Stud. Univ. Babeş-Bolyai Math.
Volume LVI, Number 1
March 2011, pp. 165–178

Simulated results for deterministic model of HIV dynamics

Marwan Taiseer Alquran, Kamel Al-Khaled and Ameen Alawneh

Abstract. In this paper, an algorithm based on He's variational iteration method (shortly, VIM) is developed to approximate the solution of a non-linear mathematical model of HIV dynamics. Using a system of ordinary differential equations, the model describes the viral dynamics of HIV-1. Some plots of the solution are depicted and used to investigate the influence of certain key parameters on the spread of the disease. The results shows that the VIM has the advantages of being more concise for numerical purposes. Furthermore, this work opens a new direction of research whereby He's VIM applications might offer more insight into the modeling of dynamical systems in life sciences.

Mathematics Subject Classification (2010): 35R99, 49M27.

Keywords: Iteration method, HIV-1 dynamics, mathematical epidemiology, ODE models.

1. Introduction

Mathematical modeling of many biological or physical systems leads to nonlinear ordinary differential equations. An effective method is required to analyze the mathematical model which provides solutions conforming to physical reality. Therefore, we must be able to solve nonlinear ordinary differential equations. Common analytic procedures linearize the system or assume that nonlinearities are relatively insignificant. Such procedures change the actual problem to make it tractable by the conventional methods. In short, the physical problem is transformed to a purely mathematical one, for which the solution is readily available. This changes, sometimes seriously, the solution, which means that the problem being solved is no longer a proper representation of the physical problem whose solution is desired. However, in spite of the extensive development in the mathematical and statistical techniques

applied to modeling infectious diseases, little has been done to apply approximate methods to solve epidemic models. We try to obtain some analytical results to the deterministic model posed in this paper. In particular, we discuss mathematical and statistical ideas representing HIV internal virus dynamics. Simulation results from initial attempts in the areas of applied mathematics and statistics will be presented.

The human immuno-deficiency virus (HIV) infection which can lead to acquired immuno-deficiency syndrome (AIDS), has become an important infectious disease in both developed and developing nations. Mathematical models have been used extensively in research into the epidemiology of HIV/AIDS, to help improve our understanding of the major contributing factors in a given epidemic.

The key markers of the disease progression due to HIV and ADIS are the CD4+ $T-$cell and viral levels in the plasma. Modeling the interaction between HIV-1 virus and CD4 cells has been a major area of research for many years [17, 3, 18]. In recent years, a few studies of HIV dynamics have been conducted to describe the effects of various epidemiological factors [1, 5, 15, 2, 19, 16]. In particular, in [1], the authors present an overview of some concepts and methodologies that are useful on modeling HIV pathogenesis. A dynamical system modeling the HIV infection was used in [5] to show the impact of the viral diversity on the immune response and disease dynamics. In [15], the authors considered a non-linear mathematical model for HIV epidemic that spreads in a variable size population through both horizontal and vertical transmission. Using stability theory and computer simulation, they showed that by controlling the rate of vertical transmission, the spread of the disease can be reduced significantly. In [4], the author introduce a novel class of HIV models that incorporates mutation, the mutation is modeled by an integral operator whose kernel describes the transition probability between different strains. Numerical aspects of computer simulations are discussed.

Instead of finding a small parameter for solving nonlinear problems through perturbation method, a new analytical method called He's variational iteration method will be used in this paper to solve the epidemic model problem. The VIM is useful to obtain exact and approximate solutions for linear and nonlinear differential equations. It has been used to solve effectively, easily and accurately a large class of nonlinear problems with approximations.

The organization of the paper is as follows: In section 2, we describe a $3-$dimensional model for internal HIV dynamics. In section 3, we review the procedure of VIM. To show the efficiency of the method, in section 4, we apply the method on the model system appeared in section 2. Simulation results are presented in section 5.

2. HIV Model System

Mathematical models have come to play an important part in biological systems. Mathematics makes it possible to make predictions about the behavior

Symbol	Description
$x(t)$	concentration of uninfected cells.
$y(t)$	concentration of infected cells.
$z(t)$	concentration of virus particles.
$(1-\gamma)$	reverse transcriptase inhibitor drug effects.
$(1-\eta)$	protease inhibitor drug effect.
λ	total rate of production of healthy cells per unit time.
κ	per capita death rate of healthy cells.
β	transmission coefficient between uninfected cells and the infective virus particles.
N	average number of infective virus particles produced by an infected cell in the absence of HAART during its entire infectious lifetime.
u	per capita death rate of infective virus particles.
a	death rate of infected cells.

TABLE 1. Variables and parameters in system (2.1)

of the system. Following [14], we introduce a 3−dimensional model to describe the viral dynamics in the presence of HIV-1 infection and Highly Active Antiretrovital Treatment (HAART). The equations of the model represents the variation rate of uninfected cells, infected cells, and virus particles. The model is thus described by the following

$$\begin{aligned} \tfrac{dx(t)}{dt} &= \lambda - \kappa x(t) - (1-\gamma)\beta x(t)z(t) \\ \tfrac{dy(t)}{dt} &= (1-\gamma)\beta x(t)z(t) - ay(t) \\ \tfrac{dz(t)}{dt} &= (1-\eta)Nay(t) - uz(t) - (1-\gamma)\beta x(t)z(t) \end{aligned} \tag{2.1}$$

with suitable initial conditions. The variables $x(t), y(t)$ and $z(t)$ are functions of time $t \in [0, \infty)$. We summarize in Table 1 the biological meaning of the variables and parameters occurring in this model. This model captures mathematically the viral dynamics of HIV-1 virus interacting with CD4 cells. It can be seen that a model of such a simple nature is able to adequately reflect the disease progression from the initial infection to an asymptomatic stage where the set-point is reached.

We assume that the cells and the virus are uniformly distributed on the organism. Note that when a single infective virus particle infects a single uninfected cell the virus particle is absorbed into the infected cell and effectively dies. Hence, the term $(1-\gamma)\beta x(t)z(t)$ appears in all the three equations. In system (2.1), the first equation represents the dynamics of the concentration of healthy cells $x(t)$; λ represents the rate (assumed to be constant) at which

new $x(t)$ cells are generated. In the case of active HIV infection, the concentration of healthy cells decreases proportionally to the product $(1-\gamma)\beta x(t)z(t)$, where β represents a coefficient that depends on various factors, including the velocity of penetration of virus into cells, and the frequency of encounters between uninfected cells and free virus. The second equation in system (2.1) describes the dynamics of the concentration of infected cells $y(t)$; $(1-\gamma)\beta$ is the rate of infections; a is the death rate of infected cells. Therefore, the average lifetime of an infected cell is $1/a$. The third equation describes the concentration of free virion $z(t)$, which are produced by the infected cells at a rate $(1-\eta)Na$, and u is the death rate of the virion. The parameters of the model and their values are defined in Tables 1 and 2. Regarding equilibrium points and stability for system (2.1), a qualitative investigation [14] of the system described by equations (2.1) reveals that the model system has a unique disease-free equilibrium given by $(\lambda/\kappa, 0, 0)$.

A value for R_0, the basic reproduction number, is also useful to study further behavior of the system. This number tells us how many secondary infective virus particle will result from the introduction of one infected cell which was infected by the original infective virus particle. Hence

$$R_0 = \frac{(1-\gamma)\beta\lambda N(1-\eta)}{\kappa u + \beta\lambda(1-\gamma)}.$$

R_0 can also be interpreted as the expected number of secondary infected particles caused by a single infected virus particle entering the disease-free population at equilibrium $(\lambda/\kappa, 0, 0)$. $R_0 = 1$, means that each infected cell will infect one uninfected cell. Usually, $R_0 < 1$ implies that an epidemic will not result from the introduction of one infected cell, whereas $R_0 > 1$ implies that an epidemic will occur, and $R_0 = 1$ requires further investigation. However, as will be seen, the model (2.1) may imply something further, namely that the threshold value of R_0 must be brought far below one in order to avoid an epidemic, and if this does not happen, an endemic equilibrium may be established. R_0 is also useful for establishing the existence of equilibrium points, and in performing stability analysis for the system. To discuss the local behavior of the system around the equilibrium point, we introduce the following theorem

Theorem 2.1. *The solution of the model system (*2.1*) is asymptotically stable at the equilibrium point* $(\lambda/\kappa, 0, 0)$ *provided that* $R_0 < 1$.

Proof. The Jacobian of the system (2.1) is

$$J(x,y,z) = \begin{bmatrix} -\kappa - (1-\gamma)\beta z(t) & 0 & -(1-\gamma)\beta x(t) \\ (1-\gamma)\beta z(t) & -a & (1-\gamma)\beta x(t) \\ -(1-\gamma)\beta z(t) & (1-\eta)Na & -u-(1-\gamma)\beta x(t) \end{bmatrix}$$

Substituting the equilibrium point $(\lambda/\kappa, 0, 0)$, the Jacobian matrix becomes

$$J(\lambda/\kappa, 0, 0) = \begin{bmatrix} -\kappa & 0 & -(1-\gamma)\beta\lambda/\kappa \\ 0 & -a & (1-\gamma)\beta\lambda/\kappa \\ 0 & (1-\eta)Na & -u-(1-\gamma)\beta\lambda/\kappa \end{bmatrix}$$

The eigenvalues of this matrix are $\lambda_1 = -\kappa$,

$$\lambda_2 = \frac{-a\kappa - \beta\lambda + \beta\gamma\lambda - \kappa u + \sqrt{M - 4a\kappa(-\beta(-1+\gamma)\lambda(1+(-1+\eta)N) + \kappa u)}}{2\kappa}$$

and,

$$\lambda_3 = -\frac{a\kappa + \beta\lambda - \beta\gamma\lambda + \kappa u + \sqrt{M - 4a\kappa(-\beta(-1+\gamma)\lambda(1+(-1+\eta)N) + \kappa u)}}{2\kappa}$$

where $M = (a\kappa + \beta(\lambda - \gamma\lambda) + \kappa u)^2$. λ_1 is clearly real and negative. Also, as

$$R_0 = \frac{(1-\gamma)\beta\lambda N(1-\eta)}{\kappa u + \beta\lambda(1-\gamma)} < 1,$$

then $(1-\gamma)\beta\lambda N(1-\eta)$ is less than $\kappa u + \lambda(1-\gamma)\beta\lambda$, and so λ_2, λ_3 meets the necessary criteria. The system (2.1) shows local asymptotic stability at the equilibrium point $(\lambda/\kappa, 0, 0)$.

To examine the sensitivity of R_0 to the parameters, say N and u, the normalized forward sensitivity index [6] with respect to the parameters N, u are calculated as

$$\mu_N = \frac{\frac{\partial R_0}{R_0}}{\frac{\partial N}{N}} = \frac{N}{R_0}\frac{\partial R_0}{\partial N} = \frac{N}{R_0}\frac{(1-\gamma)\beta\lambda(1-\eta)}{\kappa u} = 1.$$

Thus, R_0 and N are directly proportional. Also,

$$|\mu_u| = |\frac{\frac{\partial R_0}{R_0}}{\frac{\partial u}{u}}| = |\frac{u}{R_0}\frac{\partial R_0}{\partial u}| = |\frac{-\kappa u}{\kappa u + \beta\lambda(1-\gamma)}| < 1.$$

Therefore, R_0 is most sensitive to changes in N. So, in section 5, we choose to focus on changing the parameters N and u.

3. Basic Idea of VIM

In 1978, Inokuti et al [8] proposed a general Lagrange multiplier method to solve nonlinear problems. Ji-Huan He has modified the method of Inokuti, and propose the variational iteration method (VIM) [9, 12]. This method has been employed to solve a large variety of linear and nonlinear problems with approximations converging rapidly to accurate solutions. Some advantages of this technique are

1. The initial condition can be chosen freely with some unknown parameters.
2. The unknown parameters in the initial condition can be easily identified.
3. The calculation is simple and straightforward.

This approach is successfully and effectively applied to various equations, see for example [9, 12, 13], and the reference therein.

The idea of this method is constructing a correction functional by a general Lagrange multiplier. The multiplier in the functional should be chosen such that its correction solution is superior to its initial approximation, called trial function, and is the best within the flexibility of trial function,

accordingly we can identify the multiplier by the variational theory [9, 12]. A complete review of the VIM is available in [10].

The initial approximation can be freely chosen with possible unknowns, which can be determined by imposing the boundary/initial conditions. To illustrate the procedure of this approach, we consider the following general differential equation

$$\mathbf{L}u(t) + \mathbf{N}u(t) = f(t). \tag{3.1}$$

where $\mathbf{L}$ is a linear operator, $\mathbf{N}$ is a nonlinear operator, and $f(t)$ is an inhomogeneous term. According to the variational iteration method [9, 12], the terms of a sequence $\{u_n\}$ are constructed such that this sequence converges to the exact solution, u_n's are calculated by a correction functional as follows:

$$u_{n+1}(t) = u_n(t) + \int_0^t \lambda(\tau) \left\{ \mathbf{L}u_n(\tau) + \mathbf{N}(\tilde{u})(\tau) - f(\tau) \right\} d\tau \tag{3.2}$$

where λ is general Lagrangian multipliers, which can be identified optimally via the variational theory [9], the subscript n denotes the nth order approximation. The second term, involving the integral, on the right-hand side of equation (3.2) is called the correction. Under suitable restricted variational assumption (i.e.,$\tilde{u}_n$ is considered as a restricted variation), we can assume that the above correctional functional are stationary (i.e., $\delta\tilde{u}_n = 0$). The successive approximations $u_{n+1}(t), n \geq 0$ of the solution $u(t)$ will be readily obtained upon using Lagrange multipliers, and by using the selective function u_0. The initial condition $u(0)$ is usually used for selecting the zeroth approximation u_0. With λ determined, then several approximations $u_n(t), n \geq 0$, follow immediately, the exact solution may be obtained by using

$$u(t) = \lim_{n \to \infty} u_n(t).$$

For linear problems, its exact solution can be obtained by only one iteration step, this is due to the fact that the Lagrange multipliers can be exactly identified, see [9]. He's technique provides a sequence of functions which converges to the exact solution of the problem [12].

In fact, the solution of the differential equation (3.1) is considered as the fixed point of the functional (3.2) under suitable choice of the initial approximation. For the convergence proof of (3.2), we state the following known result that is useful to support the convergence of our iteration.

Theorem 3.1. [7] *For a Banach space X, suppose the nonlinear mapping $A : X \to X$ satisfy*

$$\| A[u] - A[\bar{u}] \| \leq \gamma \| u - \bar{u} \|, \quad u, \bar{u} \in X$$

for some constant $\gamma < 1$. Then A has a unique fixed point. Furthermore, the sequence $u_{n+1} = A[u_n]$ with arbitrary choice of $u_0 \in X$, converges to the fixed point of A, and

$$\| u_k - u_j \| \leq \| u_1 - u_0 \| \sum_{\ell=j-1}^{k-2} \gamma^{\ell}.$$

According to this Theorem, for the nonlinear mapping

$$A[u] = u(t) + \int_0^t \Big[\mathbf{L}u(\tau) + \mathbf{N}(u(\tau)) - f(\tau)\Big]d\tau.$$

A sufficient condition for the convergence of the VIM is strictly contraction of A. Furthermore, the sequence (3.2) converges to the fixed point of A, which is also the solution of the differential equation in Equation (3.1). In what follows, we will apply the VIM to solve the epidemic model (2.1), to illustrate the strength of the method and to establish approximations of high accuracy for these models.

4. Applications

To show the efficiency of the method described in the previous section, in this section, we apply the VIM to solve the system of nonlinear ordinary differential equations (2.1). According to the VIM, we can construct the correction functionals as follows:

$$x_{n+1}(t) = x_n(t) + \int_0^t \lambda_1(\tau)\Big\{x_n'(\tau) - \lambda + \kappa x_n(\tau) + (1-\gamma)\beta x_n(\tau)\tilde{z}_n(\tau)\Big\}d\tau$$

$$y_{n+1}(t) = y_n(t) + \int_0^t \lambda_2(\tau)\Big\{y_n'(\tau) - (1-\gamma)\beta\tilde{x}_n(\tau)\tilde{z}_n(\tau) + ay(\tau)\Big\}d\tau$$

$$z_{n+1}(t) = z_n(t)$$

$$+ \int_0^t \lambda_3(\tau)\Big\{z_n'(\tau) - (1-\eta)Na\tilde{y}_n(\tau) + uz_n(\tau) + (1-\gamma)\beta\tilde{x}_n(\tau)\tilde{z}_n(\tau)\Big\}d\tau \quad (4.1)$$

where λ_1, λ_2 and λ_3 are the general Lagrange multipliers, and $\tilde{x}_n, \tilde{y}_n$ and $\tilde{z}_n$ denote restricted variations, i.e., $\delta\tilde{x}_n = \delta\tilde{y}_n = \delta\tilde{z}_n = 0$. Making the above correction functional stationary

$$\delta x_{n+1}(t) = \delta x_n(t) + \delta\int_0^t \lambda_1(\tau)\Big\{x_n'(\tau) - \lambda + \kappa x_n(\tau) + (1-\gamma)\beta x_n(\tau)\tilde{z}_n(\tau)\Big\}d\tau$$

$$= \delta x_n(t) + \delta\int_0^t \lambda_1(\tau)\Big\{x_n'(\tau) + \kappa x_n(\tau)\Big\}d\tau$$

$$= \delta x_n(t) + \lambda_1(\tau)\delta x_n(\tau)\Big|_{\tau=t} + \int_0^t (\kappa\lambda_1 - \lambda_1')(\tau)\delta x_n(\tau)d\tau = 0,$$

also,

$$\delta y_{n+1}(t) = \delta y_n(t) + \delta \int_0^t \lambda_2(\tau)\Big\{y_n'(\tau) - (1-\gamma)\beta\tilde{x}_n(\tau)\tilde{z}_n(\tau) + ay(\tau)\Big\}d\tau$$

$$= \delta y_n(t) + \delta \int_0^t \lambda_2(\tau)\Big\{y_n'(\tau) + ay_n(\tau)\Big\}d\tau$$

$$= \delta y_n(t) + \lambda_2(\tau)\delta y_n(\tau)\Big|_{\tau=t} + \int_0^t (a\lambda_2 - \lambda_2')(\tau)\delta y_n(\tau)d\tau = 0,$$

and,

$$\delta z_{n+1}(t) = \delta z_n(t)$$
$$+\delta \int_0^t \lambda_3(\tau)\Big\{z_n'(\tau) - (1-\eta)Na\tilde{y}_n(\tau) + uz_n(\tau) + (1-\gamma)\beta\tilde{x}_n(\tau)\tilde{z}_n(\tau)\Big\}d\tau$$
$$= \delta z_n(t) + \delta \int_0^t \lambda_3(\tau)\Big\{z_n'(\tau) + uz_n(\tau)\Big\}d\tau$$
$$= \delta z_n(t) + \lambda_3(\tau)\delta z_n(\tau)\Big|_{\tau=t} + \int_0^t (u\lambda_3 - \lambda_3')(\tau)\delta z_n(\tau)d\tau = 0,$$

yield the following stationary conditions

$$\begin{aligned} &\lambda_1'(\tau) - \kappa\lambda_1(\tau) = 0, \;\; 1 + \lambda_1(\tau)\Big|_{\tau=t} = 0 \\ &\lambda_2'(\tau) - a\lambda_2(\tau) = 0, \;\; 1 + \lambda_2(\tau)\Big|_{\tau=t} = 0 \\ &\lambda_3'(\tau) - u\lambda_3(\tau) = 0, \;\; 1 + \lambda_3(\tau)\Big|_{\tau=t} = 0 \end{aligned} \tag{4.2}$$

The general Lagrange multipliers can be identified by solving the system of equations in (4.2), to obtain $\lambda_1(\tau) = -e^{\kappa(\tau-t)}$, $\lambda_2(\tau) = -e^{a(\tau-t)}$, $\lambda_3(\tau) = -e^{u(\tau-t)}$. Substituting these values back into the correction functional Equation (4.1) results into the following iteration formula:

$$x_{n+1}(t) = x_n(t) - \int_0^t e^{\kappa(\tau-t)}\Big\{x_n'(\tau) - \lambda + \kappa x_n(\tau) + (1-\gamma)\beta x_n(\tau)z_n(\tau)\Big\}d\tau$$
$$y_{n+1}(t) = y_n(t) - \int_0^t e^{a(\tau-t)}\Big\{y_n'(\tau) - (1-\gamma)\beta x_n(\tau)z_n(\tau) + ay(\tau)\Big\}d\tau$$
$$z_{n+1}(t) = z_n(t)$$
$$-\int_0^t e^{u(\tau-t)}\Big\{z_n'(\tau) - (1-\eta)Nay_n(\tau) + uz_n(\tau) + (1-\gamma)\beta x_n(\tau)z_n(\tau)\Big\}d\tau. \tag{4.3}$$

We start with initial approximations $x_0(t) = N_1, y_0(t) = N_2, z_0(t) = N_3$. We can use $x_{n+1}(t)$ obtained in the first equation of (4.3) into the second equation of (4.3), and so on for other variables, this increases the convergence

rate. By the above iteration formula (4.3), we can obtain a few first terms being calculated.

$$\begin{aligned} x_1(t) &= 9.999995 \times 10^7 - 9.989995 \times 10^6 \ e^{-0.1t} \\ y_1(t) &= 1. + 9999e^{-0.5t} \\ z_1(t) &= 49999.9 - 39999.9e^{-5t} \end{aligned} \tag{4.4}$$

While,

$$\begin{aligned} x_2(t) &= 1 \times 10^7 + 399.6e^{-5.1t} - 408.162e^{-5t} - 9.99 \times 10^6 e^{-0.1t} \\ &\quad - 24994.9e^{-3.60822\times 10^{-16}t} - e^{-0.1t}(-25003.5 - 2497.5t) \\ y_2(t) &= 4999.99 - 434.347e^{-5.1t} + 444.443e^{-5t} + 11233.7e^{-0.5t} - 6243.73e^{-0.1t} \\ z_2(t) &= -494.9 + 19979.9e^{-5.1t} - 39999.9e^{-5t} + 55550e^{-0.5t} + 509.693e^{-0.1t} \\ &\quad - e^{-5t}(25544.7 - 1999.9t) \end{aligned}$$

Continuing in this manner, the rest of components of the iteration formulas can be obtained using symbolic packages such as *Mathematica*. In our case, only three terms from the iteration formula are used to obtain the approximation for our solutions.

5. Simulation Results and Discussion

To illustrate the use of the VIM, we describe some numerical experiments made to get a better understanding of the solutions behavior for the model system (2.1). The parameter values used here have all been taken from a published paper [14] and the reference therein, which are quoted here as in Table 2. The computer simulations were performed using the first three iterations $(x_3(t), y_3(t), z_3(t))$ for each variable, with the parameters values appeared in Table 2. Simulation results for the model, are displayed in Figures $1-6$. As can be clearly seen, Figure 1 shows the uninfected cells, it is found that uninfected cells first increases with time, and then after almost 40 days reaches it equilibrium position, which is $\lambda/\kappa = 1 \times 10^7$. As seen from Figure 2 that infected cells decreases exponentially as all infectives will develop AIDS and will die out. Figure 3, show the virus particles, we observe that immediately after infection, the amount of virus particles rises dramatically. After a few days (usually six to eight days), the virus concentration falls to the virus particles. Our further graphs $4-6$ dealing mainly with the existence of steady state for some values of $R_0 < 1$.

It should be pointed out that the parameters in the model are independent of each other, since each of them plays an independent role. These parameters have definite meaning, so the results of simulation can hardly coincide with the actual situation of the epidemic if the parameters cannot be adjusted to proper values.

Parameter	Values in Simulation
λ	10^6 day^{-1} dm^3
κ	0.1 day^{-1}
u	5 day^{-1}
a	0.5 day^{-1}
η	0.5
β	1×10^{-8} day^{-1} dm^3
N	100 per cell
γ	0.5
$N_1 = N_2 = N_3$	10000

TABLE 2. Parameters in system (2.1) with their values

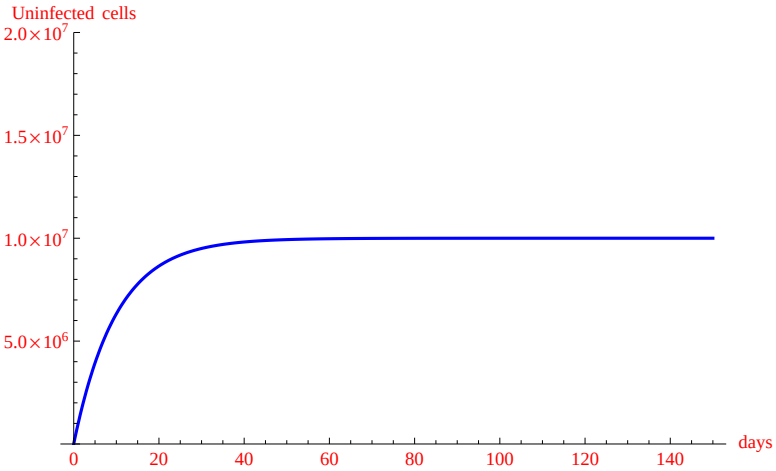

FIGURE 1. Simulated behavior of uninfected cells with parameter values given in Table 2, $R_0 = 0.49$, the steady state $(\lambda/\kappa, 0, 0)$ is asymptotically stable.

References

[1] Adams, B. M., Banks, H. T., Davidian, M., Hee-Dae Kwon, Tran, H. T., Wynne, S. N., Rosenberg, E. S., *HIV dynamics, Modeling, data analysis, and optimal treatment protocols*, Comput. Math. Appl., **184**(2005), no. 1, 10-49.

[2] Tripathi, A., *Ram Naresh and Dileep Sharma: Modeling the effect of screening of unaware infectives on the spread of HIV infection*, Appl. Math. Comput., **184**(2007), 1053-1068.

[3] Bhatia, N. P., Szego, G. P., *Stability Theory of Dynamical Systems*, Springer, New York, 1970.

[4] Pastore, D. H., *On the numerical simulations of a class of HIV models*, Math. Comp. Mode., in press, 2008.

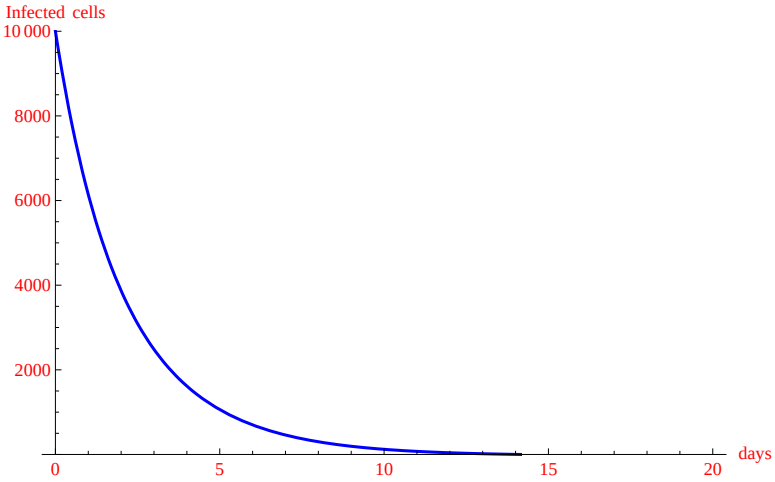

FIGURE 2. Simulated behavior of infected cells with parameter values as in Table 2, $R_0 = 0.49$

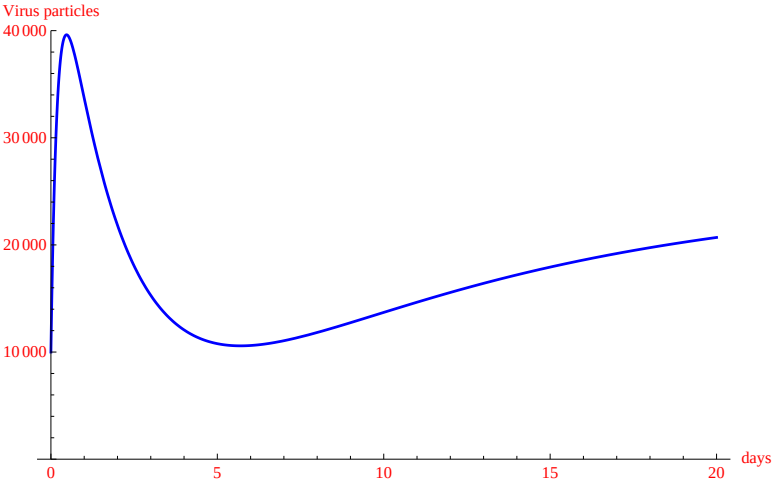

FIGURE 3. Simulated behavior of particles cells with parameter values given in Table 2, $R_0 = 0.49$

[5] Vergu, E., Mallet, A., Golmard, J. L., *A modeling approach to the impact of HIV mutations on the immune system*, Comp. Bio. Med., **35**(2005), 1-24.

[6] White, E., Comiskey, C., *Heroin epidemics, treatment and ODE modelling*, Mathematical Biosciences, **208**(2007), 312-324.

[7] Evans, L. C., *Partial Differential Equations*, AMS, Providence, RI, 1998.

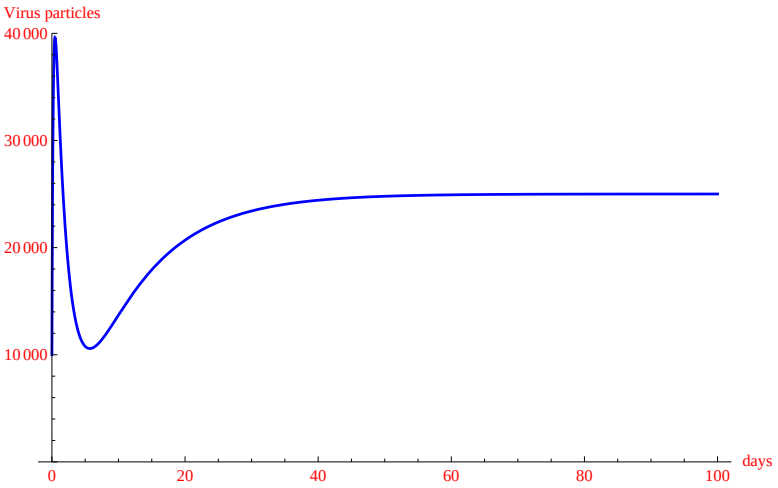

FIGURE 4. Simulated behavior of particles cells with parameter values given in Table 2, $R_0 = 0.49$, and $0 < t < 100$

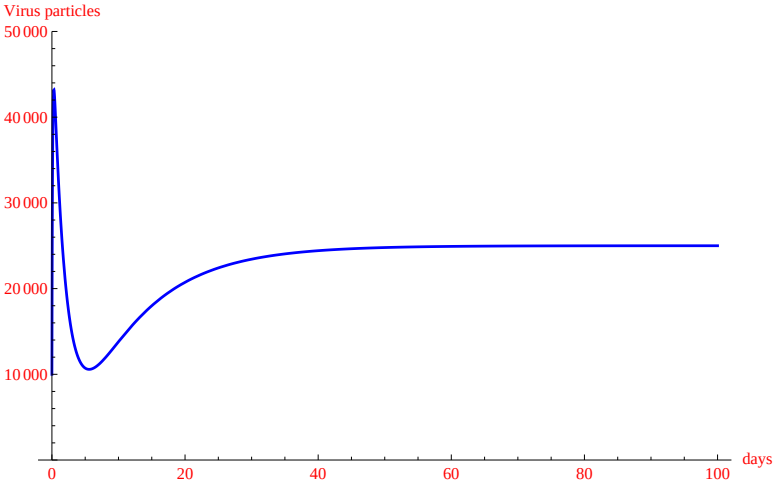

FIGURE 5. Simulated behavior for virus particles, the values of the parameters are the same as those in Table 2 except $N = 200, u = 10$

[8] Inokuti, M., Sekine, H., Mura, T., *General use of the Lagrange multiplier in nonlinear mathematics*, Nemat-Nassers (Ed.) Variational Method in the Mechanics of Solids. Pergamon Press, Oxford, 1978, 156-162.

[9] Ji-Huan He, *Variational iteration method − a kind of non-linear analytical technique: Some examples*, International J. of Nonlinear Mechanics, **34**(1999), no. 4, 699-708.

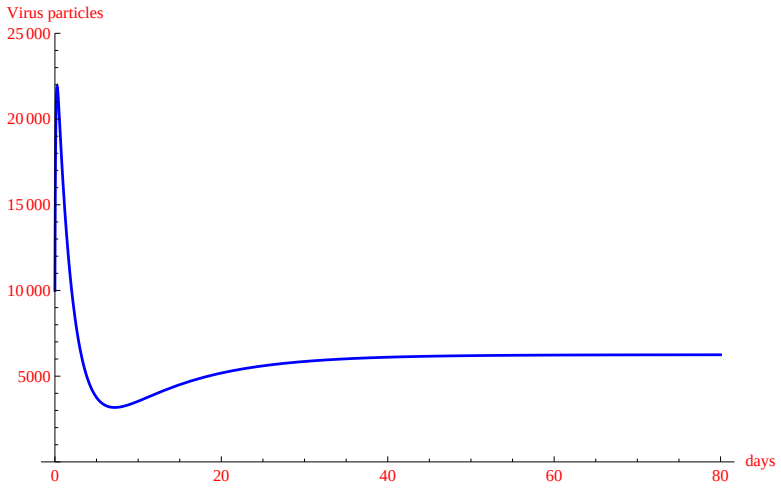

FIGURE 6. Simulated behavior for virus particles, the values of the parameters are the same as those in Table 2 except $u = 10$

[10] Ji-Huan He, *Variational iteration method for autonomous ordinary differential systems*, Comput. Appl. Math., **114**(2000), no. 2-3, 115-123.

[11] Ji-Huan He, *Some asymptotic methods for strongly nonlinear equations*, Intern J. of Modern Physics, **20**(2006), 1141-1199.

[12] Ji-Huan He, *Variational iteration method – Some recent results and new interpretations*, Comput. Appl. Math., **207**(2007), no. 1, 3-17.

[13] Dehghan, M., Tatari, M., *Identifying an unknown function in a parabolic equation with overspecified data via He's variational iteration method*, Chaos, Solitons and Fractals, **36**(2008), no. 1, 157-166.

[14] Dalal, N., Greenhalgh, D., Xuerong Mao, *A stochastic model for internal HIV dynamics*, Math. Analy. Appl. (in press).

[15] Naresh, R., Tripathi, A., Omar, S., *Modelling the spread of AIDS epidemic with vertical transmision*, Appl. Math. Comput., **178**(2006), 262-272.

[16] Khalili, S., Armaou, A., *Sensitivity analysis of HIV infection response to treatment via stochastic modeling*, Chem. Eng. Sci. (in press).

[17] Verhulst, F., *Nonlinear Differential Equations and Dynamical Systems*, Springer, London, 1990.

[18] Wu, H., Tan, W. Y., *Deterministic and Stochastic Models of AIDS Epidemics and HIV Infections with Intervention*, World Scientific Publishing Co., 2005.

[19] Xiaohua Xia, *Modelling of HIV infection, Vaccine readiness, drug effectiveness and therapeutical failures*, Journal of Process Control, **17**(2007), 253-260.

Marwan Taiseer Alquran
Jordan University of Science and Technology
Department of Mathematics and Statistics
P. O. Box 3030, Irbid-22110, Jordan
e-mail: marwan04@just.edu.jo

Kamel Al-Khaled
Jordan University of Science and Technology
Department of Mathematics and Statistics
P. O. Box 3030, Irbid-22110, Jordan
e-mail: kamel@just.edu.jo

Ameen Alawneh
Qatar University
Department of Mathematics, Statistics and Physics
On Sabbatical Leave from Jordan University of Science and Technology
e-mail: aalawneh@qu.edu.qa