



Numerical Technique for Solving Non-Linear Deterministic Hepatitis B Model

Pratibha Rani^{1,2*}, Arunodaya Raj Mishra³

¹ Faculty of Business and Communications, INTI International University, Nilai, Negeri Sembilan, Malaysia

² Saveetha School of Engineering, Saveetha Institute of Medical and Technical Sciences (SIMATS), Chennai, Tamil Nadu, India

³ Department of Mathematics, Government College Raigaon, Satna, Madhya Pradesh, India

ARTICLE INFO

Article history:

Received 10 May 2025

Received in revised form 14 June 2025

Accepted 16 June 2025

Available online 17 June 2025

Keywords: Hepatitis B; SEIVR model; Initial value problem; Homotopy Analysis method.

ABSTRACT

Epidemic models classify the process of infectious disease into a series of discrete stages, with transition from susceptible to different states. In this paper, a non-linear epidemic model is proposed to control and understand the dynamics of hepatitis B disease. Epidemic model describes a set of equations where computational tools are used to study the spread of transmissible pathogens in host populations. This model considers the effect of vaccination during the formulation of proposed model. In the work, the numerical solution of the model is further attained through Homotopy Analysis Method, which provides an effective and flexible way for controlling and adjusting the convergence region of the infinite series solution by means of an auxiliary parameter. The outcomes of this paper display that the hepatitis B disease has the inclination over a period of time but it is under control after vaccination.

1. Introduction

In ecology, the sizes of plant and animal populations are generally affected and constrained by foraging, predatism, competition, and inadequate resources [1-4]. In 1979, Anderson and May [5] studied the effects of diseases in laboratory, household and undomesticated populations such as parasites, butterflies, birds, vertebrates, etc. In reality, transmissible diseases have impacted the sizes of human population and chronological past events [6,7]. The parasitic diseases such as babesiosis, filariasis, myiasis, sleeping sickness and so many others concerning viruses, bacteria, arthropods, and protozoans combined with low nutritional diet are the major reasons for discrepancies in the age-dependent existence possibilities in the whole world [8-10]. Consequently, it is important to investigate the epidemic models for understanding the transmission dynamics of infectious diseases with different demographic structures.

Mathematical modelling (MM) is one of the significant branches of mathematical area, which helps us to understand real-life problems and formulate them to the mathematical models and interpret the solutions to the real world [11-14]. MM in biology is the application of mathematical

* pratibha138@gmail.com

<https://doi.org/10.59543/kadsa.v1i.14901>

models to problems arising in biology and life sciences, which acquires a knowledge both in mathematics and biology [15]. Over the past one hundred years, mathematics has been used to understand and forecast the transmission dynamics of diseases related to public health problems [16]. Many epidemiological models [17-20] have been developed in order to study the spread of infectious diseases in constant or variable size populations. These models with constant size population are easier to investigate than the variable size population models and has been more practical for human diseases. The assumption for a fixed size population with births are approximately balanced by the natural deaths (Patwa and Wahl [21]) and a constant size population in which disease rarely causes deaths is reasonable in an endemic modelling (Naik *et al.* [18]), but if there are significant number of disease related deaths which can affect the population size, then it is not reasonable to consider the constant population size.

Hepatitis B virus (HBV) infection is a major public health problem, caused by the Hepadene virus with DNA genome which infects the liver of hominoidea including human [22-25]. This virus is transmitted through contact with blood and other bodily fluids, which could lead to develop viral persistence in the individual in the absence of strong antibody or some immune depression. It can cause a variety of symptoms such as scarring of the liver, liver cancer, gastrointestinal upset and malaise. Transmission of HBV infection are the same as those for the human immunodeficiency virus (HIV), but the hepatitis B virus is 50 to 100 times more transmittable than HIV. In HBV infection, the incubation period of virus is 90 days on average but can vary from about 30 to 180 days. From outside the body, the hepatitis B virus can live for at least seven days and it still causes infection at that time (Nelson *et al.* [26]). The occurrence of HBV infection differs from country to country and depends upon a complex mix of behavioral, environmental and host factors (Villa and Navas [27]).

In this paper, we propose a deterministic model of the form susceptible-exposed-infectious-vaccinated-recovered (SEIVR) type for Hepatitis of type B and find out the numerical solution using Homotopy Analysis Method (HAM). Infectious diseases have an exposed period after transmission of infection from susceptible to probably actual infective and then these infectives can transmit infection. If the exposure period is very short, then the potential infection can be ignored in the model, while in case of relatively long exposure period, the exposed compartment should be included in the model. In case of vaccination before the occurrence of an epidemic, the exposed period and period of treatment should be considered [8-10].

The HAM was firstly proposed by Liao [28,29], which is an analytical approach to acquire the series solutions of several linear and non-linear ordinary/partial differential equations. It is based on the basic homotopy topology theory, which is a fundamental part of topology and differential geometry. This approach provides a freedom to choose initial approximations and auxiliary linear operators, which often helps to transfer the complex non-linear problem into its simpler form. Rani *et al.* [8] applied the HAM for solving deterministic mathematical model for HIV/AIDS disease by considering the stable and unstable stages of the disease. Bakare *et al.* [30] provided the solution of interval-based uncertain susceptible-infectious-recovered (SIR) epidemic model. With the use of HAM, Naik *et al.* [18] studied the stability analysis and obtained the numerical solution of SIR epidemic model with Crowley-Martin type functional response and Holling type-II treatment rate. In a study, Pareek *et al.* [31] used the HAM for solving fractional deterministic Lotka-Volterra model and investigates the convergence region of infinite solution. In a study, Geethamalini *et al.* [32] proposed a set of differential equations for the study of epidemic and further used the HAM for solving the developed epidemic model. As per the authors' information, no one has proposed a deterministic SEIVR model for hepatitis of type B disease with an analytical solution using HAM.

The main contributions of this work are listed as follows:

- A deterministic mathematical model is proposed of the form SEIVR for the hepatitis of type B, which depends on various parameters.
- To attain the analytical solution of SEIVR model, the HAM is applied and the numerical results are presented for different particular cases.

The rest part of this paper is organized as follows: Section 2 proposes a deterministic SEIVR model for hepatitis of type B. The proposed model analyses the effect of different parameters during epidemic period. Section 3 finds the analytical solution of proposed SEIVR model by means of HAM. Further, Section 4 presents the numerical results with respect to different sizes of population and analyses the convergence region of infinite series solution. Lastly, Section 5 concludes the whole work and recommends for further studies.

2. Proposed Model

The non-linear ordinary differential equations for the SEIVR model are presented as follows:

$$\frac{dS}{dt} = -\beta S(t)I(t) + \lambda N(t) - mS(t) - \mu S(t), \quad (1a)$$

$$\frac{dE}{dt} = \beta S(t)I(t) - (\mu + \alpha + (1-\alpha)k)E(t), \quad (1b)$$

$$\frac{dI}{dt} = \beta \sigma V(t)I(t) + (1-\alpha)kE(t) - (\mu + \gamma)I(t), \quad (1c)$$

$$\frac{dV}{dt} = -\beta \sigma V(t)I(t) + \alpha E(t) + mS(t) - \eta V(t) - \mu V(t), \quad (1d)$$

$$\frac{dR}{dt} = \gamma f I(t) + \eta f_v V(t) - \mu R(t), \quad (1e)$$

$$\frac{dN}{dt} = -(1-f)\gamma I(t) - (1-f_v)\eta V(t) + \lambda N(t) - \mu N(t). \quad (1f)$$

with initial conditions $S(0)=S_0$, $E(0)=E_0$, $I(0)=I_0$, $V(0)=V_0$, $R(0)=R_0$ and $N(0)=N_0$.

In this model, $S(t)$, $E(t)$, $I(t)$, $V(t)$ and $R(t)$ denote the numbers of susceptible, exposed, infected, vaccinated and recovered individuals as a function of time t , respectively and $N(t)$ represents the total population size in time t . In this work, we assume that all the parameters are constant.

The parameters used in this model are given as follows:

β	Transmission rate
λ	Birth rate
μ	Mortality rate
k	Leaving rate of the exposed class
η	Removal rate from vaccinated class
γ	Recovery rate
m	Susceptible individuals selected for vaccination per unit time

α	Individuals in the exposed compartment selected for vaccination per unit time
σ	Fraction of vaccinated members who became infected
f_v	Fraction of ηV members leave the vaccinated class at the time t
f	Fraction of γI members leave the infected class at time t and enter into the recovery class
$(1-f)$	Remaining fraction dies due to the HBV infection

3. Solution of the Proposed Deterministic Model by HAM

To solve equation (1) by HAM, we choose the initial approximation $S(0)=S_0, E(0)=E_0, I(0)=I_0, V(0)=V_0, R(0)=R_0, N(0)=N_0$ and the linear operators are

$$L[\psi_1(t; q)] = \frac{d\psi_1(t; q)}{dt}, \quad (2a)$$

$$L[\psi_2(t; q)] = \frac{d\psi_2(t; q)}{dt}, \quad (2b)$$

$$L[\psi_3(t; q)] = \frac{d\psi_3(t; q)}{dt}, \quad (2c)$$

$$L[\psi_4(t; q)] = \frac{d\psi_4(t; q)}{dt}, \quad (2d)$$

$$L[\psi_5(t; q)] = \frac{d\psi_5(t; q)}{dt}, \quad (2e)$$

$$L[\psi_6(t; q)] = \frac{d\psi_6(t; q)}{dt}, \quad (2f)$$

with the property that $L[c_i]=0$, where c_i ($i=1,2,3,4,5,6$) is the integral constant and 'q' is the embedding (or homotopy) parameter. Next, from Eqs (1a)-(1f), we define the equations of nonlinear operator and given as

$$N_1[\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6] = \frac{d\psi_1(t; q)}{dt} + \beta \psi_1(t; q) \psi_3(t; q) - \lambda \psi_6(t; q) + m \psi_1(t; q) + \mu \psi_1(t; q), \quad (3a)$$

$$N_2[\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6] = \frac{d\psi_2(t; q)}{dt} - \beta \psi_1(t; q) \psi_3(t; q) + (\mu + \alpha + (1 - \alpha)k) \psi_2(t; q), \quad (3b)$$

$$N_3[\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6] = \frac{d\psi_3(t; q)}{dt} - \beta \sigma \psi_4(t; q) \psi_3(t; q) - (1 - \alpha)k \psi_2(t; q) + (\mu + \gamma) \psi_3(t; q), \quad (3c)$$

$$N_4[\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6] = \frac{d\psi_4(t; q)}{dt} + \beta \sigma \psi_4(t; q) \psi_3(t; q) - \alpha \psi_2(t; q) - m \psi_1(t; q) + \eta \psi_4(t; q) + \mu \psi_4(t; q), \quad (3d)$$

$$N_5[\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6] = \frac{d\psi_5(t; q)}{dt} - \gamma f \psi_3(t; q) - \eta f_v \psi_4(t; q) + \mu \psi_5(t; q), \quad (3e)$$

$$N_6[\psi_1, \psi_2, \psi_3, \psi_4, \psi_5, \psi_6] = \frac{d\psi_6(t; q)}{dt} + (1 - f) \gamma \psi_3(t; q) + (1 - f_v) \eta \psi_4(t; q) - \lambda \psi_6(t; q) + \mu \psi_6(t; q). \quad (3f)$$

Now, we construct the zeroth-order deformation equations as follows:

$$(1-q)L[\psi_1(t;q) - S_0(t)] = \hbar q N_1[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q), \psi_4(t;q), \psi_5(t;q), \psi_6(t;q)], \quad (4a)$$

$$(1-q)L[\psi_2(t;q) - E_0(t)] = \hbar q N_2[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q), \psi_4(t;q), \psi_5(t;q), \psi_6(t;q)], \quad (4b)$$

$$(1-q)L[\psi_3(t;q) - I_0(t)] = \hbar q N_3[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q), \psi_4(t;q), \psi_5(t;q), \psi_6(t;q)], \quad (4c)$$

$$(1-q)L[\psi_4(t;q) - V_0(t)] = \hbar q N_4[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q), \psi_4(t;q), \psi_5(t;q), \psi_6(t;q)], \quad (4d)$$

$$(1-q)L[\psi_5(t;q) - R_0(t)] = \hbar q N_5[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q), \psi_4(t;q), \psi_5(t;q), \psi_6(t;q)], \quad (4e)$$

$$(1-q)L[\psi_6(t;q) - N_0(t)] = \hbar q N_6[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q), \psi_4(t;q), \psi_5(t;q), \psi_6(t;q)]. \quad (4f)$$

Obviously, when $q=0$ and $q=1$, we have

$$\psi_1(t;0)=S_0(t) \text{ and } \psi_1(t;1)=S(t), \quad (5a)$$

$$\psi_2(t;0)=E_0(t) \text{ and } \psi_2(t;1)=E(t), \quad (5b)$$

$$\psi_3(t;0)=I_0(t) \text{ and } \psi_3(t;1)=I(t), \quad (5c)$$

$$\psi_4(t;0)=V_0(t) \text{ and } \psi_4(t;1)=V(t), \quad (5d)$$

$$\psi_5(t;0)=R_0(t) \text{ and } \psi_5(t;1)=R(t), \quad (5e)$$

$$\psi_6(t;0)=N_0(t) \text{ and } \psi_6(t;1)=N(t). \quad (5f)$$

Therefore, as the embedding parameter q increases from zero to unity, $\psi_i(t;q): i=1, 2, 3, 4, 5, 6$ varies from the initial guess $S_0(t), E_0(t), I_0(t), V_0(t), R_0(t), N_0(t)$ to the exact solution $S(t), E(t), I(t), V(t), R(t), N(t)$, correspondingly. Next, expand the function $\psi_i(t;q): i=1, 2, 3, 4, 5, 6$ using Taylor's series with respect to q , we have

$$\psi_1(t;q)=S_0(t)+\sum_{m=1}^{\infty} S_m(t) q^m, \quad \psi_2(t;q)=E_0(t)+\sum_{m=1}^{\infty} E_m(t) q^m, \quad \psi_3(t;q)=I_0(t)+\sum_{m=1}^{\infty} I_m(t) q^m,$$

$$\psi_4(t;q)=V_0(t)+\sum_{m=1}^{\infty} V_m(t) q^m, \quad \psi_5(t;q)=R_0(t)+\sum_{m=1}^{\infty} R_m(t) q^m, \quad \psi_6(t;q)=N_0(t)+\sum_{m=1}^{\infty} N_m(t) q^m, \quad \text{where}$$

$$S_m(t)=\frac{1}{m!} \frac{d^m \psi_1(t;q)}{dq^m} \text{ at } q=0, \quad E_m(t)=\frac{1}{m!} \frac{d^m \psi_2(t;q)}{dq^m} \text{ at } q=0, \quad I_m(t)=\frac{1}{m!} \frac{d^m \psi_3(t;q)}{dq^m} \text{ at } q=0,$$

$$V_m(t)=\frac{1}{m!} \frac{d^m \psi_4(t;q)}{dq^m} \text{ at } q=0, \quad R_m(t)=\frac{1}{m!} \frac{d^m \psi_5(t;q)}{dq^m} \text{ at } q=0 \text{ and } N_m(t)=\frac{1}{m!} \frac{d^m \psi_6(t;q)}{dq^m} \text{ at } q=0.$$

If the initial approximation, the auxiliary parameter \hbar and auxiliary linear operators are appropriately selected, then the aforesaid series is convergent at $q=1$, so that

$$S(t)=S_0(t)+\sum_{m=1}^{\infty} S_m(t), \quad E(t)=E_0(t)+\sum_{m=1}^{\infty} E_m(t), \quad I(t)=I_0(t)+\sum_{m=1}^{\infty} I_m(t), \quad V(t)=V_0(t)+\sum_{m=1}^{\infty} V_m(t),$$

$$R(t)=R_0(t)+\sum_{m=1}^{\infty} R_m(t) \text{ and } N(t)=N_0(t)+\sum_{m=1}^{\infty} N_m(t),$$
 which is one of the solutions of the original non-linear equation.

Now, we define the vectors $\overrightarrow{S}_n(t)=[S_0(t), S_1(t), \dots, S_n(t)]$, $\overrightarrow{E}_n(t)=[E_0(t), E_1(t), \dots, E_n(t)]$,
 $\overrightarrow{I}_n(t)=[I_0(t), I_1(t), \dots, I_n(t)]$, $\overrightarrow{V}_n(t)=[V_0(t), V_1(t), \dots, V_n(t)]$, $\overrightarrow{R}_n(t)=[R_0(t), R_1(t), \dots, R_n(t)]$ and
 $\overrightarrow{N}_n(t)=[N_0(t), N_1(t), \dots, N_n(t)]$.

Then the m^{th} order deformation equations are

$$L[S_m(t)-\chi_m S_{m-1}(t)]=\hbar N_1[S_{m-1}(t;q), E_{m-1}(t;q), I_{m-1}(t;q), V_{m-1}(t;q), R_{m-1}(t;q), N_{m-1}(t;q)], \quad (6a)$$

$$L[E_m(t)-\chi_m E_{m-1}(t)]=\hbar N_2[S_{m-1}(t;q), E_{m-1}(t;q), I_{m-1}(t;q), V_{m-1}(t;q), R_{m-1}(t;q), N_{m-1}(t;q)], \quad (6b)$$

$$L[I_m(t)-\chi_m I_{m-1}(t)]=\hbar N_3[S_{m-1}(t;q), E_{m-1}(t;q), I_{m-1}(t;q), V_{m-1}(t;q), R_{m-1}(t;q), N_{m-1}(t;q)], \quad (6c)$$

$$L[V_m(t)-\chi_m V_{m-1}(t)]=\hbar N_4[S_{m-1}(t;q), E_{m-1}(t;q), I_{m-1}(t;q), V_{m-1}(t;q), R_{m-1}(t;q), N_{m-1}(t;q)], \quad (6d)$$

$$L[R_m(t)-\chi_m R_{m-1}(t)]=\hbar N_5[S_{m-1}(t;q), E_{m-1}(t;q), I_{m-1}(t;q), V_{m-1}(t;q), R_{m-1}(t;q), N_{m-1}(t;q)], \quad (6e)$$

$$L[N_m(t)-\chi_m N_{m-1}(t)]=\hbar N_6[S_{m-1}(t;q), E_{m-1}(t;q), I_{m-1}(t;q), V_{m-1}(t;q), R_{m-1}(t;q), N_{m-1}(t;q)], \quad (6f)$$

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

Now, the solution of m^{th} order deformation Eqs (6a)-(6f) for $m \geq 1$ becomes

$$S_m(t)=\chi_m S_{m-1}(t)+\hbar \int_0^t N_1[S_{m-1}(\xi;q), E_{m-1}(\xi;q), I_{m-1}(\xi;q), V_{m-1}(\xi;q), R_{m-1}(\xi;q), N_{m-1}(\xi;q)] d\xi, \quad (7a)$$

$$E_m(t)=\chi_m E_{m-1}(t)+\hbar \int_0^t N_2[S_{m-1}(\xi;q), E_{m-1}(\xi;q), I_{m-1}(\xi;q), V_{m-1}(\xi;q), R_{m-1}(\xi;q), N_{m-1}(\xi;q)] d\xi, \quad (7b)$$

$$I_m(t)=\chi_m I_{m-1}(t)+\hbar \int_0^t N_3[S_{m-1}(\xi;q), E_{m-1}(\xi;q), I_{m-1}(\xi;q), V_{m-1}(\xi;q), R_{m-1}(\xi;q), N_{m-1}(\xi;q)] d\xi, \quad (7c)$$

$$V_m(t)=\chi_m V_{m-1}(t)+\hbar \int_0^t N_4[S_{m-1}(\xi;q), E_{m-1}(\xi;q), I_{m-1}(\xi;q), V_{m-1}(\xi;q), R_{m-1}(\xi;q), N_{m-1}(\xi;q)] d\xi, \quad (7d)$$

$$R_m(t)=\chi_m R_{m-1}(t)+\hbar \int_0^t N_5[S_{m-1}(\xi;q), E_{m-1}(\xi;q), I_{m-1}(\xi;q), V_{m-1}(\xi;q), R_{m-1}(\xi;q), N_{m-1}(\xi;q)] d\xi, \quad (7e)$$

$$N_m(t) = \chi_m N_{m-1}(t) + \hbar \int_0^t N_6[S_{m-1}(\xi; q), E_{m-1}(\xi; q), I_{m-1}(\xi; q), V_{m-1}(\xi; q), R_{m-1}(\xi; q), N_{m-1}(\xi; q)] d\xi. \quad (7f)$$

Here, the associated homotopy series solution is presented as $S(t) = \sum_{k=0}^{\infty} S_k(t)$, $E(t) = \sum_{k=0}^{\infty} E_k(t)$,

$$I(t) = \sum_{k=0}^{\infty} I_k(t), \quad V(t) = \sum_{k=0}^{\infty} V_k(t), \quad R(t) = \sum_{k=0}^{\infty} R_k(t), \quad N(t) = \sum_{k=0}^{\infty} N_k(t),$$

which is convergent for any value of

\hbar in the convergence region. Thus, the series solution to the second approximation is obtained and given as follows:

$$\begin{aligned} S(t) = & [S_0 + \hbar((2 + \hbar)(-\lambda N_0 + m S_0 + \beta S_0 I_0 + \mu S_0))t + \hbar^2(m^2 S_0 + \beta(-1 + \alpha)k S_0 E_0 \\ & + \beta^2 S_0 I_0^2 + \beta \gamma S_0 I_0 - \beta \lambda N_0 I_0 - \gamma \lambda I_0 + \gamma \lambda f I_0 - \eta \lambda V_0 + \lambda^2 N_0 \\ & + 3\beta \mu S_0 I_0 - 2\lambda \mu N_0 + \mu^2 S_0 + \lambda^2 N_0 + 3\beta \mu S_0 I_0 - 2\lambda \mu N_0 + \mu^2 S_0 \\ & + m(-\lambda N_0 + 2\beta S_0 I_0 + 2\mu S_0) - \beta^2 \sigma S_0 V_0 I_0 + \eta \lambda f_v V_0) \frac{t^2}{2}], \end{aligned} \quad (8a)$$

$$\begin{aligned} E(t) = & [E_0 - \hbar((2 + \hbar)(\beta S_0 I_0 + k E_0(-1 + \alpha) - (\alpha + \mu) E_0))t \\ & + \hbar^2((k + \alpha - k \alpha + \mu)(-\beta S_0 I_0 + k E_0 + \alpha E_0 - k \alpha E_0 + \mu E_0) \\ & - \beta(k S_0 E_0(-1 + \alpha) + I_0(-\lambda N_0 + m S_0 + \beta S_0 I_0 + \gamma S_0 + 2\mu S_0 - \beta \sigma S_0 V_0))) \frac{t^2}{2}], \end{aligned} \quad (8b)$$

$$\begin{aligned} I(t) = & [I_0 + \hbar((2 + \hbar)(k E_0(-1 + \alpha) + I_0(\gamma + \mu - \beta \sigma V_0)))t \\ & + \hbar^2(k(1 - \alpha) E_0(-k(1 - \alpha) - \alpha - \gamma - 2\mu) + \beta \sigma E_0(\alpha I_0 + k V_0(1 - \alpha)) \\ & + I_0((\gamma + \mu)^2 + \beta k S_0(1 - \alpha) + \beta \sigma(m S_0 - V_0(2\gamma + \eta + 3\mu) + \beta \sigma V_0(V_0 - I_0)))) \frac{t^2}{2}], \end{aligned} \quad (8c)$$

$$\begin{aligned} V(t) = & [V_0 - \hbar((2 + \hbar)(m S_0 + \alpha E_0 - \eta V_0 - \mu V_0 - \beta \sigma V_0 I_0))t \\ & + \hbar^2(-m^2 S_0 + \alpha E_0(k(-1 + \alpha) - \alpha - \eta - 2\mu) + (\eta + \mu)^2 V_0 + \beta \sigma E_0(k V_0(-1 + \alpha) - \alpha I_0) \\ & + \beta I_0(\alpha S_0 + \sigma V_0(\gamma + 2\eta + 3\mu + \beta \sigma(I_0 - V_0))) - m(-\lambda N_0 + S_0(\eta + 2\mu + \beta I_0(1 + \sigma)))) \frac{t^2}{2}], \end{aligned} \quad (8d)$$

$$\begin{aligned} R(t) = & [R_0 - \hbar(2 + \hbar)(\gamma f I_0 - \mu R_0 + \eta f_v V_0)t + \hbar^2(\gamma(1 - \alpha)f k E_0 + \mu^2 R_0 \\ & - \gamma f I_0(\gamma + 2\mu - \beta \sigma V_0) + \eta f_v(m S_0 + \alpha E_0 - \eta V_0 - 2\mu V_0 - \beta \sigma V_0 I_0)) \frac{t^2}{2}], \end{aligned} \quad (8e)$$

$$\begin{aligned}
 N(t) = & [N_0 - \hbar((2 + \hbar)((-1 + f)\gamma I_0 - \eta V_0 + \lambda N_0 - \mu N_0 + \eta f_v V_0))t \\
 & + \hbar^2((1 - f)(\gamma k E_0(-1 + \alpha) + \gamma I_0(\gamma + \mu - \beta \sigma V_0)) \\
 & - \eta(-m S_0 - \alpha E_0 + V_0(\eta + \mu + \beta \sigma I_0))(-1 + f_v) + \mu(\gamma(1 - f)I_0 + \eta V_0 \\
 & - \lambda N_0 + \mu N_0 - \eta f_v V_0) + \lambda(-1 + f)\gamma I_0 - \lambda \eta V_0 + \lambda^2 N_0 - \lambda \mu N_0 + \eta \lambda f_v V_0) \frac{t^2}{2}].
 \end{aligned} \tag{8f}$$

4. Numerical Results

The main aim of this work is to develop SEIVR model for the Hepatitis of type B and obtain the approximate analytical solution by HAM. Moreover, we investigate the impact of auxiliary parameter on the convergence of the series solution by means of \hbar – curve analysis. In the following, we take four cases with the different values of considered parameters and analyse the influence of convergence-control parameter \hbar . For all the cases, we consider the initial approximation as $S_0=50$, $E_0=20$, $I_0=10$, $V_0=10$, $R_0=10$, $N_0=100$.

Case I:

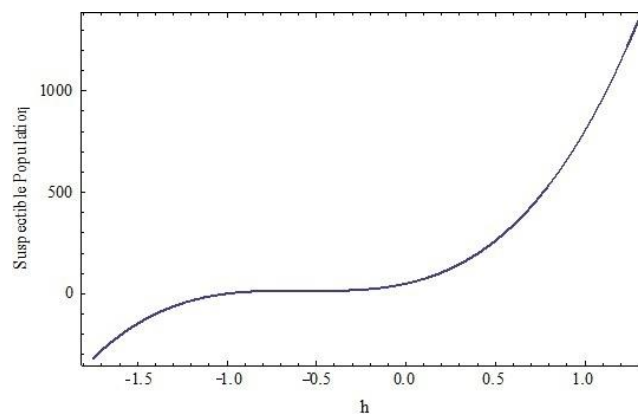


Figure 1. \hbar - curve for the HAM approximate solution for third term approximation of susceptible (S) populations over the parameter \hbar

In this case, Figure 1 presents the region of convergence of the series solution for susceptible population with respect to \hbar and various values of the parameters, which are $f=0.7$, $\gamma=0.6$, $\alpha=0.4$, $\beta=0.3$, $k=0.7$, $\lambda=0.2$, $f_v=0.8$, $m=0.4$, $\sigma=0.01$, $\mu=0.1$ and $\eta=0.7$. It presents the validity of region of convergence of the series solution for susceptible population at $\hbar=-1.75$ to 1.3.

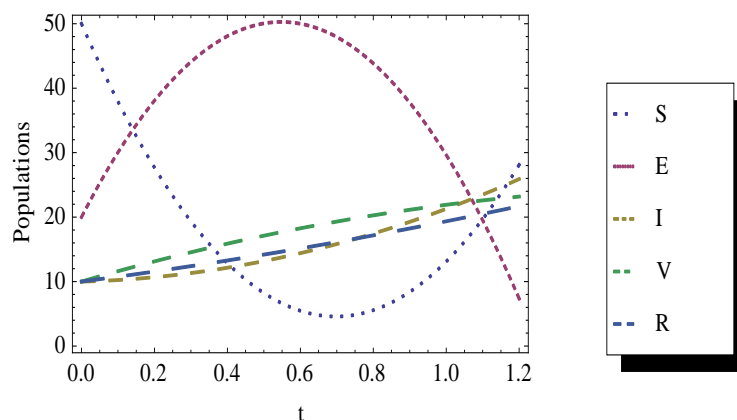


Figure 2. For Case-I, plot of susceptible (S), exposed (E), infected (I), vaccinated (V) and recovered (R) populations with respect to time t

Figure 2 graphically represents the plot of populations (susceptible, exposed, infectious, vaccinated and recovered) over the time ' t ' and different values of parameters $f=0.7$, $\gamma=0.6$, $\alpha=0.4$, $\beta=0.3$, $k=0.7$, $\lambda=0.2$, $f_v=0.8$, $m=0.4$, $\sigma=0.01$, $\mu=0.1$, $\eta=0.7$, $h=-0.6$. From Figure 2, it can be seen that initially the number of susceptible population is quickly decreasing with respect to time t ; afterwards its nature changes rapidly. In a similar way, the exposed population is increasing initially over the time t ; afterwards it is decreasing with respect to time ' t '. Moreover, the number of infectious, recovered and vaccinated group populations are slowly increasing as compared to exposed population with respect to time ' t '. So, there is an epidemic in the population, which is physically justified.

Case II:

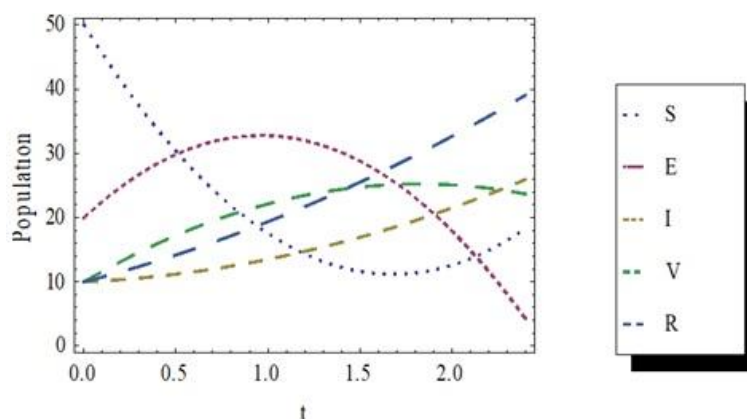


Figure 3. For Case-II, plot of susceptible (S), exposed (E), infected (I), vaccinated (V) and recovered (R) populations with respect to time t

In this Case, we assume that the transmission rate β is less than the Case-I and the other parameters are similar as the previous case. From Figure 3, we can easily see that the number of susceptible populations is initially decreasing slowly than the first case with respect to time t and after that, it behaves opposite. For the exposed population, initially the number of individuals is increasing slowly over the time t and later, it is decreasing. Similarly, the numbers of vaccinated, infectious and recovered populations are increasing slowly as compared to expected population with

respect to time t and different values of the parameters $f=0.7$, $\gamma=0.6$, $\alpha=0.4$, $\beta=0.1$, $k=0.7$, $\lambda=0.2$, $\eta=0.7$, $f_v=0.8$, $m=0.4$, $\sigma=0.01$, $\mu=0.1$ and $\hbar=-0.6$. In this case, initially the infected population are increasing very slowly than the previous case, then there is no epidemic in the population.

Case III:

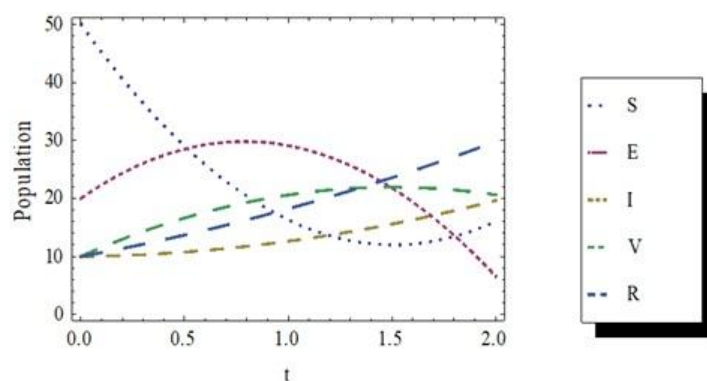


Figure 4. For Case-III, plot of susceptible (S), exposed (E), infected (I), vaccinated (V) and recovered (R) populations with respect to time t

In this case, we assume that the mortality rate and the birth rate are equal and all other parameters are similar to the second case. Figure 4 shows that initially the number of susceptible populations is decreasing with respect to time t and later, it behaves opposite. For the exposed populations, the number of individuals is increasing slowly over the time t and further, it is decreasing with respect to time t . Next, the numbers of infected, vaccinated and recovered populations are increasing slowly with respect to time t and different values of the parameters $f=0.7$, $\gamma=0.6$, $\alpha=0.4$, $\beta=0.1$, $k=0.7$, $\lambda=0.2$, $f_v=0.8$, $m=0.4$, $\sigma=0.01$, $\mu=0.2$, $\eta=0.7$ and $\hbar=-0.6$.

Case IV:

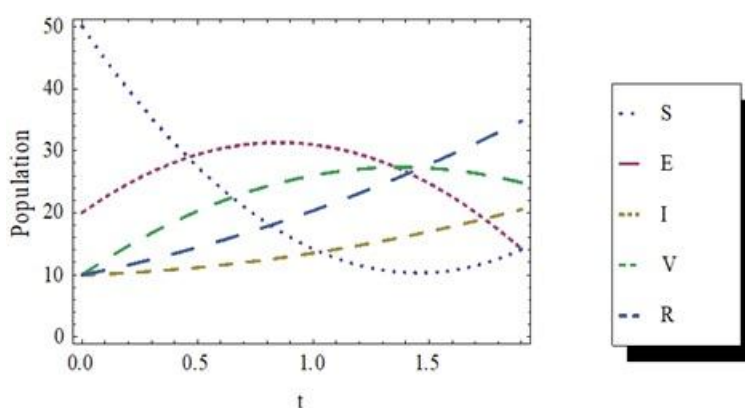


Figure 5. For Case-IV, plot of susceptible (S), exposed (E), infected (I), vaccinated (V) and recovered (R) populations with respect to time t

In this case, we assume that the number of susceptible people who receives the vaccine is more than the previous cases and all other parameters are same as the second case. Figure 5 shows that the number of susceptible populations is decreasing initially with respect to time and later, it behaves

opposite. For the exposed population, the number of individuals is increasing slowly over the time and later, it is decreasing with respect to time. In a similar way, the numbers of infected and vaccinated populations are increasing slowly as compared to exposed population over the time t but the recovered population is increasing rapidly with respect to time t and different values of the parameters $f=0.7$, $\gamma=0.6$, $\alpha=0.4$, $\beta=0.1$, $k=0.7$, $\lambda=0.2$, $f_v=0.8$, $m=0.6$, $\sigma=0.01$, $\mu=0.1$, $\mu=0.1$, $\eta=0.7$ and $\hbar=-0.6$. Since the vaccination parameter for susceptible population is larger than the previous cases, therefore most of the people in the total population are susceptible and vaccinated.

4. Conclusions

This paper has developed a deterministic SEIVR model to study the epidemic of hepatitis B. Further, the homotopy analysis method has applied to acquire the solution of nonlinear equations of the proposed SEIVR model, depending on auxiliary/convergence-control parameter \hbar . In this work, the \hbar -curves are plotted to see the convergence region of the series solution of the problem. For the taken values of \hbar , the obtained solution is applicable for the various values of parameters. In future, we would extend this work by taking fuzzy parameters instead of exact numerical values. Moreover, we can combine the HAM model with machine learning techniques to forecast and analyze the behavior of the proposed model.

Author Contributions

“Conceptualization, P.R. and A.R.M.; methodology, P.R. and A.R.M.; software, P.R. and A.R.M.; validation, P.R. and A.R.M.; formal analysis, P.R. and A.R.M.; investigation, P.R. and A.R.M.; resources, P.R. and A.R.M.; data curation, P.R. and A.R.M.; writing—original draft preparation, P.R. and A.R.M.; writing—review and editing, P.R. and A.R.M.; visualization, P.R. and A.R.M.; All authors have read and agreed to the published version of the manuscript.”

Funding

This research received no external funding.

Data Availability Statement

The data used to support the findings of this study are included in this article. However, the reader may contact the corresponding author for more details on the data.

Conflicts of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

This research was not funded by any grant.

References

- [1] Hethcote, H. W., & Mena-Lorca, J., (1992). Dynamic models of infectious diseases as regulators of population sizes. *Journal of Mathematical Biology*, 30(7), 693-716. doi: 10.1007/BF00173264
- [2] Schmitz, O. (2017). Predator and prey functional traits: understanding the adaptive machinery driving predator–prey interactions. *F1000 Research*, 6, 1-10. doi: 10.12688/f1000research.11813.1
- [3] Sheriff, M. J., Peacor, S. D., Hawlena, D., & Thaker, M. (2020). Non-consumptive predator effects on prey population size: A dearth of evidence. *Journal of Animal Ecology*, 89(6), 1302-1316. <https://doi.org/10.1111/1365-2656.13213>
- [4] Boucekkine, R., Chakraborty, S., Goenka, A., & Liu, L. (2024). Economic epidemiological modelling: A progress report. *Journal of Mathematical Economics*, 113, 103011; <https://doi.org/10.1016/j.jmateco.2024.103011>
- [5] Anderson, R.M., & May, R.M., (1979). Population biology of infectious diseases I. *Nature*, 280, 361-367. <https://doi.org/10.1038/280361a0>
- [6] Fu, H., & Zhu, C. (2024). The impact of population influx on infectious diseases – from the mediating effect of polluted air transmission. *Frontiers in Public Health*, 12, 1344306; doi: 10.3389/fpubh.2024.1344306
- [7] Jesse, J. A., Agnew, M. V., Arai, K., Armstrong, C. T., Hood, S. M., Kachmar, M. L., Long, J. T., McCarty, A. J., Ross, M. O., Rubalcava, K. D., Shaner, J., Tanaka, S., Wood, L., Schott, E. J., & Wilberg, M. J. (2021). Effects of infectious diseases on population dynamics of marine organisms in Chesapeake Bay. *Estuaries and Coasts*, 44, 2334-2349. <https://doi.org/10.1007/s12237-021-00915-4>
- [8] Rani, P., Jain, D., & Saxena, V. P. (2016). Approximate analytical solution with stability analysis of HIV/AIDS model. *Cogent Mathematics*, 3(1), 1-14. <https://doi.org/10.1080/23311835.2016.1206692>
- [9] Rani, P., Jain, D., & Saxena, V. P. (2017). Stability Analysis of HIV/AIDS Transmission with Treatment and Role of Female Sex Workers. *International Journal of Nonlinear Sciences and Numerical Simulation*, 18(6), 457-467. <https://doi.org/10.1515/ijnsns-2015-0147>
- [10] Shah, N. H., & Sheoran, N. (2022). Homotopy perturbation method for Pneumonia–HIV co-infection. *Foundations*, 2(4), 1101-1113. <https://doi.org/10.3390/foundations2040072>
- [11] Badi, I., Bayane Bouraima, M., & Zonon, B. I. P. (2025). A multi-criteria decision-making model for prioritizing the causes of inflation in Libya using the best-worst method and analytic hierarchy process. *Knowledge and Decision Systems with Applications*, 1, 165–176. <https://doi.org/10.59543/kadsa.v1i.14259>
- [12] Dağıstanlı, H. A. (2025). Weapon System Selection for Capability-Based Defense Planning using Lanchester Models integrated with Fuzzy MCDM in Computer Assisted Military Experiment. *Knowledge and Decision Systems with Applications*, 1, 11–23. <https://doi.org/10.59543/kadsa.v1i.13601>
- [13] Gokasar, I., & Aytekin, K. (2025). Modelling and evaluation of work zone queues: Istanbul case study. *Journal of Soft Computing and Decision Analytics*, 3(1), 33-49. <https://doi.org/10.31181/jsda31202552>
- [14] Seker, S., & Ergün, M. T. (2023). Investigation the effect of Covid-19 pandemic in the sales for online education using machine learning methods. *Journal of Soft Computing and Decision Analytics*, 1(1), 273-282. <https://doi.org/10.31181/jsda11202322>
- [15] Divya, B., & Kavitha (2020). A review on mathematical modelling in biology and medicine. *Advances in Mathematics Scientific Journal*, 9(8), 5869-5879. DOI:10.37418/amsj.9.8.54
- [16] Yavuz, M., & Usta, F. (2023). Importance of modelling and simulation in biophysical applications. *AIMS Biophysics*, 10(3), 258-262. doi: 10.3934/biophy.2023017
- [17] Hethcote, H. W., Levin, S.A., & Liu, W.M., (1987). Dynamical behaviour of epidemiological models with nonlinear incidence rates. *Journal of Mathematical Biology*, 25, 359-380. <https://doi.org/10.1007/BF00277162>
- [18] Naik, P. A., Zu, J., & Ghoreishi, M. (2020). Stability analysis and approximate solution of sir epidemic model with Crowley-Martin type functional response and Holling type-II treatment rate by using homotopy analysis method. *Journal of Applied Analysis & Computation*, 10(4), 1482-1515. doi: 10.11948/20190239
- [19] Chakir, Y. (2023). Global approximate solution of SIR epidemic model with constant vaccination strategy. *Chaos, Solitons & Fractals*, 169, 113323. <https://doi.org/10.1016/j.chaos.2023.113323>
- [20] Duru, E. C., & Mbah, G. C. E. (2025). Approximate solution for a malaria model using the homotopy analysis method. *Biometrical letters*, <https://doi.org/10.2478/bile-2025-0001>.
- [21] Patwa, Z., & Wahl L.M. (2008). The fixation probability of beneficial mutations. *Journal of The Royal Society Interface*, 5(28), 1279-1289. doi: 10.1098/rsif.2008.0248
- [22] Wodajo, F. A., Gebbru, D.M., & Alemneh, H. T. (2023). Mathematical model analysis of effective intervention strategies on transmission dynamics of hepatitis B virus. *Scientific Reports*, 13, 1-21. <https://doi.org/10.1038/s41598-023-35815-z>
- [23] Ciupe, S.M., Dahari, H., & Ploss, A. (2024). Mathematical models of early hepatitis B virus dynamics in humanized mice. *Bulletin of Mathematical Biology*, 86, 1-20. <https://doi.org/10.1007/s11538-024-01284-2>

- [24] Xu, C., Wang, Y., Cheng, K., Yang, X., Wang, X., Guo, S., Liu, M., & Liu, X. (2023) A mathematical model to study the potential hepatitis B virus infections and effects of vaccination strategies in China. *Vaccines*, 11(10), 1-20. doi: 10.3390/vaccines11101530
- [25] Kim, K. (2023). PPlases Par14/Par17 affect HBV replication in multiple ways. *Viruses*, 15(2), 1-12. <https://doi.org/10.3390/v15020457>
- [26] Nelson, N. P., Easterbrook, P. J., & McMahon, B. J. (2016). Epidemiology of hepatitis B virus infection and impact of vaccination on disease. *Clinical Liver Disease*, 20(4), 607-628. doi: 10.1016/j.cld.2016.06.006.
- [27] Villa, D. D. F., & Navas, M.-C. (2023). Vertical transmission of hepatitis B virus—An update. *Microorganisms*, 11(5), 1-20. <https://doi.org/10.3390/microorganisms11051140>
- [28] Liao, S. J. (1992). *The proposed homotopy analysis technique for the solution of nonlinear problems*. Ph.D. Thesis, Shanghai Jiao Tong University.
- [29] Liao, S.J., (2004). *Beyond Perturbation: Introduction to homotopy analysis method*. CRC Press/ Chapman and Hall, Boca Raton.
- [30] Bakare, E. A., Chakraverty, S., & Potucek, R. (2021). Numerical solution of an interval-based uncertain SIR (Susceptible–Infected–Recovered) epidemic model by homotopy analysis method. *Axioms*, 10(2), 1-19. <https://doi.org/10.3390/axioms10020114>
- [31] Pareek, N., Gupta, A., Suthar, D. L., Agarwal, G., & Nisar, K. S. (2022). Homotopy analysis approach to study the dynamics of fractional deterministic Lotka-Volterra model. *Arab Journal of Basic and Applied Sciences*, 29(1), 121–128. <https://doi.org/10.1080/25765299.2022.2071027>
- [32] Geethamalini, S., Sangeetha, S., & Venkataraman, P. (2025). Homotopy Analysis Method for the Approximate Solution of the SIRC Epidemic Model. *Communications on Applied Nonlinear Analysis*, 32(6s), 427-438. <https://doi.org/10.52783/cana.v32.3307>