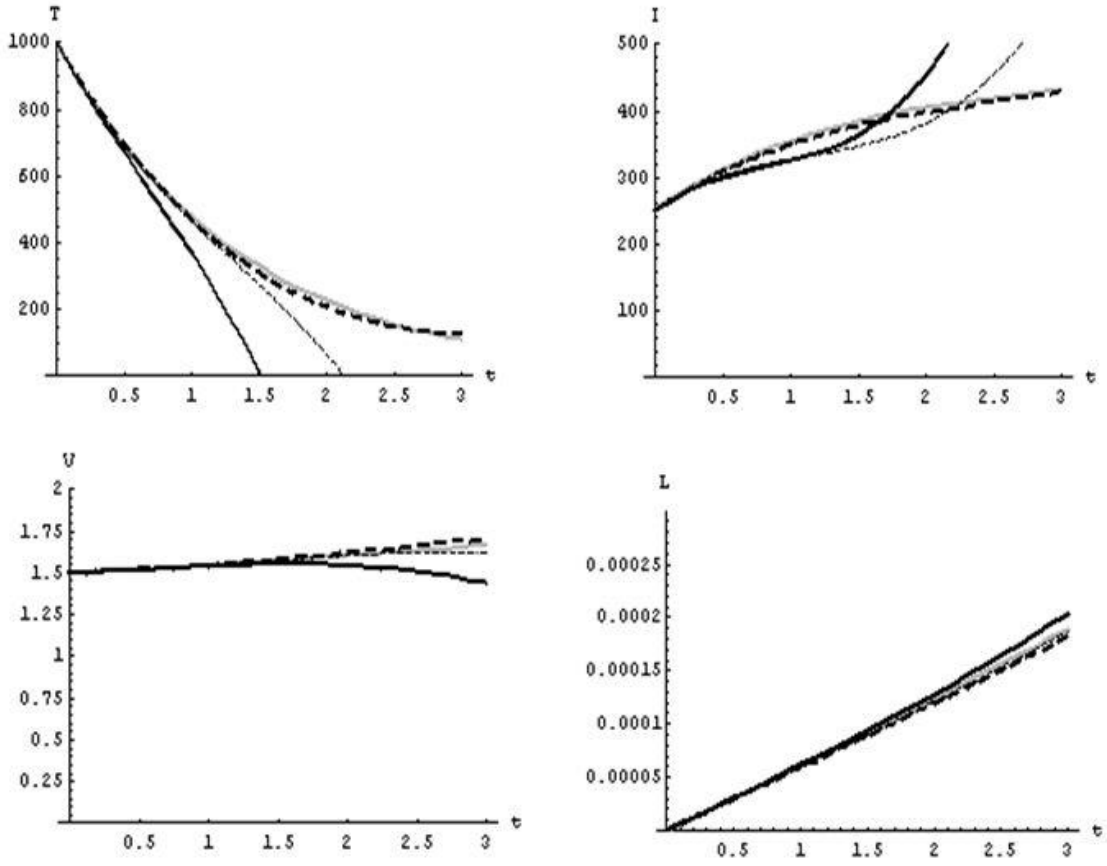

Figure 2: Plots of three terms approximations for human T-cell lymphotropic virus I (HTLV) infection of CD4$^+$T-cells model at $\kappa_1 = \kappa = 0.1$, $\alpha = 1$: Gray solid line ($RK4$), Dashed line ($h = -0.7$), Dotted line ($h = -1$), Black solid line ($h = -1.2$).

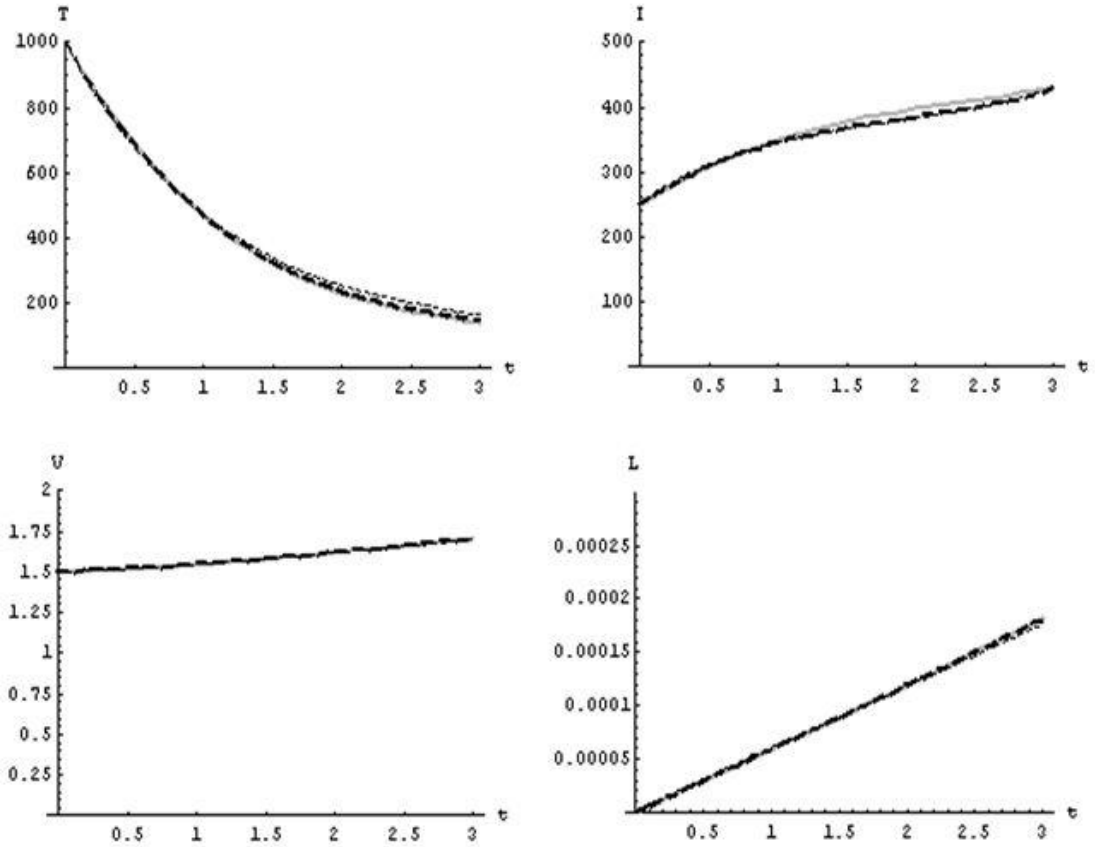

Figure 3**:** Plots of three terms approximations for human T-cell lymphotropic virus I (HTLV) infection of $CD4^+$T-cells model at $\kappa_1 = \kappa = 0.1$, $h = -0.7$: Gray solid line ($\alpha = 1$), Dashed line ($\alpha = 0.99$), Dotted line ($\alpha = 0.95$).

6 Conclusion

This paper attempts to find an approximate analytic solution for a general class of fractional order model of childhood diseases (18). For this purpose, we employed homotopy analysis method as a reasonable basis for studying the solution of the fractional model of human T-cell lymphotropic virus I (HTLV-I) infection of $CD4^+$ T-cells. We have solved the model for different values of the convergence controller h. The best value is $h = -0.7$, at which the HAM solution has an excellent agreement with the solution of classical Runge-Kutta method (RK4) for step size 0.01.

From the obtained results in the presented figures and tables, it is clear that varying the values of κ andκ_1 will alter the number of uninfected $CD4^+$T-cells, infected cells, and leukemic cells.

All parameters in the presented model are assumed to be positive constants. In [15], a threshold parameter

$$R_0 = \frac{\gamma\lambda\kappa}{\mu_T(\mu_L + \gamma)(\mu_A + \rho)}$$

is derived. It represents the average number of secondary infections caused by a single primary actively infected T cell introduced into a pool of susceptible T cells during its entire infection period. R_0 is typically called a basic reproduction number or the contact number in the literature of epidemiological models. If $R_0 > 1$, the HTLV infection persists in the T-cell population and infected T cells persists but if $R_0 < 1$ always die out. For example, if $k_1 = k = 0.1$, then $R_0 = 1.2$. The numerical results show that increasing the value of κ and $\kappa_1$makes the number of healthy $CD4^+$T-cells decreases dramatically, while the numbers of latently infected cells and leukemic cells increase substantially (see fig. 2). We modified the integer-order model (1) into a fractional-order model (18). The reason of using fractional order differential equations (FOD) is that FOD are naturally related to systems with memory which exists in most biological systems. Also they are closely related to fractals which are abundant in biological systems. The results derived of the fractional system (9) are of a more general nature. However, the fundamental solutions of these equations still exhibit useful scaling properties that make them attractive for applications. We would like to put your attention that time fractional derivatives change also the solutions we usually get in standard system (1).The results show that the solution continuously depends on the time-fractional derivative. GEM provides more realistic solutions series solutions which generally converge very rapidly. The concentration of healthy $CD4^+$T-cells $T(t)$, the concentration of latently infected $CD4^+$ T-cells $I(t)$, the concentration of actively infected $CD4^+$ T-cells $V(t)$, and the concentration of leukemic cells $L(t)$ have been obtained, therefore when $\alpha \to 1$ the solution of the fractional model (9) $T_\alpha(t)$, $I_\alpha(t)$, $V_\alpha(t)$, $L_\alpha(t)$ reduce to the standard solution $T(t), I(t), V(t), L(t)$ (see fig. 3).

References

[1] A.A.M. Arafa, S.Z. Rida and M. Khalil, Fractional modeling dynamics of HIV and CD4+ T-cells during primary infection, Nonlinear Biomedical Physics 6(2012) 1-7.

[2] T.J. Anastasio, The fractional-order dynamics of bainstem vestibulo-oculomotor neurons, Biological Cybernetics 72 (1994) 69_79.

[3] K.S. Cole, Electric conductance of biological systems, in: Proc. Cold Spring Harbor Symp. Quant. Biol, Cold Spring Harbor, New York, (1993) 107-116.

[4] R.V. Culshaw, S. Ruan, A delay-differential equation model of HIV infection of CD4C T-cells, Mathematical Bioscience 165 (2000) 27-39.

[5] A.M.A. El-Sayed, S.Z. Rida, A.A.M. Arafa, On the Solutions of Time-fractional Bacterial Chemotaxis in a Diffusion Gradient Chamber, International Journal of Nonlinear Science , 7(2009) 485–492.

[6] Hany N. Hassan, and Magdy A. El-Tawil, A new technique of using homotopy analysis method for solving high-order nonlinear differential equations, Math. Meth. Appl. Sci. 34 (2011) 728–742.
[7] Hany N. Hassan, and Magdy A. El-Tawil, An efficient analytic approach for solving two-point nonlinear boundary value problems by homotopy analysis method, Math. Meth. Appl. Sci. 34 (2011) 977–989.
[8] I. Hashim, O. Abdulaziz, S. Momani, Homotopy analysis method for fractional IVPs, Communications in Nonlinear Science and Numerical Simulation 14 (2009) 674–684.
[9] G. Jumarie, New stochastic fractional models for Malthusian growth, the Poissonian birth process and optimal management of populations, Mathematical and Computer Modelling 44 (2006) 231-254.
[10] S.J. Liao, A kind of approximate solution technique which does not depend upon small parameters: a special example, Int. J. Non-Linear Mech. 30(1995) 371–380.
[11] S.J. Liao, An approximate solution technique which does not depend upon small parameters (part 2): an application in fluid mechanics, Int. J. Non-Linear Mech. 32 (1997) 815–822.
[12] S.J. Liao, Y. Tan, A general approach to obtain series solutions of nonlinear differential equations, Stud. Appl. Math. 119 (2007) 297–354.
[13] D. Matignon, Stability results for fractional differential equations with applications to control processing, in: Computational Eng. in Sys. Appl., vol. 2, Lille, France, 1996, p. 963.
[14] I. Podlubny, Fractional Differential Equations, Academic Press, New York, 1999.
[15] Patricia Katri, Shigui Ruan , Dynamics of human T-cell lymphotropic virus I (HTLV-I) infection of CD4+ T-cells, C. R. Biologies 327 (2004) 1009–1016.
[16] L.M. Petrovic, D.T. Spasic, T.M. Atanackovic, On a mathematical model of a human root dentin, Dental Materials 21 (2005) 125_128.
[17] N.I. Stilianakis, J. Seydel, Modeling the T-cell dynamics and pathogenesis of HTLV-I infection, Bull. Math. Biol. 61 (1999) 935–947.
[18] L. Wang, M.Y. Li, D. Kirschner, Mathematical analysis of the global dynamics of a model for HTLV-I infection and ATL progression, Math. Biosci. 179 (2002) 207–217.