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Application of modified homotopy perturbation method for EIAV infection

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Abstract : Equine Infectious Anemia Virus (EIAV) is a retrovirus that establishes a persistent infection in horses. The virus is characterized by acute and chronic recurring clinical signs including fever, anemia, edema and cachexia in some animals. The virus is in the same lentivirus subgroup that includes human immunodeficiency virus (HIV). The similarities between these two viruses make the study of the immune response to EIAV make it relevant to the research on HIV. In this paper, a modification of the homotopy perturbation method is applied to solve a mathematical model of within-host EIAV infection dynamics which contain both humoral and cell-mediated immune. This model is a nonlinear ordinary differential equations. Example is provided to show the ability and reliability of the method. The results reveal that the method is very effective and simple.

Keywords: Modified homotopy perturbation method, Equine Infectious Anemia Virus, Nonlinear ordinary differential equations
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1 Introduction

One of the most significant gaps in our knowledge of virus-host dynamics involves how different immune responses work together to counteract the pathogen. Understanding viral dynamics in the context of immune responses is essential for developing our knowledge of host-pathogen interactions as well as developing control strategies. Mathematical modelling has been applied in the study of viral dynamics and has increased our understanding of basic pathogenic interactions for infections, including human immunodeficiency virus, hepatitis B virus, hepatitis C virus, and influenza virus. EIAV is a useful model for investigating the correlation of immune control. EIAV is a retrovirus that is distantly related to the human immunodeficiency virus (HIV), the causative agent of acquired immunodeficiency syndrome. Like HIV, EIAV establishes a persistent infection in the host, with blood cells remaining infectious after disease free intervals for many years [2], [6].
The goal of this study is to create a mathematical model of EIAV and immune system dynamics in order to predict conditions that correlate with viral control.We use a five-equation model.
The homotopy perturbation method (HPM) introduced by He [4] has come to be accepted as an elegant tool in the hands of researchers looking for simple highly effective solutions to complicated problems in diverse arenas of science and technology. In a series of papers He has outlined and refined the HPM, showing

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its usefulness by solving algebraic, nonlinear ordinary differential equations, partial differential equations and problems involving discontinuities [3] In this paper, we present a modification of the HPM to solve a model for EIAV infection. The modification demonstrates a rapid convergence of the series solution if compared with standard HPM. In addition this method may give the exact solution for nonlinear equations by using only two iterations.

2 Mathematical Model

In this section we present a five-equation model that builds upon earlier studies [8]. Our model explicitly contains the dynamics of cytotoxic T lymphocytes (CTLs) and antibodies, including an equation for antibody dynamics that models antibody production in direct proportion to virus. CTLs are important for controlling equine infectious anemia virus (EIAV). Consider the system of ordinary differential equations [7]:

$$\begin{aligned} u_1^* &= \lambda - \rho u_1 - \beta u_1 u_3 \\ u_2^* &= \beta u_1 u_3 - \delta u_2 - k u_2 u_4 \\ u_3^* &= b u_2 - \gamma u_3 - f u_3 u_5 \\ u_4^* &= \psi u_2 u_4 - \omega u_4 \\ u_5^* &= \alpha u_3 - \mu u_5 \end{aligned} \tag{2.1}$$

where $\frac{du}{dt} = u^*$. In this model, the target cells of EIAV infection are monocyte-derived tissue macrophages. The number of uninfected target cells is represented by u_1. These cells become infected cells u_2 following contact with virus u_3 at rate β. Uninfected target cells are generated at rate λ and die at rate ρ. The infected cell death rate is δ. Infected cells are killed by CTLs u_4, at rate k.Virus is produced by infected cells at rate b and cleared at rate γ. The virus is neutralized by antibodies u_5 at rate f. CTLs proliferate in response to contact with infected cells at rate ψ and die at rate ω. The antibody population grows in proportion to virus at rate α and is cleared at rate μ. The initial conditions for the model are $u_1(0) = u_1, u_3(0) = u_3, u_4(0) = u_4$ and $u_2(0) = u_5(0) = 0$. We assume all parameters are nonnegative.

3 Analysis of the method

To clarify the basic ideas of the HPM, we consider the following nonlinear differential equation:

$$L(u) + N(u) - f(r) = 0, \qquad r \in \Omega \tag{3.1}$$

with boundary conditions $B(u, \frac{du}{dn}), \quad r \in \Gamma$, where L is linear operator, while N is nonlinear operator, B is a boundary operator, u is a known analytic function, Γ is the boundary of the domain Ω and $f(r)$ is a known analytic function.
The modified form of the HPM can be established based on the assumption that the function $f(r)$ can be

replaced by Taylor series as follow [5] :

$$f(r) = \sum_{n=0}^{\infty} f_n(r). \tag{3.2}$$

If we consider:

$$f(r) = f_0(r) + f_1(r), \tag{3.3}$$

we can construct the homotopy $U(r,p) : \Omega \times [0,1] \to R$ which satisfies:

$$H(U,p) = L(U) - L(u_0) + pL(u_0) + p[N(U) - f_1(r)] = f_0(r).$$

Here, a slight variation was proposed only on the components u_0 and u_1. The suggestion was that only the part f_0 be assigned to the zeroth component u_0, whereas the remaining part f_1 be combined with the component u_1.
According to the assumption 3.3 we can construct the homotopy $U(r,p) : \Omega \times [0,1] \to R$ which satisfies:

$$H(U,p) = L(U) - L(u_0) + pL(u_0) + pN(U) = \sum_{n=0}^{\infty} p^n f_n(r).$$

In this case the term f_0 is combined with the component u_0, f_1 is combined with the component u_1, f_2 is combined with the component u_2 and so on.This suggestion will facilitate the calculations of the terms $u_0, u_1, ...$ and hence accelerate the rapid convergence of the series solution. It observes that the modification of the HPM, reduces the number of terms involved in each component and hence the size of calculations is minimized compared to the HPM. In this section, we will apply modification HPM to nonlinear ordinary differential equations 2.1, we derive the equations:

$$\begin{aligned}
&U_1^* - u_{10}^* + p[u_{10}^* + \rho U_1 + \beta U_1 U_3] = \lambda \\
&U_2^* - u_{20}^* + p[u_{20}^* - \beta U_1 U_3 + \delta U_2 + k U_2 U_4] = 0 \\
&U_3^* - u_{30}^* + p[u_{30}^* - b U_2 + \gamma U_3 + f U_3 U_5] = 0 \\
&U_4^* - u_{40}^* + p[u_{40}^* - \psi U_2 U_4 + \omega U_4] = 0 \\
&U_5^* - u_{50}^* + p[u_{50}^* - \alpha U_3 + \mu U_5] = 0
\end{aligned} \tag{3.4}$$

Let's present the solution of the system 3.4 as the following:

$$U_j = U_{j0} + pU_{j1} + p^2 U_{j2} + ..., \;\; j = 1, ..., 5 \tag{3.5}$$

Substituting 3.5 in to 3.4, and equating the coefficients of the terms with the identical powers of p leads to:

$$p^0 : \begin{cases} U_{10}^* - u_{10}^* = \lambda \\ U_{20}^* - u_{20}^* = 0 \\ U_{30}^* - u_{30}^* = 0 \\ U_{40}^* - u_{40}^* = 0 \\ U_{50}^* - u_{50}^* = 0 \end{cases}$$

$$p^1 : \begin{cases} U_{11}^* + u_{10}^* + \rho U_{10} + \beta U_{10}U_{30} = 0 \\ U_{21}^* + u_{20}^* - \beta U_{10}U_{30} + \delta U_{20} + kU_{20}U_{40} = 0 \\ U_{31}^* + u_{30}^* - bU_{20} + \gamma U_{30} + fU_{30}U_{50} = 0 \qquad ... \\ U_{41}^* + u_{40}^* - \psi U_{20}U_{40} + \omega U_{40} = 0 \\ U_{51}^* + u_{50}^* - \alpha U_{30} + \mu U_{50} = 0 \end{cases}$$

$$p^j : \begin{cases} U_{1j}^* + \rho U_{1j-1} + \beta[U_{10}U_{3j-1} + U_{11}U_{3j-2} + ... + U_{1j-1}U_{30}] = \\ U_{2j}^* - \beta[U_{10}U_{3j-1} + U_{11}U_{3j-2} + ... + U_{1j-1}U_{30}] + \delta U_{2j-1} \\ +k[U_{20}U_{4j-1} + U_{21}U_{4j-2} + ... + U_{2j-1}U_{40}] = 0 \\ U_{3j}^* - bU_{jj-1} + \gamma U_{jj-1} + f[U_{30}U_{5j-1} + U_{31}U_{5j-2} + ... + U_{3j-1}U_{50}] = 0 \\ U_{4j}^* - \psi[U_{20}U_{4j-1} + U_{21}U_{4j-2} + ... + U_{2j-1}U_{40}] + \omega U_{4j-1} = 0 \\ U_{5j}^* - \alpha U_{3j-1} + \mu U_{5j-1} = 0 \end{cases}$$

where $j = 2, 3, 4, 5$.
Therefore, the approximation solution can be obtained as follows:

$$U_1 = \lambda t + [-\rho - \beta u_3]\frac{\lambda t^2}{2} + [\rho^2 + 2b\beta u_2 - 2\beta\gamma u_3 - 2f\beta u_3 u_5 - \beta^2 u_3^2]\frac{\lambda t^3}{6} + ...$$

$$U_2 = u_2 + [-\delta u_2 - ku_2u_4]t + [\beta u_3\lambda + \delta^2 u_2 + \delta k u_2 u_4 - k\psi u_2^2 u_4 + k\omega u_2 u_4 + k\delta u_2 u_4$$

$$+ k^2 u_2 u_4^2]\frac{t^2}{2} + [2\beta b u_2 - 2\beta\gamma u_3 - 2f\beta u_3 u_5 - \rho\beta u_3 - \beta^2 u_3^2 - \beta\delta u_3 - k\beta u_3 u_4]\frac{\lambda t^3}{6} + ...$$

$$U_3 = u_3 + [bu_2 - \gamma u_3 - fu_3u_5]t + [-\delta b u_2 - kbu_2u_4 - \gamma b u_2 + \gamma^2 u_3 + f\gamma u_3 u_5 - f\alpha u_3^2 \tag{3.6}$$

$$+ f\mu u_3 u_5 - bfu_2u_5 + f\gamma u_3 u_5 + f^2 u_3 u_5^2]\frac{t^2}{2} + b\beta u_3\frac{\lambda t^3}{6} + ...$$

$$U_4 = u_4 + [\psi u_2 u_4 - \omega u_4]t + [\psi^2 u_2^2 u_4 - \psi\omega u_2 u_4 - \delta\psi u_2 u_4 - k\psi u_2 u_4^2 + \omega\psi u_2 u_4$$

$$- \omega^2 u_4]\frac{t^2}{2} + \beta\psi u_3 u_4\frac{\lambda t^3}{6} + ...$$

$$U_5 = u_5 + [\alpha u_3 - \mu u_5]t + [\alpha b u_2 - \alpha\gamma u_3 - f\alpha u_3 u_5 - \mu\alpha u_3 + \mu^2 u_5]\frac{t^2}{2} + ...$$

Regarding to the convergence theorem of the homotopy perturbation method [1], the convergence of our method is clear.

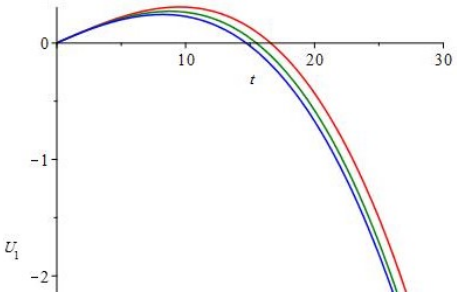

Figure 1: Parameter estimation of U_1: Variation of ρ

4 Numerical example

One example is provided here. This example is considered to illustrate the method for system of ordinary differential equations.

Example 4.1. *Consider the system of ordinary differential equations :*

$$u_1^* = 0.05 - 0.01u_1 - 0.0001u_1u_3$$

$$u_2^* = 0.0001u_1u_3 - 0.5u_2 - 0.01u_2u_4$$

$$u_3^* = 10000u_2 - 20u_3 - 3u_3u_5$$

$$u_4^* = 0.75u_2u_4 - 5u_4$$

$$u_5^* = 150u_3 - 20u_5$$

with initial conditions $u_1(0) = 10, u_3(0) = 5, u_4(0) = 7$ *and* $u_2(0) = u_5(0) = 0$. *From eqns.(6) we get:*

$$U_1 = 0.05t - 2.625 \times 10^{-4}t^2 - 1.658 \times 10^{-4}t^3 + ...$$

$$U_2 = 1.25 \times 10^{-5}t^2 - 1.690 \times 10^{-4}t^3 + ...$$

$$U_3 = 5 - 100t - 4625t^2 + 4.166 \times 10^{-2}t^3 + ...$$

$$U_4 = 7 - 35t - 87.5t^2 + 2.187 \times 10^{-5}t^3 + ...$$

$$U_5 = 750t - 15000t^2 + ...$$

In figure 1 if death rate of uninfected cells (ρ) *increases, uninfected cells* (U_1) *will decrease. Figure 2. shows that, if rate of killing by CTLs* (k) *increases, infected cells* (U_2) *decrease. Figure 3 shows that, if clearance rate of virus* (γ) *increases, infected cells* (U_2) *decrease. In figure 4 if neutralization of virus by*

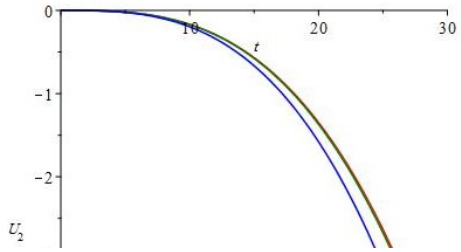

Figure 2: Parameter estimation of U_2: Variation of k

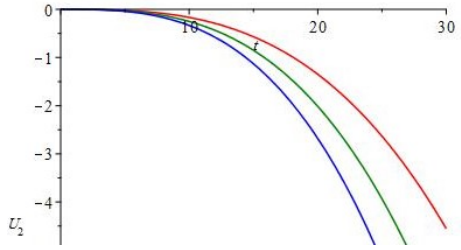

Figure 3: Parameter estimation of U_2: Variation of γ

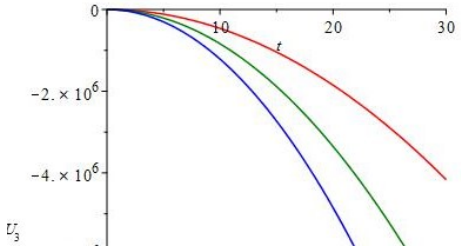

Figure 4: Parameter estimation of U_3: Variation of f

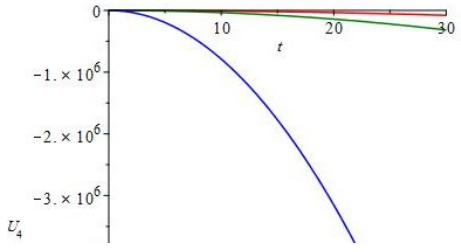

Figure 5: Parameter estimation of U_4: Variation of ω

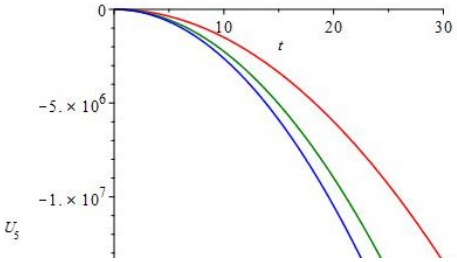

Figure 6: Parameter estimation of U_5: Variation of μ

antibodies (f) *increases, virus* (U_3) *will decrease. Figure 5 shows that, if death rate of CLTs* (ω) *increases, CTLs* (U_4) *decrease and in figure 6 if clearance rate of antibodies* (μ) *decrease, antibodies* (U_5) *will decrease.*

5 Conclusion

In this paper, we have applied modified homotopy perturbation method for the solving systems of ordinary differential equations as a new application of this method. Example shows that the analytical approximations to the solutions are reliable. Ability of modified homotopy perturbation method as an easy device for computing the solution of the systems of ordinary differential equations as well as some of the functional equations.

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