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Mathematical Analysis of the Transmission Dynamics of Hepatitis B Virus

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Abstract
Hepatitis B is a life-threatening hepatic illness induced by the Hepatitis B virus (HBV). This is a major worldwide health issue, especially in low- and middle-income nations in Africa and the Western Pacific, where prevalence rates are the greatest. Nevertheless, the existence of an efficacious vaccination, Hepatitis B persists in inflicting significant morbidity and death owing to a deficiency of awareness regarding the illness. Thus, we developed a deterministic mathematical model to elucidate the transmission dynamics of Hepatitis B, integrating elements such as vertical transmission, re-infection, and environmental viral concentration. The study starts with the calculation of the basic reproduction number and the assessment of the local stability of the disease-free equilibrium employing the Routh-Hurwitz criteria. A comprehensive examination of the model indicates that the model may experience backward bifurcation phenomena under some specific conditions. This trait presents considerable challenges in the proper management of Hepatitis B infection among the population. Assuming no re-infection of Hepatitis B post-recovery, the disease-free equilibrium point is globally asymptotically stable when the basic reproduction number is less than or equal to one. The sensitivity analysis of the basic reproduction number was conducted to assess the influence of each fundamental parameter in the model that contributes to disease transmission. Utilizing the optimal control theory to effectively curb the spread of Hepatitis B, we incorporated two time-varying control strategies, namely the prevention of susceptible individuals from acquiring HBV (through safe sex practice, regular washing of hands, and using protective hand gloves when handling blood, body fluid and semen) and the sensitization on individuals on personal hygiene, sterilization and proper disposal of medical and dental equipment like syringes in order to reduce the shedding of HBV in the environment. The numerical simulations indicated that Hepatitis B infection may be effectively managed and mitigated within the community if both control measures are correctly implemented.

Keywords Hepatitis B · Basic reproduction number · Stability · Sensitivity analysis · Bifurcation

1 Introduction

The Hepatitis B virus (HBV) causes an acute infection in humans that may evolve into a chronic condition, potentially resulting in liver cirrhosis and hepatocellular carcinoma (HCC), with the risk contingent upon the patient's age at the time of infection [1]. Worldwide, HBV is a major health issue, impacting around 250–260 million persons with chronic infections and resulting in roughly 1.1 million deaths each year from associated consequences [2]. As an oncogenic DNA virus from the Hepadnaviridae family, HBV infects liver hepatocytes by binding its surface antigen (HBsAg) to the sodium taurocholate co-transporting polypeptide (NTCP) receptor [3, 4]. This dangerous and possibly life-threatening disease can be averted with vaccination and effective therapies. It is typically spread through percutaneous or mucosal exposure to contaminated blood and body fluids [5]. The principal ways of HBV transmission vary according to the virus's frequency in different places. In places with high endemicity, the virus is mostly transmitted vertically from infected mothers to their babies at delivery. Horizontal transmission through close contact among children is widespread in locations with high or intermediate incidence citeHBV6. In low-endemicity areas, HBV infection often occurs during adolescence and early adulthood, commonly linked with high-risk behaviours such as unprotected sex and injectable drug use [5]. High HBV prevalence is notably widespread in many regions of the Asia-Pacific and sub-Saharan Africa.

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Approximately 45% of the world population inhabits these high-prevalence regions [7]. Regions exhibiting intermediate HBV prevalence (2–7%) encompass North Africa, the Middle East, certain areas of Eastern and Southern Europe, segments of Latin America, and South Asia, collectively representing a comparable share of the global population to that of high-prevalence regions (slightly exceeding 40%) [8]. In contrast, individuals in low-prevalence areas, such as Australia, parts of Asia, Northern and Western Europe, Japan, North America, and some South American countries, make up a minority of the global population (around 12%) [7]. Moreover, HBV may persist and retain its infectiousness on contaminated surfaces at ambient temperature for several weeks [9, 10]. HBV can be transmitted vertically from an infected mother to her child, through sexual contact with an infected partner, and by exposure to contaminated needles or sharp objects; however, it is not transmitted through breastfeeding, hugging, kissing, coughing, sneezing, or sharing food and beverages [11].

The HBV vaccine is the most efficacious and secure means of preventing exposure to the virus [11]. Since its introduction in 1982, the HBV vaccine has significantly reduced global HBV infection rates [12]. The vaccine can be administered from birth through adulthood. In the USA, three single-antigen vaccines-ENGERIX-B, RECOMBIVAX HB, and HEPLISAV-are licensed, along with two combination vaccines, PEDIARIX and TWINRIX [11]. Hepatitis B continues to be a significant global health concern, resulting in acute and chronic infections, severe liver failure, and malignancy, all of which contribute to high morbidity and mortality rates, despite the availability of effective vaccines and treatment strategies. Approximately 2 billion people worldwide have been infected with HBV, and an estimated 292 million are living with chronic HBV infection. HBV and associated disorders cause approximately 887,000 deaths annually, largely from advanced liver fibrosis and cirrhosis [11]. Many people with Hepatitis B are asymptomatic and may not be aware of their illness. Symptoms may include fever, exhaustion, lack of appetite, nausea, vomiting, dark urine, gray-colored faeces, joint pain, and skin and eye yellowing.

Various researchers, including mathematicians, have done studies to assist the community in preventing the widespread transmission of HBV. Zhang et al. [13] developed and qualitatively examined an epidemic model incorporating a time delay to investigate the transmission dynamics of Hepatitis B in Xinjiang, China. Their results indicated that, in the absence of vertical transmission, the endemic equilibrium is globally asymptotically stable. Additionally, they projected that, without more effective preventive measures, the cumulative number of Hepatitis B cases in Xinjiang could reach approximately 700,000 by the end of 2028. Xu et al. [14] developed and meticulously examined a mathematical model to evaluate prospective Hepatitis B infections and the effects of vaccination regimens in China. Their research indicated that raising the Hepatitis B vaccination rate markedly decreases the incidence of illnesses. Nevertheless, they determined that the prospective incidence of HBV infections remained sufficiently elevated, resulting in a continual increase in the number of Hepatitis B patients in China. Ullah et al. [15] developed and analyzed a mathematical model to investigate the dynamics of HBV, incorporating optimal control analysis with a focus on the hospitalized population. They introduced three time-varying control strategies: isolation of infected individuals, intensive public education and awareness campaigns, and treatment of hospitalized patients, along with early vaccination against HBV. Their findings demonstrated that the concurrent execution of all three possible controls might markedly decrease the incidence of infections. Khan et al. [16] introduced a mathematical model to analyse the transmission patterns of acute and chronic Hepatitis B in a community. Their research shows that interventions including treatment, isolation, and vaccination are efficacious in eliminating HBV. Khan et al. [17] developed and qualitatively examined a mathematical model for HBV transmission with a convex incidence rate. Liu et al. [18] introduced a numerical and fractional model for the dynamics of HBV transmission, employing non-singular and non-local kernels. They constructed an Atangana-Baleanu fractional HBV model that integrates the impacts of vaccination. Their findings indicated that vaccination is an efficient method for mitigating the transmission of Hepatitis B in the general population. Khatun et al. [19] developed a mathematical model for Hepatitis B infection incorporating cytotoxic T lymphocyte (CTL) immunological responses. Their mathematical study and numerical simulations demonstrated that CTL immune responses are essential for disease elimination. Tchinda et al. [20] proposed a mathematical model for HBV transmission that includes two concentrated delays modelled by a gamma distribution. Their investigation demonstrated that the model displays backward bifurcation, suggesting that lowering the basic reproduction number below one may be inadequate to manage the HBV pandemic. Wodajo and Mekonnen [21] introduced and examined a compartmental, non-linear deterministic model for the dynamics of HBV transmission. Their research shows that the synergistic effects of vaccination, efficient treatment, and disruption of transmission render the elimination of the illness attainable, perhaps culminating in the eradication of the virus. Several new research provide broader perspectives on epidemic dynamics, including the effects of sociocultural determinants, hospitalization, and public response [40–42, 45]. Other papers address periodic interventions and asymptomatic transmission [43, 44], which provide essential context to disease modeling efforts.

In this study, we extended the works of Ullah et al. [15] and Khan et al. [16] by incorporating vertical transmission

(i.e., mother-to-child transmission) and introducing a compartment to represent the concentration of Hepatitis B virus in the environment, considering that HBV can survive and remain infectious on moist surfaces at room temperature for several weeks [9, 10]. We conducted a comprehensive analysis of the model. Additionally, we introduced two time-dependent control measures: first, the prevention of HBV transmission among susceptible individuals through safe sex practices, regular handwashing, and the use of protective gloves when handling blood, body fluids, and semen; second, public sensitization on personal hygiene, sterilization, and proper disposal of medical and dental equipment such as syringes to reduce the environmental shedding of HBV. We then investigated the impact of these control measures on the HBV transmission dynamics. Notably, this study marks the first inclusion of a compartment for environmental HBV concentration in an epidemiological model in the literature. The subsequent sections of the paper are structured as follows: Sect. 2 delineates the model formulation and its essential features, Sect. 3 elucidates the model analysis, Sect. 4 examines the optimum control analysis, and Sect. 5 provides the final conclusion.

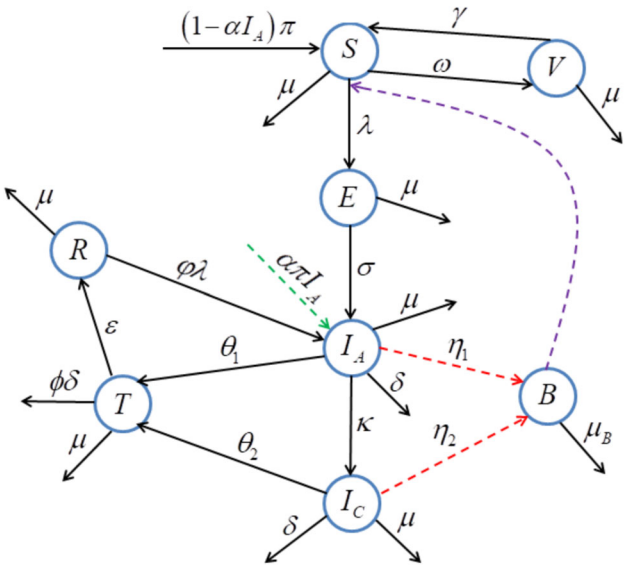


Fig. 1 Flowchart of the Hepatitis B model in (1)

2 Model Formulation and It Basic Properties

2.1 Model Formulation

At time t, we divide the Hepatitis B population into two namely, the human population, denoted by N, and the concentration of Hepatitis B virus in the environment, denoted by $B(t)$. The human population N is further sub-divided into seven mutual exclusive compartments of susceptible individuals $S(t)$, vaccinated individuals $V(t)$, exposed individuals $E(t)$, infected individuals with acute HBV infection $I_A(t)$, infected individuals with chronic HBV infection $I_C(t)$, treated individuals $T(t)$, and recovered individuals $R(t)$. So that

$$N(t) = S(t) + V(t) + E(t) + I_A(t) + I_C(t) + T(t) + R(t).$$

The model construction is based on the transmission diagram given in Fig. 1, and explained as follows. The population of susceptible individuals is increased by the recruitment of individuals via birth at a constant rate of $(1 - \alpha I_A)$, where α represents the proportion of infants infected with HBV. Vulnerable people are immunised against HBV at the rate ω. Susceptible people acquire HBV infection by effective contact with either infected persons or environmental concentrations of HBV at a rate λ, expressed as

$$\lambda = \frac{\beta(I_A + I_C)}{N} + \frac{\beta_e B}{K_B + B},$$

where $beta$ denotes the transmission probability from infected to susceptible persons, $beta_e$ represents the transmission probability from environmental HBV concentration to susceptible individuals, and K_B signifies the carrying capacity of the Hepatitis B virus in the environment. The rate γ represents the fading rate of the Hepatitis B vaccination. Susceptible individuals who encounter infected individuals or are exposed to a high concentration of the virus in the environment transition to the exposed class at a rate of λ, thereafter advancing to the acute infected compartment at a rate of σ. Individuals infected with acute HBV progressed to the chronic stage at a rate of κ. The rate μ represents the natural mortality rate of humans and is uniform across all human compartments. The parameter θ_1 denotes the treatment rate for persons with acute HBV infection, whereas θ_2 represents the treatment rate for those with chronic HBV infection. The rate δ represents the disease-induced mortality rate of persons infected with HBV. Individuals receiving therapy succumb to the illness at a rate of $\phi\delta$, where ϕ is the modification parameter that quantifies the decrease in HBV mortality attributable to treatment. An individual undergoing treatment recovers from HBV infection at a rate of ε. Individuals who have recovered are re-infected with HBV at a rate of $\varphi\lambda$, where φ is the parameter that adjusts for re-infection. The environmental concentration of Hepatitis B virus is produced by the shedding of the virus from persons infected with acute and chronic HBV at rates η_1 and η_2, respectively. The rate μ_B denotes the decay rate of the Hepatitis B virus in the environment (Table 1).

Base on the above formulations and assumptions, the dynamics of the Hepatitis model is governed by a system

Table 1 Description of the model variables and parameters

Variable	Description
S	Group of susceptible individuals
V	Group of vaccinated individuals against Hepatitis B
E	Group of exposed individuals to Hepatitis B
I_A	Group of infected individuals with acute Hepatitis B
I_C	Group of infected individuals with chronic Hepatitis B
T	Group of treated individuals
R	Group of recovered individuals
B	Concentration of Hepatitis B virus in the environment
Parameter	**Description**
π	Recruitment rate
α	Proportion of individuals infected with Hepatitis B at birth
β	Human to human transmission rate
β_e	Rate of successful of transmission from the environment to susceptible persons
μ	Human natural death rate
θ_1	Treatment rate of individual with acute Hepatitis B
θ_2	Treatment rate of individual with chronic Hepatitis B
ω	Vaccination rate
γ	Waning rate of Hepatitis B vaccine
σ	Transition rate from E to I_A
δ	Disease-induced death rate of individuals with Hepatitis B
κ	Transition rate from I_A to I_C
ϕ	Modification parameter accounting for the reduction in Hepatitis B mortality within the treatment class
ε	Recovery rate individuals in T
μ_B	Decay rate of hepatitis B virus
φ	Modification parameter addressing re-infection in recovered individuals
η_1	Shedding rate of Hepatitis B virus by acute infected individuals in the environment
η_2	Shedding rate of Hepatitis B virus by chronic infected individuals in the environment
K_B	Carrying capacity

of non-linear differential equations given by

$$\begin{aligned}
\frac{dS}{dt} &= (1-\alpha I_A)\,\pi - \lambda S - (\omega+\mu)S + \gamma V,\\
\frac{dV}{dt} &= \omega S - (\gamma+\mu)V,\\
\frac{dE}{dt} &= \lambda S - (\sigma+\mu)E,\\
\frac{dI_A}{dt} &= \sigma E + \alpha\pi I_A - (\theta_1+\kappa+\delta+\mu)I_A + \varphi\lambda R,\\
\frac{dI_C}{dt} &= \kappa I_A - (\theta_2+\delta+\mu)I_C,\\
\frac{dT}{dt} &= \theta_1 I_A + \theta_2 I_C - (\varepsilon+\phi\delta+\mu)T,\\
\frac{dR}{dt} &= \varepsilon T - (\varphi\lambda+\mu)R,\\
\frac{dB}{dt} &= \eta_1 I_A + \eta_2 I_C - \mu_B B,
\end{aligned} \tag{1}$$

where

$$\lambda = \frac{\beta(I_A+I_C)}{N} + \frac{\beta_e B}{K_B+B}.$$

It is essential to highlight several key assumptions made during the model formulation:

1. HBV can be transmitted vertically from an infected mother to her child during pregnancy. ([11]).
2. HBV can be transmitted from the environment to vulnerable persons. Hepatitis B can remain viable outside the body for as long as 7 days [9, 10].
3. Natural mortality is uniform across all epidemiological compartments.
4. The population is homogenous, suggesting that all individuals have an identical probability of catching the virus with adequate exposure to infected individuals.

2.2 Basic Properties of the Model

2.2.1 Positivity of Solution

For the Hepatitis B model (1) to possess a biological meaning, the solution of the system must be non-negative for all time values t. Consequently, it is imperative to establish that all state variables of the Hepatitis B model (1) remain positive for all $t > 0$ inside the feasible area Ω, defined as follows:

$$\Omega = \Omega_H \cup \Omega_B \subset \Re_+^7 \times \Re_+^1. \tag{2}$$

where

$$\Omega_H = \left\{ (S, V, E, I_A, I_C, T, R) \in \Re_+^7 : N \leq \frac{\pi}{\mu} \right\},$$

and

$$\Omega_B = \left\{ B \in \Re_+^1 : B \leq \frac{\eta^*}{\mu_B} \left(\frac{\pi}{\mu} \right) \right\}.$$

Theorem 1 *Let the initial data for the Hepatitis B model (1) be $S(0) > 0$, $V(0) \geq 0$, $E(0) \geq 0$, $I_A(0) \geq 0$, $I_C(0) \geq 0$, $T(0) \geq 0$, $R(0) \geq 0$, and $B(0) \geq 0$. Then, the solution $(S, V, E, I_A, I_C, T, R, B)$ of the Hepatitis B model (1) is non-negative for all time $t > 0$.*

Proof Let $t_f = \sup\{t > 0 : (S > 0, V > 0, E > 0, I_A > 0, I_C > 0, T > 0, R > 0, B > 0) \in [0, t]\}$. Thus, $t_f > 0$.

From the first equation of Hepatitis B model system (1), we have

$$\frac{dS}{dt} = (1 - \alpha I_A)\pi - \lambda S - (\omega + \mu)S + \gamma V,$$

Solving the above equation, we obtained

$$\begin{aligned} &\frac{d}{dt}\left\{ S(t)\left[\exp\left(\int_0^t \lambda(x)dx + (\omega + \mu)t \right) \right] \right\} \\ &= ((1 - \alpha I_A)\pi + \gamma V) \exp\left(\int_0^t \lambda(x)\, dx + (\omega + \mu)t \right) \end{aligned} \tag{3}$$

Integrating the above equation at the range $[0, t_f]$, we obtained

$$\begin{aligned} &\left\{ S(t) \exp\left[\int_0^{t_f} \lambda(x)dx + (\omega + \mu)t_f \right] \right\} - S(0) \\ &= ((1 - \alpha I_A)\pi + \gamma V) \int_0^{t_f} \exp\left[\int_0^y \lambda(x)dx + (\omega + \mu)y \right] dy \end{aligned} \tag{4}$$

So that

$$\begin{aligned} S(t) &= S(0) \exp\left[-\left(\int_0^{t_f} \lambda(x)dx + (\omega + \mu)t_f \right) \right] \\ &+ \exp\left[-\left(\int_0^{t_f} \lambda(x)dx + (\omega + \mu)t_f \right) \right] \times \\ &((1 - \alpha I_A)\pi + \gamma V) \int_0^{t_f} \exp\left[\int_0^y \lambda(x)dx + (\omega + \mu)y \right] dy > 0 \end{aligned} \tag{5}$$

Similarly, it can be shown that $V > 0$, $E > 0$, $I_A > 0$, $I_C > 0$, $T > 0$, $R > 0$, $B > 0$. □

2.2.2 Invariant Region

Lemma 1 *The region $\Omega = \Omega_H \cup \Omega_B \subset \Re_+^7 \times \Re_+^1$ is positively invariant and attracts all solution in $\Re_+^8$.*

Proof By summing the equations for the human population and the concentration of Hepatitis B virus in the environmental compartments of the Hepatitis B model (1), the rates of change for the human population and the concentration of the Hepatitis B virus in the environment are expressed as follows:

$$\frac{dN}{dt} = \pi - \mu N - \delta(I_A + I_C) - \phi\delta T,$$

and

$$\frac{dB}{dt} = \eta_1 I_A + \eta_2 I_C - \mu_B B.$$

We have that

$$\frac{dN}{dt} \leq \pi - \mu N,$$

and

$$\frac{dB}{dt} \leq \eta^* \left(\frac{\pi}{\mu} \right) - \mu_B B.$$

Where $\eta^* = \max(\eta_1, \eta_2)$.

A standard comparison theorem described in [22] can be used to show that

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}\left(1 - e^{-\mu t}\right),$$

and

$$B(t) \leq B(0)e^{-\mu_B t} + \frac{\eta^*}{\mu_B}\left(\frac{\pi}{\mu} \right)\left(1 - e^{-\mu_B t}\right).$$

Consequently, if $N(0) \leq \frac{\pi}{\mu}$ and $B(0) \leq \frac{\eta^*}{\mu_B}\left(\frac{\pi}{\mu}\right)$, then it follows that $N(t) \leq \frac{\pi}{\mu}$ and $B(t) \leq \frac{\eta^*}{\mu_B}\left(\frac{\pi}{\mu}\right)$. Consequently, the area Ω is positively invariant and attracts all solutions in $\mathbb{R}^8_+$. Consequently, the Hepatitis B model (1) is both biologically and mathematically well-posed inside the domain Ω. Therefore, it is crucial to analyze the dynamics of the Hepatitis B model (1) inside the region Ω [23]. □

3 Model Analysis

3.1 Disease-Free Equilibrium

The disease-free equilibrium of system (1) is given by the following:

$$\begin{aligned}\xi_0 &= \left(S^*, V^*, E^*, I_A^*, I_C^*, T^*, R^*, B^*\right)\\ &= \left(\frac{\pi(\gamma+\mu)}{\mu(\gamma+\omega+\mu)}, \frac{\pi\omega}{\mu(\gamma+\omega+\mu)}, 0, 0, 0, 0, 0, 0\right).\end{aligned} \tag{6}$$

By adding S^* and V^* at ξ_0, the total population in the disease-free equilibrium is precisely defined as $\frac{\pi}{\mu}$, which only depends on the recruitment and natural death rates.

3.2 Basic Reproduction Number

The basic reproduction number of Hepatitis B, $\mathscr{R}_0$, quantifies the potential for HBV transmission within a population. Mathematically, $\mathscr{R}_0$ serves as a threshold parameter for the stability of a disease-free equilibrium and is associated with the peak and ultimate magnitude of an epidemic. It is defined as the anticipated number of recurring infections arising from a single incidence among a fully susceptible population. If $\mathscr{R}_0 < 1$, a few number of infected people introduced into a wholly susceptible population would, on average, be unable to sustain transmission, hence inhibiting the spread of the illness. When $\mathscr{R}_0 > 1$, the population of infected people increases with each generation, resulting in the propagation of the infections. The basic reproduction number of HBV may be determined using the next-generation operator method as outlined in [24]. Utilising the methodology outlined in [24], the non-negative matrix $\mathcal{F}$ and the non-singular matrix $\mathcal{V}$, corresponding to the new infection and the residual transition terms respectively, at the disease-free equilibrium are presented as follows:

$$\mathcal{F} = \begin{bmatrix} 0 & \frac{\beta S^*}{S^*+V^*} & \frac{\beta S^*}{S^*+V^*} & 0 & \frac{\beta S^*}{K_B} \\ 0 & \alpha\pi & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \quad \text{and}$$

$$\mathcal{V} = \begin{bmatrix} (\sigma+\mu) & 0 & 0 & 0 & 0 \\ -\sigma & (\theta_1+\kappa+\delta+\mu) & 0 & 0 & 0 \\ 0 & -\kappa & (\theta_2+\delta+\mu) & 0 & 0 \\ 0 & -\theta_1 & -\theta_2 & (\varepsilon+\phi\delta+\mu) & 0 \\ 0 & -\eta_1 & -\eta_2 & 0 & \mu_B \end{bmatrix}$$

Thus, as stated in [24], it follows that $\mathscr{R}_0 = \rho\left(\mathcal{F}\mathcal{V}^{-1}\right)$, where ρ denotes the spectral radius or the largest eigenvalue of the matrix $\mathcal{F}\mathcal{V}^{-1}$.

Thus,

$$\begin{aligned}\mathscr{R}_0 &= \frac{\beta\sigma(\gamma+\mu)(\kappa+P_5)}{(\gamma+\omega+\mu)P_3P_4P_5} + \frac{\alpha\pi}{P_4}\\ &\quad + \frac{\beta_e\pi\sigma(\gamma+\mu)(\eta_1P_5+\eta_2\kappa)}{K_B\mu(\gamma+\omega+\mu)P_3P_4P_5\mu_B}\end{aligned} \tag{7}$$

where

$P_1 = \omega+\mu$, $P_2 = \gamma+\mu$, $P_3 = \sigma+\mu$, $P_4 = \theta_1+\kappa+\delta+\mu$, $P_5 = \theta_2+\delta+\mu$, and $P_6 = \varepsilon+\phi\delta+\mu$.

It is important to emphasize that the Hepatitis B basic reproduction number ($\mathscr{R}_0$) comprises three components: the HBV horizontal basic reproduction number, the HBV vertical basic reproduction number, and the HBV basic reproduction number from the environment. This can be expressed as follows:

$$\mathscr{R}_0 = \mathscr{R}_0^H + \mathscr{R}_0^V + \mathscr{R}_0^B, \tag{8}$$

where $\mathscr{R}_0^H = \frac{\beta\sigma(\gamma+\mu)(\kappa+P_5)}{(\gamma+\omega+\mu)P_3P_4P_5}$ is the horizontal basic reproduction number, $\mathscr{R}_0^V = \frac{\alpha\pi}{P_4}$ is the vertical basic reproduction number, and $\mathscr{R}_0^B = \frac{\beta_e\pi\sigma(\gamma+\mu)(\eta_1P_5+\eta_2\kappa)}{K_B\mu(\gamma+\omega+\mu)P_3P_4P_5\mu_B}$ is the basic reproduction number from the environment.

3.3 Local Stability of the Disease-Free Equilibrium

Theorem 2 *Model (1) is locally asymptotically stable if* $\mathscr{R}_0 < 1$, *and unstable otherwise at disease-free equilibrium.*

Proof The evaluation of the model's Jacobian matrix (1) at the disease-free equilibrium (ξ_0) is provided by

$$\mathcal{J}(\xi_0) = \begin{bmatrix} -P_1 & \gamma & 0 & -\frac{\beta P_2}{(\gamma+\omega+\mu)} & -\frac{\beta P_2}{(\gamma+\omega+\mu)} & 0 & 0 & -\frac{\beta_e \pi P_2}{K_B \mu(\gamma+\omega+\mu)} \\ \omega & -P_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -P_3 & \frac{\beta P_2}{(\gamma+\omega+\mu)} & \frac{\beta P_2}{(\gamma+\omega+\mu)} & 0 & 0 & \frac{\beta_e \pi P_2}{K_B \mu(\gamma+\omega+\mu)} \\ 0 & 0 & \sigma & \alpha\pi - P_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \kappa & -P_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & \theta_1 & \theta_2 & -P_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \varepsilon & -\mu & 0 \\ 0 & 0 & 0 & \eta_1 & \eta_2 & 0 & 0 & -\mu_B \end{bmatrix}$$

where

$P_1 = \omega+\mu$, $P_2 = \gamma+\mu$, $P_3 = \sigma+\mu$, $P_4 = \theta_1+\kappa+\delta+\mu$, $P_5 = \theta_2 + \delta + \mu$, and $P_6 = \varepsilon + \phi\delta + \mu$.

The Jacobian matrix $\mathcal{J}(\xi_0)$ above has the following eigenvalues: $\lambda_1 = -\mu$, $\lambda_2 = -P_6$, and the characteristic polynomial's root is provided below.

$$\mathcal{G}(\lambda) = \lambda^6 + \mathscr{A}_1\lambda^5 + \mathscr{A}_2\lambda^4 + \mathscr{A}_3\lambda^3 + \mathscr{A}_4\lambda^2 + \mathscr{A}_5\lambda + \mathscr{A}_6 = 0 \tag{9}$$

where the values of $\mathscr{A}_1$, $\mathscr{A}_2$, $\mathscr{A}_3$, $\mathscr{A}_4$, $\mathscr{A}_5$, and $\mathscr{A}_6$ are in Appendix 1

All roots of the polynomial (9) contain negative real parts if and only if the coefficient $\mathscr{A}_i > 0$, for $i = 1, 2, 3, 4, 5, 6$, according to the Routh-Hurwitz criterion [25]. It goes without saying that if $\mathscr{A}_6 > 0$, then $\mathscr{R}_0 < 1$. According to the Routh-Hurwitz criterion, the Hepatitis B model's disease-free equilibrium (1) is thus locally asymptotically stable when $\mathscr{R}_0 < 1$. □

Epidemiologically, Theorem 2 indicates that a small influx of HBV-infected individuals into the population will not result in an outbreak if $\mathscr{R}_0 < 1$. It is important to note that this conclusion is influenced by the initial sizes of the infected individuals within the population.

3.4 Endemic Equilibrium

This section examined the presence of the endemic equilibrium point of our model in system (1). The endemic equilibrium point is a positive steady-state solution characterized by non-zero infected variables, indicating the persistent presence of the disease within the population. We analyze the model system (1) with respect to the force of infection (λ) to calculate the endemic equilibrium point:

$$\lambda^{**} = \frac{\beta(I_A^{**} + I_C^{**})}{N^{**}} + \frac{\beta_e B^{**}}{K_B + B^{**}} \tag{10}$$

where

$$N^{**} = S^{**} + V^{**} + E^{**} + I_A^{**} + I_C^{**} + T^{**} + R^{**}$$

with

$$\begin{aligned}
S^{**} &= \frac{\pi P_2 P_3 \left((\varphi\lambda^{**}+\mu)(P_4-\alpha\pi)P_5P_6 - \lambda^{**}\varphi\varepsilon(\theta_1 P_5+\theta_2\kappa)\right)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)} \\
V^{**} &= \frac{\pi\omega P_3 \left((\varphi\lambda^{**}+\mu)(P_4-\alpha\pi)P_5P_6 - \lambda^{**}\varphi\varepsilon(\theta_1 P_5+\theta_2\kappa)\right)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)} \\
E^{**} &= \frac{\lambda\pi P_2 \left((\varphi\lambda^{**}+\mu)(P_4-\alpha\pi)P_5P_6 - \lambda^{**}\varphi\varepsilon(\theta_1 P_5+\theta_2\kappa)\right)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)} \\
I_A^{**} &= \frac{\lambda\pi\sigma P_2P_5P_6(\varphi\lambda^{**}+\mu)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)} \\
I_C^{**} &= \frac{\lambda\pi\sigma\kappa P_2P_6(\varphi\lambda^{**}+\mu)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)} \\
T^{**} &= \frac{\lambda\pi\sigma P_2(\varphi\lambda^{**}+\mu)(\theta_1P_5+\theta_2\kappa)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)} \\
R^{**} &= \frac{\lambda\pi\sigma\varepsilon P_2(\theta_1P_5+\theta_2\kappa)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)} \\
B^{**} &= \frac{\lambda\pi\sigma P_2P_6(\varphi\lambda^{**}+\mu)(\eta_1P_5+\eta_2\kappa)}{(\varphi\lambda^{**}+\mu)\left(((\lambda^{**}+P_1)P_2-\gamma\omega)(P_4-\alpha\pi)P_3+\lambda^{**}\pi\alpha\sigma P_2\right)P_5P_6 - \lambda^{**}\varphi\varepsilon P_3(\theta_1P_5+\theta_2\kappa)((\lambda^{**}+P_1)P_2-\gamma\omega)}
\end{aligned} \tag{11}$$

and

$P_1 = \omega+\mu$, $P_2 = \gamma+\mu$, $P_3 = \sigma+\mu$, $P_4 = \theta_1+\kappa+\delta+\mu$, $P_5 = \theta_2 + \delta + \mu$, and $P_6 = \varepsilon + \phi\delta + \mu$.

Substituting Eq. (11) into (10), we obtained (40) in Appendix 2. From Eq. (40), it is evident that $U_1 > 0$ (since all model parameters are non-negative). Additionally, $U_5 > 0$ whenever $\mathscr{R}_0 < 1$. Therefore, the number of possible positive real roots of the polynomial (40) is contingent on the signs of U_2, U_3, and U_4. This can be analyzed using Descartes' rule of signs applied to the quartic equation $f(x) = U_1x^4+U_2x^3+U_3x^2+U_4x+U_5 = 0$ (with $x = \lambda^{**}$). Consequently, the following results are established.

Theorem 3 *The Hepatitis B model (1)*

1. *Has a unique endemic equilibrium if $\mathscr{R}_0 > 1$ and either of the following holds:*

   (i) $U_2 > 0, U_3 > 0, U_4 > 0$;
   (ii) $U_2 < 0, U_3 < 0, U_4 < 0$;
   (iii) $U_2 > 0, U_3 < 0, U_4 < 0$;
   (iv) $U_2 > 0, U_3 > 0, U_4 < 0$;

2. *Could have more than one endemic equilibrium if $\mathscr{R}_0 > 1$ and either of the following:*

   (i) $U_2 < 0, U_3 > 0, U_4 < 0$;
   (ii) $U_2 < 0, U_3 < 0, U_4 > 0$;
   (iii) $U_2 > 0, U_3 < 0, U_4 > 0$;
   (iv) $U_2 < 0, U_3 > 0, U_4 > 0$;

3. *Could have 2 or more endemic equilibria if $\mathscr{R}_0 < 1$ and any, or all, U_2, U_3, and U_4 are negative.*

Point 3 of Theorem 3 demonstrates the possibility of several endemic equilibria when $\mathscr{R}_0 < 1$, which often implies the occurrence of backward bifurcation [26–29]. Backward bifurcation refers to the simultaneous occurrence of a stable disease-free equilibrium (DFE) and a stable endemic equilibrium when the model's reproduction number is below one. This behaviour has been recorded in many disease transmission scenarios, encompassing vector-borne illnesses, inadequate immunisation, and multi-group interactions. The occurrence of this phenomenon in our model in(1) meticulously examined in the subsequent section.

3.5 Possibility of the Existence of Backward Bifurcation

This section will examine the potential for a backward bifurcation, utilising the centre manifold theorem as extensively analyzed by Castillo-Chavez and Song in their study [29].

Theorem 4 ***(Castillo-Chavez and Song theorem).*** *Let us consider a general system of ordinary differential equations with a parameter φ;*

$$\frac{dx}{dt} = f(x, \varphi), \; f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n, \; f \in \mathbf{C}^2\left(\mathbb{R}^n \times \mathbb{R}\right) \quad (12)$$

where $x = 0$ is an equilibrium point for the system in equation (12). That is, $f(0, \varphi) \equiv 0 \; \forall \; \varphi$.

Assume the following:

H_1: $A = D_x f(0, 0) = \left[\frac{\partial f}{\partial x}(0, 0)\right]$ is the linearization matrix of the system given by equation (12) around the equilibrium 0 with φ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts.

H_2: Matrix A has a non-negative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.

Let f_k be the k^{th} component of f and

$$\begin{aligned} a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0, 0), \\ b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(0, 0). \end{aligned} \quad (13)$$

The local dynamics of (12) around 0 are totally determined by the sign of a and b.

1. $a > 0, b > 0$: When $\varphi < 0$ with $|\varphi| << 1$, 0 is locally asymptotically stable and there exist a positive unstable equilibrium; when $0 < \varphi << 1$, 0 is unstable and there exist a negative, locally asymptotically stable equilibrium.
2. $a < 0, b < 0$: When $\varphi < 0$ with $|\varphi| << 1$, 0 is unstable; when $0 < \varphi << 1$, 0 is locally asymptotically stable equilibrium, and there exist a positive unstable equilibrium.
3. $a > 0, b < 0$: When $\varphi < 0$ with $|\varphi| << 1$, 0 is unstable, and there exist a locally asymptotically stable negative equilibrium; when $0 < \varphi << 1$, 0 is stable and a positive unstable equilibrium appears.
4. $a < 0, b > 0$: When φ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.

In particular, if $a < 0$ and $b > 0$, then the bifurcation is forward; if $a > 0$ and $b > 0$, then the bifurcation is backward. Using this approach, the following result may be obtained.

Theorem 5 *The model of system (1) exhibits backward bifurcation at $\mathscr{R}_0 = 1$.*

Proof To apply the centre manifold theorem, we will modify the state variables of the Hepatitis B model (1). We define the variables as follows: $S = x_1$, $V = x_2$, $E = x_3$, $I_A = x_4$, $I_C = x_5$, $T = x_6$, $R = x_7$, and $B = x_8$. Using vector notation, we express the state as $x = (x_1, x_2, x_3, \ldots, x_8)^T$ and the system of equations as $\frac{dx}{dt} = F(x)$, where $F = (f_1, f_2, f_3, \ldots, f_8)^T$. Thus, the Hepatitis B model (1) can

be rewritten as follows:

$$\begin{aligned}
\frac{dx_1}{dt} &\equiv f_1 = (1-\alpha x_4)\,\pi - \lambda x_1 - (\omega+\mu)x_1 + \gamma x_2,\\
\frac{dx_2}{dt} &\equiv f_2 = \omega x_1 - (\gamma+\mu)x_2,\\
\frac{dx_3}{dt} &\equiv f_3 = \lambda x_1 - (\sigma+\mu)x_3,\\
\frac{dx_4}{dt} &\equiv f_4 = \sigma x_3 + \alpha\pi x_4 - (\theta_1+\kappa+\delta+\mu)x_4 + \varphi\lambda x_7,\\
\frac{dx_5}{dt} &\equiv f_5 = \kappa x_4 - (\theta_2+\delta+\mu)x_5,\\
\frac{dx_6}{dt} &\equiv f_6 = \theta_1 x_4 + \theta_2 x_5 - (\varepsilon+\phi\delta+\mu)x_6,\\
\frac{dx_7}{dt} &\equiv f_7 = \varepsilon x_6 - (\varphi\lambda+\mu)x_7,\\
\frac{dx_8}{dt} &\equiv f_8 = \eta_1 x_4 + \eta_2 x_5 - \mu_B x_8.
\end{aligned} \tag{14}$$

where

$$\lambda = \frac{\beta(x_4+x_5)}{x_1+x_2+x_3+x_4+x_5+x_6+x_7} + \frac{\beta_e x_8}{K_B+x_8}.$$

Considering $\beta = \beta^*$, as the bifurcation parameter at $\mathscr{R}_0 = 1$, we have

$$\beta^* = \frac{K_B\mu(\gamma+\omega+\mu)P_3P_4P_5\mu_B - \alpha\pi K_B\mu(\gamma+\omega+\mu)P_3P_5\mu_B - \beta_e\pi\sigma(\gamma+\mu)(\eta_1P_5+\eta_2\kappa)}{K_B\mu\sigma\mu_B(\gamma+\mu)(\kappa+P_5)} \tag{15}$$

Evaluating the Jacobian of the transformed system (14) at disease-free equilibrium (ξ_0) with $\beta = \beta^*$, we have

$$\mathcal{J}(\xi_0)|_{\beta=\beta^*} = \begin{bmatrix}
-P_1 & \gamma & 0 & -\frac{\beta^*P_2}{(\gamma+\omega+\mu)} & -\frac{\beta^*P_2}{(\gamma+\omega+\mu)} & 0 & 0 & -\frac{\beta_e\pi P_2}{K_B\mu(\gamma+\omega+\mu)}\\
\omega & -P_2 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & -P_3 & \frac{\beta^*P_2}{(\gamma+\omega+\mu)} & \frac{\beta^*P_2}{(\gamma+\omega+\mu)} & 0 & 0 & \frac{\beta_e\pi P_2}{K_B\mu(\gamma+\omega+\mu)}\\
0 & 0 & \sigma & \alpha\pi - P_4 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \kappa & -P_5 & 0 & 0 & 0\\
0 & 0 & 0 & \theta_1 & \theta_2 & -P_6 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & \varepsilon & -\mu & 0\\
0 & 0 & 0 & \eta_1 & \eta_2 & 0 & 0 & -\mu_B
\end{bmatrix}$$

where

$P_1 = \omega+\mu$, $P_2 = \gamma+\mu$, $P_3 = \sigma+\mu$, $P_4 = \theta_1+\kappa+\delta+\mu$, $P_5 = \theta_2+\delta+\mu$, and $P_6 = \varepsilon+\phi\delta+\mu$.

The right eigenvector, denoted as $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8)^T$, corresponding to the simple zero eigenvalue, can be derived from $\mathcal{J}(\xi_0)|_{\beta=\beta^*} w = 0$, given by

$$\begin{aligned}
&-P_1w_1 + \gamma w_2 - \frac{\beta^*P_2}{(\gamma+\omega+\mu)}w_4 - \frac{\beta^*P_2}{(\gamma+\omega+\mu)}w_5\\
&- \frac{\beta_e\pi P_2}{K_B\mu(\gamma+\omega+\mu)}w_8 = 0\\
&\omega w_1 - P_2w_2 = 0\\
&-P_3w_3 + \frac{\beta^*P_2}{(\gamma+\omega+\mu)}w_4 + \frac{\beta^*P_2}{(\gamma+\omega+\mu)}w_5\\
&+ \frac{\beta_e\pi P_2}{K_B\mu(\gamma+\omega+\mu)}w_8 = 0\\
&\sigma w_3 + (\alpha\pi - P_4)w_4 = 0\\
&\kappa w_4 - P_5w_5 = 0\\
&\theta_1w_4 + \theta_2w_5 - P_6w_6 = 0\\
&\varepsilon w_6 - \mu w_7 = 0\\
&\eta_1w_4 + \eta_2w_5 - \mu_Bw_8 = 0
\end{aligned} \tag{16}$$

From Eq. (16), we obtained

$$\begin{aligned}
&w_1 = w_1 > 0,\ w_2 = \frac{\omega w_1}{P_2},\ w_3 = \frac{(P_4-\alpha\pi)w_4}{\sigma},\\
&w_4 = w_4 > 0,\ w_5 = \frac{\kappa w_4}{P_5},\\
&w_6 = \frac{(\theta_1P_5+\theta_2\kappa)w_4}{P_5P_6},\ w_7 = \frac{\varepsilon(\theta_1P_5+\theta_2\kappa)w_4}{\mu P_5P_6},\ \textit{and}\\
&w_8 = \frac{\varepsilon(\eta_1P_5+\eta_2\kappa)w_4}{\mu P_5}.
\end{aligned} \tag{17}$$

Likewise, the left eigenvector, denoted as $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8)$, satisfying $v.w = 1$, associated with the simple zero eigenvalue, can be determined from $vJ\,(\xi_0)\,|_{\beta=\beta^*} = 0$, given by

$$\begin{aligned}
&-P_1v_1 + \omega v_2 = 0\\
&\gamma v_1 - P_2v_2 = 0\\
&-P_3v_3 + \sigma v_4 = 0\\
&-\frac{\beta^* P_2}{(\gamma+\omega+\mu)}v_1 + \frac{\beta^* P_2}{(\gamma+\omega+\mu)}v_3 + (\alpha\pi - P_4)v_4 + \kappa v_5 + \theta_1 v_6 + \eta_1 v_8 = 0\\
&-\frac{\beta^* P_2}{(\gamma+\omega+\mu)}v_1 + \frac{\beta^* P_2}{(\gamma+\omega+\mu)}v_3 - P_5v_5 + \theta_2 v_6 + \eta_2 v_8 = 0\\
&-P_6v_6 + \varepsilon v_7 = 0\\
&-\mu v_7 = 0\\
&-\frac{\beta_e \pi P_2}{K_B\mu(\gamma+\omega+\mu)}v_1 + \frac{\beta_e \pi P_2}{K_B\mu(\gamma+\omega+\mu)}v_3 - \mu v_8 = 0
\end{aligned} \tag{18}$$

solving the above equation (18) above, we obtained

$$\begin{aligned}
&v_6 = v_7 = 0,\ v_1 = v_1 > 0,\ v_2 = \frac{(\gamma - P_1)v_1}{P_2 - \omega},\ v_3 = v_3 > 0,\ v_4 = \frac{P_3v_3}{\sigma},\\
&v_5 = \frac{(P_4 - \alpha\pi)P_3v_3}{(\kappa + P_5)\sigma} + \frac{\beta_e\pi P_2(\eta_2 - \eta_1)(v_3 - v_1)}{K_B\mu\mu_B(\gamma+\omega+\mu)(\kappa+P_5)},\ v_8 = \frac{\beta_e\pi P_2(v_3 - v_1)}{K_B\mu\mu_B(\gamma+\omega+\mu)}.
\end{aligned} \tag{19}$$

Computation of *a* and *b*

Since $v_6 = v_7 = 0$ for $k = 1, 2, 3, ..., 8$, the only non-zero partial derivatives are given in Appendix 3.

But

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}\,(\xi_0)$$

$$\begin{aligned}
a = {} & \frac{2(v_1 - v_3)w_4^2\beta^*\mu(\gamma+\mu)(P_5+\kappa)\left((P_4 - \alpha\pi)\mu P_5P_6 + \sigma(\theta_1P_5 + \theta_2\kappa)(\varepsilon+\mu)\right)}{\pi\sigma\mu P_5^2P_6(\gamma+\omega+\mu)} +\\
& \frac{2(v_1 - v_3)w_1w_4\beta^*\omega\mu(\gamma+\mu)(P_5+\kappa)}{\pi P_2P_5(\gamma+\omega+\mu)} + \frac{2(v_3 - v_1)w_1w_4\beta^*\omega\mu(P_5+\kappa)}{\pi P_5(\gamma+\omega+\mu)} +\\
& \frac{2(v_3 - v_1)w_1w_4\beta_e\varepsilon(\eta_1P_5 + \eta_2\kappa)}{K_B\mu_BP_5} + \frac{4(v_1 - v_3)w_4^2\beta^*\kappa\mu(\gamma+\mu)}{\pi P_5(\gamma+\omega+\mu)} +\\
& \frac{2(v_1 - v_3)w_4^2\beta^*\mu(\gamma+\mu)(P_5^2+\kappa^2)}{\pi P_5^2(\gamma+\omega+\mu)} + \frac{2(v_1 - v_3)w_4^2\beta_e\pi(\gamma+\mu)\varepsilon^2(\eta_1P_5 + \eta_2\kappa)^2}{K_B\mu\mu_B^2P_5^2(\gamma+\omega+\mu)}.
\end{aligned} \tag{20}$$

and

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha_d^*}\,(\xi_0)$$

$$b = \frac{(v_3 - v_1)w_4(\gamma+\mu)(P_5+\kappa)}{P_5(\gamma+\omega+\mu)} > 0 \tag{21}$$

Given that the bifurcation coefficient b is positive, Theorem 4.1 in [29] indicates that the Hepatitis B model in (1) demonstrates a backward bifurcation at $\mathscr{R}_0 = 1$ whenever the bifurcation coefficient a is also positive. Please see Fig. 2 for the illustration of backward bifurcation. □

3.6 Global Stability of the Disease-Free Equilibrium: Special Case

Several key factors contributing to backward bifurcation include ineffective vaccination, vector-borne infections, and the recurrence of disease in previously recovered individuals. Models accounting for re-infection in recovered populations tend to lose the backward bifurcation trait when re-infection

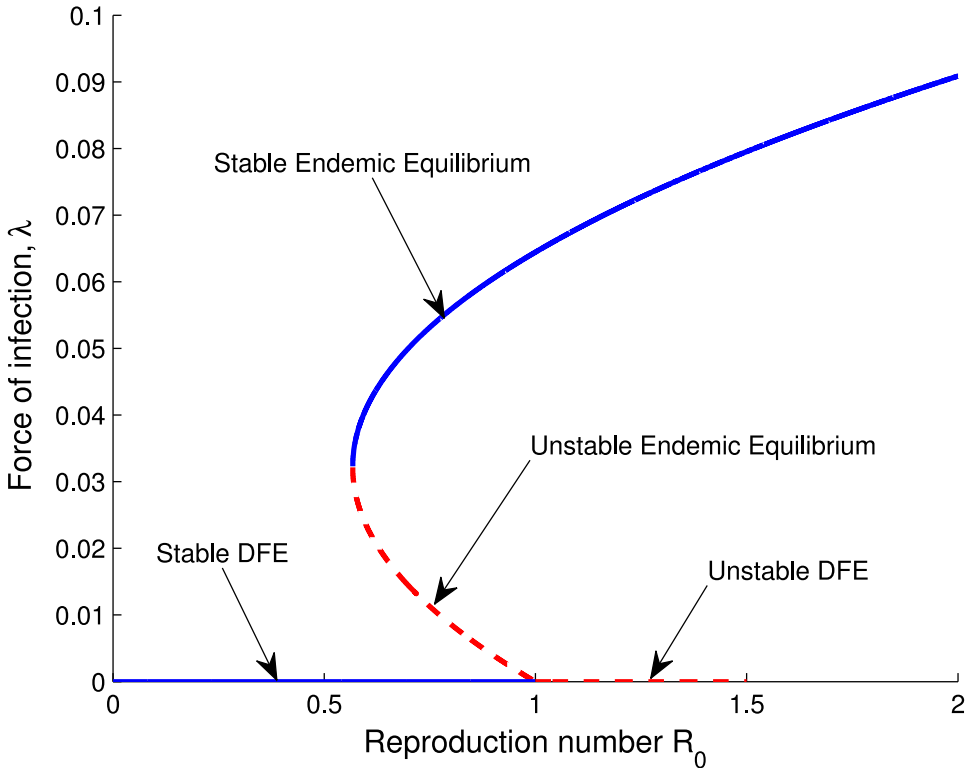


Fig. 2 Backward bifurcation of the Hepatitis B model

parameters are nullified. Therefore, this study will examine the influence of re-infection (φ) on backward bifurcation. Accordingly, the HBV model can be reformulated as follows.

$$\begin{aligned} \frac{dX}{dt} &= \mathcal{P}(X, Y), \\ \frac{dY}{dt} &= \mathcal{Q}(X, Y) \end{aligned} \tag{22}$$

where $X = (S, V, R)$ and $Y = (E, I_A, I_C, T, B)$ with $X \in \mathbb{R}+^3$ indicating the count of uninfected compartments and $Y \in \mathbb{R}+^5$ representing the total infected compartments. Let $\xi_0 = (X^*, 0)$ denote the disease-free equilibrium state of the system. ξ_0 is a globally asymptotically stable equilibrium for the model if it fulfills the conditions $\mathcal{W}1$ and $\mathcal{W}2$ described below.

$$\begin{aligned} &\mathcal{W}_1 : \frac{dX}{dt} = \mathcal{P}(X, 0), X^* \quad is \quad globally \quad asymptotically \quad stable \\ &\mathcal{W}_2 : \frac{dY}{dt} = \mathcal{D}_Y \mathcal{Q}(X^*, 0)Y - \hat{\mathcal{Q}}(X, Y), \hat{\mathcal{Q}}(X, Y) \geq 0 \quad \forall \quad (X, Y) \in \Omega, \end{aligned} \tag{23}$$

where $\mathcal{D}Y\mathcal{Q}(X^*, 0)$ represents the Jacobian matrix of $Q(X, Y)$ with respect to (E, IA, I_C, T, B), evaluated at $(X^*, 0) = \left(\left(\frac{\pi(\gamma+\mu)}{\mu(\gamma+\omega+\mu)}, \frac{\pi\omega}{\mu(\gamma+\omega+\mu)}, 0\right), 0\right)$. If the system (22) meets these conditions, then the theorem below is valid.

Theorem 6 *The equilibrium points $\xi_0 = (X^*, 0)$ of the system (22) is globally asymptotically stable if $\mathscr{R}_0 \leq 1$, and condition $\mathcal{W}_1$ and $\mathcal{W}_2$ are satisfied.*

Proof From the system (1), we obtained $\mathcal{P}(X, Y)$ and $\mathcal{Q}(X, Y)$

$$\mathcal{P}(X, Y) = \begin{bmatrix} (1 - \alpha I_A)\pi - \left(\frac{\beta(I_A+I_C)}{N} + \frac{\beta_e B}{K_B+B}\right) S - (\omega + \mu)S + \gamma V \\ \omega S - (\gamma + \mu)V \\ \varepsilon T - \mu R, \end{bmatrix}$$

$$\mathcal{Q}(X, Y) = \begin{bmatrix} \left(\frac{\beta(I_A+I_C)}{N} + \frac{\beta_e B}{K_B+B}\right) S - (\sigma + \mu)E \\ \sigma E + \alpha\pi I_A - (\theta_1 + \kappa + \delta + \mu)I_A \\ \kappa I_A - (\theta_2 + \delta + \mu)I_C \\ \theta_1 I_A + \theta_2 I_C - (\varepsilon + \phi\delta + \mu)T \\ \eta_1 I_A + \eta_2 I_C - \mu_B B \end{bmatrix}$$

Now, we examine $\frac{dX}{dt} = \mathcal{P}(X^*, 0)$, which represents the reduced system derived from the condition $\mathcal{W}_1$:

$$\begin{aligned} \frac{dS}{dt} &= \pi - (\omega + \mu)S + \gamma V, \\ \frac{dV}{dt} &= \omega S - (\gamma + \mu)V, \\ \frac{dR}{dt} &= 0. \end{aligned} \tag{24}$$

$X = \left(\frac{\pi(\gamma+\mu)}{\mu(\gamma+\omega+\mu)}, \frac{\pi\omega}{\mu(\gamma+\omega+\mu)}, 0\right)$ is a globally asymptotically stable equilibrium for the reduced system $\frac{dX}{dt} = \mathcal{P}(X, 0)$ in (24). This asymptotic behaviour remains invariant to the starting condition inside Ω. Thus, the solution of the simplified system (24) demonstrates global convergence in Ω. We compute

$$\mathcal{D}_Y \mathcal{Q}(X^*, 0) = \begin{bmatrix} -(\sigma+\mu) & \frac{\beta(\gamma+\mu)}{(\gamma+\omega+\mu)} & \frac{\beta(\gamma+\mu)}{(\gamma+\omega+\mu)} & 0 & \frac{\beta_e \pi(\gamma+\mu)}{K_B \mu(\gamma+\omega+\mu)} \\ \sigma & \alpha\pi - (\theta_1+\kappa+\delta+\mu) & 0 & 0 & 0 \\ 0 & \kappa & -(\theta_2+\delta+\mu) & 0 & 0 \\ 0 & \theta_1 & \theta_2 & -(\varepsilon+\phi\delta+\mu) & 0 \\ 0 & \eta_1 & \eta_2 & 0 & -\mu_B \end{bmatrix}$$

The sensitivity index of the basic reproduction number for the Hepatitis B model (1) concerning the parameter 'q' can be expressed as

$$\mathcal{S}q^{\mathcal{R}0} = \frac{\partial \mathcal{R}0}{\partial q} \times \frac{q}{\mathcal{R}0}. \tag{26}$$

Thus, the sensitivity indices for the basic reproduction number in relation to the key parameters β are computed as follows:

$$\mathcal{S}_\beta^{\mathcal{R}_0} = \frac{\beta\sigma(\gamma+\mu)(\kappa+\theta_2+\delta+\mu)K_B\mu\mu_B}{K_B\mu\mu_B\left(\beta\sigma(\gamma+\mu)(\kappa+\theta_2+\delta+\mu)+\alpha\pi(\gamma+\omega+\mu)(\sigma+\mu)(\theta_2+\delta+\mu)\right)+\beta_e\pi\sigma(\gamma+\mu)(\eta_1(\theta_2+\delta+\mu)+\eta_2\kappa)}$$

$$\hat{\mathcal{Q}}(X, Y) = \begin{bmatrix} \beta(I_A+I_C)\left(\frac{(\gamma+\mu)}{(\gamma+\omega+\mu)} - \frac{S}{N}\right) + \beta_e B\left(\frac{\pi(\gamma+\mu)}{K_B\mu(\gamma+\omega+\mu)} - \frac{S}{K_B+B}\right) \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

It is apparent that $\hat{\mathcal{Q}}(X, Y)$ holds for every $(X, Y) \in \Omega$, given that $\frac{S}{N} \leq \frac{(\gamma+\mu)}{(\gamma+\omega+\mu)}$ and $\frac{S}{K_B+B} \leq \frac{\pi(\gamma+\mu)}{K_B\mu(\gamma+\omega+\mu)}$. Consequently, by utilizing Lasalle's invariance principle [30], it can be concluded that the disease-free equilibrium is globally asymptotically stable when $\mathcal{R}_0 \leq 1$. This result indicates that HBV eradication is not contingent upon the initial quantity of infected persons in the population. □

3.7 Sensitivity Analysis

This section concentrates on doing a sensitivity analysis of the important parameters affecting the basic reproduction number of the Hepatitis B model (8). The objective is to assess the importance of each element in influencing the transmission dynamics of Hepatitis B described in equation (1). To achieve this objective, we employ the methodology outlined in [31]. Utilising the methods outlined in [31], we use the normalised forward sensitivity index of a variable represented by 'H', which differentially varies on the parameter 'q', and is articulated as

$$\mathcal{S}_q^H = \frac{\partial H}{\partial q} \times \frac{q}{H}. \tag{25}$$

Substituting the parameter values described in previous section in to $\mathcal{S}_\beta^{\mathcal{R}_0}$ gives

$$\mathcal{S}_\beta^{\mathcal{R}_0} = 0.9806. \tag{27}$$

In similar reasoning, we obtained the sensitivity indices of the remaining basic parameters that constitute the basic reproduction number and are given by

$$\begin{aligned} &\mathcal{S}_{\beta_e}^{\mathcal{R}_0} = 0.0001161,\ \mathcal{S}_\gamma^{\mathcal{R}_0} = 0.7575,\ \mathcal{S}_\kappa^{\mathcal{R}_0} = 0.0646,\ \mathcal{S}_{K_B}^{\mathcal{R}_0} = -0.0001161, \\ &\mathcal{S}_{\eta_1}^{\mathcal{R}_0} = 0.0000471,\ \mathcal{S}_{\eta_2}^{\mathcal{R}_0} = 0.0000691,\ \mathcal{S}_\delta^{\mathcal{R}_0} = -0.1293,\ \mathcal{S}_\sigma^{\mathcal{R}_0} = 0.05633, \\ &\mathcal{S}_{\theta_1}^{\mathcal{R}_0} = -0.4308,\ \mathcal{S}_{\theta_2}^{\mathcal{R}_0} = -0.4839,\ \mathcal{S}_\alpha^{\mathcal{R}_0} = 0.0193,\ \mathcal{S}_\pi^{\mathcal{R}_0} = 0.0194, \\ &\mathcal{S}_\omega^{\mathcal{R}_0} = -0.8129,\ \mathcal{S}_{\mu_B}^{\mathcal{R}_0} = -0.0001161,\ \mathcal{S}_\mu^{\mathcal{R}_0} = -0.0216. \end{aligned} \tag{28}$$

The results are illustrated to provide a clearer perspective on comparing the sensitivity indices of each parameter, as shown in Fig. 3.

3.8 Interpretation of the Sensitivity Indices

Figure 3 illustrates the sensitivity indices of the basic reproduction number for the Hepatitis B model outlined in (1). Parameters with positive indices substantially influence the dissemination of the illness. An increase in these parameters, with other aspects held constant, leads to a significant elevation in the basic reproduction number. Thus, elevated levels

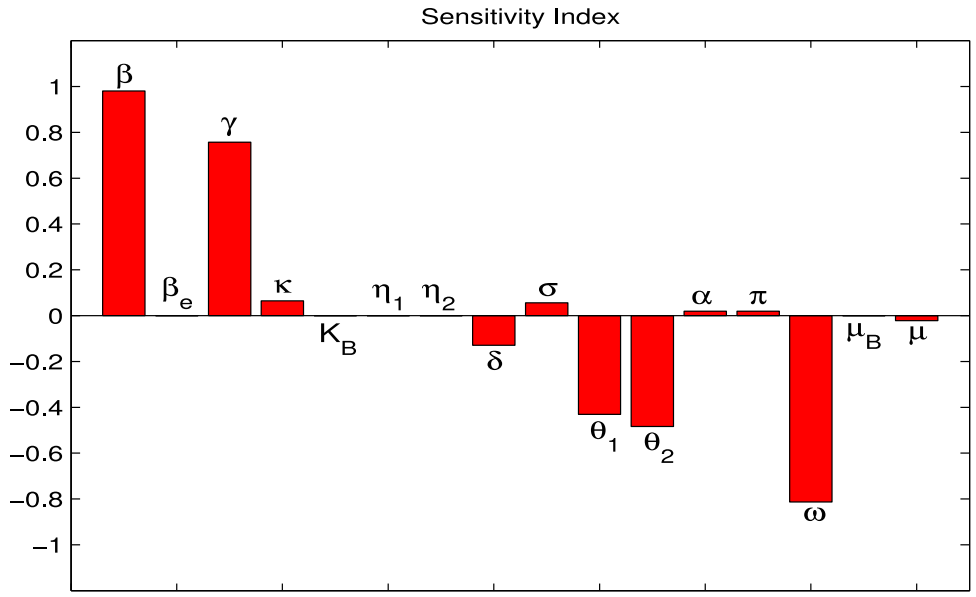


Fig. 3 Sensitivity index of the basic reproduction number of the Hepatitis B model

of these factors facilitate disease transmission. In contrast, factors with negative indices are essential for alleviating the illness load. An escalation in these components results in a diminution of the basic reproduction number, signifying a decline in the transmission of the illness.

Figure 4a, b, and c depicts the effects of the vaccination rate (ω), the treatment rate for individuals with acute HBV infection (θ_1), and the treatment rate for individuals with chronic HBV infection (θ_2) on the cumulative incidence of new HBV infections, respectively. The findings indicate that an elevation in the vaccination rate (ω) and the treatment rates (θ_1 and θ_2) resulted in a substantial decrease in the total new cases of HBV infection. Moreover, it is clear that the vaccination rate (ω) significantly influences the reduction of cumulative HBV cases more than the treatment rates (θ_1 and θ_2). These results align with the sensitivity analysis outcomes of the basic reproduction number detailed in Sect. 3.7.

4 Optimal Control Theory

Optimal control theory serves as a vital approach in epidemiological modeling, aiding in the identification of effective strategies to mitigate the spread of infections, as highlighted in [32]. In this analysis, we will explore an optimal intervention strategy involving two time-varying controls: the prevention of susceptible individuals from acquiring HBV (through safe sex practices, regular handwashing, and the use of protective gloves when handling blood, bodily fluids, and semen), denoted as $u_1(t)$, and the sensitization of individuals regarding personal hygiene, sterilization, and proper disposal of medical and dental equipment, such as syringes, to reduce the shedding of HBV in the environment, denoted as $u_2(t)$. With these controls, the Hepatitis B model can be expressed as follows:

$$\begin{aligned}
\frac{dS}{dt} &= (1-\alpha I_A)\,\pi - (1-u_1(t))\lambda S - (\omega+\mu)S + \gamma V,\\
\frac{dV}{dt} &= \omega S - (\gamma+\mu)V,\\
\frac{dE}{dt} &= (1-u_1(t))\lambda S - (\sigma+\mu)E,\\
\frac{dI_A}{dt} &= \sigma E + \alpha\pi I_A - (\theta_1+\kappa+\delta+\mu)I_A + (1-u_1(t))\varphi\lambda R,\\
\frac{dI_C}{dt} &= \kappa I_A - (\theta_2+\delta+\mu)I_C,\\
\frac{dT}{dt} &= \theta_1 I_A + \theta_2 I_C - (\varepsilon+\phi\delta+\mu)T,\\
\frac{dR}{dt} &= \varepsilon T - ((1-u_1(t))\varphi\lambda+\mu)R,\\
\frac{dB}{dt} &= (1-u_2(t))(\eta_1 I_A + \eta_2 I_C) - \mu_B B,
\end{aligned} \tag{29}$$

where

$$\lambda = \frac{\beta(I_A+I_C)}{N} + \frac{\beta_e B}{K_B+B}.$$

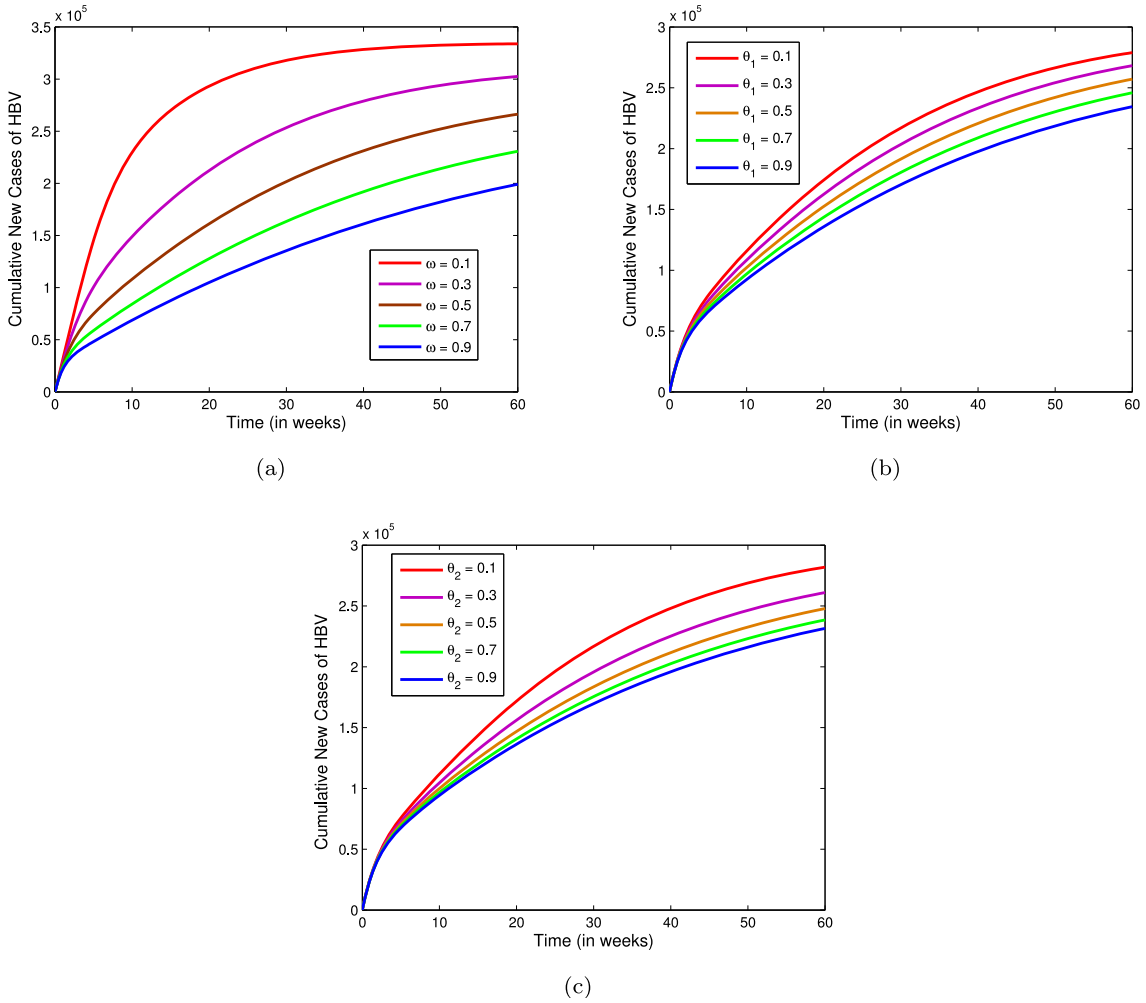


Fig. 4 Simulation of **a** the effect of the vaccination rate (ω) on the cumulative new cases of HBV, **b** the effect of the treatment rate of acute HBV-infected individuals (θ_1) on the cumulative new cases of HBV, and **c** the effect of the treatment rate of chronic HBV-infected individuals (θ_2) on the cumulative new cases of HBV

For this, we consider the objective functional

$$J[u_1, u_2] = \int_0^{t_f} \left[D_1 I_A + D_2 I_C + \frac{1}{2}\left(D_3 u_1^2(t) + D_4 u_2^2(t) \right) \right] dt, \tag{30}$$

With t_f denoting the final time, the coefficients D_1, D_2, D_3, and D_4 function as weight factors to maintain equilibrium among the terms in the integrand (30), therefore averting the predominance of any single term. The term $D_1 I_A + D_2 I_C$ encompasses the expenses associated with the management of infected individuals across all phases, whereas $D_3 u_1^2(t) + D_4 u_2^2(t)$ denotes the costs related to public health education initiatives designed to enhance awareness regarding personal hygiene and preventive strategies for Hepatitis B infection. Our aim is to reduce both infected individuals and the expenses associated with deploying control measures. The objective is to determine an ideal pair of controls, $u_1(t)$ and $u_2(t)$, such that

$$J[u_1^*, u_2^*] = \min_{u_1, u_2 \in \Omega^*} J[u_1, u_2] \tag{31}$$

where the control set (Ω^*) is defined as

$$\Omega^* = \{(u_1(t), u_2(t)) \in \mathcal{L}^1(0, t_f) | a_1 \leq u_1(t) \geq b_1, a_2 \leq u_2(t) \geq b_2\} \tag{32}$$

is lesbgue measurable.

4.1 Characterization of the Optimal Control Model

The necessary condition for an optimal control pair to be satisfied is derived from Pontryagin's maximum principle

[33]. This principle transforms the problem defined in (29) and (30) into one of pointwise minimization of a Hamiltonian ($\mathcal{H}$) with respect to the control pair $u_1(t)$ and $u_2(t)$. The Hamiltonian is expressed as follows:

$$\begin{aligned}
\mathcal{H} &= D_1 I_A + D_2 I_C + \frac{1}{2}\left(D_3 u_1^2(t) + D_4 u_2^2(t)\right) + \\
&\Delta_1\left[(1-\alpha I_A)\pi - (1-u_1(t))\left(\frac{\beta(I_A+I_C)}{N}\right.\right. \\
&\left.\left. + \frac{\beta_e B}{K_B+B}\right) S - (\omega+\mu)S + \gamma V\right] + \\
&\Delta_2\left[\omega S - (\gamma+\mu)V\right] + \Delta_3\left[(1-u_1(t))\left(\frac{\beta(I_A+I_C)}{N}\right.\right. \\
&\left.\left. + \frac{\beta_e B}{K_B+B}\right) S - (\sigma+\mu)E\right] + \\
&\Delta_4\left[\sigma E + \alpha\pi I_A - (\theta_1+\kappa+\delta+\mu)I_A\right. \\
&\left. + (1-u_1(t))\varphi\left(\frac{\beta(I_A+I_C)}{N} + \frac{\beta_e B}{K_B+B}\right) R\right] + \\
&\Delta_5\left[\kappa I_A - (\theta_2+\delta+\mu)I_C\right] + \Delta_6\left[\theta_1 I_A\right. \\
&\left. + \theta_2 I_C - (\varepsilon+\phi\delta+\mu)T\right] + \\
&\Delta_7\left[\varepsilon T - (1-u_1(t))\varphi\left(\frac{\beta(I_A+I_C)}{N}\right.\right. \\
&\left.\left. + \frac{\beta_e B}{K_B+B}\right) R - \mu R\right] + \\
&\Delta_8\left[(1-u_2(t))(\eta_1 I_A + \eta_2 I_C) - \mu_B B\right],
\end{aligned} \tag{33}$$

where Δ_1, Δ_2, Δ_3, Δ_4, Δ_5, Δ_6, Δ_7, and Δ_8 are the adjoint functions associated with the state variables of the model (29). By employing Pontryagin's Maximum Principle [33] and leveraging the existence results for the optimal control pair $u_1(t)$ and $u_2(t)$, the following theorem established:

Theorem 7 *There exists an optimal control pair $u_1^*(t)$ and $u_2^*(t)$, and corresponding solution (S^*, V^*, E^*, I_A^*, I_C^*, T^*, R^*, and B^*) that minimize $J(u_1(t), u_2(t))$ over Ω. Furthermore, there exist adjoint functions, Δ_i, ($i = 1, 2, 3, ..., 8$) such that*

$$\begin{aligned}
\frac{d\Delta_1}{dt} &= \left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)}{N^*} - \frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right.\right. \\
&\left.\left. + \frac{\beta_e B^*}{K_B+B^*}\right) + (\omega+\mu)\right]\Delta_1 - \omega\Delta_2 \\
&- \left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)}{N^*} - \frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right.\right. \\
&\left.\left. + \frac{\beta_e B^*}{K_B+B^*}\right)\right]\Delta_3 \\
&+ \left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_4 \\
&- \left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_7, \\
\frac{d\Delta_2}{dt} &= -\left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right) - \gamma\right]\Delta_1 \\
&+ (\gamma+\mu)\Delta_2 + \left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_3 + \\
&\left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_4 \\
&- \left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_7, \\
\frac{d\Delta_3}{dt} &= -\left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right) - \gamma\right]\Delta_1 \\
&+ \left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right) + (\sigma+\mu)\right]\Delta_3 - \\
&\left[\sigma - (1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_4 \\
&- \left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_7, \\
\frac{d\Delta_4}{dt} &= -D_1 + \left[\alpha\pi + (1-u_1(t))\left(\frac{\beta S^*}{N^*}\right.\right. \\
&\left.\left. - \frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_1 - \kappa\Delta_5 - \theta_1\Delta_6 - \\
&\left[(1-u_1(t))\left(\frac{\beta S^*}{N^*} - \frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_3 \\
&+ \left[(1-u_1(t))\varphi\left(\frac{\beta R^*}{N^*} - \frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_7 + \\
&\left[(\theta_1+\kappa+\delta+\mu) - \alpha\pi - (1-u_1(t))\varphi\left(\frac{\beta R^*}{N^*}\right.\right. \\
&\left.\left. - \frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_4 - (1-u_2(t))\eta_1\Delta_8, \\
\frac{d\Delta_5}{dt} &= -D_2 + \left[(1-u_1(t))\left(\frac{\beta S^*}{N^*} - \frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_1 \\
&+ (\theta_2+\delta+\mu)\Delta_5 - \theta_2\Delta_6 - \\
&\left[(1-u_1(t))\left(\frac{\beta S^*}{N^*} - \frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_3 \\
&- \left[(1-u_1(t))\varphi\left(\frac{\beta R^*}{N^*} - \frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_4 \\
&+ \left[(1-u_1(t))\varphi\left(\frac{\beta R^*}{N^*} - \frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_7 \\
&- (1-u_2(t))\eta_2\Delta_8,
\end{aligned}$$

$$
\begin{aligned}
\frac{d\Delta_6}{dt} =& -\left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_1\\
&+\left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_3+\\
&\left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_4\\
&-\left[\varepsilon+(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right)\right]\Delta_7+\\
&(\varepsilon+\phi\delta+\mu)\Delta_6,\\
\frac{d\Delta_7}{dt} =& -\left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_1\\
&+\left[(1-u_1(t))\left(\frac{\beta(I_A^*+I_C^*)S^*}{N^{*2}}\right)\right]\Delta_3-\\
&\left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)}{N^*}-\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right.\right.\\
&\left.\left.+\frac{\beta_e B^*}{K_B+B^*}\right)\right]\Delta_4+\\
&\left[(1-u_1(t))\varphi\left(\frac{\beta(I_A^*+I_C^*)}{N^*}-\frac{\beta(I_A^*+I_C^*)R^*}{N^{*2}}\right.\right.\\
&\left.\left.+\frac{\beta_e B^*}{K_B+B^*}\right)+\mu\right]\Delta_7,\\
\frac{d\Delta_8}{dt} =& \left[(1-u_1(t))\left(\frac{\beta_e K_B S^*}{(K_B+B^*)^2}\right)\right]\Delta_1\\
&-\left[(1-u_1(t))\left(\frac{\beta_e K_B S^*}{(K_B+B^*)^2}\right)\right]\Delta_3-\\
&\left[(1-u_1(t))\varphi\left(\frac{\beta_e K_B R^*}{(K_B+B^*)^2}\right)\right]\Delta_4\\
&+\left[(1-u_1(t))\varphi\left(\frac{\beta_e K_B R^*}{(K_B+B^*)^2}\right)\right]\Delta_7+\mu_B\Delta_8.
\end{aligned}
\tag{34}
$$

With transversality conditions

$$
\Delta_i(t_f)=0, \qquad i=1,2,3,...,8. \tag{35}
$$

The following characterization holds:

$$
u_1^*(t)=\min\left\{\max\left(a_1,\mathscr{C}_1\right),b_1\right\} \tag{36}
$$

where

$$
\begin{aligned}
\mathscr{C}_1 =& \frac{1}{D_3}\left((\Delta_3-\Delta_1)\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)S^*\right.\\
&\left.+(\Delta_4-\Delta_7)\varphi\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)R^*\right).
\end{aligned}
$$

Furthermore,

$$
u_2^*(t)=\min\left\{\max\left(a_1,\frac{1}{D_4}(\eta_1 I_A^*+\eta_2 I_C^*)\Delta_8\right),b_1\right\}. \tag{37}
$$

Proposition 1 *Fleming et al. [34] establish the existence of an optimal control pair $u_1(t)$ and $u_2(t)$ based on the convexity of the integrand of J with respect to $u_1(t)$ and $u_2(t)$, the prior boundedness of the state solutions, and the local Lipschitz continuity of the model (29) with respect to its variables.*

Proof We apply the Pontryagin's maximum principles to have

$$
\begin{aligned}
\frac{d\Delta_1}{dt} &= -\frac{\partial\mathcal{H}}{\partial S}, & \Delta_1(t_f)&=0,\\
\frac{d\Delta_2}{dt} &= -\frac{\partial\mathcal{H}}{\partial V}, & \Delta_2(t_f)&=0,\\
\frac{d\Delta_3}{dt} &= -\frac{\partial\mathcal{H}}{\partial E}, & \Delta_3(t_f)&=0,\\
\frac{d\Delta_4}{dt} &= -\frac{\partial\mathcal{H}}{\partial I_A}, & \Delta_4(t_f)&=0,\\
\frac{d\Delta_5}{dt} &= -\frac{\partial\mathcal{H}}{\partial I_C}, & \Delta_5(t_f)&=0,\\
\frac{d\Delta_6}{dt} &= -\frac{\partial\mathcal{H}}{\partial T}, & \Delta_6(t_f)&=0,\\
\frac{d\Delta_7}{dt} &= -\frac{\partial\mathcal{H}}{\partial R}, & \Delta_7(t_f)&=0,\\
\frac{d\Delta_8}{dt} &= -\frac{\partial\mathcal{H}}{\partial B}, & \Delta_8(t_f)&=0,
\end{aligned}
\tag{38}
$$

Considering the optimality condition,

$$
\frac{\partial\mathcal{H}}{\partial u_1}=0, \textit{and} \quad \frac{\partial\mathcal{H}}{\partial u_2}=0. \tag{39}
$$

The optimal control pair u_1^* and u_2^* can be solved by solving $\frac{\partial\mathcal{H}}{\partial u_1}=0$ and $\frac{\partial\mathcal{H}}{\partial u_2}=0$, respectively, and combined with it lower and upper bound. The partial derivative of Hamiltonian function respect to u_1 and u_2 gives the following:

$$
\begin{aligned}
\frac{\partial\mathcal{H}}{\partial u_1} =& D_3u_1(t)+\Delta_1\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)S^*\\
&-\Delta_3\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)S^*\\
&-\Delta_4\varphi\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)R^*
\end{aligned}
$$

$$+\Delta_7\varphi\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)R^*,$$

$$\frac{\partial\mathcal{H}}{\partial u_2}=D_4u_2(t)-(\eta_1I_A^*+\eta_2I_C^*)\Delta_8.$$

Solving above equations respect to each control variables gives the following:

$$u_1^*(t)=\frac{1}{D_3}\left((\Delta_3-\Delta_1)\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)S^*\right.$$
$$\left.+(\Delta_4-\Delta_7)\varphi\left(\frac{\beta(I_A^*+I_C^*)}{N^*}+\frac{\beta_e B^*}{K_B+B}\right)R^*\right)$$

$$u_2^*(t)=\frac{1}{D_4}(\eta_1I_A^*+\eta_2I_C^*)\Delta_8.$$

Combined it with it lower and upper bound (a_i, b_i, respectively), gives (35) and (36). This end the proof. □

4.2 Numerical Illustration

We conduct numerical simulations for the optimal control model using the forward-backward sweep approach, as detailed in [35, 36], with parameter values specified in Table 2. The process starts with an initial guess of the control variables. The state system (29) is solved forward in time direction using the fourth-order Runge–Kutta technique, while the adjoint system (34) is resolved in a backward time direction. The controls are subsequently revised by including the new state and adjoint values into the control characterization. This iterative procedure is continued until convergence is attained. The initial conditions for the state variables used are $S(0)=360,000$, $V(0)=10,000$, $E(0)=60,000$, $I_A(0)=7000$, $I_C(0)=3000$, $T(0)=600$, $R(0)=300$, and $B(0)=5000$.

Table 2 Parameter values for model (1)

Parameter	Value	Source
π	0.056	[37]
α	0.11	[14]
β	0.8	[38]
β_e	0.21	Assumed
μ	0.007313	Estimated from [39]
γ	0.1	[14]
ω	0.52	[21]
σ	0.12	Assumed
κ	0.475	[21]
μ_B	0.57	Assumed
θ_1	0.4	[21]
θ_2	0.28	[21]
η_1	0.33	Assumed
η_2	0.34	Assumed
ε	0.32	[21]
ϕ	0.05	Assumed
δ	0.0461	[14]
φ	0.45	Assumed
K_B	10,000	Assumed

Strategy A: Control with u_1 only

Strategy B: Control with u_2 only

Strategy C: Control with Both u_1 and u_2.

Discussion of Results

Figure 5 is the simulations of the effect of the control $u_1(t)$ on the human population and the concentration of HBV in the environment. In the presence of the control $u_1(t)$, it is observed that the number of susceptible individuals increases above 50,000 within 20 weeks, the vaccinated individuals increases above 200,000 within 80 weeks, the exposed individuals reduces below 10,000 in 40 weeks, the infected individuals with acute HBV infection declined below 4,000 in 20 weeks, the infected individuals with chronic HBV infection decreases below 2,000 within 60 weeks, the treated individuals reduces below 5,000 in 50 weeks, the recovered individuals reduces below 60,000 within 40 weeks due to less individuals being exposed to HBV infection, and the concentration of HBV in the environment reduces below 4000 within 40 weeks.

Figure 6 depicts the simulations of the effect of the control $u_2(t)$ on the human population and the concentration of HBV in the environment. In the presence of the control $u_2(t)$, it is observed that the number of susceptible individuals increases above 40,000 within 20 weeks, the vaccinated individuals increases above 150,000 within 80 weeks, the exposed individuals reduces below 30,000 in 40 weeks, the infected individuals with acute HBV infection declined below 9000 in 20 weeks, the infected individuals with chronic HBV infection decreases below 6000 within 60 weeks, the treated individuals reduces below 15,000 in 50 weeks, the recovered individuals increases above 70,000 within 40 weeks due to less individuals being exposed to HBV infection, and the concentration of HBV in the environment reduces below 1800 within 40 weeks.

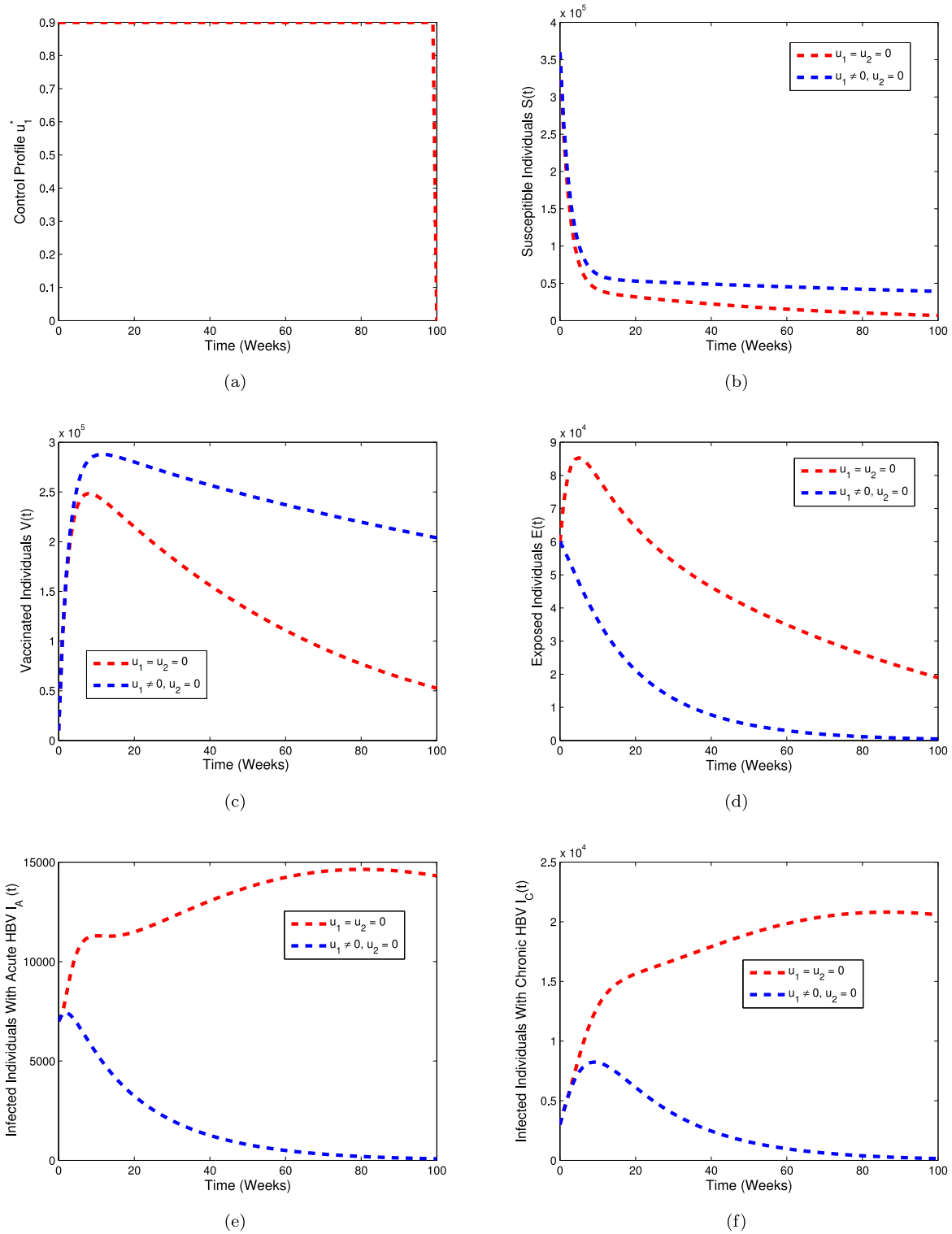
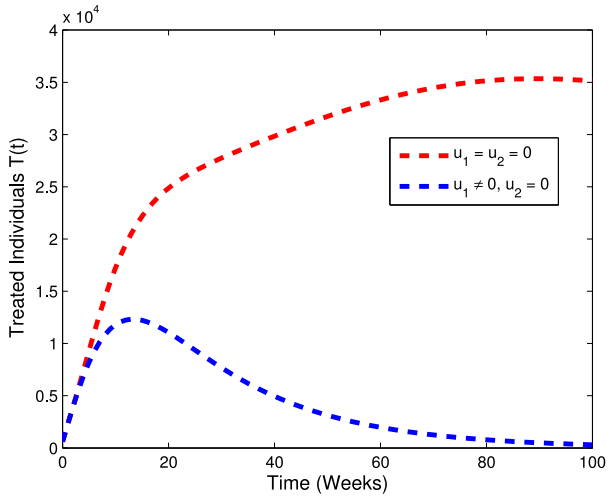
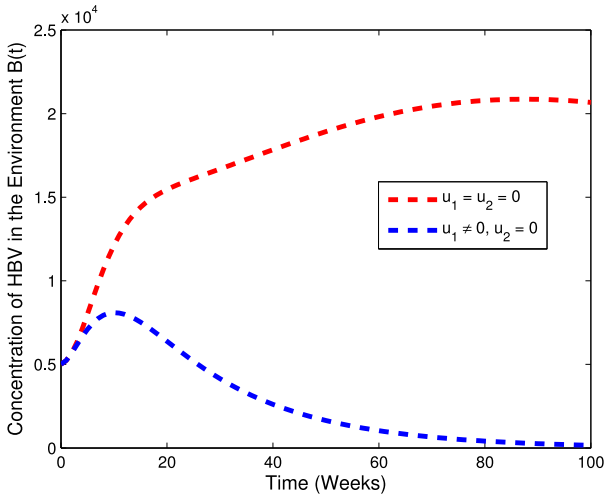


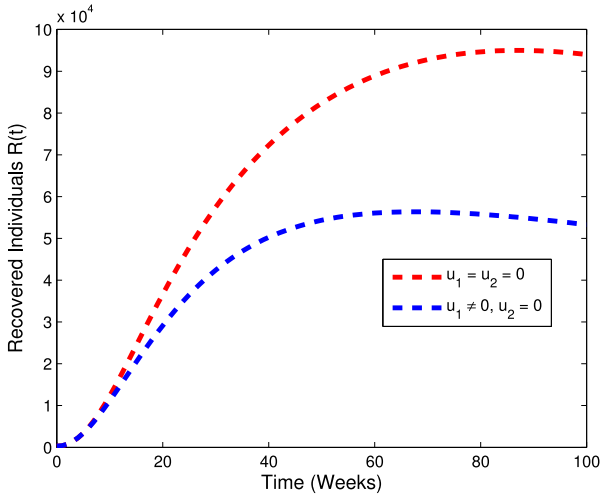
Fig. 5 Simulation of the **a** control profile u_1, **b** effect of u_1 on the susceptible individuals, **c** effect of u_1 on the vaccinated individuals, **d** effect of u_1 on the exposed individuals, **e** effect of u_1 on infected individuals with acute HBV infection, **f** effect of u_1 on infected individuals with chronic HBV infection, **g** effect of u_1 on the treated individuals, **h** effect of u_1 on the recovered individuals, and **i** effect of u_1 on concentration of HBV in the environment



(g)



(h)



(i)

Fig. 5 continued

Figure 7 is the simulations of the effect of both the controls $u_1(t)$ and $u_2(t)$ on the human population and the concentration of HBV in the environment. In the presence of both the controls $u_1(t)$ and $u_2(t)$, it is observed that the number of susceptible individuals increases above 50,000 within 20 weeks, the vaccinated individuals increases above 230,000 within 80 weeks, the exposed individuals reduces below 10,000 in 40 weeks, the infected individuals with acute HBV infection declined below 3000 in 20 weeks, the infected individuals with chronic HBV infection decreases below 1000 within 60 weeks, the treated individuals reduces below 4000 in 50 weeks, the recovered individuals reduces below 50,000 within 40 weeks due to less individuals being exposed to HBV infection, and the concentration of HBV in the environment reduces below 1000 within 40 weeks.

Based on the outcome of the numerical simulations, it is revealed that strategy C which encapsulates the combination of the controls $u_1(t)$ and $u_2(t)$, has greater impact in increasing the number of vaccinated individuals, as well as reducing the number of exposed individuals, infected individuals with acute HBV infection, infected individuals with chronic HBV infection, and the concentration of HBV in the environment when compared to strategy A and strategy B, which involves the standalone implementation of the prevention of susceptible individuals from acquiring HBV (through safe sex practice, regular washing of hands, and using protective hand glove when handling blood, body fluid, and semen) and the sensitization on individual on personal hygiene, sterilization and proper disposal of medical and dental equipment like syringes in order to reduce the shedding of HBV in the

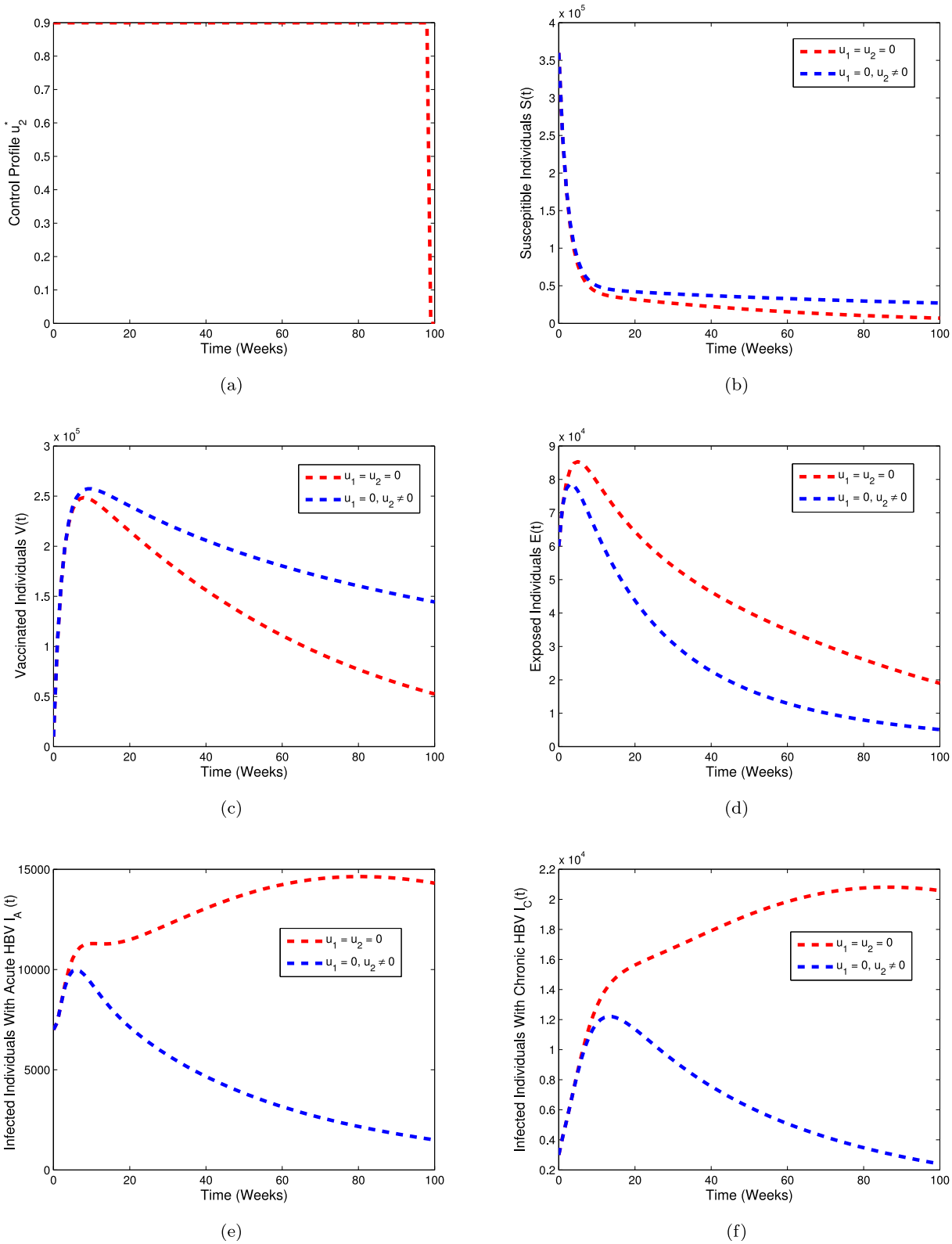
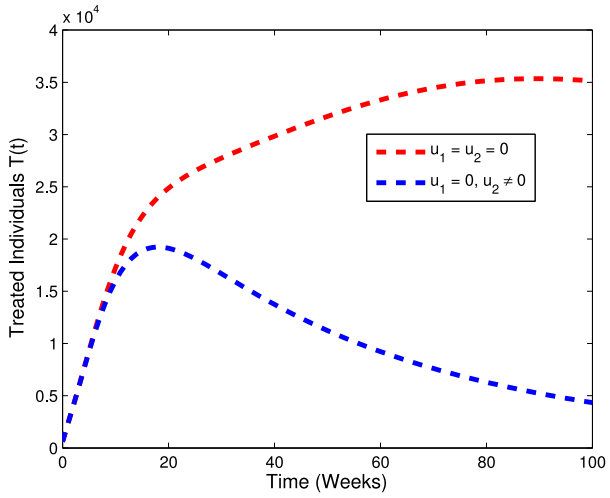
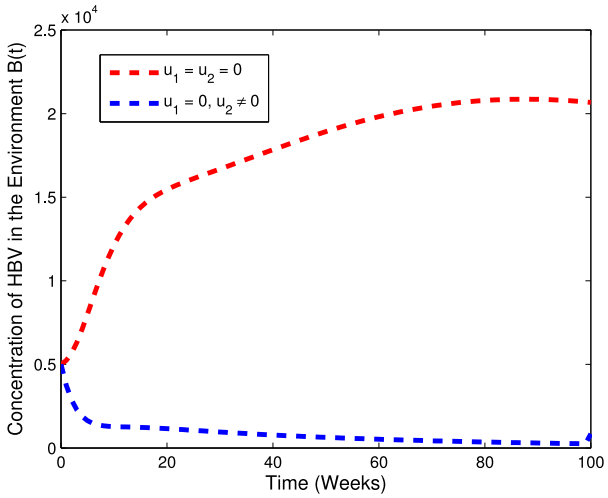


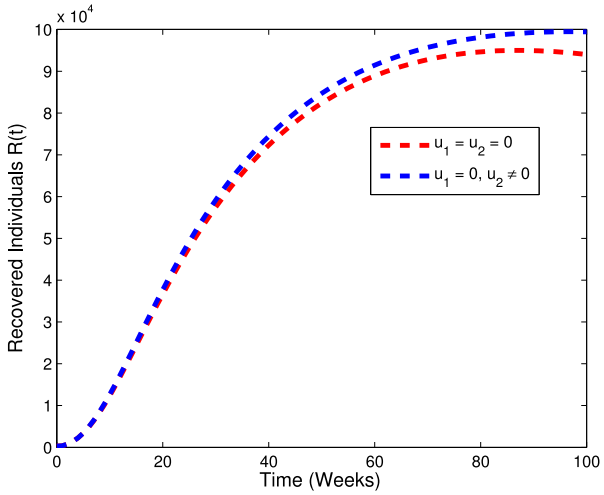
Fig. 6 Simulation of the **a** control profile u_2, **b** effect of u_2 on the susceptible individuals, **c** effect of u_2 on the vaccinated individuals, **d** effect of u_2 on the exposed individuals, **e** effect of u_2 on infected individuals with acute HBV infection, **f** Effect of u_2 on infected individuals with chronic HBV infection, **g** effect of u_2 on the treated individuals, **h** effect of u_2 on the recovered individuals, and **i** effect of u_2 on concentration of HBV in the environment



(g)



(h)



(i)

Fig. 6 continued

environment. Thus, Hepatitis B infection can successfully be controlled and mitigated within the population if the combination of both control strategies is properly and effectively implemented.

5 Conclusion

This work introduced the construction and analysis of a deterministic mathematical model to elucidate the transmission dynamics of Hepatitis B infection, including both vertical transmission and transmission affected by environmental viral concentration. A qualitative investigation of the model revealed two equilibrium points: the disease-free equilibrium and the endemic equilibrium. The basic reproduction number was calculated with the next-generation approach. When the basic reproduction number is below one, the disease-free equilibrium is locally asymptotically stable; conversely, it becomes unstable when this number surpasses one.

The existence the endemic equilibrium point analyzed analytically. Furthermore, we use the centre manifold theorem to analyzed the local stability of the endemic equilibrium. The model exhibits a backward bifurcation when the basic reproduction number is equal to one, posing substantial difficulties in substantially diminishing the prevalence of Hepatitis B in the community. Assuming no re-infection in the model, the disease-free equilibrium is globally asymptotically stable when the basic reproduction number is less than one.

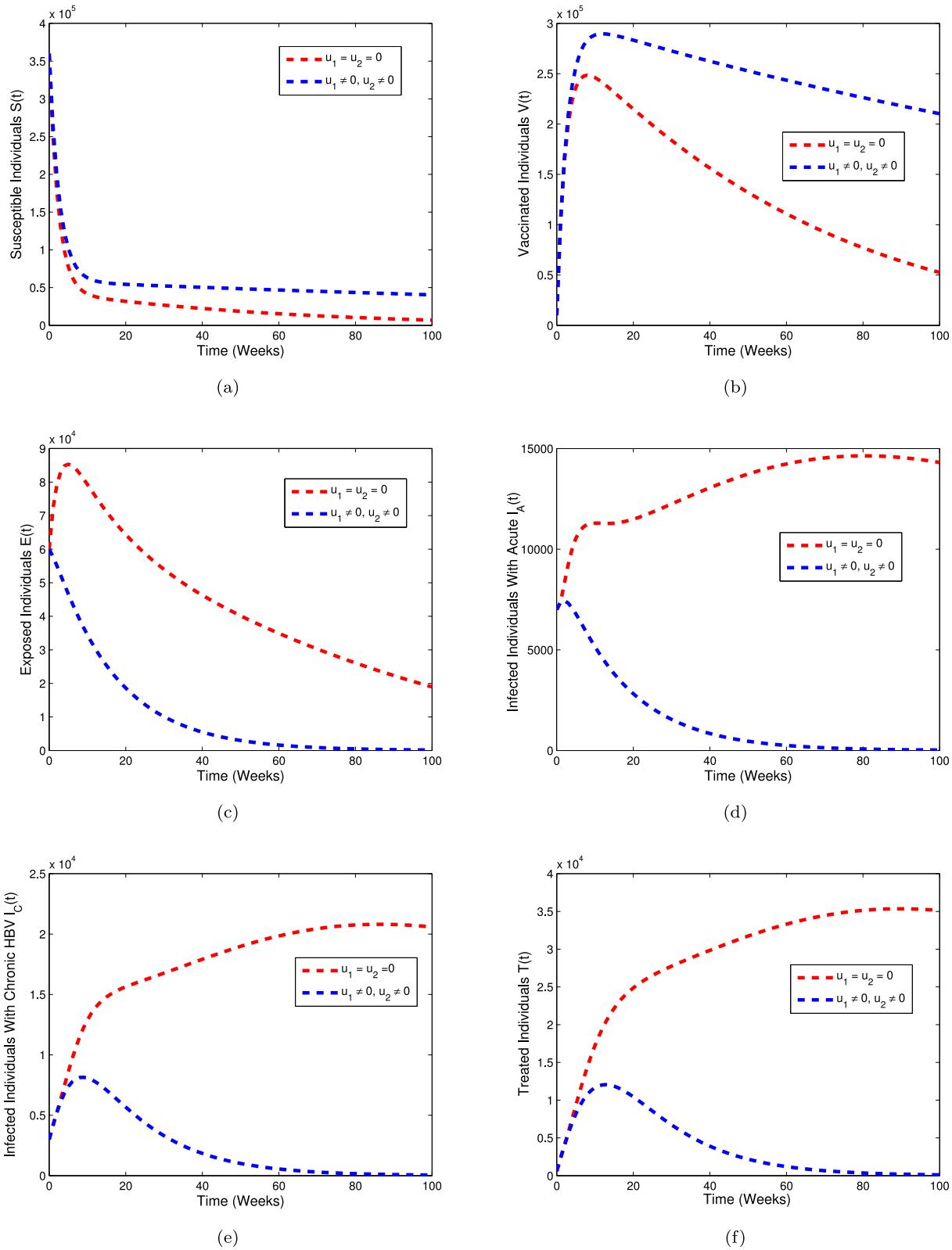
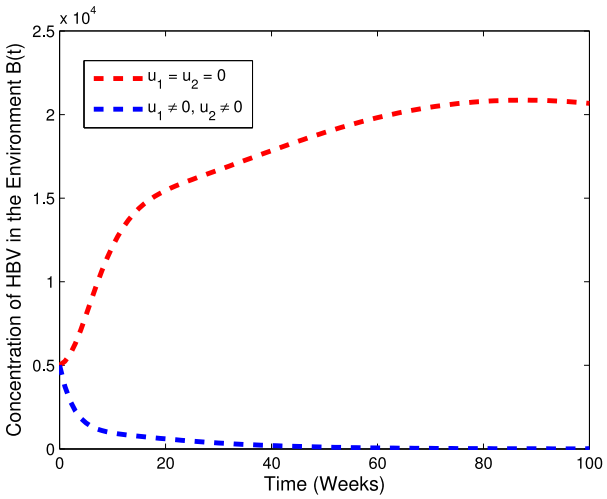
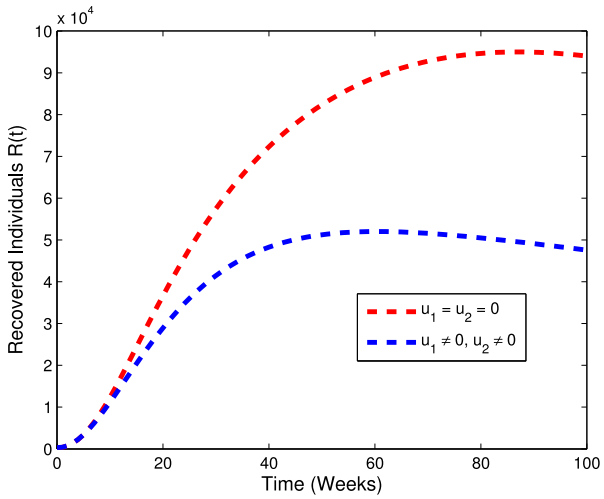


Fig. 7 Simulation of the **a** effect of u_1 and u_2 on the susceptible individuals, **b** effect of u_1 and u_2 on the vaccinated individuals, **c** effect of u_1 and u_2 on the exposed individuals, **d** effect of u_1 and u_2 on infected individuals with acute HBV infection, **e** effect of u_1 and u_2 on infected individuals with chronic HBV infection, **f** effect of u_1 and u_2 on the treated individuals, **g** effect of u_1 and u_2 on the recovered individuals, and **h** effect of u_1 and u_2 on concentration of HBV in the environment



(g)



(h)

Fig. 7 continued

A sensitivity analysis of the basic reproduction number was conducted to assess the impact of the model's critical parameters on disease transmission. Parameters exhibiting advantageous indices—particularly those with negative values—were recognized as factors that alleviate the illness burden when their values rise while others stay unchanged.

Optimal control approach was utilized to identify the most efficient scenario for reducing disease transmission by combining possible control strategies. Two time-dependent control measures were evaluated: inhibiting susceptible individuals from contracting HBV (via practices such as safe sexual conduct, consistent hand hygiene, and the utilization of protective gloves when managing blood, bodily fluids, or semen) and instructing individuals on personal hygiene, sterilization, and the appropriate disposal of medical and dental instruments (e.g., syringes) to mitigate HBV environmental contamination. Numerical experiments indicate that if both control techniques are executed appropriately and efficiently, Hepatitis B infection may be effectively managed and substantially diminished within the population.

Based on the findings of this study, the following recommendations are proposed for healthcare policymakers to effectively combat and prevent the spread of Hepatitis B:

1. **Enhance vaccination programs:** Broaden access to the Hepatitis B vaccine, prioritizing high-risk groups, newborns, and healthcare personnel, to curb the spread of new infections.
2. **Raise public awareness and knowledge:** Initiate educational campaigns to inform the public about transmission modes, prevention methods, the significance of vaccination, regular health screenings, and maintaining good personal hygiene.
3. **Increase availability of antiviral therapies:** Guarantee that antiviral treatments are affordable and readily available to manage chronic Hepatitis B, slow disease progression, and mitigate related complications.

Appendix 1

Where

$$\mathscr{A}_1 = P_1 + P_2 + P_3 + P_4 + P_5 + \mu_B - \alpha\pi,$$

$$\mathscr{A}_2 = P_1(P_2 + P_3 + P_4 + P_5 + \mu_B) + P_2(P_3 + P_4 + P_5 + \mu_B) + P_3(P_4 + P_5 + \mu_B) + P_4(P_5 + \mu_B) + P_5\mu_B - \alpha\pi(P_1 + P_2 + P_3 + P_4 + P_5 + \mu_B) - \gamma\omega - \frac{\beta\sigma P_2}{(\gamma + \omega + \mu)},$$

$$\mathscr{A}_3 = P_1P_2(P_3 + P_4 + P_5 + \mu_B) + P_3(P_1 + P_2)(P_4 + P_5 + \mu_B) + P_4(P_1 + P_2 + P_3)(P_5 + \mu_B) + \alpha\pi\gamma\omega + P_5\mu_B(P_1 + P_2 + P_3 + P_4) - \gamma\omega(P_3 + P_4 + P_5 + \mu_B) - \alpha\pi P_1(P_2 + P_3 + P_5 + \mu_B) - \alpha\pi\,(P_2(P_3 + P_5 + \mu_B) + P_3(P_5 + \mu_B) + P_5\mu_B) - \frac{\beta\sigma P_2(\kappa + P_1 + P_2 + P_5 + \mu_B)}{(\gamma + \omega + \mu)} - \frac{\beta_e\pi\sigma\eta_1 P_2}{K_B\mu(\gamma + \omega + \mu)},$$

$$\mathscr{A}_4 = P_1P_2P_3(P_4 + P_5 + \mu_B) + P_4(P_1P_2 + P_1P_3 + P_2P_3 - \gamma\omega)(P_5 + \mu_B) + P_5\mu_B(P_1 + P_2)(P_3 + P_4) + P_5\mu_B(P_1P_2 + P_3P_4) + \alpha\pi(\gamma\omega - P_1P_2)(P_3 + P_5 + \mu_B) + \frac{\beta\sigma\gamma\omega P_2}{(\gamma + \omega + \mu)} - \gamma\omega(P_3(P_4 + P_5 + \mu_B) + P_5\mu_B) - \frac{\beta\sigma P_2(\kappa(P_1 + P_2 + \mu_B) + P_1(P_2 + P_5 + \mu_B) + P_2(P_5 + \mu_B) + P_5\mu_B)}{(\gamma + \omega + \mu)} - \alpha\pi P_5\mu_B(P_1 + P_2 + P_3) - \frac{\beta_e\pi\sigma P_2(\eta_1(P_1 + P_2 + P_5) + \kappa\eta_2)}{K_B\mu(\gamma + \omega + \mu)} - \alpha\pi P_3(P_1 + P_2)(P_5 + \mu_B),$$

$$
\begin{aligned}
\mathscr{A}_5 &= P_1P_2P_3\,(P_4P_5+\mu_B(P_4+P_5))+P_1P_4P_5\mu_B(P_2+P_3)\\
&+\alpha\pi\gamma\omega\,(P_3P_5+\mu_B(P_3+P_5))+\\
&P_2P_3P_4P_5\mu_B+\frac{\beta\sigma\gamma\omega P_2(\kappa+P_5+\mu_B)}{(\gamma+\omega+\mu)}+\frac{\beta_e\pi\sigma\gamma\omega\eta_1P_2}{K_B\mu(\gamma+\omega+\mu)}\\
&-\gamma\omega\,(P_3P_4(P_5+\mu_B)+P_5\mu_B(P_3+P_4))\\
&-\frac{\beta\sigma P_2(\kappa(P_1P_2+\mu_B(P_1+P_2))+P_1P_2(P_5+\mu_B)+P_5\mu_B(P_1+P_2))}{(\gamma+\omega+\mu)}\\
&-\alpha\pi P_2P_3P_5\mu_B-\\
&\frac{\beta_e\pi P_2(\eta_1(P_1P_2+P_5(P_1+P_2))+\kappa\eta_2(P_1+P_2))}{K_B\mu(\gamma+\omega+\mu)}-\alpha\pi P_1\,(P_2P_3(P_5\\
&+\mu_B)+P_5\mu_B(P_2+P_3))\,,\\
\mathscr{A}_6 &= P_3P_4P_5\mu_B(P_1P_2-\gamma\omega)\,(1-\mathscr{R}_0)\,.
\end{aligned}
$$

Appendix 2

$$f(\lambda^{**})=U_1\lambda^{**4}+U_2\lambda^{**3}+U_3\lambda^{**2}+U_4\lambda^{**}+U_5 \quad (40)$$

where

$$
\begin{aligned}
&U_1=Z_3Z_8,\,U_2=Z_3Z_9+Z_4Z_8-(Z_1Z_8+Z_3Z_6),\\
&U_3=Z_3Z_{10}+Z_4Z_9+Z_5Z_8\\
&\quad-(Z_1Z_9+Z_2Z_8+Z_4Z_6+Z_3Z_7),\\
&U_4=Z_4Z_{10}+Z_5Z_9-(Z_1Z_{10}+Z_2Z_9+Z_5Z_6+Z_4Z_7),\\
&U_5=Z_5Z_{10}-(Z_2Z_{10}+Z_5Z_7)\\
&\quad=\pi\mu^2P_3^2P_4P_5^2P_6^2\mu_BK_B(P_1P_2-\gamma\omega)\\
&\quad\times(P_4-\alpha\pi)(\omega+P_2)(1-\mathscr{R}_0).
\end{aligned}
$$

With

$$
\begin{aligned}
&Z_1=\pi\beta\sigma\varphi P_2P_6(P_5+\kappa),\,Z_2=\pi\beta\sigma\mu P_2P_6(P_5+\kappa),\\
&Z_3=\pi\varphi P_2\,(\sigma P_6(P_5+\kappa)+P_5P_6(P_4-\alpha\pi)\\
&\quad+(\theta_1P_5+\theta_2\kappa)(\sigma-\varepsilon))\,,\\
&Z_4=P_5P_6(P_4-\alpha\pi)\,(\varphi P_3(\omega+P_2)+\mu P_2)\\
&\quad+(\theta_1P_5+\theta_2\kappa)\,(\sigma P_2(\varepsilon+\mu)-\varphi\varepsilon P_3(\omega+P_2))\\
&\quad+\mu\sigma P_2P_6(P_5+\kappa),\\
&Z_5=\pi\mu P_3P_5P_6(P_4-\alpha\pi)(\omega+P_2),\,Z_6\\
&\quad=\pi\beta_e\varphi\sigma P_2P_6(\eta_1P_5+\eta_2\kappa),\,Z_7\\
&\quad=\pi\beta_e\mu\sigma P_2P_6(\eta_1P_5+\eta_2\kappa),\\
&Z_8=\pi\sigma\varphi P_2P_6\,(\alpha P_5\mu_BK_B+\eta_1P_5+\eta_2\kappa)\\
&\quad+\varphi P_2P_3K_B\,(\mu_BP_2P_6(P_4-\alpha\pi)-\varepsilon(\theta_1P_5+\theta_2\kappa))\,,\\
&Z_9=\mu_BK_BP_3P_5P_6(P_4-\alpha\pi)(\varphi(P_1P_2-\gamma\omega)+P_2\mu)\\
&\quad+\pi\sigma\mu P_2P_6(\alpha P_5\mu_BK_B+\eta_1P_5+\eta_2\kappa)-\\
&\quad\varphi\varepsilon P_3K_B(\theta_1P_5+\theta_2\kappa)(P_1P_2-\gamma\omega),\\
&Z_{10}=\mu\mu_BP_3P_5P_6K_B(P_1P_2-\gamma\omega)(P_4-\alpha\pi).
\end{aligned}
$$

Appendix 3

$$
\begin{aligned}
&\frac{\partial^2f_1}{\partial x_1\partial x_4}=\frac{\partial^2f_1}{\partial x_4\partial x_1}=\frac{\partial^2f_1}{\partial x_1\partial x_5}=\frac{\partial^2f_1}{\partial x_5\partial x_1}=\frac{\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)}\\
&-\frac{\beta^*\mu}{\pi},\,\frac{\partial^2f_1}{\partial x_1\partial x_8}=\frac{\partial^2f_1}{\partial x_8\partial x_1}=-\frac{\beta_e}{K_B},\\
&\frac{\partial^2f_1}{\partial x_2\partial x_4}=\frac{\partial^2f_1}{\partial x_4\partial x_2}=\frac{\partial^2f_1}{\partial x_3\partial x_4}=\frac{\partial^2f_1}{\partial x_4\partial x_3}=\frac{\partial^2f_1}{\partial x_4\partial x_6}\\
&=\frac{\partial^2f_1}{\partial x_6\partial x_4}=\frac{\partial^2f_1}{\partial x_4\partial x_7}=\frac{\partial^2f_1}{\partial x_7\partial x_4}=\frac{\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_1}{\partial x_2\partial x_5}=\frac{\partial^2f_1}{\partial x_5\partial x_2}=\frac{\partial^2f_1}{\partial x_3\partial x_5}=\frac{\partial^2f_1}{\partial x_5\partial x_3}=\frac{\partial^2f_1}{\partial x_5\partial x_6}\\
&=\frac{\partial^2f_1}{\partial x_6\partial x_5}=\frac{\partial^2f_1}{\partial x_5\partial x_7}=\frac{\partial^2f_1}{\partial x_7\partial x_5}=\frac{\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_1}{\partial x_4\partial x_5}=\frac{\partial^2f_1}{\partial x_5\partial x_4}=\frac{\partial^2f_1}{\partial x_4^2}=\frac{\partial^2f_1}{\partial x_5^2}=\frac{2\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_1}{\partial x_8^2}=\frac{2\beta_e\pi(\gamma+\mu)}{K_B\mu(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_3}{\partial x_1\partial x_4}=\frac{\partial^2f_3}{\partial x_4\partial x_1}=\frac{\partial^2f_3}{\partial x_1\partial x_5}=\frac{\partial^2f_3}{\partial x_5\partial x_1}=\frac{\beta^*\mu}{\pi}\\
&-\frac{\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)},\,\frac{\partial^2f_3}{\partial x_1\partial x_8}=\frac{\partial^2f_3}{\partial x_8\partial x_1}=\frac{\beta_e}{K_B},\\
&\frac{\partial^2f_3}{\partial x_2\partial x_4}=\frac{\partial^2f_3}{\partial x_4\partial x_2}=\frac{\partial^2f_3}{\partial x_3\partial x_4}=\frac{\partial^2f_3}{\partial x_4\partial x_3}=\frac{\partial^2f_3}{\partial x_4\partial x_6}\\
&=\frac{\partial^2f_3}{\partial x_6\partial x_4}=\frac{\partial^2f_3}{\partial x_4\partial x_7}=\frac{\partial^2f_3}{\partial x_7\partial x_4}=-\frac{\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_3}{\partial x_2\partial x_5}=\frac{\partial^2f_3}{\partial x_5\partial x_2}=\frac{\partial^2f_3}{\partial x_3\partial x_5}=\frac{\partial^2f_3}{\partial x_5\partial x_3}=\frac{\partial^2f_3}{\partial x_5\partial x_6}\\
&=\frac{\partial^2f_3}{\partial x_6\partial x_5}=\frac{\partial^2f_3}{\partial x_5\partial x_7}=\frac{\partial^2f_3}{\partial x_7\partial x_5}=-\frac{\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_3}{\partial x_4\partial x_5}=\frac{\partial^2f_3}{\partial x_5\partial x_4}=\frac{\partial^2f_3}{\partial x_4^2}=\frac{\partial^2f_3}{\partial x_5^2}=-\frac{2\beta^*\mu(\gamma+\mu)}{\pi(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_3}{\partial x_8^2}=-\frac{2\beta_e\pi(\gamma+\mu)}{K_B\mu(\gamma+\omega+\mu)},\\
&\frac{\partial^2f_1}{\partial x_4\partial\beta^*}=\frac{\partial^2f_1}{\partial x_5\partial\beta^*}=-\frac{(\gamma+\mu)}{(\gamma+\omega+\mu)},\,\frac{\partial^2f_3}{\partial x_4\partial\beta^*}=\frac{\partial^2f_3}{\partial x_5\partial\beta^*}\\
&=\frac{(\gamma+\mu)}{(\gamma+\omega+\mu)}.
\end{aligned}
\quad (41)
$$

Author Contribution F.A.O. and O.J.P. wrote the main manuscript text. D.A. and G.B.B. conducted data analysis and prepared figures. O.P.O. and B.I.O. contributed to the research design and methodology. All authors reviewed and approved the final manuscript.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Conflict of Interest The authors declare no competing interests.

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