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Stability and Bifurcation of a Cholera Epidemic Model with Saturated Recovery Rate

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Abstract:

In this paper, a Cholera epidemic model is proposed and studied analytically as well as numerically. It is assumed that the disease is transmitted by contact with Vibrio cholerae and infected person according to dose-response function. However, the saturated treatment function is used to describe the recovery process. Moreover, the vaccine against the disease is assumed to be utterly ineffective. The existence, uniqueness and boundedness of the solution of the proposed model are discussed. All possible equilibrium points and the basic reproduction number are determined. The local stability and persistence conditions are established. Lyapunov method and the second additive compound matrix are used to study the global stability of the system. The conditions that guarantee the occurrence of local bifurcation and backward bifurcation are determined. Finally, numerical simulation is used to investigate the global dynamical behavior of the Cholera epidemic model and understand the effects of parameters on evolution of the disease in the environment. It is observed that the solution of the model is very sensitive to varying in parameters values and different types of bifurcations are obtained including backward bifurcation.

Keywords: Cholera; Stability; Persistence; Local bifurcation; Second additive compound matrix

MSC 2010 No.: 92D30, 34D23

1. Introduction

Mathematical modeling of infectious diseases played a vital role in our understanding of disease dynamics and developing effective prevention and control measures against epidemics. The

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occurrence of various infectious diseases represents a major challenge in modern society. The event of infectious disease causes a large loss of lives and other resources. Although there is an increased understanding of the mechanisms of infectious diseases from one side and the development of medical sciences from another side, infectious diseases caused millions of deaths and disabilities across the globe. Hence, people from different branches of science and medicine are working together to find an effective mechanism to stop the spread of infectious diseases, see Upadhyay et al. (2019) and Roy et al. (2020) and the references therein. Waterborne diseases (such as Cholera, typhoid, hepatitis) are the result of a lack of safe drinking water. The possibility of multiple transmission ways of the disease makes the study of waterborne disease more important. Among all the diseases which belong to this class, Cholera attracts more attention. Therefore, Cholera which is known as severe water and food-borne infectious disease caused by the gram-negative bacterium Vibrio cholera remains a significant public health burden in the developing world. This is due to the limited understanding at present on the complex infection dynamics of Cholera, which involve both direct (human to human) and indirect (environment to human) transmission pathways, see Cheng (2012).

Accordingly, a number of research papers dealing with modeling and simulating the dynamical behavior of Cholera have been done. Capasso and Paveri-Fontana (1979) constructed a simple Cholera model, which represents the first epidemic model that simulating the indirect transmission of the disease in the European Mediterranean region through the year 1973. They used two equations to describe the dynamics of infective people in the community and the dynamics of the bacteria population in the sea. Codeço (2001) extended the Cholera model of Capasso and Paveri-Fontana so that the dynamics of the susceptible individuals in the host population are included. Later on, Hartley et al. (2006) extended Codeço's work through incorporated a hyper infectious stage of the pathogen Vibrio cholerae based on the laboratory results. Joh et al. (2009) considered the dynamics of Cholera disease so that the primary mode of transmission is indirect and happened by contact with a contaminated reservoir. They also evaluated the realistic scenario in which the number of ingested pathogens must be above a critical threshold to cause infection in susceptible individuals. Mukandavire et al. (2011) explored the utility of mathematical models in understanding transmission (direct and indirect) dynamics of Cholera and in assessing the magnitude of interventions necessary to control the epidemic disease. Zhou et al. (2012) considered a Cholera model with imperfect vaccination. Cheng et al. (2012) discussed and investigated a global stability analysis for a generalized Cholera epidemiological model. Agarwal and Verma, (2012) proposed and analyzed a nonlinear delayed mathematical model with immigration for the spread of Cholera disease with carriers in the environment. Mondal and Kar (2013) proposed and analyzed a water-borne disease model involving water-to-person and person-to-person transmission and saturated incidence. However, Cui et al. (2014) considered an SVR-B Cholera model with imperfect vaccination. They performed a sensitivity analysis of the reproduction number on the parameters to determine their relative importance to disease transmission. After that, Wang et al. (2015) studied a Cholera model so that it explicitly includes disease prevalence dependent contact rates (direct and indirect) and host shedding rate, and then analyzed the resulting dynamics. While Zhou et al. (2016) studied a Cholera epidemic model with a saturated recovery rate.

Recently, Ayoade et al. (2018) studied the Cholera model so that it incorporates vaccination and therapeutic treatment as prevention and control measures for Cholera outbreaks. They considered

the possibility of re-infection after recovery too. However, Sisodiya et al. (2018) proposed a delayed SEIRB epidemic model with impulsive vaccination and disinfection. They studied the pulse vaccination strategy and sanitation to control the Cholera disease. While Subchan et al. (2019) have been used mathematical modeling and dynamics optimization to study the spread of Cholera disease. They proposed a SEIQR type of epidemic model considering the bacterial concentration of the Cholera spread dynamics. Later on, Meszaros et al. (2020) constructed a mathematical model of Cholera, which incorporates transmission within and between households. They observed that variation in the magnitude of household transmission changes multiple features of disease dynamics, including the severity and duration of outbreaks. However, Kwasi-Do and Afriyie (2020) developed a mathematical model that simulates the transmission mechanism of Cholera considering the role of control measures and the environment in the transmission of the disease. They formulated their model depending on two populations: the human population and the bacteria population.

In this paper, Cui et al. (2014) model is modified so that it's using a saturated recovery rate, in addition to that, a portion of the imperfect vaccine population becomes infected too through contact with an infected person or with contaminated environment resources.

2. Model formulation

In this section, a mathematical model for the transmission of Cholera epidemic disease is formulated mathematically with the help of a first-order nonlinear differential equations system. The following hypotheses are adopted in the following system:

1. It is assumed that the total human population at a certain time t is represented by $N(T)$. The disease divided the total population into four mutually exclusive compartments, namely susceptible population $S(t)$, vaccine population $V(t)$, infected population $I(t)$, and recovered population $R(t)$. Furthermore, the pathogen population is represented by $B(t)$.
2. It is assumed further that all new entrants will join the susceptible class at rate A. While the infected people contribute to the concentration of Vibrio cholerae at a rate η.
3. The susceptible people become infected at rates $\frac{\beta_e B}{K_1+B}$ and $\frac{\beta_h I}{K_2+I}$, which known as dose-response function, see Codeço (2001), where β_e and β_h are the rates of contact with the environment represented by untreated water or food and humane represented by infected people respectively. While K_1 and K_2 are the concentration of Vibrio cholerae in the environment and infected persons respectively.
4. The rate at which the susceptible population is vaccinated is γ_1, and the rate at which the vaccine wears off is γ_2. Moreover, the vaccine has the effect of reducing infection by a factor of σ so that $\sigma = 0$ means that the vaccine is completely effective in preventing infection, while $\sigma = 1$ means that the vaccine is utterly ineffective, therefore the vaccinated people become infected at the same rates given in (3) for $\sigma \in (0,1)$.
5. Since the Cholera outbreak occurs in developing countries at which the availability of hospitals and other medical facilities is limited. Hence, the saturated treatment function $\frac{cI}{b+I}$ is used, where c represents the rate at which the infected population being treated, while b

represents the rate of preventing treatment to reach the infected population for different reasons.

6. Finally, the natural death rate of humans is given by μ_1, while μ_2 is the natural death rate of Vibrio cholerae. However, the model considers the disease-related death rate as d.

According to the above hypotheses, the dynamics of the Cholera epidemic disease within the human been can be described in the following set of differential equations.

$$\begin{aligned}
\frac{dS}{dt} &= A - \frac{\beta_e SB}{K_1+B} - \frac{\beta_h SI}{K_2+I} - \gamma_1 S + \gamma_2 V - \mu_1 S,\\
\frac{dV}{dt} &= \gamma_1 S - \gamma_2 V - \frac{\sigma\beta_e VB}{K_1+B} - \frac{\sigma\beta_h VI}{K_2+I} - \mu_1 V,\\
\frac{dI}{dt} &= \frac{\beta_e SB}{K_1+B} + \frac{\beta_h SI}{K_2+I} + \frac{\sigma\beta_e VB}{K_1+B} + \frac{\sigma\beta_h VI}{K_2+I} - (d+\mu_1)I - \frac{cI}{b+I},\\
\frac{dR}{dt} &= \frac{cI}{b+I} - \mu_1 R,\\
\frac{dB}{dt} &= \eta I - \mu_2 B.
\end{aligned} \tag{1}$$

All the parameters are assumed to be positive, while σ is a nonnegative parameter. Since the first three equations along with the last equation in the system (1) are independent of the variable R, it suffices to consider the following model:

$$\begin{aligned}
\frac{dS}{dt} &= A - \frac{\beta_e SB}{K_1+B} - \frac{\beta_h SI}{K_2+I} - \gamma_1 S + \gamma_2 V - \mu_1 S,\\
\frac{dV}{dt} &= \gamma_1 S - \gamma_2 V - \frac{\sigma\beta_e VB}{K_1+B} - \frac{\sigma\beta_h VI}{K_2+I} - \mu_1 V,\\
\frac{dI}{dt} &= \frac{\beta_e SB}{K_1+B} + \frac{\beta_h SI}{K_2+I} + \frac{\sigma\beta_e VB}{K_1+B} + \frac{\sigma\beta_h VI}{K_2+I} - (d+\mu_1)I - \frac{cI}{b+I},\\
\frac{dB}{dt} &= \eta I - \mu_2 B.
\end{aligned} \tag{2}$$

The initial conditions of the system (2) are assumed as following:

$$S(0) \geq 0, V(0) \geq 0, I(0) \geq 0, B(0) \geq 0.$$

According to the equations in the system (1), and hence those given in system (2), it is clear that all the interaction functions are continuous and continuously differentiable functions. Hence, they are Lipschitz functions; therefore these systems have unique solutions. Further, the uniformly bounded of those solutions can be shown in the following theorem.

Theorem 1.

All solutions of the system (2) are uniformly bounded.
Proof*:*

Let $w = S + V + I$, then according to the first three equations in system (2) we obtain

$$\frac{dw}{dt} \leq A - \mu_1 w \xrightarrow{yields} \frac{dw}{dt} + \mu_1 w \leq A.$$

Then, $lim\ sup_{t\to\infty} w \leq \frac{A}{\mu_1}$. It follows form the fourth equation that:

$$\frac{dB}{dt} \leq \eta\frac{A}{\mu_1} - \mu_2 B \xrightarrow{yields} li\, m\, sup_{t\to\infty} B \leq \frac{\eta A}{\mu_1\mu_2}.$$

Therefore, all the variables are bounded and hence the proof is follows. ■

3. Existence of equilibrium points and basic reproduction number

The basic reproduction number, also it is known as basic reproductive rate or basic reproductive ratio, is one of the most useful threshold parameter that characterize mathematical problems concerning infection diseases. In fact, the infection will disappear and the system approaches asymptotically to the disease free equilibrium point when the basic reproduction number value is less than one, while the infection is outbreak and the disease will spread throughout all the system if it's bigger than one in most of epidemiological systems. Now, in order to determine the basic reproduction number of system (2), we began by computing the disease-free equilibrium point of system (2), which denoted by $P_0 = (S_0, V_0, 0,0)$. Straightforward computation when there is no disease in the system (2), i.e., $I = B = 0$, gives that

$$S_0 = \frac{A[\mu_1+\gamma_2]}{\mu_1(\gamma_1+\mu_1+\gamma_2)}, V_0 = \frac{\gamma_1 A}{\mu_1(\gamma_1+\mu_1+\gamma_2)}. \quad (3)$$

Now before determining the endemic equilibrium point and their existence conditions, the basic reproduction number R_0 at the disease free equilibrium point is determined.
Let $x = (I, B, S, V)^T$, then system (2) can be written as $\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x)$, where

$$\mathcal{F}(x) = \begin{bmatrix} \frac{\beta_e SB}{K_1+B} + \frac{\sigma\beta_e VB}{K_1+B} + \frac{\beta_h SI}{K_2+I} + \frac{\sigma\beta_h VI}{K_2+I} \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

and

$$\mathcal{V}(x) = \begin{bmatrix} (d+\mu_1)I + \frac{cI}{b+I} \\ \mu_2 B - \eta I \\ \frac{\beta_h SI}{K_2+I} + \frac{\beta_e SB}{K_1+B} + \gamma_1 S + \mu_1 S - A - \gamma_2 V \\ \frac{\sigma\beta_h VI}{K_2+I} + \frac{\sigma\beta_e VB}{K_1+B} + \gamma_2 V + \mu_1 V - \gamma_1 S \end{bmatrix}.$$

Consequently, from the definition of the basic reproduction number we obtain

$$F = \begin{bmatrix} \frac{\beta_h(S_0+\sigma V_0)}{K_2} & \frac{\beta_e(S_0+\sigma V_0)}{K_1} \\ 0 & 0 \end{bmatrix}, \quad \text{and} \quad \mathrm{V}^{-1} = \begin{bmatrix} \frac{1}{(d+\mu_1+\frac{c}{b})} & 0 \\ \frac{\eta}{\mu_2(d+\mu_1+\frac{c}{b})} & \frac{1}{\mu_2} \end{bmatrix}.$$

Hence, FV^{-1}, which represents the next generation matrix for system (2), can be determined and their spectral radius can be obtained as

$$\rho(FV^{-1}) = \max\left\{\frac{(S_0+\sigma V_0)}{(d+\mu_1+\frac{c}{b})}\left[\frac{\mu_2 K_1\beta_h + K_2\eta\beta_e}{K_1K_2\mu_2}\right], 0\right\}.$$

Accordingly, the basic reproduction number of system (2) is given by

$$R_0 = \frac{A(\gamma_2+\mu_1+\sigma\gamma_1)}{(d+\mu_1+\frac{c}{b})(\gamma_1+\mu_1+\gamma_2)}\left[\frac{\mu_2 K_1\beta_h + K_2\eta\beta_e}{K_1K_2\mu_1\mu_2}\right]. \tag{4}$$

On the other hand the endemic equilibrium point of system (2) can be determined as

$$S^* = \frac{AR_1R_2R_3}{R_3R_4-\gamma_1\gamma_2{R_1}^2{R_2}^2}, V^* = \frac{A\gamma_1{R_1}^2{R_2}^2}{R_3R_4-\gamma_1\gamma_2{R_1}^2{R_2}^2}, B^* = \frac{\eta I^*}{\mu_2}, \tag{5}$$

while $I^* > 0$ represents the positive root of the following five order polynomial equation

$$\psi_5 I^5 + \psi_4 I^4 + \psi_3 I^3 + \psi_2 I^2 + \psi_1 I + \psi_0 = 0, \tag{6}$$

here

$$\begin{aligned}
\psi_5 &= -(d+\mu_1)[\theta_1\eta\sigma_1 + \sigma{\sigma_1}^2 + \theta_3\eta^2] < 0,\\
\psi_4 &= \alpha_1\sigma_1 + A\gamma_1\sigma\eta\sigma_1 - \varsigma_4,\\
\psi_3 &= \alpha_1\sigma_2 + \alpha_2\sigma_1 + A\gamma_1\sigma[(R_5+\eta b)\sigma_1 + \eta R_6) - \varsigma_3,\\
\psi_2 &= \alpha_1\sigma_3 + \alpha_2\sigma_2 + \alpha_3\sigma_1 + A\gamma_1\sigma[(R_5 b + \mu_2K_1K_2)\sigma_1 + (R_5+\eta b)R_6) - \varsigma_2,\\
\psi_1 &= \alpha_2\sigma_3 + \alpha_3\sigma_1 + A\gamma_1\sigma(b\mu_2K_1K_2\sigma_1 + (R_5 b + \mu_2K_1K_2)R_6) - \varsigma_1,\\
\psi_0 &= \alpha_3\sigma_3 + A\gamma_1\sigma\mu_2K_1K_2R_5 b\ - \varsigma_0,
\end{aligned}$$

with

$$\begin{aligned}
\varsigma_4 &= \big[[(d+\mu_1)b+c][\theta_1\eta\sigma_1 + \sigma{\sigma_1}^2 + \theta_3\eta^2]\\
&\qquad + [d+\mu_1][2\sigma\sigma_1R_6 + \theta_1\eta(R_7+2R_6) + 2\theta_3\eta R_5]\big],\\
\varsigma_3 &= \Big[[(d+\mu_1)b+c][2\sigma\sigma_1R_6 + \theta_1\eta(R_7+2R_6) + 2\theta_3\eta R_5] + [d\\
&\qquad + \mu_1]\big[\sigma{R_6}^2 + \theta_1(2\mu_2K_1K_2\sigma_1 + R_8) + \theta_3\big(2\mu_2K_1K_2\eta + {R_5}^2\big)\big]\Big],\\
\varsigma_2 &= \Big[[(d+\mu_1)b+c]\big[\sigma{R_6}^2 + \theta_1(2\mu_2K_1K_2\sigma_1 + R_8) + \theta_3\big(2\mu_2K_1K_2\eta + {R_5}^2\big)\big]\\
&\qquad + [d+\mu_1][\theta_1\mu_2K_1K_2R_6 + 2\theta_3\mu_2K_1K_2R_5]\Big],\\
\varsigma_1 &= \big[[(d+\mu_1)b+c][\theta_1\mu_2K_1K_2R_6 + 2\theta_3\mu_2K_1K_2R_5] + [d+\mu_1]\theta_3(\mu_2K_1K_2)^2\big],\\
\varsigma_0 &= [(d+\mu_1)b+c]\theta_3(\mu_2K_1K_2)^2,\\
R_1 &= \mu_2K_1 + \eta I > 0,\\
R_2 &= K_2 + I > 0,\\
R_3 &= \gamma_2R_1R_2 + \sigma\beta_e\eta R_2 I + \sigma\beta_h R_1 I + \mu_1R_1R_2 > 0,\\
R_4 &= \beta_e\eta R_2 I + \beta_h R_1 I + \gamma_1R_1R_2 + \mu_1R_1R_2 > 0,
\end{aligned}$$

$$R_5 = \mu_2 K_1 + \eta K_2 > 0,$$
$$R_6 = \beta_h \mu_2 K_1 + \beta_e \eta K_2 > 0,$$
$$R_7 = \beta_e \mu_2 K_1 + \beta_h \eta K_2 > 0,$$
$$R_8 = \eta^2 \beta_e {K_2}^2 + {\mu_2}^2 \beta_h {K_1}^2 > 0,$$

and

$$\alpha_1 = A\eta(\gamma_2 + \sigma\beta_e + \sigma\beta_h + \mu_1) > 0,$$
$$\alpha_2 = A(\gamma_2 R_5 + \sigma R_6 + \mu_1 R_5) > 0,$$
$$\alpha_3 = AK_1 K_2 \mu_2(\gamma_2 + \mu_1) > 0,$$
$$\sigma_1 = \eta(\beta_h + \beta_e) > 0,$$
$$\sigma_2 = b\sigma_1 + R_6 > 0,$$
$$\sigma_3 = bR_6 > 0,$$
$$\theta_1 = \gamma_2 + \sigma(\gamma_1 + \mu_1) + \mu_1 > 0,$$
$$\theta_2 = \gamma_1\gamma_2 + \theta_3 > 0,$$
$$\theta_3 = \mu_1\gamma_2 + (\gamma_1 + \mu_1)\mu_1 > 0.$$

Since ψ_5 is negative in equation (6) then equation (6) has at least one positive root provided that $\psi_0 > 0$, which gives the following condition

$$\psi_0 = b\left[d + \mu_1 + \frac{c}{b}\right]\mu_1(\gamma_2 + \gamma_1 + \mu_1)(\mu_2 K_1 K_2)^2[R_0 - 1] > 0. \tag{7a}$$

Therefore, in order to have a positive equilibrium point, denoted by P_1, we have to have in addition to condition (7a) the following condition

$$R_3 R_4 > \gamma_1\gamma_2 {R_1}^2 {R_2}^2. \tag{7b}$$

4. Local stability Analysis and Persistence

In the following section, the local stability around each equilibrium point is studied using Linearization method and then the persistence conditions of the system are established. The general Jacobian matrix of system (2) at the point (S, V, I, B), can be written as

$$J = \left[c_{ij}\right]_{4\times 4}, \tag{8}$$

here

$$c_{11} = -\frac{\beta_e B}{K_1+B} - \frac{\beta_h I}{K_2+I} - \gamma_1 - \mu_1\,,\ c_{12} = \gamma_2,\ c_{13} = \frac{-K_2\beta_h S}{(K_2+I)^2}\,,\ c_{14} = \frac{-K_1\beta_e S}{(K_1+B)^2},$$
$$c_{21} = \gamma_1\,,\ c_{22} = -\gamma_2 - \mu_1 - \frac{\sigma\beta_e B}{K_1+B} - \frac{\sigma\beta_h I}{K_2+I},\ c_{23} = \frac{-K_2\sigma\beta_h V}{(K_2+I)^2},\ c_{24} = \frac{-K_1\sigma\beta_e V}{(K_1+B)^2},$$
$$c_{31} = \frac{\beta_e B}{K_1+B} + \frac{\beta_h I}{K_2+I}\,,\ c_{32} = \frac{\sigma\beta_e B}{K_1+B} + \frac{\sigma\beta_h I}{K_2+I},\ c_{33} = \frac{K_2\beta_h S}{(K_2+I)^2} + \frac{K_2\sigma\beta_h V}{(K_2+I)^2} - (d+\mu_1) - \frac{bc}{(b+I)^2},$$
$$c_{34} = \frac{K_1\beta_e S}{(K_1+B)^2} + \frac{K_1\sigma\beta_e V}{(K_1+B)^2},\ c_{41} = 0,\ c_{42} = 0,\ c_{43} = \eta, \text{ and } c_{44} = -\mu_2.$$

Accordingly, the following theorems can be proved directly using Equation (8) at each equilibrium point for system (2).

Theorem 2.

The disease-free equilibrium P_0 is local asymptotically for $R_0 < 1$ and unstable for $R_0 > 1$.

Proof:

By substituting the equilibrium point P_0 in the Jacobian matrix, we obtain the following characteristic equation

$$[\lambda^2 + (\gamma_1 + \gamma_2 + 2\mu_1)\lambda + \mu_1(\gamma_1 + \gamma_2 + \mu_1)](\lambda^2 - T\lambda + D) = 0, \tag{9}$$

where

$$T = \frac{\beta_h}{K_2}(S_0 + \sigma V_0) - (d + \mu_1 + \frac{c}{b} + \mu_2),\ D = -\mu_2\left(d + \mu_1 + \frac{c}{b}\right)(R_0 - 1).$$

For $R_0 < 1$, we obtain that $D > 0$ and $T < 0$. Hence, the roots of the, $\lambda^2 - T\lambda + D = 0$, have negative real parts. Also since the coefficients of the first term of equation (9) are positive, then its follows that all the eigenvalues have negative real parts. Therefore, the disease free equilibrium point P_0 is locally asymptotically stable. However, for $R_0 > 1$ we have $D < 0$, which leads to have positive eigenvalue for equation (9). Hence, the disease free equilibrium point P_0 is unstable.

■

Theorem 3.

The endemic equilibrium point $P_1 = (S^*, V^*, I^*, B^*)$ is locally asymptotically stable provided that

$$(d + \mu_1) + \frac{bc}{(b+I)^2} > \frac{K_2\beta_h}{(K_2+I)^2}(S^* + \sigma V^*), \tag{10a}$$

$$\mu_2\left[\frac{K_2\beta_h S}{(K_2+I)^2} + \frac{K_2\sigma\beta_h V}{(K_2+I)^2} - (d + \mu_1) - \frac{bc}{(b+I)^2}\right] > \eta\left(\frac{K_1\beta_e S}{(K_1+B)^2} + \frac{K_1\sigma\beta_e V}{(K_1+B)^2}\right), \tag{10b}$$

$$\begin{aligned}&\Gamma_1\Gamma_2(\Gamma_3 - \Gamma_4)^2 + (\Gamma_1 + \Gamma_2)(\Gamma_1\Gamma_2 - \Gamma_5)[\Gamma_2\Gamma_3 + \Gamma_1\Gamma_4 - \Gamma_{10} - \Gamma_{11}] > \\ &\quad(\Gamma_{10} - \Gamma_{11})^2 + (\Gamma_1 + \Gamma_2)^2(\Gamma_6\Gamma_7 + \Gamma_8\Gamma_9) + (\Gamma_{10} - \Gamma_{11})(\Gamma_3 - \Gamma_4)(\Gamma_2 - \Gamma_1),\end{aligned} \tag{10c}$$

where all symbols are clearly described in the proof.

Proof:

Clearly the Jacobian matrix around the endemic equilibrium point is written as

$$J(P_1) = \left[d_{ij}\right]_{4\times4}, \tag{11}$$

here d_{ij} follows directly from equation (8) by substituting, $P_1 = (S^*, V^*, I^*, B^*)$, instead of the point (S, V, I, B). Hence, the characteristic equation of $J(P_1)$ can be written as

$$\lambda^4 + A_1\lambda^3 + A_2\lambda^2 + A_3\lambda + A_4 = 0, \tag{12}$$

where

$$A_1 = -(\Gamma_1 + \Gamma_2),\ A_2 = \Gamma_3 + \Gamma_1\Gamma_2 + \Gamma_4 - \Gamma_5,$$
$$A_3 = -\Gamma_2\Gamma_3 - \Gamma_1\Gamma_4 - \Gamma_{10} + \Gamma_{11},\ A_4 = \Gamma_3\Gamma_4 + \Gamma_8\Gamma_9 + \Gamma_6\Gamma_7,$$

with

$$\Gamma_1 = d_{11} + d_{22}, \Gamma_2 = d_{33} + d_{44}, \Gamma_3 = d_{11}d_{22} - d_{12}d_{21}, \Gamma_4 = d_{33}d_{44} - d_{34}d_{43},$$
$$\Gamma_5 = d_{23}d_{32} + d_{13}d_{31}, \Gamma_6 = d_{21}d_{32} - d_{22}d_{31}, \Gamma_7 = d_{13}d_{44} - d_{14}d_{43},$$
$$\Gamma_8 = d_{44}d_{23} - d_{24}d_{43}, \Gamma_9 = d_{12}d_{31} - d_{11}d_{32}, \Gamma_{10} = d_{13}\Gamma_6 - d_{31}\Gamma_7,$$
$$\Gamma_{11} = d_{32}\Gamma_8 - d_{23}\Gamma_9.$$

Moreover, it's easy to verify that

$$\begin{aligned} &A_1A_2A_3 - {A_3}^2 - {A_1}^2A_4 \\ &\quad = \Gamma_1\Gamma_2(\Gamma_3 - \Gamma_4)^2 + (\Gamma_1 + \Gamma_2)(\Gamma_1\Gamma_2 - \Gamma_5)(\Gamma_2\Gamma_3 + \Gamma_1\Gamma_4 + \Gamma_{10} - \Gamma_{11}) \\ &\quad -(\Gamma_{10} - \Gamma_{11})(\Gamma_3 - \Gamma_4)(\Gamma_2 - \Gamma_1) - (\Gamma_{10} - \Gamma_{11})^2 - (\Gamma_1 + \Gamma_2)^2(\Gamma_6\Gamma_7 + \Gamma_8\Gamma_9). \end{aligned}$$

Now straightforward computation shows that conditions (10a) - (10b) guarantee that $\Gamma_1 < 0$, $\Gamma_2 < 0$, $\Gamma_3 > 0$, $\Gamma_4 > 0$, $\Gamma_5 < 0$, $\Gamma_6 > 0$, $\Gamma_7 > 0$, $\Gamma_8 > 0$, $\Gamma_9 > 0$, $\Gamma_{10} < 0$, and $\Gamma_{11} > 0$. Therefore, it's follows that $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, and $A_4 > 0$. However, the sufficient conditions (10a) - (10c) guarantee that $A_1A_2A_3 - {A_3}^2 - {A_1}^2A_4 > 0$. Hence, the endemic equilibrium point P_1 is locally asymptotically stable.

In the following, we will present the persistence of the system (2). The disease is endemic if the infected population remains above a certain positive level for a sufficiently large time. This definition of the endemic concept has been characterized with the help of the notion of uniform persistence in several epidemiological models, see Thiem (1993). Accordingly system (2) can be defined to be uniformly persistent if

$$\min\left\{\lim_{t\to\infty}\inf S(t), \lim_{t\to\infty}\inf V(t), \lim_{t\to\infty}\inf I(t), \lim_{t\to\infty}\inf B(t)\right\} > \varepsilon, \tag{13}$$

for some $\varepsilon > 0$ and all initial points in interior of positive domain. Note that it's easy to show using Bendixson theorem that there is no periodic dynamics in the interior of SV −plane and then the only possible invariant set in that plane is the disease free equilibrium point. Hence, for any initial point in the interior of SV −plane with $R_0 < 1$ the disease free equilibrium point P_0 is a globally asymptotically stable.

Theorem 4.

Assume that $R_0 > 1$ then system (2) is uniformly persistent.

Proof:

Suppose that u is a point in the interior of $\mathbb{R}^4_+$, and $O(u)$ is the orbit through u. Let $\Omega(u)$ is the omega limit set of $O(u)$. Further, since $\Omega(u)$ is bounded, due to the boundedness of the system (2), then we first show that $P_0 \notin \Omega(u)$. Assume the contrary, since P_0 is a saddle point under $R_0 > 1$, then P_0 cannot be the only point in $\Omega(u)$, and hence by Butler-McGhee lemma, see Freedman

and Waltman (1984), there is at least one other point v such that $v \in \omega^s(P_0) \bigcap \Omega(u)$, where $\omega^s(P_0)$ is the stable manifold of P_0.

Now, since the stable manifold of P_0 is given by $\mathbb{R}^3_+$ with the directions of S, V, and B respectively and the entire orbit through v, say $O(v)$, is contained in $\Omega(u)$. Hence, if v is on either boundary axes of $\mathbb{R}^3_+$ with the directions of S, V, and B, then we obtain that the positive specific axis (that containing v) is contained in $\Omega(u)$, which contradicting its boundedness.

Now, let v belongs to the interior of $\mathbb{R}^3_+$ with the directions of S, V, and B. Since there is no equilibrium point in the interior of $\mathbb{R}^3_+$ with the directions of S, V, and B; the orbit through v, which is contained in $\Omega(u)$ must be unbounded. Giving a contradiction too and this shows that $P_0 \notin \Omega(u)$. Thus $\Omega(u)$ must be in the interior of $\mathbb{R}^4_+$, which guarantee the uniform persistence of system (2). ■

5. Global stability

The global stability of the equilibrium points of system (2) is studied. It's well known that an equilibrium point $P_i; i = 0,1$ is said to be a globally asymptotically stable with respect to an open set, say Λ, if it's locally asymptotically stable and its basin of attraction contains Λ, see Li and Muldowney (1996). Consequently, in the following theorems the global stability of the free-disease equilibrium point starting from any initial point in the interior of $\mathbb{R}^4_+$ is discussed with the help of suitable Lyapunov function, while the stability of endemic equilibrium point P_1 is discussed with the help of second additive compound matrix.

Theorem 5.

For any initial point in the interior of $\mathbb{R}^4_+$ the disease-free equilibrium P_0 is global asymptotically stable provided that $R_0 < 1$.

Proof:

Consider the following Lyapunov function $L = I + mB$ with $m > 0$ constant. Clearly $L: \mathbb{R}^4 \to \mathbb{R}$ is a positive definite real valued function so that

$$\frac{dL}{dt} = \frac{dL}{dt} + m\frac{dB}{dt}.$$

Then, we obtain that

$$\frac{dL}{dt} = \frac{\beta_e SB}{K_1+B} + \frac{\beta_h SI}{K_2+I} + \frac{\sigma\beta_e VB}{K_1+B} + \frac{\sigma\beta_h VI}{K_2+I} - (d+\mu_1)I - \frac{cI}{b+I} + m\eta I - m\mu_2 B.$$

Hence,

$$\frac{dL}{dt} \leq \left[\frac{\beta_e}{K_1}(S_0 + \sigma V_0) - m\mu_2\right]B + \left[\frac{\beta_h}{K_2}(S_0 + \sigma V_0) - \left(d + \mu_1 + \frac{c}{b}\right) + m\eta\right]I.$$

So by choosing the value of the constant m as $m = \frac{\beta_e K_2 (d+\mu_1+\frac{c}{b})}{K_1\mu_2\beta_h+\eta K_2\beta_e}$, its obtain that

$$\frac{dL}{dt} \leq \frac{\beta_e K_2\left(d+\mu_1+\frac{c}{b}\right)}{K_1\mu_2\beta_h+\eta K_2\beta_e}(R_0-1)B + \left(\frac{K_1\mu_2\beta_h\left(d+\mu_1+\frac{c}{b}\right)}{K_1\mu_2\beta_h+\eta K_2\beta_e}\right)(R_0-1)I.$$

Then, $\frac{dL}{dt} < 0$ for $R_0 < 1$ and $\frac{dL}{dt} = 0$ for all points $(S, V, 0,0)$ including $(S_0, V_0, 0,0)$. Therefore, the disease-free equilibrium P_0 is a stable point. Now since the only invariant set that satisfies $\frac{dL}{dt} = 0$ is given by P_0 then according to the LaSalle's invariance principle, its attracting. Hence, P_0 is a globally asymptotically stable. ■

Now in order to study the global dynamics around the endemic equilibrium point P_1 of system (2), the Li and Muldowney (1996), approach is used as shown in the following theorem.

Theorem 6.

The endemic equilibrium point P_1 is globally asymptotically stable in the $intD \subset \mathbb{R}^4$ provided that

$$\pi < \mu_1, \tag{14}$$

here π is given in the proof.

Proof:

Rewrite system (2) in the form of autonomous dynamical system given by

$$\frac{d\boldsymbol{X}}{dt} = \boldsymbol{f}(\boldsymbol{X}), \tag{15}$$

here $\boldsymbol{f}: D \to \mathbb{R}^n, D \subset \mathbb{R}^n$ is a simplify connected open set, and $\boldsymbol{f} \in C^1(D)$.

Let $\boldsymbol{x}^*$ be equilibrium point of system (15). The point $\boldsymbol{x}^*$ is said to be a globally stable in D provided that it's locally stable and all the trajectories approach to $\boldsymbol{x}^*$.

Let $\boldsymbol{x} \longmapsto Q(x)$ be an $\binom{n}{2} \times \binom{n}{2}$ matrix valued function that is C^1 for $\boldsymbol{x} \in D$. Assume that $Q^{-1}(\boldsymbol{x})$ exists and is continuous for $\boldsymbol{x} \in K$, the compact absorbing set.

Consider

$$B = Q_f Q^{-1} + QJ^{[2]}Q^{-1}, \tag{16}$$

here $Q_f = (DQ)(f)$ or simply Q_f is the matrix obtained by replacing each entry q_{ij} of Q by its directional derivative in the direction of f and $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix for system (15).

Consider the Lozinskiĭ measure μ of B with respect to a vector norm $|\cdot|$ on $\mathbb{R}^N$, $N = \binom{n}{2}$, which defined

$$\mu(B) = \lim_{h\to 0^+} \frac{\|I+hB\|-1}{h}. \tag{17a}$$

Then, according to Li and Muldowney, the Lozinskiĭ measure μ of B, can be written by

$$\mu(B) = inf\{c: D_+ \| z \| \leq c \| z \|\}, \tag{17b}$$

for all solutions of $z' = Az$, with D_+ is the righthand derivative. Hence, if we can find a norm on $\mathbb{R}^N$ for which the associated Lozinskiĭ measure satisfied $\mu(B) < 0$ for all $x \in int\, D$ then the endemic equilibrium is globally asymptotically stable for $R_0 > 1$.

Now, since the Jacobian matrix J of the autonomous dynamical system given by system (2) at an arbitrary point (S, V, I, B) is given in equation (8), then the second compound matrix of J can be written as follows

$$M = [M_{ij}]_{6\times 6}, \tag{18}$$

where

$$M_{11} = -(1+\sigma)\frac{\beta_e B}{K_1+B} - (1+\sigma)\frac{\beta_h I}{K_2+I} - \gamma_1 - \gamma_2 - 2\mu_1,$$
$$M_{12} = \frac{-K_2\sigma\beta_h V}{(K_2+I)^2}, M_{13} = \frac{-K_1\sigma\beta_e V}{(K_1+B)^2}, M_{14} = \frac{K_2\beta_h S}{(K_2+I)^2},$$
$$M_{15} = \frac{K_1\beta_e S}{(K_1+B)^2}, M_{21} = \frac{\sigma\beta_e B}{K_1+B} + \frac{\sigma\beta_h I}{K_2+I},$$
$$M_{22} = -\frac{\beta_e B}{K_1+B} - \frac{\beta_h I}{K_2+I} - \gamma_1 - \mu_1 + \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V) - (d+\mu_1) - \frac{bc}{(b+I)^2},$$
$$M_{23} = \frac{K_1\beta_e}{(K_1+B)^2}(S+\sigma V), M_{24} = \gamma_2, M_{26} = \frac{K_1\beta_e S}{(K_1+B)^2},$$
$$M_{32} = \eta, M_{33} = -\frac{\beta_e B}{K_1+B} - \frac{\beta_h I}{K_2+I} - \gamma_1 - \mu_1 - \mu_2,$$
$$M_{35} = \gamma_2, M_{36} = -\frac{K_2\beta_h S}{(K_2+I)^2}, M_{41} = -\frac{\beta_e B}{K_1+B} - \frac{\beta_h I}{K_2+I}, M_{42} = \gamma_1,$$
$$M_{44} = -\frac{\sigma\beta_e B}{K_1+B} - \frac{\sigma\beta_h I}{K_2+I} - \gamma_2 - \mu_1 + \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V) - (d+\mu_1) - \frac{bc}{(b+I)^2},$$
$$M_{45} = \frac{K_1\beta_e}{(K_1+B)^2}(S+\sigma V), M_{46} = \frac{K_1\sigma\beta_e V}{(K_1+B)^2}, M_{53} = \gamma_1, M_{54} = \eta,$$
$$M_{55} = -\frac{\sigma\beta_e B}{K_1+B} - \frac{\sigma\beta_h I}{K_2+I} - \gamma_2 - \mu_1 - \mu_2, M_{56} = -\frac{K_2\sigma\beta_h V}{(K_2+I)^2},$$
$$M_{63} = \frac{\beta_e B}{K_1+B} + \frac{\beta_h I}{K_2+I}, M_{65} = \frac{\sigma\beta_e B}{K_1+B} + \frac{\sigma\beta_h I}{K_2+I},$$
$$M_{66} = \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V) - (d+\mu_1) - \frac{bc}{(b+I)^2} - \mu_2,$$

while

$$M_{16} = M_{25} = M_{31} = M_{34} = M_{43} = M_{51} = M_{52} = M_{61} = M_{62} = M_{64} = 0.$$

Let

$$Q = \begin{bmatrix} 1/I & 0 & 0 & 0 & 0 & 0 \\ 0 & 1/I & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1/I & 0 & 0 \\ 0 & 0 & 1/B & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1/B & 0 \\ 0 & 0 & 0 & 0 & 0 & 1/B \end{bmatrix}.$$

Then, we obtain

$$Q^{-1} = \begin{bmatrix} I & 0 & 0 & 0 & 0 & 0 \\ 0 & I & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & B & 0 & 0 \\ 0 & 0 & I & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & B & 0 \\ 0 & 0 & 0 & 0 & 0 & B \end{bmatrix}.$$

Also from the definition of Q_f with the help of system (2), its obtain that

$$Q_f = \begin{bmatrix} -I'/I^2 & 0 & 0 & 0 & 0 & 0 \\ 0 & -I'/I^2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -I'/I^2 & 0 & 0 \\ 0 & 0 & -B'/B^2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -B'/B^2 & 0 \\ 0 & 0 & 0 & 0 & 0 & -B'/B^2 \end{bmatrix}.$$

Consequently,

$$B = Q_f Q^{-1} + QJ^{[2]}Q^{-1} = \left[b_{ij}\right]_{6\times 6},$$

where

$$b_{11} = M_{11} - (I'/I) = -(1+\sigma)\frac{\beta_e B}{K_1+B} - (1+\sigma)\frac{\beta_h I}{K_2+I} - \frac{\beta_e B}{I(K_1+B)}(S+\sigma V) - \frac{\beta_h}{K_2+I}(S+\sigma V) - \gamma_1 - \gamma_2 - \mu_1 + d + \frac{c}{b+I},$$

$$b_{12} = M_{12},\ b_{13} = M_{14},\ b_{14} = M_{13}\frac{B}{I},\ b_{15} = M_{15}\frac{B}{I},$$

$$b_{22} = M_{22} - (I'/I) = -\frac{\beta_e B}{K_1+B} - \frac{\beta_h I}{K_2+I} + \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V) - \frac{\beta_e B}{I(K_1+B)}(S+\sigma V) - \frac{\beta_h}{K_2+I}(S+\sigma V) - \gamma_1 - \mu_1 + \frac{cI}{(b+I)^2},$$

$$b_{21} = M_{21},\ b_{23} = M_{24},\ b_{24} = M_{23}\frac{B}{I},\ b_{26} = M_{26}\frac{B}{I},$$

$$b_{33} = M_{44} - (I'/I) = -\frac{\sigma\beta_e B}{K_1+B} - \frac{\sigma\beta_h I}{K_2+I} + \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V) - \frac{\beta_e B}{I(K_1+B)}(S+\sigma V) - \frac{\beta_h}{K_2+I}(S+\sigma V) - \gamma_2 - \mu_1 + \frac{cI}{(b+I)^2},$$

$$b_{31} = M_{41},\ b_{32} = M_{42},\ b_{35} = M_{45}\frac{B}{I},\ b_{36} = M_{46}\frac{B}{I},$$

$$b_{44} = M_{33} - (B'/B) = -\frac{\beta_e B}{(K_1+B)} - \frac{\beta_h I}{K_2+I} - \gamma_1 - \mu_1 - \frac{\eta I}{B},$$

$$b_{42} = M_{32}\frac{I}{B},\ b_{45} = M_{35},\ b_{46} = M_{36},$$

$$b_{55} = M_{55} - (B'/B) = -\frac{\sigma\beta_e B}{K_1+B} - \frac{\sigma\beta_h I}{K_2+I} - \gamma_2 - \mu_1 - \frac{\eta I}{B},$$
$$b_{53} = M_{54}\frac{I}{B}, b_{54} = M_{53}, b_{56} = M_{56}, b_{64} = M_{63}, b_{65} = M_{65}$$
$$b_{66} = M_{66} - (B'/B) = \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V) - \frac{bc}{(b+I)^2} - d - \mu_1 - \frac{\eta I}{B},$$

while

$$b_{16} = b_{25} = b_{34} = b_{41} = b_{43} = b_{51} = b_{52} = b_{61} = b_{62} = b_{63} = 0.$$

Now B can be written as a block matrix

$$B = \left[H_{ij}\right]_{4\times 4},$$

where

$$H_{11} = b_{11}, H_{12} = [b_{12} \quad b_{13}], H_{13} = [b_{14} \quad b_{15}], H_{14} = 0,$$
$$H_{21} = \begin{bmatrix} b_{21} \\ b_{31} \end{bmatrix}, H_{22} = \begin{bmatrix} b_{22} & b_{23} \\ b_{32} & b_{33} \end{bmatrix}, H_{23} = \begin{bmatrix} b_{24} & 0 \\ 0 & b_{35} \end{bmatrix}, H_{24} = \begin{bmatrix} b_{26} \\ b_{36} \end{bmatrix},$$
$$H_{31} = \begin{bmatrix} 0 \\ 0 \end{bmatrix}, H_{32} = \begin{bmatrix} b_{42} & 0 \\ 0 & b_{53} \end{bmatrix}, H_{33} = \begin{bmatrix} b_{44} & b_{45} \\ b_{54} & b_{55} \end{bmatrix}, H_{34} = \begin{bmatrix} b_{46} \\ b_{56} \end{bmatrix},$$
$$H_{41} = 0, H_{42} = [0 \quad 0], H_{43} = [b_{64} \quad b_{65}], H_{44} = b_{66}.$$

Let $Z = (Z_1, Z_2, Z_3, Z_4, Z_5, Z_6)$ be a vector in $\mathbb{R}^6$, with the norm defined as

$$\|Z\| = \max\{|Z_1|, |Z_2| + |Z_3|, |Z_4| + |Z_5|, |Z_6|\}.$$

Let $\mu(B)$ be the Lozinskiĭ measure of B with respect to this norm. So by using similar argument as in Zhou et al. (2016), we have the following estimate

$$\mu(B) \leq \sup\{g_1, g_2, g_3, g_4\},$$

here

$$g_1 = \mu(H_{11}) + |H_{12}| + |H_{13}| + |H_{14}|,$$
$$g_2 = |H_{21}| + \mu(H_{22}) + |H_{23}| + |H_{24}|,$$
$$g_3 = |H_{31}| + |H_{32}| + \mu(H_{33}) + |H_{34}|,$$
$$g_4 = |H_{41}| + |H_{42}| + |H_{43}| + \mu(H_{44}),$$

with $\left|H_{ij}\right|, i \neq j = 1,2,3,4$ are the matrix norms induced by the l_1 vector norm, and $\mu(H_{ii})$ denotes the Lozinskiĭ measure with respect to l_1 norm. Moreover we have

$$\mu(H_{11}) = -(1+\sigma)\frac{\beta_e B}{K_1+B} - (1+\sigma)\frac{\beta_h I}{K_2+I} - \frac{\beta_e B}{I(K_1+B)}(S+\sigma V)$$
$$-\frac{\beta_h}{K_2+I}(S+\sigma V) - \gamma_1 - \gamma_2 - \mu_1 + d + \frac{cI}{(b+I)},$$
$$\mu(H_{22}) = -\frac{\beta_e B}{K_1+B} - \frac{\beta_h I}{K_2+I} - \frac{\beta_h I}{(K_2+I)^2}(S+\sigma V) - \frac{\beta_e B}{I(K_1+B)}(S+\sigma V) - \mu_1 + \frac{cI}{(b+I)^2},$$
$$\mu(H_{33}) = -\frac{\beta_e B}{K_1+B} - \frac{\beta_h I}{K_2+I} - \mu_1 - \frac{\eta I}{B},$$
$$\mu(H_{44}) = \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V) - \frac{bc}{(b+I)^2} - d - \mu_1 - \frac{\eta I}{B},$$

with

$$|H_{12}| = \frac{K_2\beta_h S}{(K_2+I)^2}, |H_{13}| = \frac{K_1\beta_e S}{(K_1+B)^2}\frac{B}{I}, |H_{21}| = (1+\sigma)\frac{\beta_e B}{(K_1+B)} + (1+\sigma)\frac{\beta_h I}{K_2+I},$$
$$|H_{23}| = \frac{K_1\beta_e B}{I(K_1+B)^2}(S+\sigma V), |H_{24}| = \frac{K_1\beta_e B}{I(K_1+B)^2}(S+\sigma V), |H_{32}| = \frac{\eta I}{B},$$
$$|H_{34}| = \frac{K_2\beta_h}{(K_2+I)^2}(S+\sigma V), |H_{43}| = \frac{\beta_e B}{K_1+B} + \frac{\beta_h I}{K_2+I},$$
$$|H_{14}| = |H_{31}| = |H_{41}| = |H_{42}| = 0.$$

Accordingly, we get that

$$g_1 \le -\mu_1 + d + \frac{c}{b+\varepsilon},$$
$$g_2 \le \sigma\beta_e + \sigma\beta_h + 2\beta_e\frac{\delta}{\varepsilon} - \mu_1 + \frac{c}{b+\varepsilon},$$
$$g_3 \le -\mu_1 + \frac{\beta_h\delta}{K_2+\varepsilon},$$
$$g_4 \le \beta_e + \beta_h + \frac{\beta_h\delta}{K_2+\varepsilon} - \mu_1,$$

where $sup(S+\sigma V) = \delta, min(I) = \varepsilon$. Assume that

$$\pi = max\left\{\left(d + \frac{c}{b+\varepsilon}\right), \left(\sigma\beta_e + \sigma\beta_h + 2\beta_e\frac{\delta}{\varepsilon} + \frac{c}{b+\varepsilon}\right), \left(\beta_e + \beta_h + \frac{\beta_h\delta}{K_2+\varepsilon}\right)\right\}. \quad (19)$$

Hence, we obtain

$$g_i \le -\mu_1 + \pi = -\rho, i = 1,2,3,4.$$

Therefore, we get that $\mu(B) \le -(\mu_1 - \pi) = -\rho$, and hence the proof is complete provided that the condition (14) is satisfied. ■

6. Local bifurcation

In this section, the effect of the varying parameter values on the dynamical behavior of the system (2) around equilibrium points are considered using the Sotomayer theorem that is given in Perko (2013). The occurrence of backward bifurcation in the system (2) is also considered using Castillo-Chavez and Song (2004) theorem for backward bifurcation.

Now by using equation (14) to describe system (2) with $\boldsymbol{X} = (x_1, x_2, x_3, x_4)^T = (S, V, I, B)^T$ and $\boldsymbol{f} = (f_1, f_2, f_3, f_4)^T$, where $f_i, i = 1,2,3,4$ are given in the right hand side of system (2) then for any vector $V = (v_1, v_2, v_3, v_4)^T$ we have

$$D^2\boldsymbol{f}(\boldsymbol{X})(V,V) = \begin{bmatrix} \sum_{i,j=1}^4 \frac{\partial f_1}{\partial x_i \partial x_j}(v_i)(v_j) \\ \sum_{i,j=1}^4 \frac{\partial f_2}{\partial x_i \partial x_j}(v_i)(v_j) \\ \sum_{i,j=1}^4 \frac{\partial f_3}{\partial x_i \partial x_j}(v_i)(v_j) \\ \sum_{i,j=1}^4 \frac{\partial f_4}{\partial x_i \partial x_j}(v_i)(v_j) \end{bmatrix} = \left[a_{ij}\right]_{4\times 1}, \quad (20)$$

where

$$a_{11} = -\frac{2K_2\beta_h}{(K_2+I)^2}v_1v_3 - \frac{2K_1\beta_e}{(K_1+B)^2}v_1v_4 + \frac{2K_2\beta_hS}{(K_2+I)^3}{v_3}^2 + \frac{2K_1\beta_eS}{(K_1+B)^3}{v_4}^2,$$
$$a_{21} = -\frac{2K_2\sigma\beta_h}{(K_2+I)^2}v_2v_3 - \frac{2K_1\sigma\beta_e}{(K_1+B)^2}v_2v_4 + \frac{2K_2\sigma\beta_hV}{(K_2+I)^3}{v_3}^2 + \frac{2K_1\sigma\beta_eV}{(K_1+B)^3}{v_4}^2,$$
$$a_{31} = \frac{2K_2\beta_h}{(K_2+I)^2}v_1v_3 + \frac{2K_1\beta_e}{(K_1+B)^2}v_1v_4 + \frac{2K_2\sigma\beta_h}{(K_2+I)^2}v_2v_3 + \frac{2K_1\sigma\beta_e}{(K_1+B)^2}v_2v_4$$
$$-\left[\frac{2K_2\beta_h}{(K_2+I)^3}(S+\sigma V) - \frac{2bc}{(b+I)^3}\right]{v_3}^2 - \frac{2K_1\beta_e}{(K_1+B)^3}(S+\sigma V){v_4}^2,$$
$$a_{41} = 0.$$

Accordingly, the following theorem specifies the bifurcation parameter around the disease free equilibrium point and the type of local bifurcation occurred.

Theorem 7.

Assume that $R_0 = 1$. Then, system (2) near the equilibrium point P_0 has

1. No saddle-node bifurcation.
2. A trans-critical bifurcation provided that the following condition holds

$$\frac{\beta_h}{K_2}\gamma_3(\gamma_1+\sigma\gamma_2) + \frac{\beta_e}{K_1}(\gamma_1+\sigma\gamma_2) \neq \left[\frac{\beta_h}{{K_2}^2}(S_0+\sigma V_0) - \frac{c}{b^2}\right]{\gamma_3}^2 + \frac{\beta_e}{{K_1}^2}(S_0+\sigma V_0). \quad (21a)$$

3. A backward bifurcation provided that the following condition holds

$$\frac{c}{b^2} < \frac{\beta_h(S_0+\sigma V_0)}{{K_2}^2}. \quad (21b)$$

Proof:

Clearly from theorem (2) the characteristic equation given in equation (9) has zero eigenvalue when $R_0 = 1$ and hence by substituting the value of R_0 and simplifying the resulting terms we obtain the following positive quantity

$$\eta = \eta^* = \frac{K_1K_2\mu_2\left(d+\mu_1+\frac{c}{b}\right) - K_1\beta_h\mu_2(S_0+\sigma V_0)}{K_2\beta_e(S_0+\sigma V_0)}. \quad (22)$$

Hence, P_0 is a nonhyperbolic point at $\eta = \eta^*$.

Recall that, the Jacobain matrix of system (2) at P_0 and $\eta = \eta^*$ follows directly from equation (8) and can be represented by $J_0 = \left[\bar{c}_{ij}(P_0, \eta^*)\right]_{4\times4}$. Let $\boldsymbol{V_0} = (v_{10}, v_{20}, v_{30}, v_{40})^T$ be the eigenvector (right eigenvector) corresponding to the zero eigenvalue, say $\lambda_0(\eta^*) = 0$, then $J_0\boldsymbol{V_0} = \boldsymbol{0}$ gives that

$$V_0 = \begin{bmatrix} \gamma_1 v_{40} \\ \gamma_2 v_{40} \\ \gamma_3 v_{40} \\ v_{40} \end{bmatrix},$$

with $v_{40} \neq 0$ be any real number. While,

$$\gamma_1 = \left[\frac{(\bar{c}_{24}\bar{c}_{33}-\bar{c}_{23}\bar{c}_{34})\bar{c}_{12}+(\bar{c}_{13}\bar{c}_{34}-\bar{c}_{14}\bar{c}_{33})\bar{c}_{22}}{\bar{c}_{33}(\bar{c}_{11}\bar{c}_{22}-\bar{c}_{12}\bar{c}_{21})}\right],$$

$$\gamma_2 = \left[\frac{(\bar{c}_{11}\bar{c}_{23}-\bar{c}_{13}\bar{c}_{21})\bar{c}_{34}-(\bar{c}_{24}\bar{c}_{11}-\bar{c}_{14}\bar{c}_{21})\bar{c}_{33}}{\bar{c}_{33}(\bar{c}_{11}\bar{c}_{22}-\bar{c}_{12}\bar{c}_{21})}\right],$$

$$\gamma_3 = -\frac{\bar{c}_{34}}{\bar{c}_{33}}.$$

It is clear that under the condition $R_0 = 1$ we obtain that $\bar{c}_{33} < 0$ and hence the quantities γ_1, γ_2 are negative and γ_3 is positive. Similarly, let $\boldsymbol{\psi_0} = (\psi_{10}, \psi_{20}, \psi_{30}, \psi_{40})^T$ be the eigenvector corresponding to the zero eigenvalue of the matrix ${J_0}^T$, hence ${J_0}^T \boldsymbol{\psi_0} = \mathbf{0}$ gives that

$$\boldsymbol{\psi_0} = \begin{bmatrix} 0 \\ 0 \\ \alpha_1 \psi_{40} \\ \psi_{40} \end{bmatrix},$$

where $\psi_{40} \neq 0$ be any real number and $\alpha_1 = -\frac{\bar{c}_{43}}{\bar{c}_{33}} > 0$. Moreover, since

$$\frac{d\boldsymbol{f}}{d\eta} = \boldsymbol{f}_\eta = (0,0,I,0)^{\mathrm{T}} \rightarrow \boldsymbol{f}_\eta(P_0, \eta^*) = (0,0,0,0)^{\mathrm{T}}.$$

Then, ${\boldsymbol{\psi_0}}^T \boldsymbol{f}_\eta(P_0, \eta^*) = 0$, which leads according to Sotomayer's theorem to that system (2) near the disease free equilibrium point P_0 has no saddle node bifurcation. Further, we have

$$D\boldsymbol{f}_\eta(P_0, \eta^*) = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 \end{bmatrix}.$$

Therefore, we obtain that $Df_\eta(P_0, \eta^*)\boldsymbol{V_0} = (0,0,0,\gamma_3 v_{40})^{\mathrm{T}}$ and hence the following is obtained ${\boldsymbol{\psi_0}}^T [D\boldsymbol{f}_\eta(P_0, \eta^*)\boldsymbol{V_0}] = \psi_{40}\gamma_3 v_{40} \neq 0$. Moreover according to equation (20) with $\boldsymbol{V} = \boldsymbol{V_0}$ we have

$$D^2\boldsymbol{f}(P_0, \eta^*)(\boldsymbol{V_0}, \boldsymbol{V_0}) = [\bar{a}_{i1}]_{4\times 1},$$

with

$$\bar{a}_{11} = -\frac{2\beta_h}{k_2}\gamma_1\gamma_3 {v_{40}}^2 - \frac{2\beta_e}{k_1}\gamma_1 {v_{40}}^2 + \frac{2\beta_h S_0}{{k_2}^2}{\gamma_3}^2 {v_{40}}^2 + \frac{2\beta_e S_0}{{K_1}^2}{v_{40}}^2,$$

$$\bar{a}_{21} = -\frac{2\sigma\beta_h}{K_2}\gamma_2\gamma_3 {v_{40}}^2 - \frac{2\sigma\beta_e}{K_1}\gamma_2 {v_{40}}^2 + \frac{2\sigma\beta_h V_0}{{K_2}^2}{\gamma_3}^2 {v_{40}}^2 + \frac{2\sigma\beta_e V_0}{{K_1}^2}{v_{40}}^2,$$

$$\bar{a}_{31} = \frac{2\beta_h}{K_2}\gamma_1\gamma_3 {v_{40}}^2 + \frac{2\beta_e}{K_1}\gamma_1 {v_{40}}^2 + \frac{2\sigma\beta_h}{K_2}\gamma_2\gamma_3 {v_{40}}^2 + \frac{2\sigma\beta_e}{K_1}\gamma_2 {v_{40}}^2 - \left[\frac{2\beta_h}{{K_2}^2}(S_0 + \sigma V_0) - \frac{2c}{b^2}\right]{\gamma_3}^2 {v_{40}}^2 - \frac{2\beta_e}{{K_1}^2}(S_0 + \sigma V_0){v_{40}}^2,$$

$$\bar{a}_{41} = 0.$$

Therefore,

$$\boldsymbol{\psi_0}^T D^2\boldsymbol{f}(P_0, \eta^*)(\boldsymbol{V_0}, \boldsymbol{V_0}) = 2\,\alpha_3 {v_{40}}^2 \psi_{40}\left[\frac{\beta_h}{k_2}\gamma_3(\gamma_1 + \sigma\gamma_2) + \frac{\beta_e}{k_1}(\gamma_1 + \sigma\gamma_2) - \left[\frac{\beta_h}{{k_2}^2}(S_0 + \sigma V_0) - \frac{c}{b^2}\right]{\gamma_3}^2 - \frac{\beta_e}{{k_1}^2}(S_0 + \sigma V_0)\right].$$

Obviously, $\boldsymbol{\psi_0}^T D^2\boldsymbol{f}(P_0, \eta^*)(\boldsymbol{V_0}, \boldsymbol{V_0}) \neq 0$ under the condition (21) and hence system (2) near $P_0 = (S_0, V_0, 0, 0)$ has a trans-critical bifurcation.

Now, to find the left eigenvector $\boldsymbol{\Phi_0} = (\phi_{10}, \phi_{20}, \phi_{30}, \phi_{40})$ that satisfy $\boldsymbol{\Phi_0 V_0} = 1$, we have to solve the solve $\boldsymbol{\Phi_0} J_0 = \boldsymbol{0}$. Straightforward computation gives that $\boldsymbol{\Phi_0} = (0, 0, \sigma_1\emptyset_{40}, \emptyset_{40})$, where $\sigma_1 = \frac{K_1\mu_2}{\beta_e(S_0 + \sigma V_0)} > 0$. Since we have $\boldsymbol{\Phi_0 V_0} = 1$ we get that $v_{40} = \bar{c}_{33} < 0;\ \emptyset_{40} = \frac{1}{\bar{c}_{33} + \bar{c}_{44}} < 0$.

Now by using the backward theorem given by Castillo-Chavez and Song (2004), system (2) undergoes a backward bifurcation around the disease free equilibrium point provided the following quantities are positive.

$$a = \sum_{k,i,j=1}^{4} \emptyset_{k0} v_{i0} v_{j0} \frac{\partial^2 f_k}{\partial x_i \partial x_j}(P_0, \eta^*)$$

$$b = \sum_{k,i=1}^{4} \emptyset_{k0} v_{i0} \frac{\partial^2 f_k}{\partial x_i \partial \eta}(P_0, \eta^*)$$

Therefore, direct computation gives that

$$a = \sigma_1\emptyset_{40}\, {v_{40}}^2 \left[2\gamma_1\gamma_3\frac{\beta_h}{K_2} + 2\gamma_1\frac{\beta_e}{K_1} + 2\gamma_2\gamma_3\frac{\sigma\beta_h}{K_2} + 2\gamma_2\frac{\sigma\beta_e}{K_1} + {\gamma_3}^2\left(-2\frac{\beta_h(S_0 + \sigma V_0)}{{K_2}^2} + 2\frac{c}{b^2}\right) - 2\frac{\beta_e(S_0 + \sigma V_0)}{{K_1}^2}\right],$$

$$b = \gamma_3\, v_{40}\emptyset_{40} > 0.$$

Now, since $a > 0$ under the sufficient condition (21b), hence the backward bifurcation is occurred around the disease free equilibrium point and hence the proof is complete. ■

Theorem 8.

Assume that the condition (10a) holds while (10b) is violated then system (2) near the endemic equilibrium point P_1 has a saddle-node bifurcation at $\mu_2 = {\mu_2}^*$ provided that

$$\zeta_1\psi_{41}\tilde{a}_{11} + \zeta_2\psi_{41}\tilde{a}_{21} + \zeta_3\psi_{41}\tilde{a}_{31} \neq 0, \tag{23}$$

where μ_2^* , ζ_i, $\psi_{41}, \tilde{a}_{i1}, i = 1,2,3$ are given in the proof.

Proof:

According to the characteristic equation of $J(P_1)$ that given by equation (12), there is a zero eigenvalue and hence P_1 will be nonhyperbolic point provided that $A_4 = 0$. Straightforward computation shows that this is (*i.e.,* $A_4 = 0$) equivalent to that

$$\mu_2^* = -\frac{d_{43}(d_{34}\Gamma_3+d_{24}\Gamma_9+d_{14}\Gamma_6)}{(d_{33}\Gamma_3+d_{23}\Gamma_9+d_{13}\Gamma_6)},$$

where d_{ij} and A_4 represent the elements of $J(P_1)$ and their determinant that given in equation (12) respectively, while Γ_3, Γ_6, and Γ_9 are given in equation (12). Therefore, the Jacobain matrix of system (2) around the point P_1 and $\mu_2 = \mu_2^*$, which denoted by $J_1 = J_1(P_1, \mu_2^*) = \left[\tilde{d}_{ij}(P_1, \mu_2^*)\right]_{4\times4}$, follows directly from equation (11).

Let $\boldsymbol{V_1} = (v_{11}, v_{21}, v_{31}, v_{41})^T$ be an eigenvector corresponding to the zero eigenvalue, say $\lambda_1(\mu_2^*) = 0$, then $J_1\boldsymbol{V_1} = \boldsymbol{0}$ gives that

$$\boldsymbol{V_1} = \begin{bmatrix} \rho_1\, v_{41} \\ \rho_2\, v_{41} \\ \rho_3\, v_{41} \\ v_{41} \end{bmatrix},$$

where $v_{41} \neq 0$ be any real number. While,

$$\rho_1 = -\frac{\tilde{d}_{12}\rho_2+\tilde{d}_{13}\rho_2+\tilde{d}_{14}}{\tilde{d}_{11}},$$
$$\rho_2 = -\frac{(\tilde{d}_{11}\tilde{d}_{23}-\tilde{d}_{13}\tilde{d}_{21})\rho_3+(\tilde{d}_{11}\tilde{d}_{24}-\tilde{d}_{14}\tilde{d}_{21})}{\Gamma_3},$$
$$\rho_3 = \frac{-\Gamma_9(\tilde{d}_{11}\tilde{d}_{24}-\tilde{d}_{14}\tilde{d}_{21})-\Gamma_3(\tilde{d}_{11}\tilde{d}_{34}-\tilde{d}_{14}\tilde{d}_{31})}{\Gamma_3(\tilde{d}_{11}\tilde{d}_{33}-\tilde{d}_{13}\tilde{d}_{31})+\Gamma_9(\tilde{d}_{11}\tilde{d}_{23}-\tilde{d}_{13}\tilde{d}_{21})}.$$

It is clear that under the condition (10a), we obtain that $\tilde{d}_{33} < 0$ and hence the quantities ρ_1, ρ_2 are nonzero quantities and ρ_3 is positive. Similarly, let $\boldsymbol{\psi_1} = (\psi_{11}, \psi_{21}, \psi_{31}, \psi_{41})^T$ be an eigenvector corresponding to $\lambda_1(\mu_2^*) = 0$ for of the matrix ${J_1}^T$, hence ${J_1}^T\boldsymbol{\psi_1} = \boldsymbol{0}$ gives that

$$\boldsymbol{\psi_1} = \begin{bmatrix} \zeta_1\psi_{41} \\ \zeta_2\psi_{41} \\ \zeta_3\psi_{41} \\ \psi_{41} \end{bmatrix},$$

where $\psi_{41} \neq 0$ be any real number. while

$$\zeta_1 = -\frac{(\tilde{d}_{21}\,\zeta_2+\tilde{d}_{31}\,\zeta_3)}{\tilde{d}_{11}},$$
$$\zeta_2 = -\frac{(\tilde{d}_{11}\tilde{d}_{32}-\tilde{d}_{12}\tilde{d}_{31})\,\zeta_3}{\Gamma_3},$$
$$\zeta_3 = -\frac{\tilde{d}_{11}\tilde{d}_{44}\Gamma_3}{\Gamma_3(\tilde{d}_{11}\tilde{d}_{34}-\tilde{d}_{14}\tilde{d}_{31})+\Gamma_9(\tilde{d}_{11}\tilde{d}_{24}-\tilde{d}_{14}\tilde{d}_{21})}.$$

Moreover, since

$$\frac{d\boldsymbol{f}}{d\mu_2} = \boldsymbol{f}_{\mu_2} = (0,0,0,-B)^{\mathrm{T}} \rightarrow \boldsymbol{f}_{\mu_2}(P_1,{\mu_2}^*) = (0,0,0,-B^*)^{\mathrm{T}}.$$

Then, $\boldsymbol{\psi_1}^T \boldsymbol{f}_{\mu_2}(P_1,{\mu_2}^*) = -\psi_{41}B^* \neq 0$, which leads according to Sotomayer's theorem to that system (2) near the endemic equilibrium point P_1 has a saddle node bifurcation if the following further condition holds $\boldsymbol{\psi_1}^T\, D^2\boldsymbol{f}(P_1,{\mu_2}^*)(\boldsymbol{V_1},\boldsymbol{V_1}) \neq 0$. Now straightforward computation gives that

$$D^2\boldsymbol{f}\big(P_1,{\mu_2}^*\big)(\boldsymbol{V_1},\boldsymbol{V_1}) = \big[\tilde{a}_{ij}\big]_{4\times 1},$$

with

$$\tilde{a}_{11} = -\frac{2K_2\beta_h}{(K_2+I^*)^2}\rho_1\rho_3\,{v_{41}}^2 - \frac{2K_1\beta_e}{(K_1+B^*)^2}\rho_1{v_{41}}^2 + \frac{2K_2\beta_h S^*}{(K_2+I^*)^3}{\rho_3}^2\,{v_{41}}^2 + \frac{2K_1\beta_e S^*}{(K_1+B^*)^3}{v_{41}}^2,$$
$$\tilde{a}_{21} = -\frac{2K_2\sigma\beta_h}{(K_2+I^*)^2}\rho_2\rho_3\,{v_{41}}^2 - \frac{2K_1\sigma\beta_e}{(K_1+B^*)^2}\rho_2\,{v_{41}}^2 + \frac{2K_2\sigma\beta_h V^*}{(K_2+I^*)^3}{\rho_3}^2\,{v_{41}}^2 + \frac{2K_1\sigma\beta_e V^*}{(K_1+B^*)^3}{v_{41}}^2,$$
$$\tilde{a}_{31} = \frac{2K_2\beta_h}{(K_2+I^*)^2}\rho_1\rho_3\,{v_{41}}^2 + \frac{2K_1\beta_e}{(K_1+B^*)^2}\rho_1{v_{41}}^2 + \frac{2K_2\sigma\beta_h}{(K_2+I^*)^2}\rho_2\rho_3\,{v_{41}}^2 + \frac{2K_1\sigma\beta_e}{(K_1+B^*)^2}\rho_2\,{v_{41}}^2$$
$$-\left[\frac{2K_2\beta_h}{(K_2+I^*)^3}(S^*+\sigma V^*) - \frac{2bc}{(b+I^*)^3}\right]{\rho_3}^2\,{v_{41}}^2 - \frac{2K_1\beta_e}{(K_1+B^*)^3}(S^*+\sigma V^*){v_{41}}^2,$$
$$\tilde{a}_{41} = 0.$$

Therefore, by using condition (23) we obtain that

$$\boldsymbol{\psi_1}^T\, D^2\boldsymbol{f}(P_1,{\mu_2}^*)(\boldsymbol{V_1},\boldsymbol{V_1}) = \zeta_1\psi_{41}\tilde{a}_{11} + \zeta_2\psi_{41}\tilde{a}_{21} + \zeta_3\psi_{41}\tilde{a}_{31} \neq 0.$$

Hence, the proof is complete. ■

7. Numerical simulation

In this section an investigation to the long-time behavior of the solution of system (1) is performed. The objective is to demonstrate the analytical findings numerically and study the effect of varying the parameters on the dynamical behavior of the system. In order to solve system (1) numerically and then plot the time series of the obtained solution the following hypothetical set of parameters values is adopted.

$$A = 500, K_1 = 50, K_2 = 50, c = 0.75, b = 0.1, \beta_e = 0.1, \beta_h = 0.1$$
$$\gamma_1 = 0.2, \gamma_2 = 0.1, \mu_1 = 0.01, \sigma = 0.3, d = 0.3, \eta = 0.1, \mu_2 = 0.4. \quad (24)$$

Furthermore different sets of initial values are choosing and then solve the system numerically. It is obtain that the system (1) approaches asymptotically to the endemic equilibrium point as shown in Figure 1.

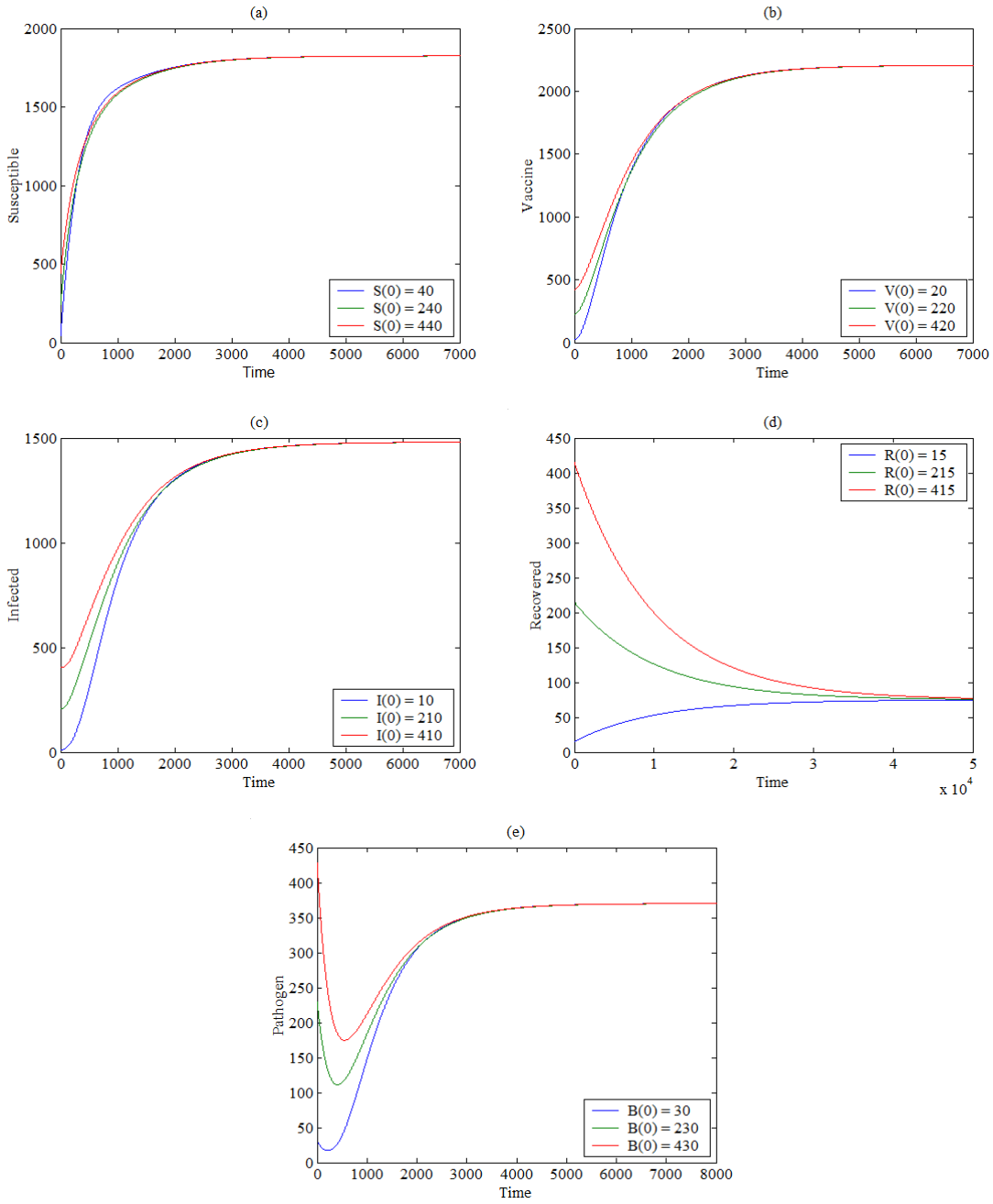

Figure 1: Time series of the solution of system (1) for the data in equation (24). (a) $S(t)$ versus time. (b) $V(t)$ versus time. (c) $I(t)$ versus time. (d) $R(t)$ versus time. (e) $B(t)$ versus time.

It is clear from Figure 1 that system (1) has a globally asymptotically stable endemic equilibrium point $(1825.16, 2206.3, 1480.43, 74.59, 370.1)$, for the data given by equation (24), where $R_0 = 8.777 > 1$, which confirm our obtained analytical findings.

Now for the data (24) with $A = 100, 50, 20$ the values of basic reproduction numbers are respectively given by $R_0 = 1.755, 0.8777, 0.351$ and the solution of system (1) plotted in Figure (2). While, for the data (24) with $\mathrm{A} = 60$ and $\eta = 0.1, 0.01$, it is observed that the values of basic reproduction numbers are $\mathrm{R}_0 = 1.05324, 0.863657$ respectively, and then the solution of system (1) is drawn in Figure 3.

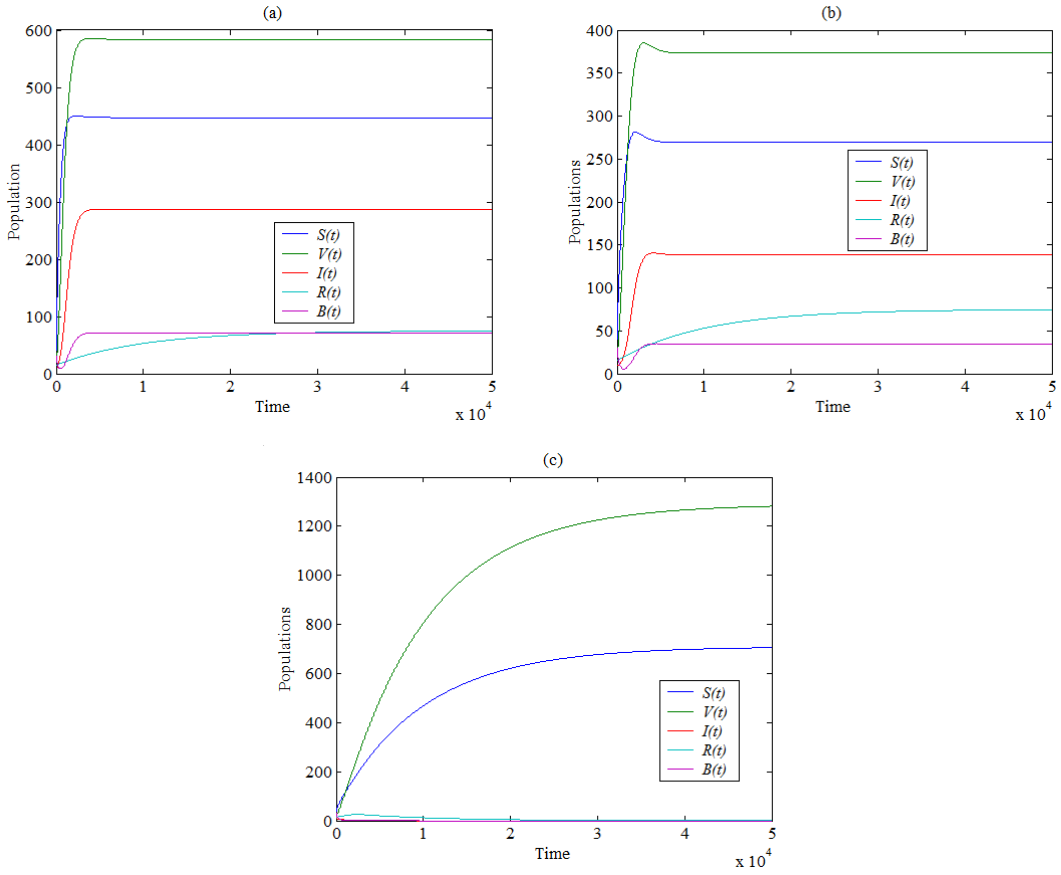

Figure 2: Time series of the solution of system (1) for the data given by equation (24). (a) When $A = 100$ with $\mathrm{R}_0 = 1.7554 > 1$. (b) When $A = 50$ with $\mathrm{R}_0 = 0.8777 < 1$. (c) When $A = 20$ with $\mathrm{R}_0 = 0.35108 < 1$.

backward bifurcation. On the other hand, the system (1) approaches to endemic equilibrium point $(447.31, 583.87, 286.89, 74.56, 71.72)$ for $A = 100$ with $\mathrm{R}_0 = 1.7554 > 1$. However, it approaches to disease free equilibrium point $(709.6, 1290.3, 0, 0, 0)$ for $A = 20$ with $\mathrm{R}_0 = 0.35108 < 1$.

Similarly, Figure (3) demonstrates that system (1) undergoes a backward bifurcation as the parameter η passes through 0.01, as shown in Figure (3b), which is confirmed our obtained analytical findings too.

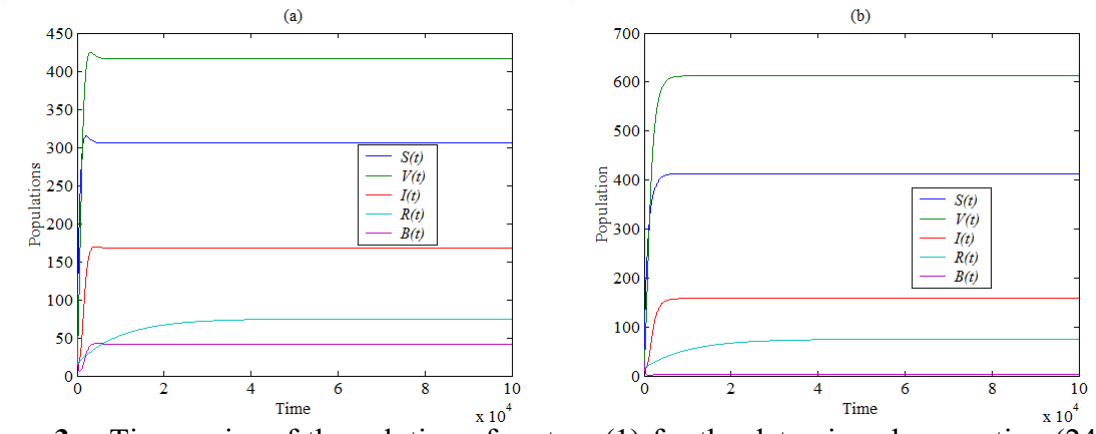

Figure 3: Time series of the solution of system (1) for the data given by equation (24), which approaches asymptotically to endemic equilibrium point. (a) When $A = 60$ with $\mathrm{R}_0 = 1.05324 > 1$. (b) When $A = 60$ and $\eta = 0.01$ with $\mathrm{R}_0 = 0.863657 < 1$.

Clearly, according to the Figure (4a) for the data given by equation (24) with $A = 60$, $K_1 = 1000$ and $K_2 = 50$, the value of basic reproduction number is less than 1 ($R_0 = 0.853125 < 1$) but the solution of the system (1) still staled at the endemic equilibrium point $(428, 639.41, 156.69, 74.95, 39.17)$, which indicates to occurrence of backward bifurcation too. Moreover, increasing the value of K_2 up to 73 reduced the value of basic reproduction number to $R_0 = 0.58768 < 1$, then the backward bifurcation disappeared, and the solution of the system (1) approached to the disease-free equilibrium point $(2128.94, 3870.78, 0, 0, 0)$, as shown in Figure (4b). Finally, for the same parameters set used in Figure (4b) with changing the initial value from (40, 20, 10, 15, 30) to (80, 60, 50, 55, 70) the solution approached again to another endemic equilibrium point $(464.11, 706.73, 153.37, 74.95, 38.34)$, as shown in Figure (4c), which indicates to occurrence of backward bifurcation too. This is happened due to the possibility of existence of both the disease free equilibrium point and many endemic equilibrium points simultaneously, in case of $R_0 < 1$ due to the higher order polynomial degree given in equation (6), for different sets of parameters values.

Finally, other parameters values are also varying and the solution of system (1) is determined and plotted. Similar results are also obtained to that demonstrated above, which indicates the higher sensitivity of system (1) for changing the parameter values.

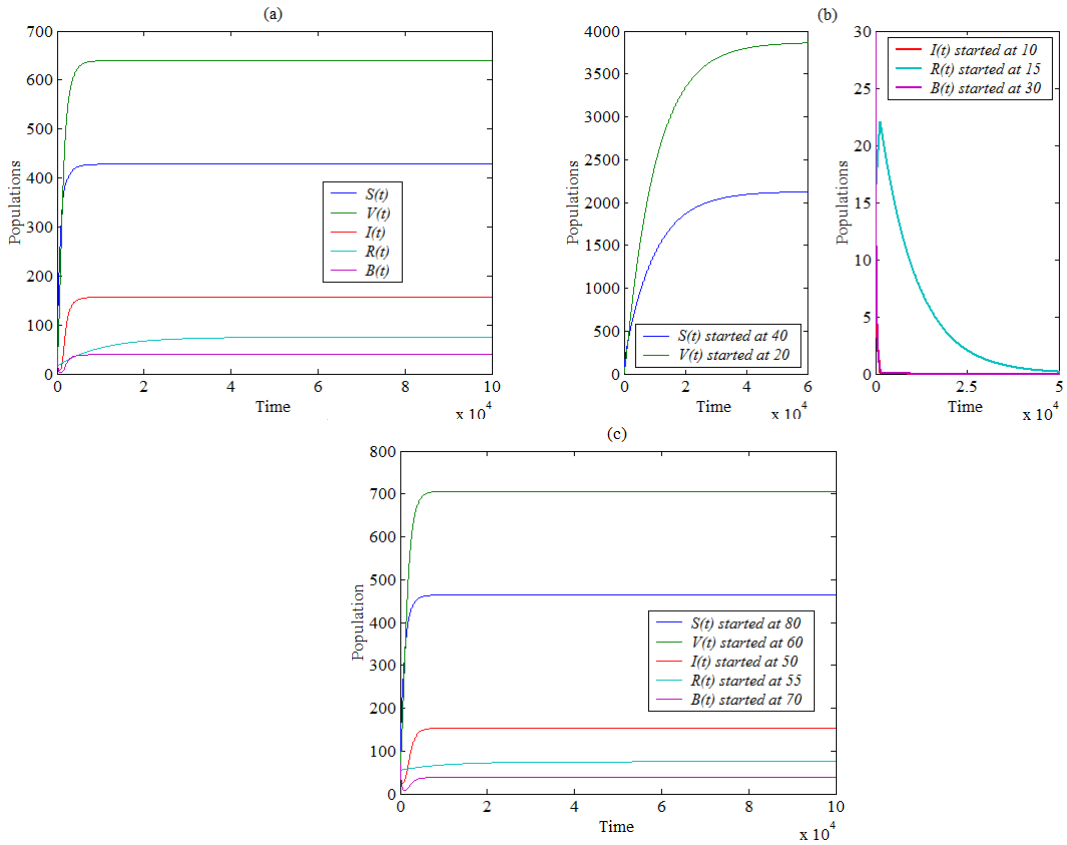

Figure 4: Time series of the solution of system (1) for the data given by equation (24) with $A = 60$, $K_1 = 1000$. (a) When $K_2 = 50$ with $\mathrm{R}_0 = 0.853125 < 1$. (b) When $K_2 = 73$ with $\mathrm{R}_0 = 0.58768 < 1$. (c) When $K_2 = 73$ with $\mathrm{R}_0 = 0.58768 < 1$, starting from different initial value.

8. Dissection

In this paper, a mathematical model of Cholera with saturated treatment function and dose-response functions as incident rates was proposed and studied. It is assumed that a portion of the imperfect vaccine population becomes infected too through contact with an infected person or with contaminated environment resources. The existence of the disease-free equilibrium point and endemic equilibrium point is discussed with the help of a basic reproduction number. It is observed that the endemic equilibrium point exists uniquely for the system (2) provided that the conditions (7a), which assumes a basic reproduction number is greater than unity, and (7b) are satisfied simultaneously. Otherwise, the system (2) may or may not have at least one endemic point. Different mathematical tools have been used to study stability, such as the linearization technique, the Lyapunov method, and Li and Muldowney geometrical approach. Furthermore, it is well known that the disease is endemic if the infected population remains above a certain positive level for a sufficiently large time. Therefore, this definition of the endemic concept has been

characterized with the help of the notion of uniform persistence using the Freedman and Waltman approach.

The local bifurcation around the equilibrium points is investigated using the Sotomayer theorem. While backward bifurcation is investigated using Castillo-Chavez and Song theorem. It is observed that, under certain conditions, the system (2) undergoes a trans-critical bifurcation near the disease-free equilibrium point, while it has a saddle-node bifurcation near an endemic equilibrium point. Finally, numerical simulation is used to investigate the global dynamics and demonstrate the results of the analytical findings.

9. Conclusion

It is well known that having a basic reproduction number (R_0) below unity eradicates the disease. But in the proposed model (1), $R_0 < 1$ is not sufficient to eliminate the Cholera from the population, and the Cholera may be still persistent, this is due high possibility of the existence of a number of endemic equilibrium point when $R_0 < 1$. Hence, it's important to find another threshold value less than one, and then R_0 should be reduced below this value to eliminate the Cholera from the population. It is observed that the model is very sensitive to varying in the values of the parameters especially those responses on the concentration of the Vibrio cholerae in the environment or infected population (K_1, K_2); liberation rate of Vibrio cholerae by infected population (η) and death rate of Vibrio cholerae (μ_2) that depends on the (η) too. Accordingly, we conclude that the basic reproduction number is not enough to describe whether Cholera will disappear or not and suggest that we should pay more attention to the initial state of Cholera.

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