

ESTIMATING THE APPROXIMATE SOLUTION OF HIV INFECTION MODEL USING HOMOTOPY PERTURBATION METHOD (HPM)

Submitted in partial fulfillment of the requirements for the award of
Master of Science degree in Mathematics

By
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(DEEMED TO BE UNIVERSITY)

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BONAFIDE CERTIFICATE

This is to certify that this Project Report is the bonafide work of **MERCY J (42630004)** who carried out the project entitled "**Estimating The Approximate Solution Of HIV Infection Model Using Homotopy Perturbation Method (HPM)**" under our supervision from November **2023** to **April 2024**.

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DECLARATION

I **MERCY J (42630004)** hereby declare that the Project Report entitled “**Estimating the Approximate Solution of HIV Infection Model Using Homotopy Perturbation Method (HPM)**” done by me under the guidance of **Dr.GEETHAMALINI S** at **Sathyabama Institute of Science and Technology (Deemed to be University)** is submitted in partial fulfillment of the requirements for the award of Master of Science degree in Mathematics.

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A handwritten signature in blue ink, appearing to read 'Mercy', with a horizontal line underneath and a vertical line extending downwards from the right side.

SIGNATURE OF THE CANDIDATE

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ABSTRACT

In the present paper, the Homotopy Perturbation Method (HPM) is applied to provide an approximate solution of the basic HIV viral dynamic model describing the viral dynamics in a susceptible population. Also, we have tested the HPM on solving different implementations which are show the efficiency and accuracy of this method. The approximated solutions of HPM for the tested problems are agree well with the numerical solutions of Runge-Kutta method.

KEYWORD: Homotopy Perturbation method, System of nonlinear differential equations.

AMS SUBJECT CLASSIFICATION: 34A34, 34G20, 46N60.

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CHAPTER 1

INTRODUCTION

1.1 VIRUS INFECTION

Virus infection and spread is generally believed to be a recursive process of the binding of free virions to permissive target cells followed by entry, replication, making multiple copies of themselves and releasing them to extracellular environment, and then moving to infect adjacent target cells. New research and findings have challenged this classical view of how viruses infect and spread, suggesting that both cell-free virus infection and cell-to-cell transfer of HIV-1 are important mechanisms in virus spread. In the classical mode of infection (cell-free virus infection), the virions spread around the body through freely circulating particles by the movement between cells and tissues of the body via fluidphase diffusion. This route of infection is undoubtedly important. However, many viruses that are pathogenic for humans can also spread between cells without diffusing through the extracellular environment by exploiting pre-existing mechanisms of physiological communication between cells.

1.2 HIV INFECTION

Human immunodeficiency virus (HIV) was discovered by French Virologist Luc Antoine Montagnier & American Physician Scientist Robert Charles Gallo in 1983.

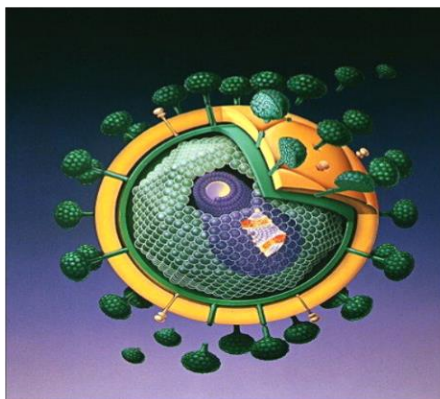


Fig:1.2.1 Image of HIV Viris

The human immunodeficiency virus (HIV) is a retrovirus that infects immune system's CD4+T cells and macrophages of the immune system. It deteriorates the person's immune system as infection progresses. The primary stage of infection takes around 10–15 years to develop into a fullblown case of acquired immunodeficiency syndrome (AIDS). HIV is transmitted through various processes involving mixing of body fluids like transfusion of contaminated blood, sharing of contaminated needles, unprotected sexual intercourse, childbirth and breastfeeding. The reasons that HIV causes so much damage stem from its biological functions and how it interacts with the human immune system. Its host cells are CD4+T-cells, which are part of the immune system component that signals to the rest of the immune system that infection is present. Unfortunately, this means that a full HIV infection means the downfall of the immune system, making the infected person susceptible to other, usually harmless, infections.

1.3 Schematic diagram of the basic HIV viral dynamics

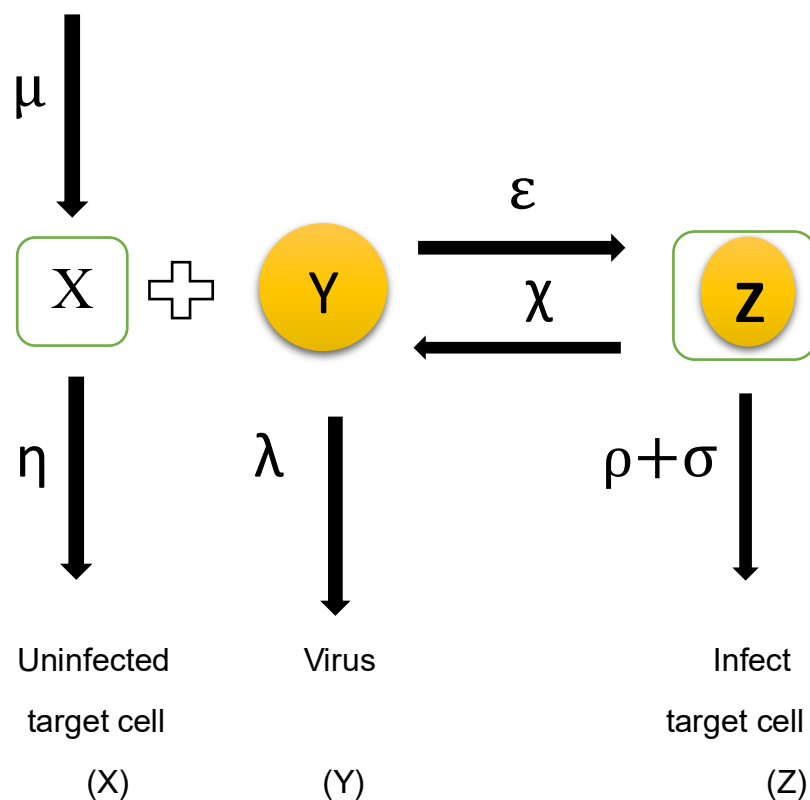


Fig.1.3.1 Schematic diagram of the basic HIV viral dynamics.

Their review illustrates the utility of mathematical models in comprehending factors influencing the prolonged immunological control of HIV, as well as in devising therapeutic strategies to transition a progressing patient into a state of long-term non-progression.

Fig.1.3.1 illustrates the fundamental principles underlying virus dynamics models. Susceptible uninfected cells, denoted as $X(t)$, become infected upon encountering free viruses, represented as $Y(t)$. Infected cells, denoted as $Z(t)$, generate new virus particles that exit the cell and seek other susceptible target cells. Iterative infection cycles contribute to the expansion of the virus population, with growth constrained by the availability of target cells. Naik proposed the model equations in his paper estimating the approximate analytical solution of HIV viral dynamic model by using homotopy analysis method

$$\left. \begin{aligned} \frac{dx}{dt} &= \mu - \eta x - \varepsilon xz \\ \frac{dy}{dt} &= \varepsilon xz - (\rho + \sigma)y \\ \frac{dz}{dt} &= \chi y - \lambda z \end{aligned} \right\} \quad (1.1)$$

The initial conditions for the above model are $x(0) = N_1, y(0) = N_2, z(0) = N_3$. Uninfected target cells are produced at a constant rate μ , from a pool of precursor cells and die at a rate εxz ηx . When these susceptible cells encounter with free virus particles, they become infected at a rate. The infected cells die at a rate $(\rho + \sigma)y$, in which ρy is the rate at which CTLs kills infected cells and σy is the viral caused cell death. Infected cells produce new virus particles with a rate χy , and the free virus particles that have been released from the cells decay with a rate λz . Thus, the average lifetime of an infected cell is $\frac{1}{\rho + \sigma}$; the average lifetime of a free virus particle is $\frac{1}{\lambda}$; the total number of virus particles produced from one infected cell is $\frac{\chi}{\rho + \sigma}$. In this paper we construct the Homotopy perturbation

method (HPM) on HIV viral dynamic model. Homotopy perturbation methods are based on the existence of small or large parameters.

1.4 Homotopy Perturbation Method

The homotopy perturbation method integrates principles from both classical perturbation techniques and homotopy techniques. To elucidate the fundamental concept of employing the homotopy perturbation method to solve nonlinear differential equations, we consider the following nonlinear differential equation:

$$A(u) - f(r) = 0, \quad r \in \Omega, \quad (1.2)$$

subject to boundary condition

$$B(u, \partial u / \partial n) = 0, \quad r \in \Gamma, \quad (1.3)$$

where A is a general differential operator, B a boundary operator, $f(r)$ is a known analytical function, C is the boundary of domain X and $\partial / \partial n$ denotes differentiation along the normal drawn outwards from X . In broad terms, one can decompose the operator A into two distinct components: a linear portion denoted as L and a nonlinear component represented as N . This is expressed in Equation (1.2) therefore can be rewritten as follows:

$$L(v) + N(v) - f(r) = 0 \quad (1.4)$$

We construct a homotopy of Equation (1.2) $v(r, p) : \Omega \times [0, 1] \rightarrow \Re$ which satisfies

$$H(v, p) = (1 - p)[L(v) - L(u_0)] + p[A(v) - f(r)] = 0, \quad p \in [0, 1], r \in \Omega \quad (1.5)$$

which is equivalent to

$$H(v, p) = L(v) - L(u_0) + pL(u_0) + p[N(v) - f(r)] = 0 \quad (1.6)$$

The parameter p , which lies in the interval $[0, 1]$, serves as an embedding parameter, while u_0 stands for an initial approximate estimation of equation (2). which satisfies the boundary conditions. It follows from (1.5) and (1.6) that

$$H(v, 0) = L(v) - L(u_0) = 0, H(v, 1) = A(v) - f(r) = 0 \quad (1.7)$$

Thus, the changing process of p from zero to unity is just that of $v(r, p)$ from $u_0(r)$ to $u(r)$. In topology, this is called deformation and $L(v) - L(u_0)$, $A(v) - f(r)$ are called homotopic.

In this instance, the embedding parameter is seamlessly introduced, devoid of any artificial influences. Moreover, it can be regarded as a diminutive parameter within the range of 0 to 1.

Consequently, it is highly reasonable to posit that the solution to equations (1.5) and (1.6) can be represented as

$$v = v_0 + pv_1 + p^2v_2 + \dots \quad (1.8)$$

The approximate solution of Equation (1.2), therefore, can be readily obtained:

$$u = \lim_{p \rightarrow 1} v = v_0 + v_1 + v_2 + v_3 + \dots \quad (1.9)$$

The convergence of series (1.9) has been proved by He in his paper Homotopy perturbation technique.

CHAPTER 2

LITERATURE SURVEY

Perelson formulated a fundamental model for the infection of the human immune system by HIV, featuring three variables: the population sizes of uninfected cells, infected cells, and free virus particles. Nowak and Bangham in their study, presented a mathematical model focusing solely on immune response CTL. Additionally, Wodarz proposed a model that incorporates both cell-mediated and humoral responses in Hepatitis C virus. Wodarz and Nowak conducted a thorough examination of mathematical frameworks pertaining to HIV pathogenesis during treatment. Culshaw R, Ruan S was investigated a delay differential equation model of HIV infection of CD4⁺ T-cells. In 2007 Merdan M was developed the HIV approximation solution of homotopy perturbation method for solving a model for HIV infection of CD4⁺ T-cells. Yuzbasi utilized the Bessel collocation method to obtain approximate solutions for the HIV infection model of CD4⁺T cells, while Yuzbasi and Karaçayır recently applied a Galerkin-type method to address a delayed model on HIV infection. Their study delved into the technique of residual correction, aiming to reduce approximation errors. Ghoreishi M, Ismail A, Almari A was proposed the mathematical modelling for application of the homotopy analysis method for HIV infection of CD4⁺ T-cells. In 2019 Ali N, Ahmad S, Aziz s, Zaman G was proposed the adomian decomposition method for solving HIV infection model of latently infected cells. In 2020 PA Naik, J. Zu, M. Ghoreishi, chaos, solitons & Fractals was developed estimating the approximate analytical solution of HIV viral dynamic model by using homotopy analysis method. In this paper introduces Estimating the Approximate Solution of HIV Infection Model Using Homotopy Perturbation Method (HPM).

CHAPTER 3

AIM AND SCOPE

3.1 AIM

Our aim is to investigate the comparison between analytical solution of HIV using HPM method and numerically by Fourth-order Runge-Kutta method (RK4).

3.2 SCOPE

The HPM is applied to provide an approximate solution of the basic HIV viral dynamic model describing the viral dynamics in a susceptible population and numerically by Fourth-order Runge-Kutta method (RK4). A comparison between the results of the analytical and numerical methods is investigated.

CHAPTER 4

Applications

Within this section, we intend to employ the Homotopy Perturbation Method (HPM) for solving the nonlinear ordinary differential system (1.1).

4.1 The epidemic model

Following the principles of the Homotopy Perturbation Method (HPM), a homotopy for the system (1.1) can be formulated in the following manner:

$$\begin{aligned}(1-p)(\dot{v}_1 - \dot{x}_0) + p(\dot{v}_1 - \mu - \eta v_1 + \varepsilon v_1 v_3) &= 0 \\ (1-p)(\dot{v}_2 - \dot{y}_0) + p(\dot{v}_2 - \varepsilon v_1 v_3 + \rho v_2 + \sigma v_2) &= 0 \\ (1-p)(\dot{v}_3 - \dot{z}_0) + p(\dot{v}_3 - \chi v_2 + \lambda v_3) &= 0\end{aligned}\tag{4.1}$$

where “dot” denotes differentiation with respect to t , and the initial approximations are as follows:

$$\begin{aligned}v_{1,0}(t) &= x_0(t) = x(0) = N_1 \\ v_{2,0}(t) &= y_0(t) = y(0) = N_2 \\ v_{3,0}(t) &= z_0(t) = z(0) = N_3\end{aligned}\tag{4.2}$$

And

$$\begin{aligned}v_1 &= v_{1,0} + p v_{1,1} + p^2 v_{1,2} + p^3 v_{1,3} + \dots \\ v_2 &= v_{2,0} + p v_{2,1} + p^2 v_{2,2} + p^3 v_{2,3} + \dots \\ v_3 &= v_{3,0} + p v_{3,1} + p^2 v_{3,2} + p^3 v_{3,3} + \dots\end{aligned}\tag{4.3}$$

where $v_{i,j}$, $i, j = 1, 2, 3, \dots$ are functions yet to be determined. Substituting Equation (4.2) and (4.3) into Equation (4.1) and arranging the coefficients of

“ p ” powers, we have

$$\begin{aligned}
& p(\dot{v}_{1,1} - \mu + \eta v_{1,0} + \varepsilon v_{1,0} v_{3,0}) + p^2(\dot{v}_{1,2} + \eta v_{1,1} + \varepsilon v_{1,0} v_{3,1} + \varepsilon v_{1,1} v_{3,0}) + \\
& p^3(\dot{v}_{1,3} + \eta v_{1,2} + \varepsilon v_{1,0} v_{3,2} + \varepsilon v_{1,1} v_{3,1} + \varepsilon v_{1,2} v_{3,0}) + \dots = 0 \\
& p(\dot{v}_{2,1} - \varepsilon v_{1,0} v_{3,0} + \rho v_{2,0} + \sigma v_{2,0}) + p^2(\dot{v}_{2,2} - \varepsilon v_{1,0} v_{3,1} + \varepsilon v_{1,1} v_{3,0} + \rho v_{2,1} + \sigma v_{2,1}) + \\
& p^3(\dot{v}_{2,3} - \varepsilon v_{1,0} v_{3,2} - \varepsilon v_{1,1} v_{3,1} - \varepsilon v_{1,2} v_{3,0} + \rho v_{2,2} + \sigma v_{2,2}) + \dots = 0
\end{aligned} \tag{4.4}$$

$$\begin{aligned}
& p(\dot{v}_{3,1} - \chi v_{2,0} + \lambda v_{3,0}) + p^2(\dot{v}_{3,2} - \chi v_{2,1} + \lambda v_{3,1}) + \\
& p^3(\dot{v}_{3,3} - \chi v_{2,2} + \lambda v_{3,2}) + \dots = 0
\end{aligned}$$

$$\begin{aligned}
& \dot{v}_{1,1} - \mu + \eta v_{1,0} + \varepsilon N_1 N_3 = 0 \\
& \dot{v}_{1,2} + \eta v_{1,1} + \varepsilon N_1 v_{3,1} + \varepsilon v_{1,1} N_3 = 0 \\
& \dot{v}_{1,3} + \eta v_{1,2} + \varepsilon N_1 v_{3,2} + \varepsilon v_{1,1} v_{3,1} + \varepsilon v_{1,2} N_3 = 0 \\
& \dot{v}_{2,1} - \varepsilon N_1 N_3 + \rho N_2 + \sigma N_2 = 0 \\
& \dot{v}_{2,2} - \varepsilon N_1 v_{3,1} + \varepsilon v_{1,1} N_3 + \rho v_{2,1} + \sigma v_{2,1} = 0 \\
& \dot{v}_{2,3} - \varepsilon N_1 v_{3,2} - \varepsilon v_{1,1} v_{3,1} - \varepsilon v_{1,2} N_3 + \rho v_{2,2} + \sigma v_{2,2} = 0 \\
& \dot{v}_{3,1} - \chi N_2 + \lambda N_3 = 0 \\
& \dot{v}_{3,2} - \chi v_{2,1} + \lambda v_{3,1} = 0 \\
& \dot{v}_{3,3} - \chi v_{2,2} + \lambda v_{3,2} = 0
\end{aligned} \tag{4.5}$$

From Equation (1.9), if the three terms approximations are sufficient, we will obtain

$$\begin{aligned}
x(t) &= \lim_{p \rightarrow 1} v_1(t) = \sum_{k=0}^{k=3} v_{1,k}(t) \\
y(t) &= \lim_{p \rightarrow 1} v_2(t) = \sum_{k=0}^{k=3} v_{2,k}(t) \\
z(t) &= \lim_{p \rightarrow 1} v_3(t) = \sum_{k=0}^{k=3} v_{3,k}(t)
\end{aligned} \tag{4.6}$$

Therefore,

$$\begin{aligned}
x(t) &= N_1 + t(\mu - \eta N_1 - \varepsilon N_1 N_3) + \frac{t^2}{2}[(\mu - \eta N_1 - \varepsilon N_1 N_3)(-\eta - \varepsilon N_3) - \varepsilon N_1(\chi N_2 - \lambda N_3)] \\
&+ \frac{t^3}{6}[(\mu - \eta N_1 - \varepsilon N_1 N_3)(-\eta - \varepsilon N_3) - \varepsilon N_1(\chi N_2 - \lambda N_3))(-\eta - \varepsilon N_3) - \\
&\varepsilon(N_1(\varepsilon \chi N_1 N_3 - \rho \chi N_2 - \sigma \chi N_2) - \lambda(\chi N_2 - \lambda N_3) + (\mu - \eta N_1 - \varepsilon N_1 N_3)(\chi N_2 - \lambda N_3))] \quad (4.7) \\
y(t) &= N_2 + t(\varepsilon N_1 N_3 - \rho N_2 - \sigma N_2) + \frac{t^2}{2}[\varepsilon(N_1(\chi N_2 - \lambda N_3) - (\mu - \eta N_1 - \varepsilon N_1 N_3)N_3) \\
&- (\varepsilon N_1 N_3 - \rho N_2 - \sigma N_2)(\rho + \sigma)] + \frac{t^3}{6}[\varepsilon[N_1(\chi(\varepsilon N_1 N_3 - \rho N_2 - \sigma N_2) - \lambda(\chi N_2 - \lambda N_3)) \\
&+ (\mu - \eta N_1 - \varepsilon N_1 N_3)(\chi N_2 - \lambda N_3) + ((\mu - \eta N_1 - \varepsilon N_1 N_3)(-\eta - \varepsilon N_3) - \varepsilon N_1(\chi N_2 - \lambda N_3))N_3] \\
&- [\varepsilon N_1(\chi N_2 - \lambda N_3) - (\mu - \eta N_1 - \varepsilon N_1 N_3)N_3] - (\varepsilon N_1 N_3 - \rho N_2 - \sigma N_2)(\rho + \sigma)](\rho + \sigma)] \\
z(t) &= N_3 + t(\chi N_2 - \lambda N_3) + \frac{t^2}{2}[\chi(\varepsilon N_1 N_3 - \rho N_2 - \sigma N_2) - \lambda(\chi N_2 - \lambda N_3)] \\
&+ \frac{t^3}{6}[\chi[\varepsilon(N_1(\chi N_2 - \lambda N_3) - (\mu - \eta N_1 - \varepsilon N_1 N_3)N_3) - (\varepsilon N_1 N_3 - \rho N_2 - \sigma N_2)(\rho + \sigma)] \\
&- \lambda[\chi(\varepsilon N_1 N_3 - \rho N_2 - \sigma N_2) - \lambda(\chi N_2 - \lambda N_3)]]
\end{aligned}$$

4.2. Numerical results and comparison with the HPM

For comparison with the results done by Biazar [30], the following values, for parameters, are considered:

$N_1 = 20$	Initial population of $x(t)$, who are susceptible
$N_2 = 15$	Initial population of $y(t)$, who are infective
$N_3 = 10$	Initial population of $y(t)$, who are immune
$\beta = 0.01$	Rate of change of susceptibles to infective population
$\gamma = 0.02$	Rate of change of infectives to immune population

Table 4.2.1 Parameters and variables with their values for HIV viral dynamic model

Parameters and functions	Meaning	Values
x	Susceptible cells at time	Variable
y	Infected cells at time t	Variable
z	Virus	Variable
μ	Inflow of target cells	10 cells $mm^{-3}day^{-1}$
ε	Infection rate	0.000024 mm^3day^{-1}
η	Decay rate for uninfected cells	0.01 day^{-1}
χ	Rate of virus production	467 virions $cells^{-1}$
λ	Clearance rate of virus	3.4 day^{-1}
σ	Rate of cell death by virus	0.16
ρ	Rate at which CTLs kills infected cells	0.01 $day^{-1} cell^{-1}$
x_0	Initially susceptible cells	1000
y_0	Initially infected cells	0.00
z_0	Initial virus	0.001

Three term approximations for $x(t)$, $y(t)$ and $z(t)$, are calculated and presented below:

$$\begin{aligned}
 x(t) &= 20 - 3t - 0.045t^2 + 0.02805t^3 \\
 y(t) &= 15 + 2.7t + 0.018t^2 - 0.02817t^3 \\
 z(t) &= 10 + 0.3t + 0.07t^2 - 0.00012t^3
 \end{aligned}
 \tag{4.8}$$

CHAPTER 5

Results and Discussion

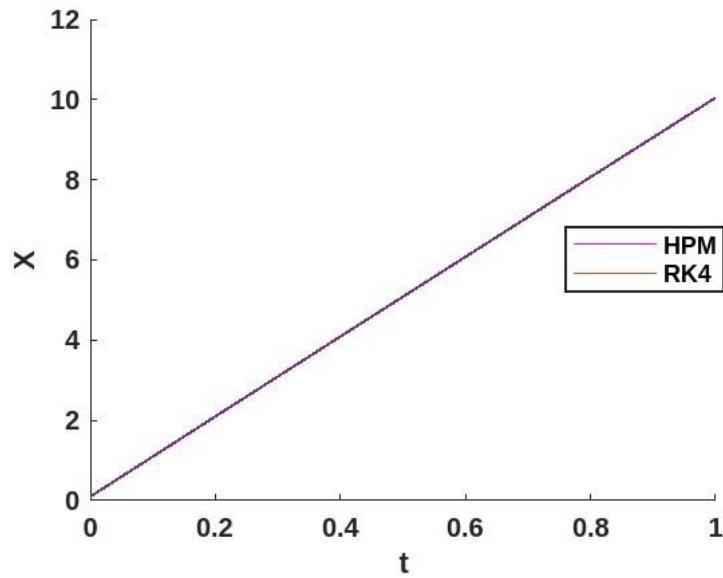


Fig. 5.1. Plots of three terms approximations for $x(t)$ versus time

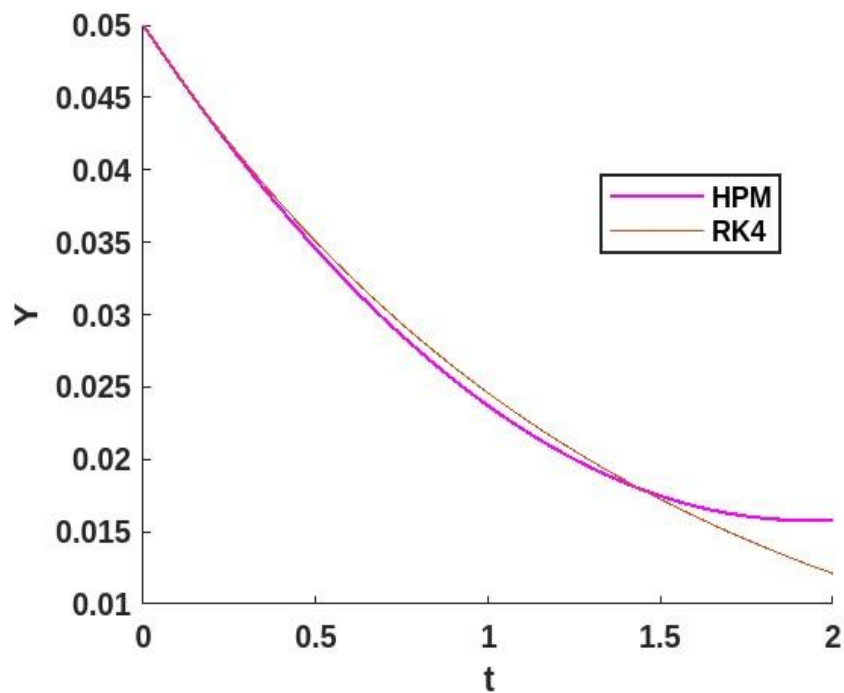


Fig.5.2. Plots of three terms approximations for $y(t)$ versus time

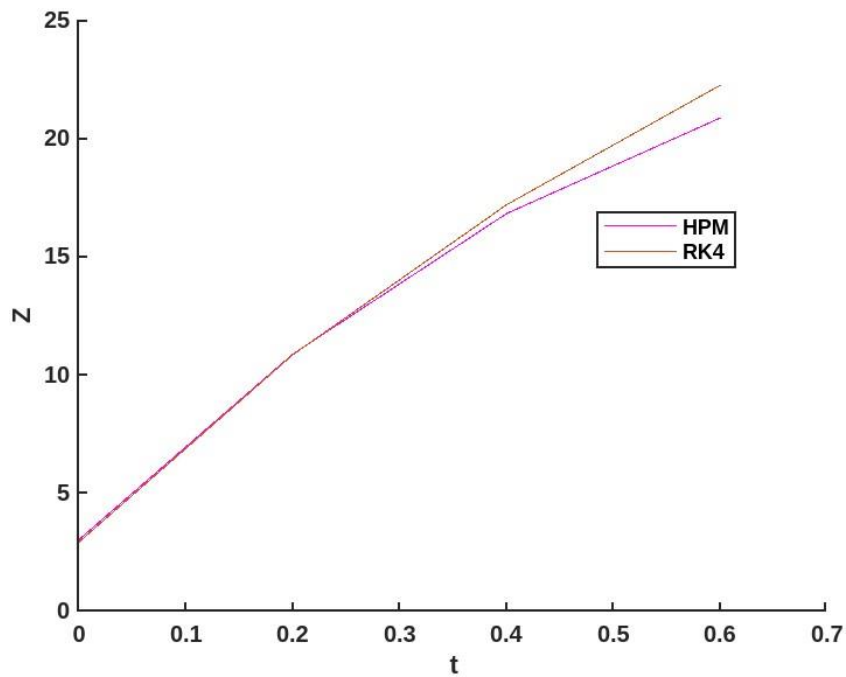


Fig. 5.3. Plots of three terms approximations for $z(t)$ versus time

comparison between the results shows that the results of three terms approximations of the HPM is same as the results of Fourth order Runge-Kutta method (RK4).

CHAPTER 6

SUMMARY AND CONCLUSIONS

The application of the Homotopy Perturbation Method (HPM) in addressing the nonlinear system of differential equations that govern the epidemic model appears notably straightforward and yields accurate and reliable results. There is less computation needed in comparison with Fourth-order Runge Kutta method (RK).

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