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EVALUATING VACCINATION AND TREATMENT IMPACTS ON CRITICAL AND LONG-TERM HEPATITIS B VIRUS INFECTIONS

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Abstract Hepatitis B is a hazardous infectious disease caused by the hepatitis B virus (HBV), posing significant global health challenges. This study introduces a modified deterministic mathematical model to evaluate the impact of vaccination campaigns and hospitalization efforts in controlling the disease's spread. By focusing on the dynamics of susceptible (S), exposed (E), chronic (C), treated (T), vaccinated (V), infected (K), acute (A), and recovered (R) populations, this model extends prior frameworks by integrating the critical roles of vaccination and treatment. We analyze the uniform boundedness of the system, perform equilibrium analysis, and determine the basic reproduction number ($\mathcal{R}_0$) using the next-generation matrix method. The homotopy perturbation method (HPM) is applied to explore analytical solutions. Numerical simulations demonstrate that increasing vaccination capacity significantly curtails infection rates, underscoring the importance of robust healthcare infrastructure in mitigating HBV outbreaks. This study offers valuable insights into designing effective HBV control and prevention strategies.

1 Introduction

Hepatitis B is a critical liver infection, infected by the hepatitis B virus. In some people, it is a short-term disease, and the cure is in six months [1, 2], but some are severe infections that last long, in more than six months, or die. Chronic hepatitis B increases the risk of liver cancer or liver failure, whereas acute hepatitis B is a short-living virus, in which patients may recover in one month. Major signs or symptoms we can see in the patient of hepatitis B are dark urine, weakness or fatigue, joint pain, fever, loss of appetite, nausea, vomiting, jaundice, etc. Hepatitis B spreads through mother to newborn, accidental needle sticks, sexual contact, and sharing of needles, but it is not spread through the following: hugging, kissing, breastfeeding, shaking hands, sneezing, or in any public activities [3]. The range of hepatitis B virus typically changes in the range of 1 to 6 months, or an average of 3 months [4, 5, 6]. In the hepatitis B virus, vaccination provides protection against infection, but it works only for a short period of time and becomes a high risk of reinfection. When people move from one place to another, then the risk of infection increases. In clinical tests, it is impossible to differentiate the hepatitis B virus from other viruses [7]. Therefore, a laboratory test is needed to confirm the result. Numerous blood tests can be used to recognize and monitor hepatitis B sick persons. In subsequent exposure to the bodily fluids of a sick person with hepatitis B, to diagnose for six months by a five-item test are the following: hepatitis B surface antigen, hepatitis B surface antibody, hepatitis B e-antigen, hepatitis B e-antibody, and hepatitis B core antibody.

The report of 2024 WHO [8] estimates that 254 million people were living with chronic hepatitis B infection in 2022, with 1.2 million new cases each year [9] whereas 1.1 million deaths by cirrhosis and hepatocellular carcinoma (primary liver cancer) in 2022. Chronic hepatitis B can be treated with medications, but there is no specific treatment for acute hepatitis B. Even though some vaccinations and medications can control this virus, sometimes it becomes a big

challenge. This is especially true in cases where the virus develops resistance to available treatments, making it more challenging to manage. Additionally, the symptoms and complications associated with hepatitis B, such as liver damage, cirrhosis, and liver cancer, further complicate the treatment process.

Numerous researchers have utilized mathematical models to investigate the transmission dynamics of the hepatitis B virus and assess the impact of preventive measures on its spread. For instance, Goldstein et al. [10] proposed a mathematical model to estimate the global hepatitis B disease burden and the impact of vaccination strategies. Li et al. [11] developed a mathematical model to analyze the pharmacological blockade of HBV transmission between mothers and infants during pregnancy. Khan et al. [12] studied the dynamics of acute and chronic hepatitis B with an optimal control framework. Recent advancements in fractional calculus and numerical methods have further enriched the study of hepatitis B modeling. Din et al. [13] conducted a stability and qualitative analysis using fixed-point theory in the Atangana-Baleanu Caputo fractional-order derivative framework. Yavuz et al. [14] performed numerical simulations employing the Adams-Bashforth numerical scheme, demonstrating the significant influence of the fractional derivative order on the dynamics of their constructed hepatitis B model. Mustapha et al. [15] developed an age-structured mathematical model incorporating backward bifurcation, fitted to real data from South Africa using the non-linear least squares curve-fitting method. Additionally, Yavuz et al. [16] explored a fractional-order differential equation system with memory effects, employing the strange processes of the model to reveal how past infection events influence future spread dynamics of hepatitis B.

Public health initiatives continue to prioritize prevention through vaccination, early detection, and innovative research aimed at developing more effective treatments. Despite these efforts, hepatitis B remains a significant global health concern, underscoring the ongoing need for awareness, education, and advancements in medical science. In response to this challenge, this study introduces a modified model that primarily examines the impact of vaccination and hospitalization. Specifically, we explore the dynamics of vaccinated individuals transitioning into the exposed category and the recovery of acute and chronic populations through treatment. This dual focus on vaccination and treatment distinguishes our work from prior studies, highlighting their combined importance in controlling the spread of the disease. By uniquely integrating these two critical components, our study provides a fresh perspective on reducing the transmission and impact of the hepatitis B virus while addressing essential gaps in existing research. The model has been analytically simulated using the Homotopy Perturbation Method (HPM) [17], with MATLAB employed to analyze event dynamics, leading to the identification of effective strategies for controlling hepatitis B virus transmission through vaccination.

This paper is organized as follows: In Section 2, we provide a detailed mathematical formulation of Hepatitis B. In Section 3, we analyze the stability of the Hepatitis B virus, along with the positivity and uniformity of the system. Section 4 presents the existence and uniqueness of the proposed model. Section 5 discusses the disease-free equilibrium points, the basic reproduction number, and the stability analysis of the disease-free equilibrium points of Hepatitis B. Section 6 uses the HPM to determine the approximate analytical expressions for each compartment in the model. Also, in Section 7, we briefly describe the numerical analysis and graphical discussion, followed by the conclusion in Section 8.

2 Model Formulation

We proposed a compartmental model to explain the dynamics of the hepatitis B Virus. The human population has been split into eight compartments namely susceptible individuals S, vaccinated individuals V, exposed individuals E, probable infectious hepatitis B infections K, long-term hepatitis B infection C, critical hepatitis B infection A, treated member T, recovered or removed R.

The meaning of each parameter symbol is as follows: Λ is the recurring rate of the susceptible population. π is the proportion of a fraction of herds susceptible and $(1-\pi)$ is the proportion of recruited people who are vaccinated. σ is the rate of the recovered population again becoming susceptible. α is the vaccination rate. β is the foundation transmission rate. ω is the transmission rate of the vaccinated population to the exposed population. ϵ is the transfer rate of A to C.

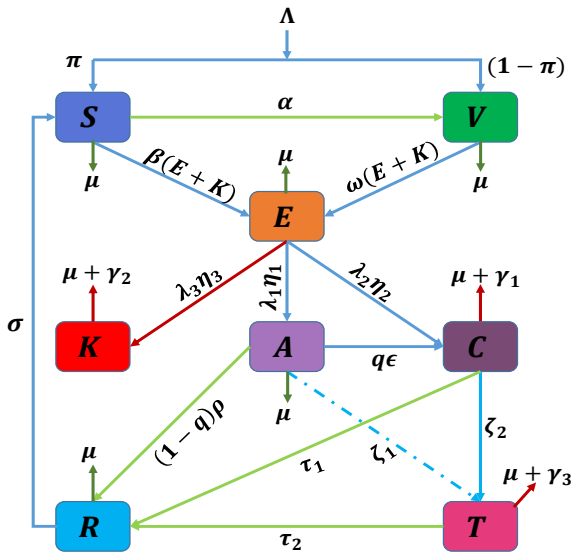

Figure 1. Schematic flow diagram of hepatitis B model.

λ_1, λ_2, and λ_3 are the transition rate of exposed population in A, C, and K respectively. η_1, η_2, and η_3 are the transfer rate of exposed population in A, C, and K respectively. γ_1, γ_2, and γ_3 are individual death rate of C, K, and T respectively. q and ρ are the ratios of people with acute infections who become chronically infected and recover from the disease respectively. ζ_1 and ζ_2 is the transfer rate of A to T and C to T respectively. τ_1 and τ_2 is the transfer rate of C to R and T to R respectively. μ is the natural death rate.

$$
\begin{aligned}
\frac{dS}{dt} &= \Lambda\pi + \sigma R - (\mu + \alpha)S - \beta S(E + K),\\
\frac{dV}{dt} &= \Lambda(1 - \pi) + \alpha S - \mu V - \omega V(E + K),\\
\frac{dE}{dt} &= \beta S(E + K) + \omega V(E + K) - (\mu + \lambda_1\eta_1 + \lambda_2\eta_2 + \lambda_3\eta_3)E,\\
\frac{dK}{dt} &= \lambda_3\eta_3 E - (\mu + \gamma_2)K,\\
\frac{dC}{dt} &= \lambda_2\eta_2 E - (\mu + \gamma_1 + \zeta_2 + \tau_1)C + q\epsilon A,\\
\frac{dA}{dt} &= \lambda_1\eta_1 E - (q\epsilon + (1 - q)\rho + \mu + \zeta_1)A,\\
\frac{dT}{dt} &= \zeta_2 C + \zeta_1 A - (\tau_2 + \mu + \gamma_3)T,\\
\frac{dR}{dt} &= \tau_1 C + (1 - q)\rho A + \tau_2 T - (\mu + \sigma)R,
\end{aligned}
\tag{2.1}
$$

with initial conditions:

$$S(0) = S_0, V(0) = V_0, E(0) = E_0, K(0) = K_0, C(0) = C_0, A(0) = A_0, T(0) = T_0, R(0) = R_0.$$

3 Qualitative Analysis of the Model

This section explores important theorems addressing the fundamental concepts of positivity and boundedness. By examining these concepts in detail, we aim to understand them comprehen-

sively. How are these properties established and validated within the HBV model's framework, ensuring the model's reliability and robustness in predicting disease dynamics?

3.1 Positivity of the System

Theorem 3.1. *Let the initial population be* $\{S_0, V_0, E_0, K_0, C_0, A_0, T_0, R_0\}$ *non-negative, then the solution set* $\{S, V, E, K, C, A, T, R\}$ *of system* (2.1) *is non-negative for all* $t > 0$.

Proof. From the first equation of the system (2.1),

$$\frac{dS}{dt} = \Lambda\pi + \sigma R - (\mu + \alpha)S - \beta S(E + K) \geq -(\mu + \alpha)S.$$

This implies

$$\frac{dS}{dt} \geq -(\mu + \alpha)S. \tag{3.1}$$

By separation of variable, (3.1) we integrated it and obtain

$$lnS \geq -(\mu + \alpha)t + M,$$

where M is a constant of integration, applying the initial conditions $S(0) = S_0$, then we obtain

$$S \geq S_0 e^{-(\mu+\alpha)t} \geq 0.$$

Similarly, it can be shown that all the system's equations (2.1) are all positive for all $t > 0$. Thus, all equation solutions are non-negative for all $t > 0$. □

3.2 Uniformly Boundedness of the System

In this section, we establish the uniform limit of our model for solutions by defining the positivity invariance of the system concerning its initial values.

Theorem 3.2. *The closed set,* $\Omega = \left\{X \in \mathbb{R}^8_+ : 0 \leq X(S, V, E, K, C, A, T, R) \leq \frac{\Lambda}{\mu}\right\}$ *is positively invariant.*

Proof. We aim to show that $\mathbb{R}^8_+$ is positively invariant, which means that all solutions of system (2.1) started in Ω do not leave Ω. So, we start by taking the total population of humans:

$$N(t) = S(t) + V(t) + E(t) + K(t) + C(t) + A(t) + T(t) + R(t),$$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dE}{dt} + \frac{dK}{dt} + \frac{dC}{dt} + \frac{dA}{dt} + \frac{dT}{dt} + \frac{dR}{dt},$$

$$\frac{dN}{dt} = \Lambda - \mu N - \gamma_1 C - \gamma_2 K - \gamma_3 T,$$

$$\frac{dN}{dt} + \mu N \leq \Lambda,$$

solving by integrating factor method yield,

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right) \exp(-\mu t),$$

as $t \longrightarrow \infty$,

$$N(t) \leq \frac{\Lambda}{\mu}.$$

Hence, Ω is positively invariant. □

4 Existence and Uniqueness of Solution of the Model

In this section, we establish the necessary conditions for the existence and uniqueness of a solution to our model. To achieve this, we apply Picard's theorem [18, 19].

Theorem 4.1. ***Picard's Theorem*** *Suppose*

$$y' = f(t, y), \;\; y(t_0) = y_0. \tag{4.1}$$

Consider a given system of ordinary differential equations (2.1), and suppose $f(t, x)$ is continuous and satisfies a Lipschitz condition in the closed and bounded domain $\|x - x_0\| \leq \ell_1$, $\|t - t_0\| \leq \ell_2$. Let $\|f(t, x)\| \leq \mathcal{M}$ within this domain.

Then the IVP (4.1) has a unique solution in the interval $\|t-t_0\| \leq h$, where $h = \min\left\{\ell_2, \frac{\ell_1}{\mathcal{M}}\right\}$.

Proof. Consider our system of equations (2.1). Let

$$\begin{aligned} x &= [S\ V\ E\ K\ C\ A\ T\ R]^{Tr}, \\ f(t, x) &= [f_1(t, x)\ f_2(t, x)\ f_3(t, x)\ f_4(t, x)\ f_5(t, x)\ f_6(t, x)\ f_7(t, x)\ f_8(t, x)]^{Tr}, \end{aligned} \tag{4.2}$$

where

$$\begin{aligned} f_1(t, x) &= \Lambda\pi + \sigma R - (\mu + \alpha)S - \beta S(E + K), \\ f_2(t, x) &= \Lambda(1 - \pi) + \alpha S - \mu V - \omega V(E + K), \\ f_3(t, x) &= \beta S(E + K) + \omega V(E + K) - (\mu + \lambda_1\eta_1 + \lambda_2\eta_2 + \lambda_3\eta_3)E, \\ f_4(t, x) &= \lambda_3\eta_3 E - (\mu + \gamma_2)K, \\ f_5(t, x) &= \lambda_2\eta_2 E - (\mu + \gamma_1 + \zeta_2 + \tau_1)C + q\epsilon A, \\ f_6(t, x) &= \lambda_1\eta_1 E - (q\epsilon + (1 - q)\rho + \mu + \zeta_1)A, \\ f_7(t, x) &= \zeta_2 C + \zeta_1 A - (\tau_2 + \mu + \gamma_3)T, \\ f_8(t, x) &= \tau_1 C + (1 - q)\rho A + \tau_2 T - (\mu + \sigma)R. \end{aligned}$$

The model equations are now in the form (4.1). Suppose the function $f(t, x)$ is defined and continuous in x, and satisfies a Lipschitz condition in the closed and bounded region.

Let $D := \{x = (S, V, E, K, C, A, T, R) : S, V, E, K, C, A, T, R \leq 1\}$.

Define $\|x - x_0\| \leq \ell_1$, $\|t\| \leq \ell_2$, $t_0 = 0$, $x_0 = (S_0, V_0, E_0, K_0, C_0, A_0, T_0, R_0)$.

We shall use Picard's theorem to prove that the solution of (2.1) exists and is unique by demonstrating the following:

(i) f is continuous,

(ii) f satisfies Lipschitz condition,

(iii) $|f| \leq \mathcal{M}$.

$f(t, x)$ is continuous because each component of f_i, $\{i = 1, 2, \cdots, 8\}$, is a continuous function of the variable $x = [S\ V\ E\ K\ C\ A\ T\ R]^{Tr}$.

To establish the Lipschitz condition for the model equations,

Let $y = [S_*\ V_*\ E_*\ K_*\ C_*\ A_*\ T_*\ R_*]^{Tr}$

$f(t, y) = [f_1(t, y)\ f_2(t, y)\ f_3(t, y)\ f_4(t, y)\ f_5(t, y)\ f_6(t, y)\ f_7(t, y)\ f_8(t, y)]^{Tr}$.

Noting that $S,\ V,\ E,\ K,\ C,\ A,\ T,\ R \leq 1$.

We have

$$\begin{aligned} |f_1(x) - f_1(y)| &= |\sigma(R - R_*) - (\mu + \alpha)(S - S_*) - \beta\{S(E - E_*) + E_*(S - S_*)\} \\ &\quad - \beta\{S(K - K_*) + K_*(S - S_*)\}| \leq \sigma|R - R_*| + |\mu + \alpha|S - S_*| \\ &\quad + \beta|S||E - E_*| + \beta|E_*||S - S_*| + \beta|S||K - K_*| + \beta|K_*||S - S_*| \\ &\leq \mathcal{U}_{11}|S - S_*| + \mathcal{U}_{12}|V - V_*| + \mathcal{U}_{13}|E - E_*| + \mathcal{U}_{14}|K - K_*| + \mathcal{U}_{15}|C - C_*| \\ &\quad + \mathcal{U}_{16}|A - A_*| + \mathcal{U}_{17}|T - T_*| + \mathcal{U}_{18}|R - R_*| \end{aligned}$$

$$|f_1(x) - f_1(y)| \leq \mathcal{U}_1|x - y|,$$

where $\mathcal{U}_1 = \max\{\mathcal{U}_{11},\mathcal{U}_{12},\mathcal{U}_{13},\mathcal{U}_{14},\mathcal{U}_{15},\mathcal{U}_{16},\mathcal{U}_{17},\mathcal{U}_{18}\}$ and $\mathcal{U}_{11} = \mu+\alpha+2\beta$, $\mathcal{U}_{12} = 0$, $\mathcal{U}_{13} = \beta$, $\mathcal{U}_{14} = \beta$, $\mathcal{U}_{15} = 0$, $\mathcal{U}_{16} = 0$, $\mathcal{U}_{17} = 0$, $\mathcal{U}_{18} = \sigma$ are constants that depend on the model's parameters.

Similarly,

$$|f_2(x) - f_2(y)| \le \mathcal{U}_{21}|S - S_*| + \mathcal{U}_{22}|V - V_*| + \mathcal{U}_{23}|E - E_*| + \mathcal{U}_{24}|K - K_*|,$$
$$|f_2(x) - f_2(y)| \le \mathcal{U}_2|x - y|,$$

where $\mathcal{U}_2 = \max\{\mathcal{U}_{21},\mathcal{U}_{22},\mathcal{U}_{23},\mathcal{U}_{24},\mathcal{U}_{25},\mathcal{U}_{26},\mathcal{U}_{27},\mathcal{U}_{28}\}$ and $\mathcal{U}_{21} = \alpha$, $\mathcal{U}_{22} = \mu+2\omega$, $\mathcal{U}_{23} = \omega$, $\mathcal{U}_{24} = \omega$, $\mathcal{U}_{25} = 0 = \mathcal{U}_{26} = \mathcal{U}_{27} = \mathcal{U}_{28}$.

$$|f_3(x) - f_3(y)| \le \mathcal{U}_{31}|S - S_*| + \mathcal{U}_{32}|V - V_*| + \mathcal{U}_{33}|E - E_*| + \mathcal{U}_{34}|K - K_*|,$$
$$|f_3(x) - f_3(y)| \le \mathcal{U}_3|x - y|,$$

where $\mathcal{U}_3 = \max\{\mathcal{U}_{31},\mathcal{U}_{32},\mathcal{U}_{33},\mathcal{U}_{34},\mathcal{U}_{35},\mathcal{U}_{36},\mathcal{U}_{37},\mathcal{U}_{38}\}$ and $\mathcal{U}_{31} = 2\beta\,\mathcal{U}_{32} = 2\omega$, $\mathcal{U}_{33} = \omega+\beta+\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3$, $\mathcal{U}_{34} = \omega+\beta$, $\mathcal{U}_{35} = 0 = \mathcal{U}_{36} = \mathcal{U}_{37} = \mathcal{U}_{38}$.

$$|f_4(x) - f_4(y)| \le \mathcal{U}_{43}|E - E_*| + \mathcal{U}_{44}|K - K_*|,$$
$$|f_4(x) - f_4(y)| \le \mathcal{U}_4|x - y|,$$

where $\mathcal{U}_4 = \max\{\mathcal{U}_{41},\mathcal{U}_{42},\mathcal{U}_{43},\mathcal{U}_{44},\mathcal{U}_{45},\mathcal{U}_{46},\mathcal{U}_{47},\mathcal{U}_{48}\}$ and $\mathcal{U}_{41} = 0 = \mathcal{U}_{42} = \mathcal{U}_{45} = \mathcal{U}_{46} = \mathcal{U}_{47} = \mathcal{U}_{48}$, $\mathcal{U}_{43} = \lambda_3\eta_3$, $\mathcal{U}_{44} = \mu+\gamma_2$.

$$|f_5(x) - f_5(y)| \le \mathcal{U}_{53}|E - E_*| + \mathcal{U}_{55}|C - C_*| + \mathcal{U}_{56}|A - A_*|,$$
$$|f_5(x) - f_5(y)| \le \mathcal{U}_5|x - y|,$$

where $\mathcal{U}_5 = \max\{\mathcal{U}_{51},\mathcal{U}_{52},\mathcal{U}_{53},\mathcal{U}_{54},\mathcal{U}_{55},\mathcal{U}_{56},\mathcal{U}_{57},\mathcal{U}_{58}\}$ and $\mathcal{U}_{51} = 0 = \mathcal{U}_{52} = \mathcal{U}_{57} = \mathcal{U}_{58}$, $\mathcal{U}_{53} = \lambda_2\eta_2$, $\mathcal{U}_{55} = \mu+\gamma_1+\zeta_2+\tau_1$, $\mathcal{U}_{56} = q\epsilon$.

$$|f_6(x) - f_6(y)| \le \mathcal{U}_{63}|E - E_*| + \mathcal{U}_{66}|A - A_*|,$$
$$|f_6(x) - f_6(y)| \le \mathcal{U}_6|x - y|,$$

where $\mathcal{U}_6 = \max\{\mathcal{U}_{61},\mathcal{U}_{62},\mathcal{U}_{63},\mathcal{U}_{64},\mathcal{U}_{65},\mathcal{U}_{66},\mathcal{U}_{67},\mathcal{U}_{68}\}$ and $\mathcal{U}_{61} = 0 = \mathcal{U}_{62} = \mathcal{U}_{64} = \mathcal{U}_{65} = \mathcal{U}_{67} = \mathcal{U}_{68}$, $\mathcal{U}_{63} = \lambda_1\eta_1$, $\mathcal{U}_{66} = q\epsilon+(1-q)\rho+\mu+\zeta_1$.

$$|f_7(x) - f_7(y)| \le \mathcal{U}_{75}|C - C_*| + \mathcal{U}_{76}|A - A_*| + \mathcal{U}_{77}|T - T_*|,$$
$$|f_7(x) - f_7(y)| \le \mathcal{U}_7|x - y|,$$

where $\mathcal{U}_7 = \max\{\mathcal{U}_{71},\mathcal{U}_{72},\mathcal{U}_{73},\mathcal{U}_{74},\mathcal{U}_{75},\mathcal{U}_{76},\mathcal{U}_{77},\mathcal{U}_{78}\}$ and $\mathcal{U}_{71} = 0 = \mathcal{U}_{72} = \mathcal{U}_{73} = \mathcal{U}_{74} = \mathcal{U}_{78}$, $\mathcal{U}_{75} = \zeta_2$, $\mathcal{U}_{76} = \zeta_1$, $\mathcal{U}_{77} = \tau_2+\mu+\gamma_3$.

$$|f_8(x) - f_8(y)| \le \mathcal{U}_{85}|C - C_*| + \mathcal{U}_{86}|A - A_*| + \mathcal{U}_{87}|T - T_*| + \mathcal{U}_{88}|R - R_*|,$$
$$|f_8(x) - f_8(y)| \le \mathcal{U}_8|x - y|,$$

where $\mathcal{U}_8 = \max\{\mathcal{U}_{81},\mathcal{U}_{82},\mathcal{U}_{83},\mathcal{U}_{84},\mathcal{U}_{85},\mathcal{U}_{86},\mathcal{U}_{87},\mathcal{U}_{88}\}$ and $\mathcal{U}_{81} = 0 = \mathcal{U}_{82} = \mathcal{U}_{83} = \mathcal{U}_{84}$, $\mathcal{U}_{85} = \tau_1$, $\mathcal{U}_{86} = (1-q)\rho$, $\mathcal{U}_{87} = \tau_2$, $\mathcal{U}_{88} = \mu+\sigma$.

Therefore $|f(x)| = \max\{\mathcal{U}_1,\mathcal{U}_2,\mathcal{U}_3,\mathcal{U}_4,\mathcal{U}_5,\mathcal{U}_6,\mathcal{U}_7,\mathcal{U}_8\} \le \mathcal{M}$.

Thus, there exists a unique solution for the IVP (4.1) in the domain, $|x - x_0| \le \ell_1$ and $|t| \le h$, where $h = \min\left\{\ell_2, \frac{\ell_1}{\mathcal{M}}\right\}$.

This complete the proof. □

5 Analysis of Equilibrium Points, Stability, and Basic Reproduction Number

In this section, the disease-free equilibrium (DFE) and the fundamental reproduction number $\mathcal{R}_0$ have been calculated. The local asymptotic stability of the infection-free equilibrium point is analyzed using the Routh-Hurwitz criterion around the equilibrium point.

5.1 Disease Free Equilibrium Point

In the absence of disease, the model system (2.1) has a disease-free equilibrium. Let $\mathcal{E}^0$ be the disease-free equilibrium state [20], then we have the disease-free equilibrium are $\left(\frac{\Lambda\pi}{\mu+\alpha}, \frac{\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)}, 0,0,0,0,0,0\right)$.

5.2 Basic Reproduction Number $\mathcal{R}_0$

The basic reproduction number is a crucial factor in determining a disease's long-term persistence or ultimate decline. In this section, we used Vandriessche [21] next generation matrix to find the reproduction number. $\frac{dZ}{dt} = f(Z) - v(Z)$, where $Z = (E, K, C, A, T)$ to calculate the reproduction number of model (2.1), let

$$F = \begin{bmatrix} \beta(E+K)S + \omega(E+K)V \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad V = \begin{bmatrix} -(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)E \\ \lambda_3\eta_3 E - (\mu+\gamma_2)K \\ \lambda_2\eta_2 E - (\mu+\gamma_1+\zeta_2+\tau_1)C + q\epsilon A \\ \lambda_1\eta_1 E - (q\epsilon+(1-q)\rho+\mu+\zeta_1)A \\ \zeta_2 C + \zeta_1 A - (\tau_2+\mu+\gamma_3)T \end{bmatrix}$$

The Jacobian matrices of F and V at the disease-free equilibrium points $\mathcal{E}^0$ are:

$$\mathbb{F} = \begin{bmatrix} \beta S^0+\omega V^0 & \beta S^0+\omega V^0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \quad \mathbb{V} = \begin{bmatrix} -L & 0 & 0 & 0 & 0 \\ \lambda_3\eta_3 & -(\mu+\gamma_2) & 0 & 0 & 0 \\ \lambda_2\eta_2 & 0 & -M & q\epsilon & 0 \\ \lambda_1\eta_1 & 0 & 0 & -N & 0 \\ 0 & 0 & \zeta_2 & \zeta_1 & -O \end{bmatrix},$$

where

$$\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3 = L, \qquad \mu+\gamma_1+\zeta_2+\tau_1 = M,$$
$$q\epsilon+(1-q)\rho+\mu+\zeta_1 = N, \qquad \tau_2+\mu+\gamma_3 = O.$$

Now, we get

$$\mathbb{F}\mathbb{V}^{-1} = \begin{bmatrix} (\beta S^0+\omega V^0)\left(-\frac{1}{L} - \frac{\lambda_3\eta_3}{L(\mu+\gamma_2)}\right) & (\beta S^0+\omega V^0)\left(-\frac{1}{\mu+\gamma_2}\right) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

So, the reproduction number be $\mathcal{R}_0 = \frac{\beta\Lambda\pi(\mu+\alpha)+\omega\Lambda(\mu+\alpha-\pi\mu)}{(\mu+\alpha)\mu L}\left(1+\frac{\lambda_3\eta_3}{\mu+\gamma_2}\right)$.

Figure 2 shows how $\mathcal{R}_0$ is affected by ω and β . As ω or β increases, $\mathcal{R}_0$ also increases. This shows that higher vaccine failure and increased transmission rates lead to a greater chance of spreading disease. Practically, higher $\mathcal{R}_0$ means that each primary infection results in more secondary infections, potentially leading to larger outbreaks.

Figure 3 explores the effects of vaccination rate (α) and vaccine failure rate (ω) on $\mathcal{R}_0$. The data shows that $\mathcal{R}_0$ decreases with higher vaccination coverage (α) and lower vaccine failure rate (ω). This implies that improving vaccination coverage and reducing the vaccine failure rate can significantly reduce the risk of disease transmission. Thus, effective vaccination strategies are essential to slow the spread of hepatitis B.

5.3 Stability Analysis of Disease-free Equilibrium

Theorem 5.1. *When $\mathcal{R}_0 < 1$ for the DFE point $\mathcal{E}^0\left(\frac{\Lambda\pi}{\mu+\alpha}, \frac{\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)}, 0,0,0,0,0,0\right)$ are exists and it is locally asymptotically stable, otherwise it is unstable.*

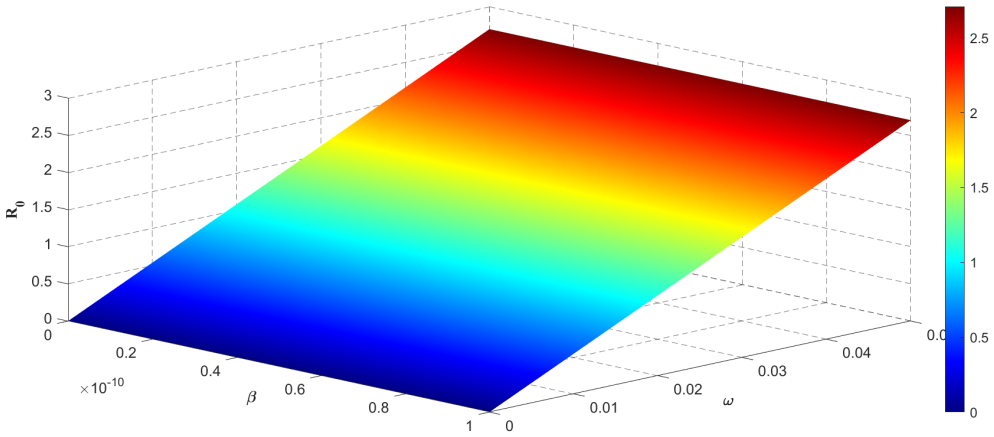

Figure 2. Effect of vaccine failure rate ω and contact rate β on $\mathcal{R}_0$.

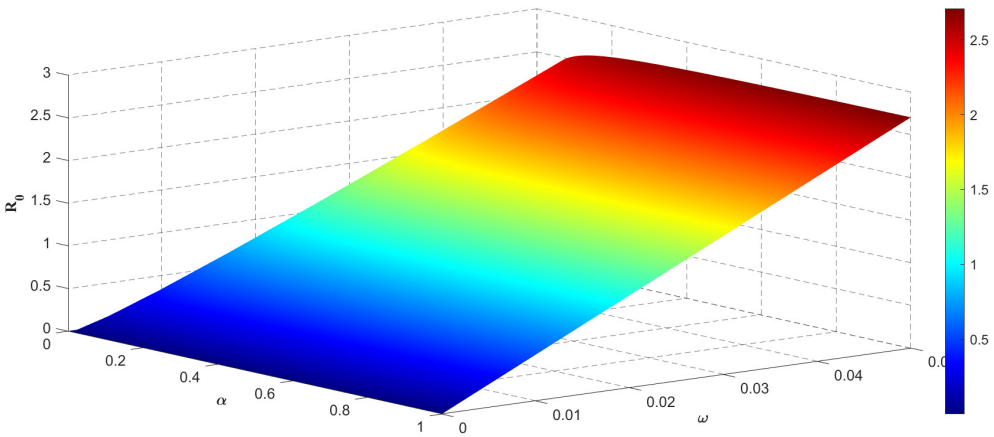

Figure 3. Effect of vaccine failure rate ω and vaccine uptake α rate on $\mathcal{R}_0$.

Proof. We calculate the Jacobian matrix of the system (2.1) for determine the local stability of $\mathcal{E}^0\left(\frac{\Lambda\pi}{\mu+\alpha}, \frac{\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)}, 0, 0, 0, 0, 0, 0\right)$ for the DFE is given by

$$J_{\mathcal{E}^0} = \begin{bmatrix} -(\mu+\alpha) & 0 & \frac{-\beta\Lambda\pi}{\mu+\alpha} & \frac{-\beta\Lambda\pi}{\mu+\alpha} & 0 & 0 & 0 & \sigma \\ \alpha & -\mu & -\omega\frac{\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)} & -\omega\frac{\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)} & 0 & 0 & 0 & 0 \\ 0 & 0 & -A_{33} & A_{34} & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_3\eta_3 & -(\mu+\gamma_2) & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_2\eta_2 & 0 & -A_{55} & q\epsilon & 0 & 0 \\ 0 & 0 & \lambda_1\eta_1 & 0 & 0 & -A_{66} & 0 & 0 \\ 0 & 0 & 0 & 0 & \zeta_2 & \zeta_1 & -A_{77} & 0 \\ 0 & 0 & 0 & 0 & \tau_1 & (1-q)\rho & \tau_2 & -(\mu+\sigma) \end{bmatrix},$$

where

$$A_{33} = (\mu + \lambda_1\eta_1 + \lambda_2\eta_2 + \lambda_3\eta_3), \quad A_{34} = \frac{\beta\Lambda\pi}{\mu+\alpha} + \omega\frac{\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)},$$
$$A_{55} = (\mu + \gamma_1 + \zeta_2 + \tau_1), \quad A_{66} = (q\epsilon + (1-q)\rho + \mu + \zeta_1), \quad A_{77} = (\tau_2 + \mu + \gamma_3).$$

From the above matrix we can easily determine eigen values as follows: $\lambda_1 = -(\mu+\alpha)$, $\lambda_2 = -\mu$, $\lambda_3 = -(\mu + \gamma_1 + \zeta_2 + \tau_1)$, $\lambda_4 = -(q\epsilon + (1-q)\rho + \mu + \zeta_1)$, $\lambda_5 = -(\tau_2 + \mu + \gamma_3)$, $\lambda_6 = -(\mu+\sigma)$. The remaining eigen values λ_i (for $i = 7, 8$) can be determined by using the provided equations,

$$\lambda^2 + \lambda(A_{33} + \mu + \gamma_2) + A_{33}(\mu+\gamma_2) - \lambda_3\eta_3\left(\frac{\beta\Lambda\pi}{\mu+\alpha} + \frac{\omega\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)}\right) = 0. \tag{5.1}$$

As $A_{33}(\mu+\gamma_2) - \lambda_3\eta_3\left(\frac{\beta\Lambda\pi}{\mu+\alpha} + \frac{\omega\Lambda(\mu+\alpha-\pi\mu)}{\mu(\mu+\alpha)}\right) > 0$, $A_{33} + (\mu+\gamma_2) > 0$, according to Routh-Hurwitz criterion, Equation (5.1) possesses two roots with a negative real part. Consequently, all the eigenvalues of the Jacobian matrix $J_{\mathcal{E}^0}$ exhibits a characteristics where their real parts are negative, given that $\mathcal{R}_0$ are less than 1. This leads us to the theorem. □

6 Homotopy Perturbation Method

The HPM introduces a homotopy parameter p, which varies continuously from 0 to 1. When $p = 0$, the given equation simplifies to a trivial or easily solvable form. As p gradually increases from 0 to 1, it generates a series of intermediate solutions, each progressively closer to the final solution. When $p = 1$, the system fully transitions to the original equation, and the solution at this stage corresponds to the desired result. Remarkably, the method achieves high accuracy with only a few iterations.

The HPM method considers that a nonlinear differential equation can be expressed as

$$\mathcal{F}(u) - \Upsilon(r) = 0, \tag{6.1}$$

with the boundary condition

$$B\left(u, \frac{\partial u}{\partial \eta}\right) = 0, \quad \text{where } r \in \Omega,\ r \in \Gamma,$$

where $\mathcal{F}$ is a general differential operator, $\Upsilon(r)$ is a known analytic function, B is a boundary operator, and Γ is the boundary of the domain Ω.

The $\mathcal{F}$ operator can usually be divided into two operators, L and N, where L is the linear operator and N is the nonlinear operator. Hence, (6.1) can be rewritten as:

$$L(u) + N(u) - f(r) = 0. \tag{6.2}$$

Now, the homotopy function is:

$$H(v,p) = (1-p)[L(v) - L(u_0)] + p[L(v) + N(v) - f(r)] = 0, \quad \text{where } p \in [0,1] \tag{6.3}$$

u_0 is the initial approximation of (6.2), which satisfies the boundary conditions, and p is known as the perturbation homotopy parameter.

Analyzing (6.3), it can be concluded that:

$$H(v,0) = L(v) - L(u_0) = 0, \quad H(v,1) = L(v) + N(v) - f(r) = 0.$$

We assume that the solution of (6.3) can be written as a power series of p:

$$v = p^0 v_0 + p^1 v_1 + p^2 v_2 + \cdots .$$

Adjusting $p = 1$ results in the approximate solution for (6.1):

$$u = \lim_{p \to 1} v = v_0 + v_1 + v_2 + \cdots . \tag{6.4}$$

Ji-Huan He, analyzed the convergence of the series in Equation (6.4) in his works [22, 23].

6.1 Solutions of the Model Equations

The solutions of the system (2.1) obtained by HPM are follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda\pi + \sigma R - (\mu + \alpha)S - \beta S(E + K),\\
\frac{dV}{dt} &= \Lambda(1 - \pi) + \alpha S - \mu V - \omega V(E + K),\\
\frac{dE}{dt} &= \beta S(E + K) + \omega V(E + K) - (\mu + \lambda_1\eta_1 + \lambda_2\eta_2 + \lambda_3\eta_3)E,\\
\frac{dK}{dt} &= \lambda_3\eta_3 E - (\mu + \gamma_2)K,\\
\frac{dC}{dt} &= \lambda_2\eta_2 E - (\mu + \gamma_1 + \zeta_2 + \tau_1)C + q\epsilon A,\\
\frac{dA}{dt} &= \lambda_1\eta_1 E - (q\epsilon + (1 - q)\rho + \mu + \zeta_1)A,\\
\frac{dT}{dt} &= \zeta_2 C + \zeta_1 A - (\tau_2 + \mu + \gamma_3)T,\\
\frac{dR}{dt} &= \tau_1 C + (1 - q)\rho A + \tau_2 T - (\mu + \sigma)R.
\end{aligned} \tag{6.5}$$

To obtain the analytical solution of system (6.5), we construct the homotopy as follows:

$$\begin{aligned}
&(1-p)\Big(\frac{dS}{dt} - \Lambda\pi + (\mu+\alpha)S\Big) + p\Big(\frac{dS}{dt} - \Lambda\pi - \sigma R + (\mu+\alpha)S + \beta S(E+K)\Big) = 0,\\
&(1-p)\Big(\frac{dV}{dt} - \Lambda(1-\pi) + \mu V\Big) + p\Big(\frac{dV}{dt} - \Lambda(1-\pi) - \alpha S + \mu V + \omega V(E+K)\Big) = 0,\\
&(1-p)\Big(\frac{dE}{dt} + (\mu + \lambda_1\eta_1 + \lambda_2\eta_2 + \lambda_3\eta_3)E\Big)\\
&\quad + p\Big(\frac{dE}{dt} - \beta S(E+K) - \omega V(E+K) + (\mu + \lambda_1\eta_1 + \lambda_2\eta_2 + \lambda_3\eta_3)E\Big) = 0,\\
&(1-p)\Big(\frac{dK}{dt} + (\mu+\gamma_2)K\Big) + p\Big(\frac{dK}{dt} - \lambda_3\eta_3 E + (\mu+\gamma_2)K\Big) = 0,\\
&(1-p)\Big(\frac{dC}{dt} + (\mu + \gamma_1 + \zeta_2 + \tau_1)C\Big) + p\Big(\frac{dC}{dt} - \lambda_2\eta_2 E + (\mu + \gamma_1 + \zeta_2 + \tau_1)C - q\epsilon A\Big) = 0,\\
&(1-p)\Big(\frac{dA}{dt} + (q\epsilon + (1-q)\rho + \mu + \zeta_1)A\Big) + p\Big(\frac{dA}{dt} - \lambda_1\eta_1 E + (q\epsilon + (1-q)\rho + \mu + \zeta_1)A\Big) = 0,\\
&(1-p)\Big(\frac{dT}{dt} + (\tau_2 + \mu + \gamma_3)T\Big) + p\Big(\frac{dT}{dt} - \zeta_2 C - \zeta_1 A + (\tau_2 + \mu + \gamma_3)T\Big) = 0,\\
&(1-p)\Big(\frac{dR}{dt} + (\mu+\sigma)R\Big) + p\Big(\frac{dR}{dt} - \tau_1 C - (1-q)\rho A - \tau_2 T + (\mu+\sigma)R\Big) = 0.
\end{aligned} \tag{6.6}$$

Equating p^0 terms on both sides of the above system of equations (6.6) we get constructing homotopy, we get

$$
\begin{aligned}
p^0 : \frac{dS_0}{dt} &= \Lambda\pi - (\mu+\alpha)S_0,\\
p^0 : \frac{dV_0}{dt} &= \Lambda(1-\pi) - \mu V_0,\\
p^0 : \frac{dE_0}{dt} &= -(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)E_0,\\
p^0 : \frac{dK_0}{dt} &= -(\mu+\gamma_2)K_0,\\
p^0 : \frac{dC_0}{dt} &= -(\mu+\gamma_1+\zeta_2+\tau_1)C_0,\\
p^0 : \frac{dA_0}{dt} &= -(q\epsilon+(1-q)\rho+\mu+\zeta_1)A_0,\\
p^0 : \frac{dT_0}{dt} &= -(\tau_2+\mu+\gamma_3)T_0,\\
p^0 : \frac{dR_0}{dt} &= -(\mu+\sigma)R_0,
\end{aligned}
$$

the solutions of these equations are given below:

$$
\begin{aligned}
S_0 &= \frac{\Lambda\pi}{(\mu+\alpha)} + M_1 e^{-(\mu+\alpha)t},\\
V_0 &= \frac{\Lambda(1-\pi)}{\mu} + M_2 e^{-\mu t},\\
E_0 &= M_3 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t},\\
K_0 &= M_4 e^{-(\mu+\gamma_2)t},\\
C_0 &= M_5 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t},\\
A_0 &= M_6 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t},\\
T_0 &= M_7 e^{-(\tau_2+\mu+\gamma_3)t},\\
R_0 &= M_8 e^{-(\mu+\sigma)t}.
\end{aligned}
$$

Applying initial conditions, $S(0)=\kappa_0$, $V(0)=\kappa_1$, $E(0)=\kappa_2$, $K(0)=\kappa_3$, $C(0)=\kappa_4$, $A(0)=\kappa_5$, $T(0)=\kappa_6$, $R(0)=\kappa_7$ for all $\kappa_i>0$, $i=0,1,2,3,4,5,6,7$ and initial approximations, $S(i)=0$, $V(i)=0$, $E(i)=0$, $K(i)=0$, $C(i)=0$, $A(i)=0$, $T(i)=0$, $R(i)=0$ for all $i=1,2,3,\cdots$.

By applying initial conditions in the solution of equations are given below:

$$M_1 = \kappa_0 - \frac{\Lambda\pi}{(\mu+\alpha)}.$$

Therefore

$$S_0 = \frac{\Lambda\pi}{(\mu+\alpha)} + \Big(\kappa_0 - \frac{\Lambda\pi}{(\mu+\alpha)}\Big)e^{-(\mu+\alpha)t}.$$

Similarly

$$M_2 = \kappa_1 - \frac{\Lambda(1-\pi)}{\mu},$$

$$V_0 = \frac{\Lambda(1-\pi)}{\mu} + \Big(\kappa_1 - \frac{\Lambda(1-\pi)}{\mu}\Big)e^{-\mu t},$$

$$M_3 = \kappa_2, \quad \text{therefore} \quad E_0 = \kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t},$$

$$M_4 = \kappa_3, \quad \text{therefore} \quad K_0 = \kappa_3 e^{-(\mu+\gamma_2)t},$$

$$M_5 = \kappa_4, \quad \text{therefore} \quad C_0 = \kappa_4 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t},$$

$$M_6 = \kappa_5, \quad \text{therefore} \quad A_0 = \kappa_5 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t},$$

$$M_7 = \kappa_6, \quad \text{therefore} \quad T_0 = \kappa_6 e^{-(\tau_2+\mu+\gamma_3)t},$$

$$M_8 = \kappa_7, \quad \text{therefore} \quad R_0 = \kappa_7 e^{-(\mu+\sigma)t}.$$

Again equating p^1 terms, we get

$$p^1 : \frac{dS_1}{dt} = \Lambda_0\pi_0 - \sigma R_0 - (\mu+\alpha)S_1 - \beta S_0 E_0 - \beta S_0 K_0,$$

$$p^1 : \frac{dV_1}{dt} = \Lambda_0(1-\pi_0) + \alpha S_0 - \mu V_1 - \omega V_0 E_0 - \omega V_0 K_0,$$

$$p^1 : \frac{dE_1}{dt} = \beta S_0(E_0+K_0) + \omega V_0(E_0+K_0) - (\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)E_1,$$

$$p^1 : \frac{dK_1}{dt} = \lambda_3\eta_3 E_0 - (\mu+\gamma_2)K_1,$$

$$p^1 : \frac{dC_1}{dt} = \lambda_2\eta_2 E_0 - (\mu+\gamma_1+\zeta_2+\tau_1)C_1 - q\epsilon A_0,$$

$$p^1 : \frac{dA_1}{dt} = \lambda_1\eta_1 E_0 - (q\epsilon+(1-q)\rho+\mu+\zeta_1)A_1,$$

$$p^1 : \frac{dT_1}{dt} = \zeta_2 C_0 + \zeta_1 A_0 - (\tau_2+\mu+\gamma_3)T_1,$$

$$p^1 : \frac{dR_1}{dt} = \tau_1 C_0 + (1-q)\rho A_0 + \tau_2 T_0 - (\mu+\sigma)R_1.$$

By applying all the initial conditions in p^1 terms we get,

$$\frac{dS_1}{dt} = \Lambda_0\pi_0 - \sigma\big(\kappa_7 e^{-(\mu+\sigma)t}\big) - (\mu+\alpha)S_1 - \beta\Big(\frac{\Lambda\pi}{(\mu+\alpha)} + \kappa_0 - \frac{\Lambda\pi}{(\mu+\alpha)}e^{-(\mu+\alpha)t}\Big)$$
$$\Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}\Big) - \beta\Big(\frac{\Lambda\pi}{(\mu+\alpha)} + \kappa_0 - \frac{\Lambda\pi}{(\mu+\alpha)}e^{-(\mu+\alpha)t)}\Big)\Big(\kappa_3 e^{-(\mu+\gamma_2)t}\Big),$$

$$S_1 = \frac{\Lambda_0\pi_0}{(\mu+\alpha)} - \frac{\sigma\kappa_7 e^{-(\mu+\sigma)t}}{(\alpha-\sigma)} - \beta\Big(\frac{\Lambda\pi}{(\mu+\alpha)^2} + \frac{\kappa_0}{(\mu+\alpha)} - \frac{\Lambda\pi}{(\mu+\alpha)}e^{-(\mu+\alpha)t}t\Big)$$
$$\Big(\frac{\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}}{\alpha-\lambda_1\eta_1-\lambda_2\eta_2-\lambda_3\eta_3} + \frac{\kappa_3 e^{-(\mu+\gamma_2)t}}{\alpha-\gamma_2}\Big) + \mathcal{C}_1 e^{-(\mu+\alpha)t}.$$

Applying initial conditions $S(0) = 0$, then we have

$$\mathcal{C}_1 = -\frac{\Lambda_0\pi_0}{\mu+\alpha} + \frac{\sigma\kappa_7}{\alpha-\sigma} + \beta\Big(\frac{\Lambda\pi}{(\mu+\alpha)^2} + \frac{\kappa_0}{\mu+\alpha}\Big)\Big(\frac{\kappa_2}{\alpha-\lambda_1\eta_1-\lambda_2\eta_2-\lambda_3\eta_3} + \frac{\kappa_3}{\alpha-\gamma_2}\Big),$$

then

$$\begin{aligned} S_1 = {} & \frac{\Lambda_0\pi_0}{(\mu+\alpha)} - \frac{\sigma\kappa_7 e^{-(\mu+\sigma)t}}{(\alpha-\sigma)} - \beta\Big(\frac{\Lambda\pi}{(\mu+\alpha)^2} + \frac{\kappa_0}{(\mu+\alpha)} - \frac{\Lambda\pi}{(\mu+\alpha)}e^{-(\mu+\alpha)t}t\Big) \\ & \Big(\frac{\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}}{\alpha-\lambda_1\eta_1-\lambda_2\eta_2-\lambda_3\eta_3} + \frac{\kappa_3 e^{-(\mu+\gamma_2)t}}{\alpha-\gamma_2}\Big) + \\ & \Big(-\frac{\Lambda_0\pi_0}{\mu+\alpha} + \frac{\sigma\kappa_7}{\alpha-\sigma} + \beta\Big(\frac{\Lambda\pi}{(\mu+\alpha)^2} + \frac{\kappa_0}{\mu+\alpha}\Big)\Big(\frac{\kappa_2}{\alpha-\lambda_1\eta_1-\lambda_2\eta_2-\lambda_3\eta_3} + \frac{\kappa_3}{\alpha-\gamma_2}\Big)\Big) \\ & \times e^{-(\mu+\alpha)t}. \end{aligned} \tag{6.7}$$

$$\begin{aligned} \frac{dV_1}{dt} = {} & \Lambda_0(1-\pi_0) - \alpha\Big(\frac{\Lambda\pi}{(\mu+\alpha)} + \kappa_0 - \frac{\Lambda\pi}{(\mu+\alpha)}e^{-(\mu+\alpha)t}\Big) - \mu V_1 \\ & - \omega\Big(\frac{\Lambda(1-\pi)}{\mu} - \frac{\Lambda(1-\pi)}{\mu}e^{-\mu t} + \kappa_1\Big)\Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}\Big) \\ & - \omega\Big(\frac{\Lambda(1-\pi)}{\mu} + \kappa_1 - \frac{\Lambda(1-\pi)}{\mu}e^{-\mu t}\Big)\Big(\kappa_3 e^{-(\mu+\gamma_2)t}\Big), \end{aligned}$$

$$\begin{aligned} V_1 = {} & \frac{\Lambda_0(1-\pi_0)}{\mu} - \alpha\Big(\frac{\Lambda\pi}{\mu(\mu+\alpha)} + \frac{\kappa_0}{\mu} + \frac{\Lambda\pi e^{-(\mu+\alpha)t}}{\alpha(\mu+\alpha)}\Big) - \omega\Big(\frac{\Lambda(1-\pi)}{\mu^2} + \frac{\kappa_1}{\mu} - \frac{\Lambda(1-\pi)e^{-\mu t}t}{\mu}\Big) \\ & \Big(-\kappa_2\frac{e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}}{(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)} - \kappa_3\frac{e^{-(\mu+\gamma_2)t}}{\gamma_2}\Big) + \mathcal{C}_2 e^{-\mu t}. \end{aligned}$$

Applying initial conditions $V(0) = 0$, then

$$\begin{aligned} \mathcal{C}_2 = {} & -\frac{\Lambda_0(1-\pi_0)}{\mu} + \alpha\Big(\frac{\Lambda\pi}{\mu(\mu+\alpha)} + \frac{\kappa_0}{\mu} + \frac{\Lambda\pi}{\alpha(\mu+\alpha)}\Big) - \omega\Big(\frac{\Lambda(1-\pi)}{\mu^2} + \frac{\kappa_1}{\mu}\Big) \\ & \times \Big(\frac{\kappa_3}{\gamma_2} + \frac{\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3}\Big). \end{aligned}$$

$$\begin{aligned} V_1 = {} & \frac{\Lambda_0(1-\pi_0)}{\mu} - \alpha\Big(\frac{\Lambda\pi}{\mu(\mu+\alpha)} + \frac{\kappa_0}{\mu} + \frac{\Lambda\pi e^{-(\mu+\alpha)t}}{\alpha(\mu+\alpha)}\Big) - \omega\Big(\frac{\Lambda(1-\pi)}{\mu^2} + \frac{\kappa_1}{\mu} - \frac{\Lambda(1-\pi)e^{-\mu t}t}{\mu}\Big) \\ & \Big(-\kappa_2\frac{e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}}{(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)} - \kappa_3\frac{e^{-(\mu+\gamma_2)t}}{\gamma_2}\Big) + \alpha\Big(\frac{\Lambda\pi}{\mu(\mu+\alpha)} + \frac{\kappa_0}{\mu} + \frac{\Lambda\pi}{\alpha(\mu+\alpha)}\Big)e^{-\mu t} \\ & \Big(-\frac{\Lambda_0(1-\pi_0)}{\mu} - \omega\Big(\frac{\Lambda(1-\pi)}{\mu^2} + \frac{\kappa_1}{\mu}\Big)\Big(\frac{\kappa_3}{\gamma_2} + \frac{\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3}\Big)\Big)e^{-\mu t}. \end{aligned} \tag{6.8}$$

$$\begin{aligned} \frac{dE_1}{dt} = {} & \beta\Big(\frac{\Lambda\pi}{(\mu+\alpha)}\Big) + \Big(\kappa_0 - \frac{\Lambda\pi}{(\mu+\alpha)}\Big)e^{-(\mu+\alpha)t}\Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t} + \kappa_3 e^{-(\mu+\gamma_2)t}\Big) \\ & + \omega\Big(\frac{\Lambda(1-\pi)}{\mu}\Big) + \Big(\kappa_1 - \frac{\Lambda(1-\pi)}{\mu}\Big)e^{-\mu t}\Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t} + \kappa_3 e^{-(\mu+\gamma_2)t}\Big) \\ & - \Big(\mu + \lambda_1\eta_1 + \lambda_2\eta_2 + \lambda_3\eta_3\Big)E_1, \end{aligned}$$

$$\begin{aligned} E_1 = {} & \frac{\beta\Lambda\pi}{(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)(\mu+\alpha)} + \Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t} + \frac{\kappa_3 e^{-(\mu+\gamma_2)t}}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}\Big) \\ & \Big(\frac{\kappa_0 e^{-(\mu+\alpha)t}}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha} - \frac{\Lambda\pi e^{-(\mu+\alpha)t}}{(\mu+\alpha)(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha)} + \frac{\kappa_1 e^{-\mu t}}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3} \\ & - \frac{\Lambda(1-\pi)e^{-\mu t}}{\mu(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)}\Big) + \frac{\omega\Lambda(1-\pi)}{\mu(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)} + \mathcal{C}_3 e^{-\mu t}. \end{aligned}$$

Applying initial conditions $E(0) = 0$, then

$$\mathcal{C}_3 = -\frac{\beta\Lambda\pi}{(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)(\mu+\alpha)} - \Big(\kappa_2 + \frac{\kappa_3}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}\Big)$$
$$\Big(\frac{\kappa_0}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha} - \frac{\Lambda\pi}{(\mu+\alpha)(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha)} + \frac{\kappa_1}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3}$$
$$- \frac{\Lambda(1-\pi)}{\mu(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)}\Big) - \frac{\omega\Lambda(1-\pi)}{\mu(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)}.$$

$$E_1 = \frac{\beta\Lambda\pi}{(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)(\mu+\alpha)} + \Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t} + \frac{\kappa_3 e^{-(\mu+\gamma_2)t}}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}\Big)$$
$$\Big(\frac{\kappa_0 e^{-(\mu+\alpha)t}}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha} - \frac{\Lambda\pi e^{-(\mu+\alpha)t}}{(\mu+\alpha)(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha)} + \frac{\kappa_1 e^{-\mu t}}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3}$$
$$- \frac{\Lambda(1-\pi)e^{-\mu t}}{\mu(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)}\Big) + \frac{\omega\Lambda(1-\pi)}{\mu(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)} - \Big[\frac{\beta\Lambda\pi}{(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)(\mu+\alpha)}$$
$$+ \Big(\kappa_2 + \frac{\kappa_3}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}\Big)\Big(\frac{\kappa_0}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha} - \frac{\Lambda\pi}{(\mu+\alpha)(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\alpha)}$$
$$+ \frac{\kappa_1}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3} - \frac{\Lambda(1-\pi)}{\mu(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)}\Big) + \frac{\omega\Lambda(1-\pi)}{\mu(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)}\Big]e^{-\mu t}. \tag{6.9}$$

$$\frac{dK_1}{dt} = \lambda_3\eta_3\Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}\Big) - (\mu+\gamma_2)K_1,$$
$$K_1 = \mathcal{C}_4 e^{-(\mu+\gamma_2)t} - \frac{\lambda_3\eta_3\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}e^{-(\lambda_1\eta_1+\lambda_2\eta_2+\mu+\lambda_3\eta_3)t}.$$

Applying initial conditions $K(0) = 0$, then

$$\mathcal{C}_4 = \frac{\lambda_3\eta_3\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}.$$

$$K_1 = \Big(\frac{\lambda_3\eta_3\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}\Big)e^{-(\mu+\gamma_2)t} - \frac{\lambda_3\eta_3\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_2}e^{-(\lambda_1\eta_1+\lambda_2\eta_2+\mu+\lambda_3\eta_3)t}. \tag{6.10}$$

$$\frac{dC_1}{dt} = \lambda_2\eta_2\Big(\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}\Big) - q\epsilon\Big(\kappa_5 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t}\Big) - (\mu+\gamma_1+\zeta_2+\tau_1)C_1,$$
$$C_1 = \mathcal{C}_5 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t} - \frac{\lambda_2\eta_2\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_1-\zeta_2-\tau_1}e^{-(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3+\mu)t}$$
$$+ \frac{q\epsilon\kappa_5}{q\epsilon+(1-q)\rho+\zeta_1-\gamma_1-\zeta_2-\tau_1}e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t}.$$

Applying initial conditions $C(0) = 0$, then

$$\mathcal{C}_5 = \frac{\lambda_2\eta_2\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_1-\zeta_2-\tau_1} - \frac{q\epsilon\kappa_5}{q\epsilon+(1-q)\rho+\zeta_1-\gamma_1-\zeta_2-\tau_1}.$$

$$C_1 = \Big(\frac{\lambda_2\eta_2\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_1-\zeta_2-\tau_1} - \frac{q\epsilon\kappa_5}{q\epsilon+(1-q)\rho+\zeta_1-\gamma_1-\zeta_2-\tau_1}\Big)e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t}$$
$$- \frac{\lambda_2\eta_2\kappa_2}{\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3-\gamma_1-\zeta_2-\tau_1}e^{-(\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3+\mu)t}. \tag{6.11}$$

$$\frac{dA_1}{dt} = \lambda_1\eta_1\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t} - (q\epsilon + (1-q)\rho + \mu + \zeta_1)A_1,$$

$$A_1 = \frac{\lambda_1\eta_1\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}}{q\epsilon + (1-q)\rho + \zeta_1 - \lambda_1\eta_1 - \lambda_2\eta_2 - \lambda_3\eta_3} + \mathcal{C}_6 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t}.$$

Applying initial conditions $A(0) = 0$, then

$$\mathcal{C}_6 = -\frac{\lambda_1\eta_1\kappa_2}{q\epsilon + (1-q)\rho + \zeta_1 - \lambda_1\eta_1 - \lambda_2\eta_2 - \lambda_3\eta_3}.$$

$$A_1 = \frac{\lambda_1\eta_1\kappa_2 e^{-(\mu+\lambda_1\eta_1+\lambda_2\eta_2+\lambda_3\eta_3)t}}{q\epsilon + (1-q)\rho + \zeta_1 - \lambda_1\eta_1 - \lambda_2\eta_2 - \lambda_3\eta_3} - \frac{\lambda_1\eta_1\kappa_2 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t}}{q\epsilon + (1-q)\rho + \zeta_1 - \lambda_1\eta_1 - \lambda_2\eta_2 - \lambda_3\eta_3}. \tag{6.12}$$

$$\frac{dT_1}{dt} = \zeta_2\kappa_4 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t} + \zeta_1\kappa_5 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t} - (\tau_2 + \mu + \gamma_3)T_1,$$

$$T_1 = \frac{\zeta_2\kappa_4 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t}}{(\tau_2 + \gamma_3 - \gamma_1 - \zeta_2 - \tau_1)} + \frac{\zeta_1\kappa_5 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t}}{(\tau_2 + \gamma_3 - q\epsilon - (1-q)\rho - \zeta_1)} + \mathcal{C}_7 e^{-(\tau_2+\mu+\gamma_3)t}.$$

Applying initial conditions $T(0) = 0$, then

$$\mathcal{C}_7 = -\frac{\zeta_2\kappa_4}{(\tau_2 + \gamma_3 - \gamma_1 - \zeta_2 - \tau_1)} - \frac{\zeta_1\kappa_5}{(\tau_2 + \gamma_3 - q\epsilon - (1-q)\rho - \zeta_1)}.$$

$$\begin{aligned} T_1 = {} & \frac{\zeta_2\kappa_4 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t}}{(\tau_2 + \gamma_3 - \gamma_1 - \zeta_2 - \tau_1)} + \frac{\zeta_1\kappa_5 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t}}{(\tau_2 + \gamma_3 - q\epsilon - (1-q)\rho - \zeta_1)} \\ & - \Big(\frac{\zeta_2\kappa_4}{(\tau_2 + \gamma_3 - \gamma_1 - \zeta_2 - \tau_1)} + \frac{\zeta_1\kappa_5}{(\tau_2 + \gamma_3 - q\epsilon - (1-q)\rho - \zeta_1)}\Big) e^{-(\tau_2+\mu+\gamma_3)t}. \end{aligned} \tag{6.13}$$

$$\frac{dR_1}{dt} = \tau_1\kappa_4 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t} + (1-q)\rho\kappa_5 e^{-(q\epsilon+(1-q)\rho+\mu+\zeta_1)t} + \tau_2\kappa_7 e^{-(\tau_2+\mu+\gamma_3)t} - (\mu + \sigma)R_1,$$

$$R_1 = -\frac{\tau_1\kappa_4 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t}}{(\sigma - \gamma_1 - \zeta_2 - \tau_1)} - \frac{(1-q)\rho\kappa_5 e^{-(q\epsilon-(1-q)\rho+\mu+\zeta_1)t}}{(\sigma - q\epsilon - (1-q)\rho - \zeta_1)} - \frac{\tau_2\kappa_7 e^{-(\tau_2+\mu+\gamma_3)t}}{(\sigma - \tau_2 - \gamma_3)} + \mathcal{C}_8 e^{-(\mu+\sigma)t}.$$

Applying initial conditions $R(0) = 0$, then

$$\mathcal{C}_8 = \frac{\tau_1\kappa_4}{(\sigma - \gamma_1 - \zeta_2 - \tau_1)} + \frac{(1-q)\rho\kappa_5}{(\sigma - q\epsilon - (1-q)\rho - \zeta_1)} + \frac{\tau_2\kappa_7}{(\sigma - \tau_2 - \gamma_3)}.$$

$$\begin{aligned} R_1 = {} & -\frac{\tau_1\kappa_4 e^{-(\mu+\gamma_1+\zeta_2+\tau_1)t}}{(\sigma - \gamma_1 - \zeta_2 - \tau_1)} - \frac{(1-q)\rho\kappa_5 e^{-(q\epsilon-(1-q)\rho+\mu+\zeta_1)t}}{(\sigma - q\epsilon - (1-q)\rho - \zeta_1)} - \frac{\tau_2\kappa_7 e^{-(\tau_2+\mu+\gamma_3)t}}{(\sigma - \tau_2 - \gamma_3)} \\ & + \Big(\frac{\tau_1\kappa_4}{(\sigma - \gamma_1 - \zeta_2 - \tau_1)} + \frac{(1-q)\rho\kappa_5}{(\sigma - q\epsilon - (1-q)\rho - \zeta_1)} + \frac{\tau_2\kappa_7}{(\sigma - \tau_2 - \gamma_3)}\Big) e^{-(\mu+\sigma)t}. \end{aligned} \tag{6.14}$$

7 Numerical Simulation

This section plays a crucial role in this study. The proposed $SVEKCATR$ model system (2.1) comprises 22 parameters detailed in Table 1, along with their respective values. The initial values for the system (2.1) are as follows: $S(0) = 1,292,270,000$, $V(0) = 6,540,000$, $E(0) = 250,000$, $K(0) = 143,802$, $C(0) = 719,011$, $A(0) = 180,000$, $T(0) = 15,000$, and $R(0) =$

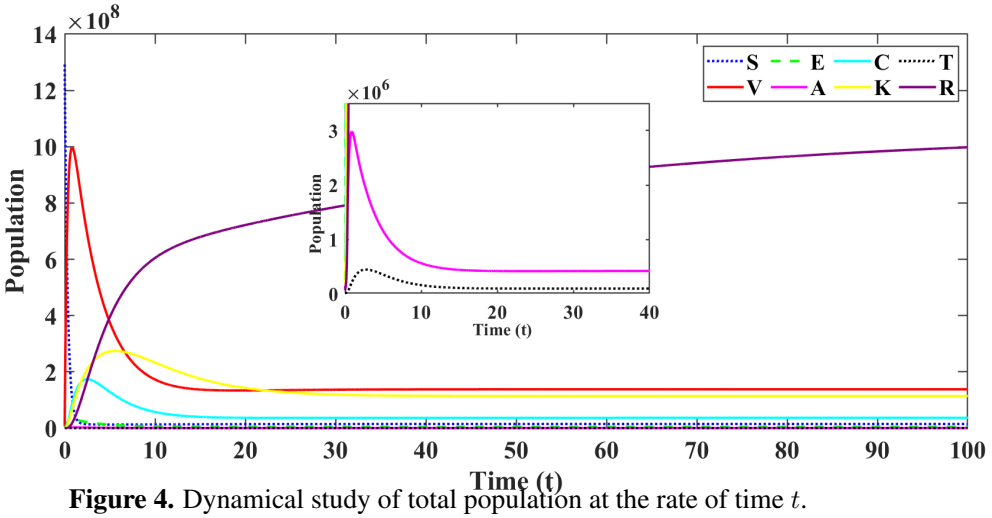

Figure 4. Dynamical study of total population at the rate of time t.

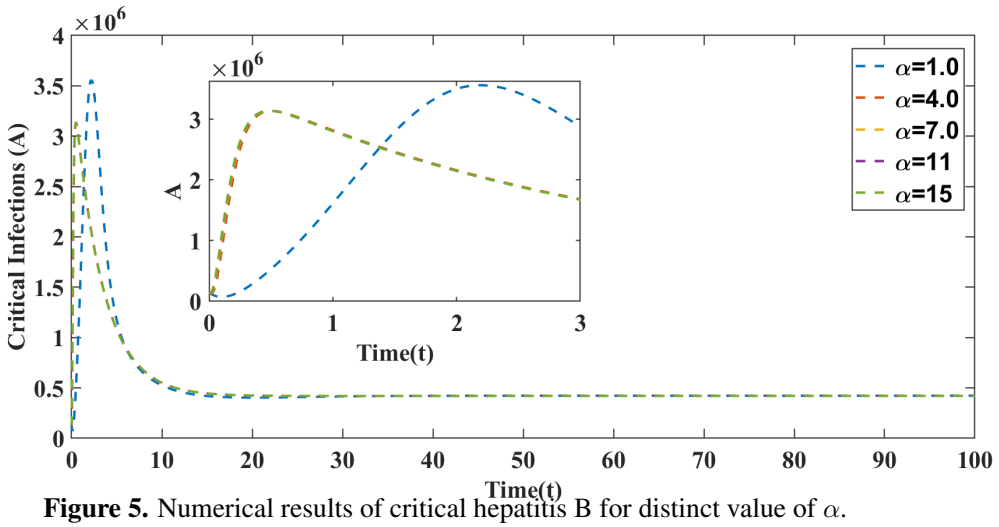

Figure 5. Numerical results of critical hepatitis B for distinct value of α.

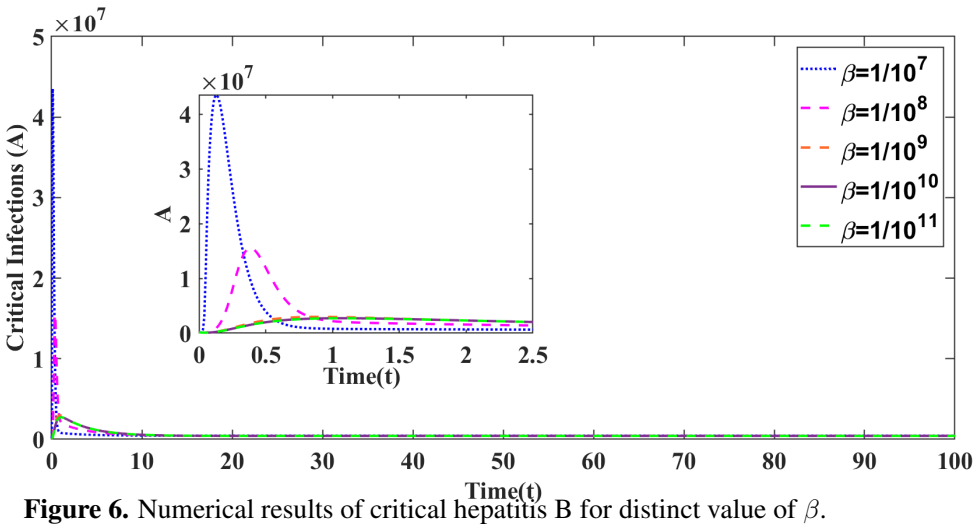

Figure 6. Numerical results of critical hepatitis B for distinct value of β.

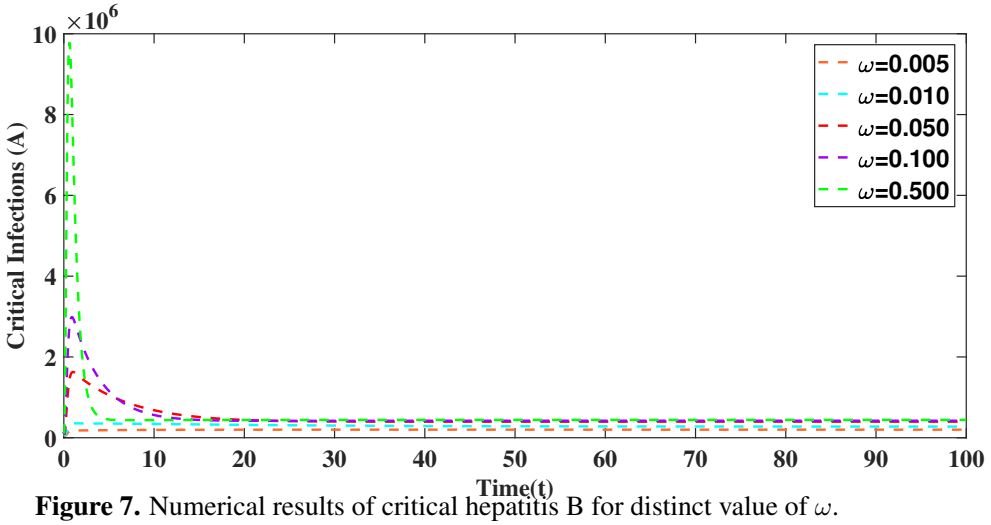

Figure 7. Numerical results of critical hepatitis B for distinct value of ω.

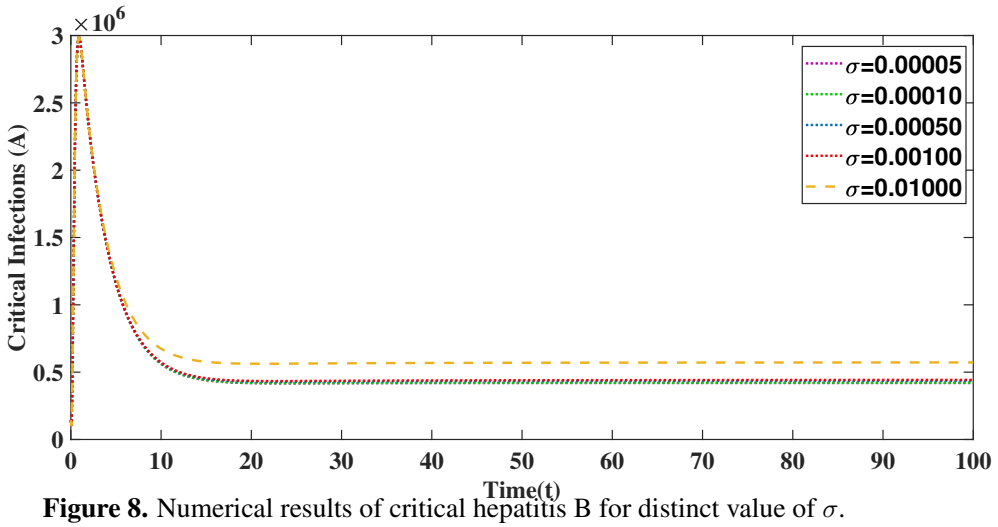

Figure 8. Numerical results of critical hepatitis B for distinct value of σ.

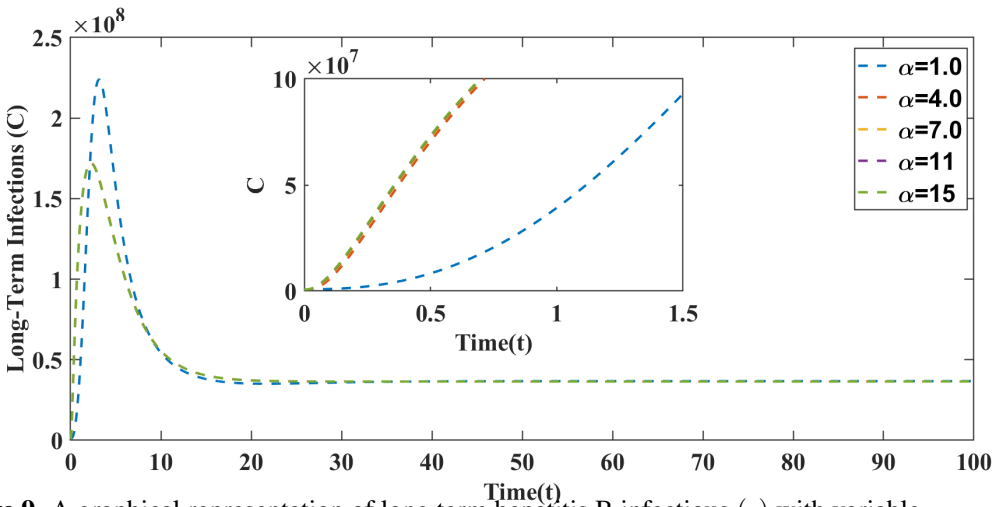

Figure 9. A graphical representation of long term hepatitis B infectious (c) with variable α.

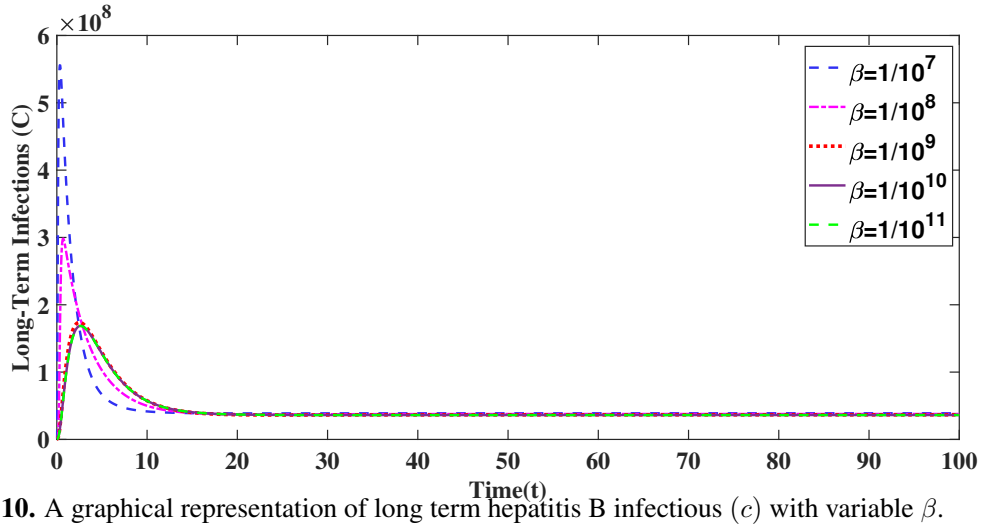

Figure 10. A graphical representation of long term hepatitis B infectious (c) with variable β.

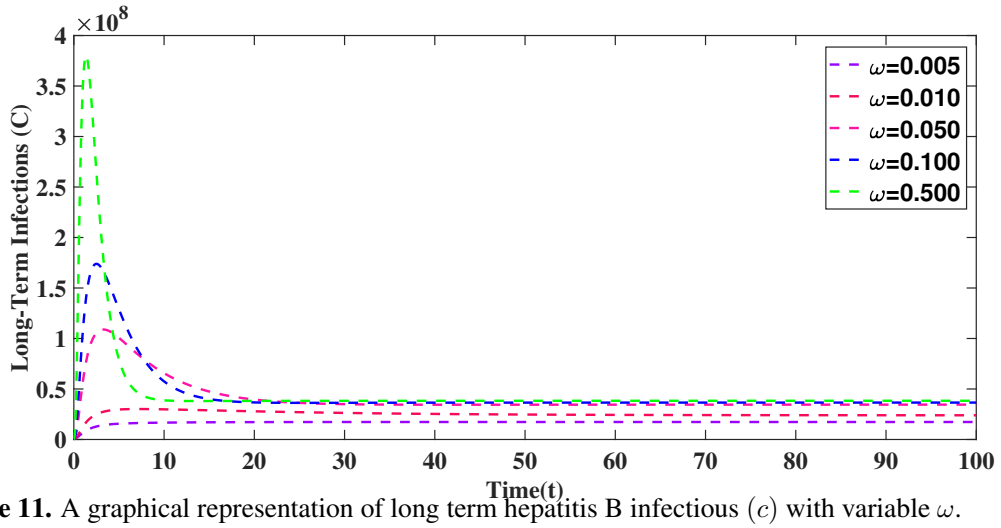

Figure 11. A graphical representation of long term hepatitis B infectious (c) with variable ω.

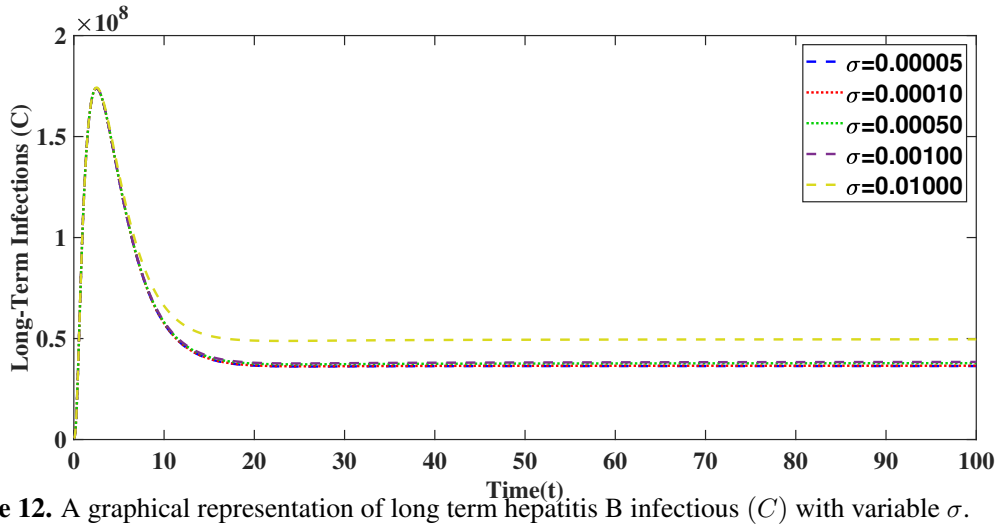

Figure 12. A graphical representation of long term hepatitis B infectious (C) with variable σ.

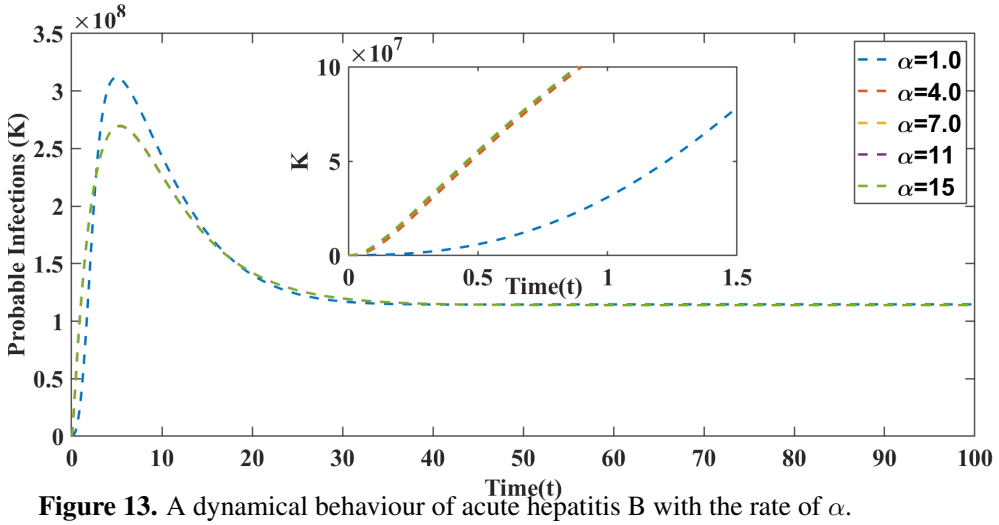

Figure 13. A dynamical behaviour of acute hepatitis B with the rate of α.

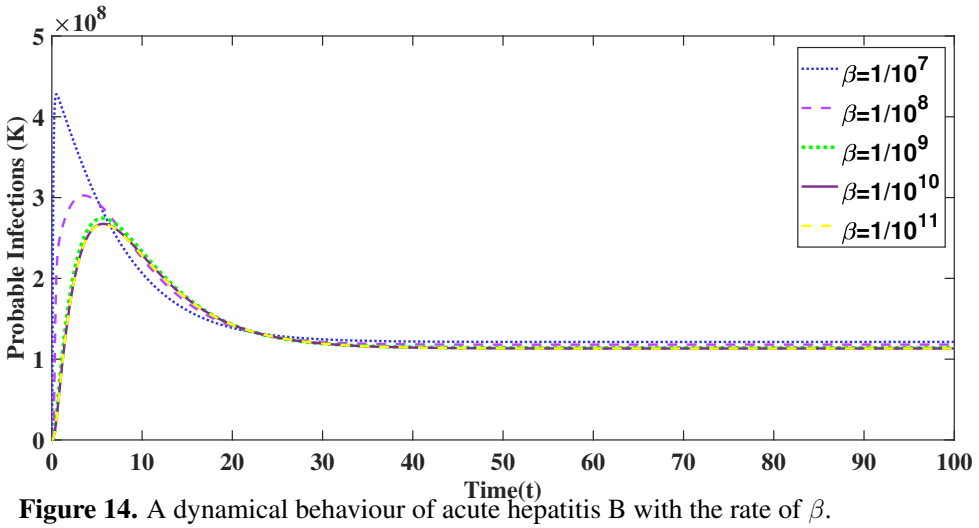

Figure 14. A dynamical behaviour of acute hepatitis B with the rate of β.

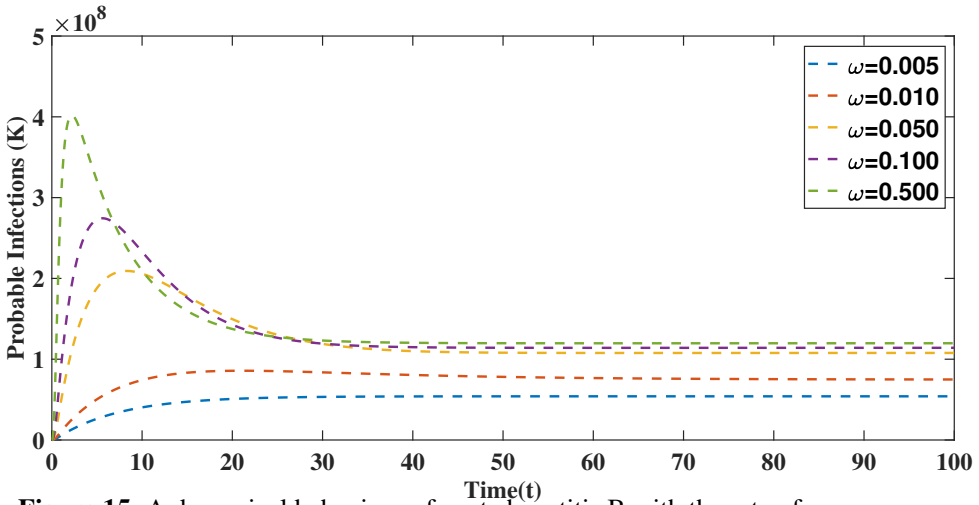

Figure 15. A dynamical behaviour of acute hepatitis B with the rate of ω.

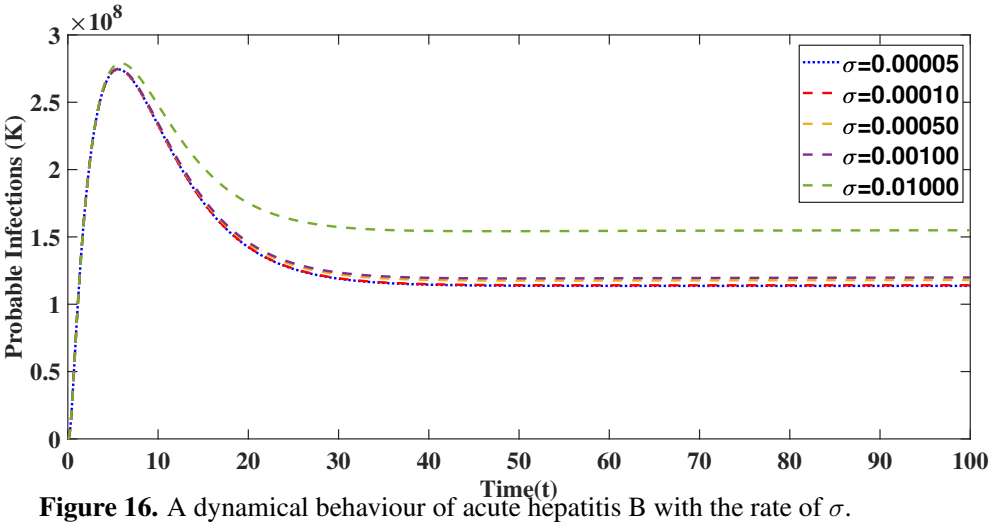

Figure 16. A dynamical behaviour of acute hepatitis B with the rate of σ.

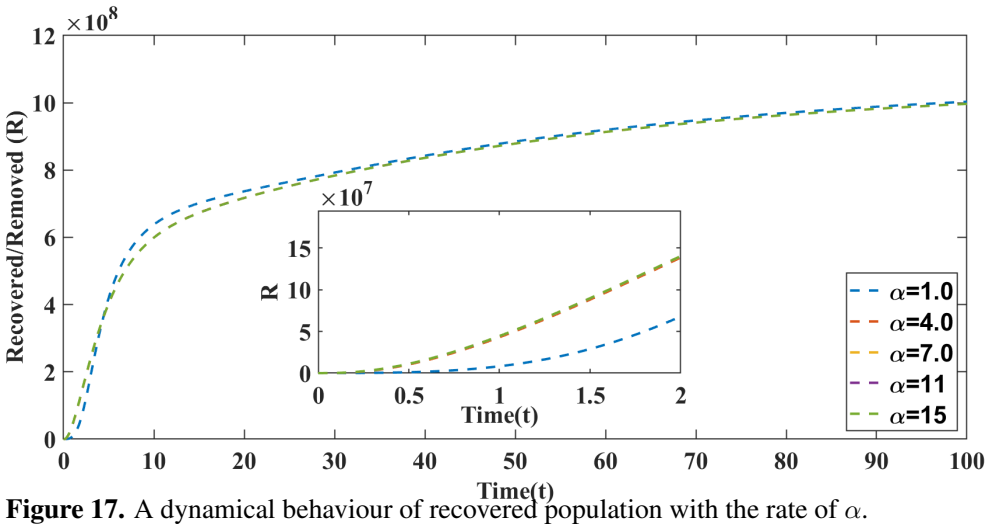

Figure 17. A dynamical behaviour of recovered population with the rate of α.

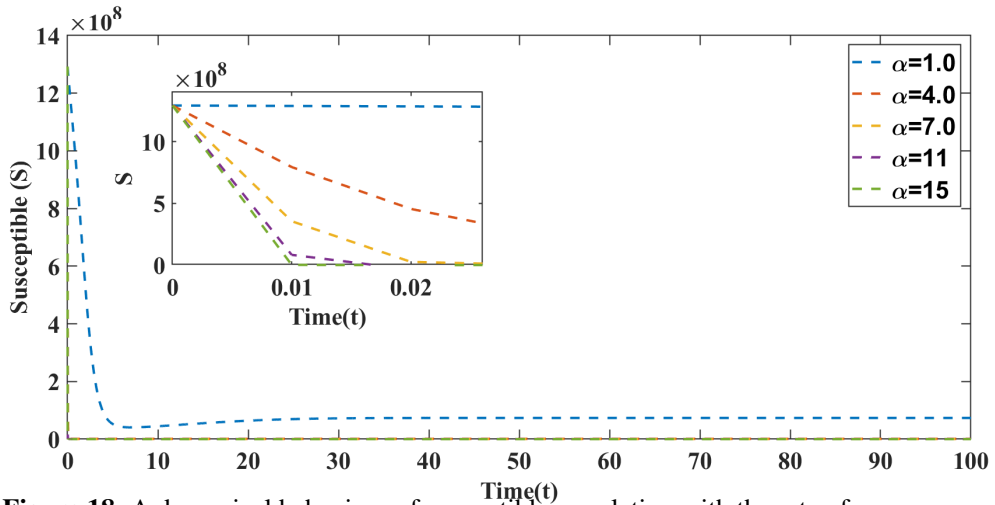

Figure 18. A dynamical behaviour of susceptible population with the rate of α.

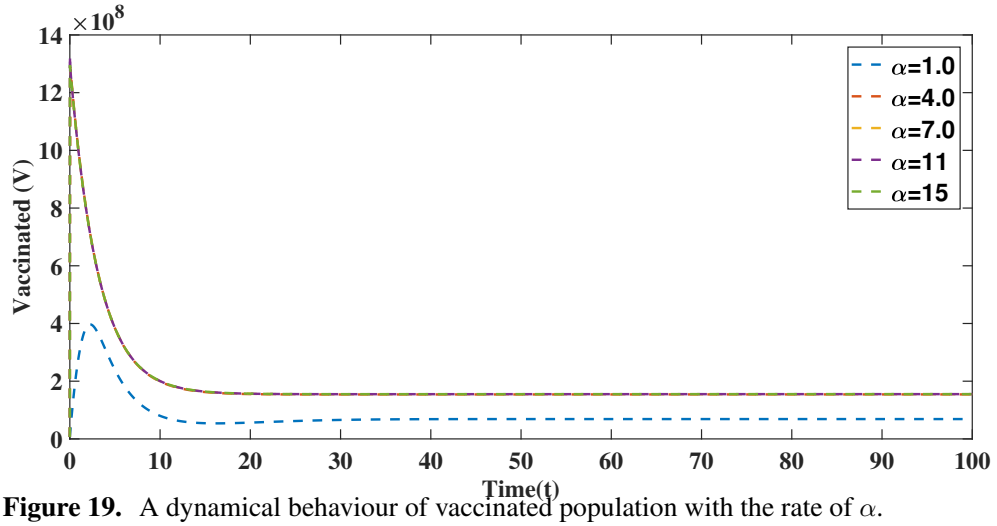

Figure 19. A dynamical behaviour of vaccinated population with the rate of α.

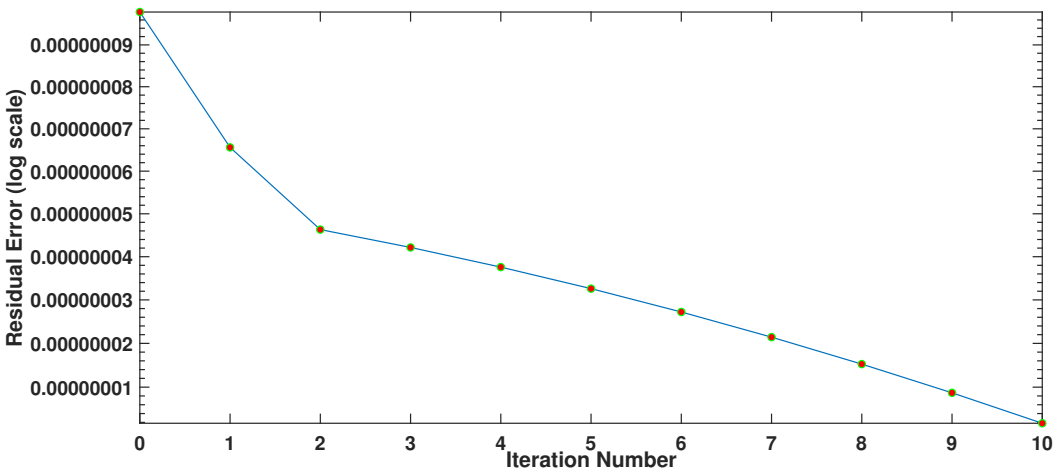

Figure 20. Convergence plot of residual error vs. iteration number for HPM.

Table 1. Values of parameters in the model.

Parameter	Value	Source	Parameter	Value	Source
Λ	16540000	[24, 3]	λ_2	0.5	[3]
π	$(0, 1)$	Fitted	λ_3	0.39	[3]
σ	0.0001	Assumed	η_1	3.4	[3]
ζ_1	0.05	Assumed	ζ_2	0.0001	Assumed
μ	0.007	[24, 3]	η_2	3.4	[3]
α	1	[3]	η_3	3.4	[3]
ω	0.1	[25, 3]	γ_2	0.0461	[3]
λ_1	0.11	[3]	γ_3	0.0461	Assumed
τ_1	0.2	Assumed	τ_2	0.2	Assumed
ρ	4	[25, 3]	q	0.08805	[25, 3]
β	1×10^{-9}	[3]	γ_1	0.0461	[3]
ϵ	4	[3]			

68, 737. We obtain numerical results by substituting all the parameters and initial values into the system using MATLAB2021. We generate graphs for the variables using the HPM.

A detailed analysis of the total population dynamics over time is shown in Figure 4. This figure provides a comprehensive view of the model, showing the changes and trends at different time intervals.

Figures 5, 6, 7, and 8 illustrate the behavior of individuals affected by hepatitis B under varying rates of vaccination uptake, contact rate, vaccination failure rate, and the rate at which recovered individuals become susceptible again (σ), respectively. These figures demonstrate that an increase in the vaccination rate (α) leads to a significant reduction in the proportion of the population critically affected by hepatitis B. Similarly, a decrease in the contact rate (β) and the vaccination failure rate (ω) further reduces the proportion of the critically affected population. Reducing the rate σ also decreases the number of critically affected individuals.

Figures 9, 10, 11, and 12 depict the long-term dynamics of hepatitis B infection under different rates of vaccination, contact, vaccination failure, and σ, respectively. These figures highlight that increasing the vaccination rate (alpha) reduces the proportion of the population with a long-term hepatitis B infection. Similarly, decreasing the contact rate (β) and the vaccination failure rate (ω) contributes to a lower proportion of long-term infections. Reducing the rate σ also decreases long-term hepatitis B cases.

Figures 13, 14, 15, and 16 show the proportion of potentially infectious hepatitis B cases under varying rates of vaccination, contact, vaccination failure, and σ, respectively. These figures indicate that an increase in the vaccination rate (α) significantly reduces the proportion of the population with potentially infectious cases. Similarly, a decrease in the contact rate (β) and the vaccination failure rate (ω) leads to a lower proportion of potentially infectious cases. A reduction in the rate σ also decreases the proportion of potentially infectious hepatitis B cases.

Figures 17, 18, and 19 represent the dynamic behavior of the vaccination uptake rate over time under varying conditions in the susceptible, vaccinated, and recovered classes, respectively. These figures illustrate how the rate of vaccination intake changes in different scenarios, highlighting the temporal variations and trends in vaccination behavior.

Figure 20 illustrates the convergence behavior of HPM applied to a compartmental model designed to capture the dynamics of HBV transmission within a human population. The x-axis represents the iteration number in the HPM, reflecting the progressive refinement of the semi-analytical solution for the model. The y-axis shows the residual error on a logarithmic scale, capturing the difference between successive approximations of the solution. The logarithmic scale highlights the rapid error reduction, signifying convergence towards a stable solution. The observed rapid decrease in residual error across iterations indicates that the HPM achieves a stable and accurate solution within a few iterations. This is crucial for computational efficiency and reliability when studying HBV dynamics. By combining the plot's geometric insights with the model parameters' sensitivity and complexity, this convergence plot substantiates the efficacy

of HPM as a powerful tool for approximating solutions in infectious disease modeling.

This study comprehensively analyzes the newly introduced parameters, offering context for the depicted dynamics. Specifically, This work illustrates the reproductive impact of π, which represents the proportion of susceptible individuals relative to vaccinated recruits $(1-\pi)$. Examined here for the first time, this parameter offers novel insights into how vaccination influences disease dynamics. Another key parameter, ω, representing the transmission rate from vaccinated individuals to the exposed population, is analyzed in Figures 2 and 3 through reproduction number diagrams. These reveal the significant effect of ω in controlling HBV. Moreover, Figures 7, 11, and 15 highlight how ω impacts the progression of the exposed class to acute, long-term, and chronic HBV populations. Collectively, these findings underscore the detailed extensions of the model, addressing important dynamics previously overlooked in the literature. By incorporating these parameters, the study reinforces the necessity of integrating vaccination and recovery processes into strategies to control the progression and impact of hepatitis B.

8 Conclusion

This paper focuses on studying the transmission dynamics of the hepatitis B virus worldwide. We formulated a mathematical model with eight compartments: susceptible, exposed, vaccinated, potentially infectious, chronic hepatitis B infectious, acute hepatitis B infectious, treated, and recovered individuals. We used the HPM to solve the dynamic model and find an analytical solution for hepatitis B. The HPM is a straightforward method that provides an approximate analytical expression, making diagnosing and interpreting the solution easy. This explicit solution is valuable for investigating the epidemic model of hepatitis B, as it helps in understanding the parameters in the numerical simulation section. Our study considered control strategies such as isolation and vaccination to prevent and intervene in the spread of the hepatitis B virus within a specific population, N. The interactions between vaccine failure rates, transmission rates, and vaccination rates significantly affect $\mathcal{R}_0$ and the dynamics of hepatitis B transmission. The insights from Figures 2 and 3 emphasize the importance of comprehensive vaccination strategies to control the disease's spread effectively. This approach can enhance population-wide immunity, reducing the potential for hepatitis B transmission.

The analysis of the proposed compartmental model for HBV dynamics identifies the transmission rates (ω and β) as the most influential parameters in determining the basic reproduction number and the overall spread of the disease. Additionally, the figures highlight the significant impact of ω, σ, and β on the system's dynamics. Effectively managing these parameters through vaccination programs, public health interventions, and strategies to minimize secondary infections is crucial for controlling the spread of HBV.

This study emphasizes the importance of increasing vaccination rates while reducing contact and vaccination failure rates to combat hepatitis B. By implementing robust vaccination initiatives, raising public awareness, enacting supportive policies, and strengthening healthcare infrastructure, communities, and governments can significantly mitigate the incidence and spread of hepatitis B, enhance public health, and safeguard future generations.

8.1 Model Advantages and Limitations

The proposed compartmental model for HBV dynamics provides a comprehensive framework to analyze disease progression by categorizing the population into eight compartments. Its adaptability allows tailoring to specific populations, enabling effective resource allocation and vaccination strategies. The integration of HPM ensures efficient semi-analytical solutions, making the model suitable for real-time applications in public health policy and epidemiological studies. However, its reliance on precise parameter estimation can be a limitation when high-quality data is unavailable. The model assumes uniform population mixing and may not capture heterogeneities like age, immunity levels, or socioeconomic factors. It must account for some biological complexities, such as immune responses or co-infections, which limits its applicability in multifactorial scenarios.

Declaration of competing 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.

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Data availability: No data was used for the research described in the article.

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