



Research article

Dynamic modeling and analysis of Hepatitis B epidemic with general incidence

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Abstract: New stochastic and deterministic Hepatitis B epidemic models with general incidence are established to study the dynamics of Hepatitis B virus (HBV) epidemic transmission. Optimal control strategies are developed to control the spread of HBV in the population. In this regard, we first calculate the basic reproduction number and the equilibrium points of the deterministic Hepatitis B model. And then the local asymptotic stability at the equilibrium point is studied. Secondly, the basic reproduction number of the stochastic Hepatitis B model is calculated. Appropriate Lyapunov functions are constructed, and the unique global positive solution of the stochastic model is verified by Itô formula. By applying a series of stochastic inequalities and strong number theorems, the moment exponential stability, the extinction and persistence of HBV at the equilibrium point are obtained. Finally, using the optimal control theory, the optimal control strategy to eliminate the spread of HBV is developed. To reduce Hepatitis B infection rates and to promote vaccination rates, three control variables are used, for instance, isolation of patients, treatment of patients, and vaccine inoculation. For the purpose of verifying the rationality of our main theoretical conclusions, the Runge-Kutta method is applied to numerical simulation.

Keywords: stochastic epidemic model; Hepatitis B; extinction; persistence; optimal control

1. Introduction

Hepatitis B is a global health problem with a high incidence rate in developing countries. According to statistics, asymptomatic HBV carriers in the world exceed 280 million, and China accounts for about 130 million. HBV infects liver cells when it enters the body. Most Hepatitis infections are caused by viruses, infections, germs or addiction to ethyl alcohol and medicines. The transmission of HBV can take place in a variety of ways, for instance, transmission of blood, bodily fluid transmission and mother-to-child vertical transmission, and so on. Vaccination against Hepatitis B is the most basic measure for preventing and controlling the disease. Many mathematicians and biologists have studied

the Hepatitis B epidemic. They built different mathematical models to analyze the dynamic behavior of HBV. Then stability theory, bifurcation phenomenon, analysis of sensitivity and optimal control strategies of the infectious disease models are studied. This not only helps to achieve a reduction in Hepatitis B transmission, but also helps to prevent Hepatitis B in daily life. For example, in [1, 2], the authors established the epidemic models with bilinear incidence for Hepatitis B. Hepatitis B can be effectively controlled through optimal control strategies. In [3], a Hepatitis B transmission model is established as follows:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \frac{\alpha_1 S I}{1 + \alpha_2 I} - (\mu_0 + \nu) S, \\ \frac{dI}{dt} = \frac{\alpha_1 S I}{1 + \alpha_2 I} - (\mu_0 + \mu_1 + \gamma) I, \\ \frac{dR}{dt} = \gamma I + \nu S - \mu_0 R, \\ S(0) \geq 0, I(0) \geq 0, R(0) \geq 0, \end{cases} \quad (1.1)$$

where α_1 represents the transmission rate of HBV, α_2 represents the saturation rate. The kinetic behavior of HBV model is discussed with stability theory. In [4, 5], the authors established the fractional HBV model. By means of the fractional Routh-Hurwitz stability criterion, the global dynamic behavior of fractional Hepatitis B model is studied.

Epidemics are strongly influenced by environmental changes. In the case of human diseases, since one person's contact with another is unpredictable, the prevalence and spread of infectious diseases is random. Therefore, it is necessary to incorporate random effects into mathematical models [6, 7]. By doing so, we are able to develop a more reasonable model. Modeling using stochastic differential equations is a very suitable method in the study of epidemic dynamics. Many researchers use stochastic infectious disease models to investigate different diseases. Here we focus on the stochastic HBV models. Khan et al. [8] proposed a stochastic HBV epidemic model with bilinear incidence as follows:

$$\begin{cases} \frac{dS}{dt} = [\Lambda - \alpha S I - (\mu_0 + \nu) S] dt - \eta S I dB(t), \\ \frac{dI}{dt} = [\alpha S I - (\mu_0 + \mu_1 + \gamma) I] dt + \eta S I dB(t), \\ \frac{dR}{dt} = (\gamma I + \nu S - \mu_0 R) dt, \\ S(0) \geq 0, I(0) \geq 0, R(0) \geq 0, \end{cases} \quad (1.2)$$

where α represents the bilinear incidence rate, $\eta^2 > 0$ represents the intensity of white noise, $B(t)$ represents the Brownian motion. Based on the theory of stochastic Lyapunov function, the dynamic behavior of Hepatitis B stochastic model is studied. Anwarud Din et al. [9–11] proposed the stochastic models of Hepatitis B with standard incidence. Wu et al. [12] established a stochastic delay model of Hepatitis B with bilinear incidence. Anwarud Din et al. [13] built a stochastic time-delay model of Hepatitis B with standard incidence. In [14], a new stochastic Hepatitis B epidemic model that includes white noise, Markov switching, and vaccination control was developed. The above studies concluded that high noise can guarantee the extinction of Hepatitis B.

To sum up, most of the current research focus on HBV models with bilinear incidence, standard incidence and saturated incidence, and few studies on HBV models with environmental noise disturbance and general incidence. The general incidence rate is more realistic than the bilinear incidence

rate, standard incidence rate and saturated incidence rate. This is the research motivation of this paper. New stochastic and deterministic models of Hepatitis B epidemic with general incidence rate are established. The dynamic behavior of HBV model is studied, and the influence of environmental white noise on the epidemic of HBV is analyzed, and the optimal control strategy for eliminating HBV is developed. The research work in this paper is an extension of the work on [3, 8–11]. The meanings of parameters in the models studied in this paper are as follows:

Λ : the birth rate;

β : infection rate from susceptible population to Hepatitis B;

μ_0 : the natural mortality rate;

μ_1 : the mortality rate from HBV;

γ : the recovery rate of HBV;

ν : the vaccination rate of HBV.

Here is a breakdown of this article's organization. In Section 2, the new models of Hepatitis B are established. In Section 3, we obtain the basic reproduction number and the equilibrium points for the deterministic epidemic model of Hepatitis B. Lyapunov function is used to prove the local asymptotic stability. In Section 4, we verify that the stochastic model has one and only one global positive solution. The extinction, persistence and moment exponential stability of stochastic model are studied by means of stochastic Lyapunov function theory. In section 5, the optimal control strategy to eliminate HBV is developed by using the optimal control theory. To reduce Hepatitis B infection rates and to promote vaccination rates, three control variables are used, for instance, isolation of patients, treatment of patients, and vaccine inoculation. In Section 6, Runge-Kutta method is used for numerical simulation to support our main theoretical conclusions. Section 7 provides a brief summary and outlook of the main findings.

2. Mathematical model formulation

New deterministic and stochastic mathematical models of HBV transmission are established. We make the following assumptions about the models:

(A₁). $N(t)$ represents the total population at time t , which is divided into three parts: susceptible persons $S(t)$, infected persons $I(t)$ and convalescent patients $R(t)$. Namely, $N(t) = S(t) + I(t) + R(t)$.

(A₂). All parameter values of the models are non-negative.

(A₃). The incidence is set as nonlinear incidence rate.

(A₄). Once successfully vaccinated or cured by treatment, immunity is considered permanent.

The supposed conditions (A₁)–(A₄) lead to Hepatitis B epidemic model as below:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \frac{\beta SI}{f(S, I)} - (\mu_0 + \nu) S, \\ \frac{dI}{dt} = \frac{\beta SI}{f(S, I)} - (\mu_0 + \mu_1 + \gamma) I, \\ \frac{dR}{dt} = \gamma I + \nu S - \mu_0 R, \end{cases} \quad (2.1)$$

with $S(0) = S_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0$, where β represents the transmission rate of Hepatitis B, $f(S, I) = 1 + a_1S + a_2I + a_3SI$. For convenience, we define $g(S, I) = \frac{\beta SI}{f(S, I)} = \frac{\beta SI}{1 + a_1S + a_2I + a_3SI}$. Here

$g(S, I)$ is the incidence rate, where $a_1, a_2, a_3 \geq 0$. Morbidity is the number of new infections per population in a given time period. To simulate the spread of disease, some scholars use bilinear incidence (see [15–17]), standard incidence (see [18–20]) and saturated incidence (see [21, 22]). However, the nonlinear morbidity $g(S, I)$ takes many forms, each of which has its own advantages, as below:

- (i) When $a_1 = a_2 = a_3 = 0$, $g(S, I)$ represents the bilinear incidence;
- (ii) When $a_1 = a_3 = 0$ or $a_2 = a_3 = 0$, $g(S, I)$ is the saturation incidence [3];
- (iii) When $a_3 = 0$, $g(S, I)$ is the Beddington-DeAngelis functional response as shown in [23];
- (iv) When $a_3 = a_1 a_2$, $g(S, I)$ is the Crowley-Martin functional response as shown in [24].

On the other hand, the following stochastic epidemic model of Hepatitis B is studied by incorporating environmental noise into the above model (2.1):

$$\begin{cases} dS = \left(\Lambda - \frac{\beta SI}{f(S, I)} - (\mu_0 + \nu) S \right) dt - \frac{\sigma SI}{f(S, I)} dB(t), \\ dI = \left(\frac{\beta SI}{f(S, I)} - (\mu_0 + \mu_1 + \gamma) I \right) dt + \frac{\sigma SI}{f(S, I)} dB(t), \\ dR = (\gamma I + \nu S - \mu_0 R) dt, \end{cases} \quad (2.2)$$

with $S(0) = S_0 \geq 0, I(0) = I_0 \geq 0, R(0) = R_0 \geq 0$, where $B(t)$ represents the Brownian motion, $\sigma > 0$ represents the white noise intensity.

3. Asymptotic behavior of deterministic Hepatitis B epidemic model (2.1)

3.1. Basic reproduction number and disease-free equilibrium point E^0

Let $\frac{dS}{dt} = 0, \frac{dI}{dt} = 0, \frac{dR}{dt} = 0$, then $E^0 = (S^0, I^0, R^0) = \left(\frac{\Lambda}{\mu_0 + \nu}, 0, \frac{\nu \Lambda}{(\mu_0 + \nu) \mu_0} \right)$, for $I^0 = 0$. The basic reproduction number of Hepatitis B model (2.1) is given below. Let $X = (S, I, R)$, (2.1) is written as $\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X)$, where

$$\mathcal{F}(X) = \begin{pmatrix} \Lambda \\ \frac{\beta SI}{f(S, I)} \\ \gamma I + \nu S \end{pmatrix}, \mathcal{V}(X) = \begin{pmatrix} \frac{\beta SI}{f(S, I)} + (\mu_0 + \nu) S \\ (\mu_0 + \mu_1 + \gamma) I \\ \mu_0 R \end{pmatrix}. \quad (3.1)$$

The Jacobian of equation (3.1) around E^0 . R_0^d can be obtained by calculating the spectral radius of $\mathcal{F}\mathcal{V}^{-1}$. Then, $R_0^d = \frac{\beta \Lambda}{(\mu_0 + \nu + a_1 \Lambda)(\mu_0 + \mu_1 + \gamma)}$.

Theorem 3.1 E^0 is locally asymptotically stable if $R_0^d < 1$, otherwise E^0 is unstable.

Proof. The Jacobian matrix of Hepatitis B epidemic model (2.1) at E^0 can be calculated as follows:

$$J|_{E^0} = \begin{pmatrix} -(\mu_0 + \nu) & \frac{\beta \Lambda}{\mu_0 + \nu + a_1 \Lambda} & 0 \\ 0 & \frac{\beta \Lambda}{\mu_0 + \nu + a_1 \Lambda} - (\mu_0 + \mu_1 + \gamma) & 0 \\ \nu & \gamma & -\mu_0 \end{pmatrix}.$$

After calculation, $J|_{E^0}$ has three eigenvalues:

$$\lambda_1 = -(\mu_0 + \nu) < 0, \lambda_2 = -\mu_0 < 0, \lambda_3 = -(\mu_0 + \mu_1 + \gamma)(1 - R_0^d).$$

Obviously, the sign of λ_3 depends on R_0^d . If $R_0^d < 1$, then the eigenvalues of $J|_{E^0}$ are all negative. Thus, HBV model (2.1) is locally asymptotically stable at E^0 . On the contrary, if $R_0^d > 1$, then λ_3 is positive, so E^0 is unstable.

3.2. Endemic equilibrium point E^* and stability

By simple calculation, we have $E^* = (S^*, I^*, R^*)$, for $I^* \neq 0$, where $t_1 = \mu_0 + \mu_1 + \gamma$, $t_2 = \mu_0 + \nu$,

$$S^* = \frac{\Lambda - t_1 I^*}{t_2}, \quad I^* = \frac{-\Delta_1 + \sqrt{\Delta_1^2 - 4t_1^2 a_3 \Delta_2}}{2t_1^2 a_3}, \quad R^* = \frac{\gamma I^* + \nu S^*}{\mu_0},$$

$$\Delta_1 = t_1^2 a_1 - \Lambda t_1 a_3 - t_1 t_2 a_2 - \beta t_1, \quad \Delta_2 = \beta \Lambda - t_1 t_2 - \Lambda t_1 a_1.$$

Lemma 3.1 If $R_0^d > 1$, then E^* exists, otherwise it does not exist.

Proof. If $R_0^d > 1$, we have

$$\begin{aligned} \Delta_1 &= t_1^2 a_1 - \Lambda t_1 a_3 - t_1 t_2 a_2 - \beta t_1 \\ &< (\mu_0 + \mu_1 + \gamma) \left[-(\mu_0 + \nu) \left(\frac{\mu_0 + \mu_1 + \gamma}{\Lambda} + a_2 \right) - \Lambda a_3 \right] < 0, \\ \Delta_2 &= \beta \Lambda - t_1 t_2 - \Lambda t_1 a_1 \\ &= (\mu_0 + \nu + a_1 \Lambda) (\mu_0 + \mu_1 + \gamma) (R_0^d - 1) > 0. \end{aligned}$$

And by calculation, we get $\Delta_1^2 - 4t_1^2 a_3 \Delta_2 > 0$. Thus, $I^* > 0$. Because $2\Lambda t_1 a_3 + \Delta_1 > \sqrt{\Delta_1^2 - 4t_1^2 a_3 \Delta_2}$, so $\Lambda - \frac{-\Delta_1 + \sqrt{\Delta_1^2 - 4t_1^2 a_3 \Delta_2}}{2t_1 a_3} > 0$. Then, $\Lambda - t_1 I^* > 0$. Hence, we have $S^* > 0$, thereby $R^* > 0$. Consequently, E^* exists, if $R_0^d > 1$.

Theorem 3.2 E^* is locally asymptotically stable if $R_0^d > 1$, otherwise E^* is unstable.

Proof. The Jacobian matrix of Hepatitis B epidemic model (2.1) at E^* can be calculated as follows:

$$J|_{E^*} = \begin{pmatrix} -(\mu_0 + \nu) - \frac{\beta I^*(1+a_2 I^*)}{f^2(S^*, I^*)} & -\frac{\beta S^*(1+a_1 S^*)}{f^2(S^*, I^*)} & 0 \\ \frac{\beta I^*(1+a_2 I^*)}{f^2(S^*, I^*)} & \frac{\beta S^*(1+a_1 S^*)}{f^2(S^*, I^*)} - (\mu_0 + \mu_1 + \gamma) & 0 \\ \nu & \gamma & -\mu_0 \end{pmatrix}.$$

By calculation, the first eigenvalue of $J|_{E^*}$ is $\lambda_1 = -\mu_0 < 0$. Take

$$A = \begin{pmatrix} -(\mu_0 + \nu) - \frac{\beta I^*(1+a_2 I^*)}{f^2(S^*, I^*)} & -\frac{\beta S^*(1+a_1 S^*)}{f^2(S^*, I^*)} \\ \frac{\beta I^*(1+a_2 I^*)}{f^2(S^*, I^*)} & \frac{\beta S^*(1+a_1 S^*)}{f^2(S^*, I^*)} - (\mu_0 + \mu_1 + \gamma) \end{pmatrix}.$$

By $\frac{dI}{dt} = 0$, one has $\frac{\beta S^*}{f(S^*, I^*)} = (\mu_0 + \mu_1 + \gamma)$. Thus, $\beta S^* = (\mu_0 + \mu_1 + \gamma) f(S^*, I^*)$. By the definition of f , we get $\beta S^* (1 + a_1 S^*) < (\mu_0 + \mu_1 + \gamma) f^2(S^*, I^*)$. So,

$$\frac{\beta S^* (1 + a_1 S^*)}{f^2(S^*, I^*)} < \mu_0 + \mu_1 + \gamma. \quad (3.2)$$

Then, by (3.2), we obtain

$$\text{trac}(A) = \left[-(\mu_0 + \nu) - \frac{\beta I^* (1 + a_2 I^*)}{f^2(S^*, I^*)} \right] + \left[\frac{\beta S^* (1 + a_1 S^*)}{f^2(S^*, I^*)} - (\mu_0 + \mu_1 + \gamma) \right] < 0$$

and

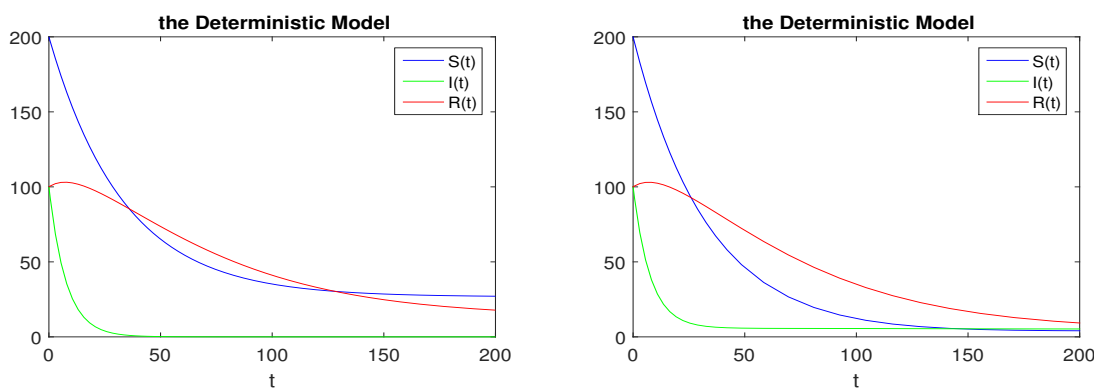
$$\begin{aligned} \det(A) &= \left((\mu_0 + \nu) + \frac{\beta I^* (1 + a_2 I^*)}{f^2(S^*, I^*)} \right) \left((\mu_0 + \mu_1 + \gamma) - \frac{\beta S^* (1 + a_1 S^*)}{f^2(S^*, I^*)} \right) \\ &\quad + \frac{\beta S^* (1 + a_1 S^*)}{f^2(S^*, I^*)} \cdot \frac{\beta I^* (1 + a_2 I^*)}{f^2(S^*, I^*)} > 0. \end{aligned}$$

Thus, $\text{trac}(A) < 0$, $\det(A) > 0$ if $R_0^d > 1$. According to Routh-Hurwitz criterion, the two eigenvalues of the matrix A are negative. It can therefore be concluded that E^* is locally asymptotically stable when $R_0^d > 1$.

3.3. Numerical simulation

Example 3.1 In model (2.1), let $\Lambda = 0.8$, $\beta = 0.01$, $a_1 = 0.1$, $a_2 = 0.2$, $a_3 = 1.2$, $\mu_0 = 0.02$, $\mu_1 = 0.1$, $\nu = 0.01$, $\gamma = 0.01$, $S(0) = 200$, $I(0) = 100$, $R(0) = 100$. After the calculation, we get $E^0 = (27, 0, 13)$ and $R_0^d = 0.56 < 1$. By observing Figure 1(a), it can be concluded that E^0 is locally asymptotically stable, which verifies the rationality of Theorem 3.1.

Example 3.2 The values of the parameters except $\beta = 0.9$ are the same as those in Example 3.1. After calculation, we get $E^* = (5, 5, 10)$ and $R_0^d = 50 > 1$. By observing Figure 1(b), it can be concluded that E^* is locally asymptotically stable, which verifies the rationality of Theorem 3.2.



(a). Simulations of $(S(t), I(t), R(t))$, when $\beta = 0.01$. (b). Simulations of $(S(t), I(t), R(t))$, when $\beta = 0.9$.

Figure 1. Simulations of $(S(t), I(t), R(t))$ in the deterministic model (2.1).

4. Dynamical behavior of the stochastic HBV model (2.2)

Let $(\Omega, \mathcal{F}, \mathbb{P})$ be one complete probability space whose filtration $\{\mathcal{F}_t\}_{t \geq 0}$ satisfies the usual conditions (i.e., $\{\mathcal{F}_t\}_{t \geq 0}$ is monotonically increasing and right-continuous while \mathcal{F}_0 contains all \mathbb{P} -null sets). We think over the following n -dimensional stochastic differential equation:

$$dx(t) = f(x(t), t)dt + g(x(t), t)dB(t), \quad \forall t \geq t_0, \tag{4.1}$$

with $x(0) = x_0 \in \mathbb{R}^n$, where $f(x(t), t) : \mathbb{R}^n \times [0, +\infty] \rightarrow \mathbb{R}^n$ and $g(x(t), t) : \mathbb{R}^n \times [0, +\infty] \rightarrow \mathbb{R}^{n \times m}$ are locally Lipschitz functions in x . $B(t)$ represents the n -dimensional Brownian motion defined on $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbb{P})$. $C^{2,1}(\mathbb{R}^n \times [0, +\infty], \mathbb{R}_+)$ is a family of all nonnegative functions $V(x, t)$ defined on $\mathbb{R}^n \times [0, +\infty]$, making them continuously differentiable twice in x and once in t . The differential operator L associated with (4.1) is defined [25] by:

$$L = \frac{\partial}{\partial t} + \sum_{i=1}^n f_i(x, t) \frac{\partial}{\partial x_i} + \frac{1}{2} \sum_{i,j=1}^n [g^T(x, t) g(x, t)]_{ij} \frac{\partial^2}{\partial x_i \partial x_j}. \tag{4.2}$$

If L acts on $V \in C^{2,1}(\mathbb{R}^n \times [t_0, \infty], \mathbb{R}_+)$, then

$$LV(x, t) = V_t(x, t) + V_x(x, t) f(x, t) + \frac{1}{2} \text{trace} [g^T(x, t) V_{xx}(x, t) g(x, t)]. \tag{4.3}$$

If $x(t) \in \mathbb{R}^n$, by Itô's formula, one has

$$dV(x, t) = LV(x, t) dt + V_x(x, t) g(x, t) dB(t). \quad (4.4)$$

Definition 4.1 [26] The equilibrium point $x = 0$ of (4.1) is considered to be p th moment exponentially stable, if there are $C_1, C_2 > 0$ so that

$$\mathbb{E}(|x(x_0, t)|^p) \leq C_1|x_0|^p e^{-C_2 t}, \quad \forall x_0 \in \mathbb{R}^n, t \geq 0. \quad (4.5)$$

4.1. The existence and uniqueness of positive solutions

A bounded set Δ is defined as below:

$$\Delta := \left\{ x = (x_1, x_2, x_3) : x_1 > 0, x_2 > 0, x_3 > 0, x_1 + x_2 + x_3 < \frac{\Lambda}{\mu_0} \text{ a.s.} \right\}.$$

Theorem 4.1 For $(S(0), I(0), R(0)) \in \Delta$, model (2.2) possesses one unique positive solution $(S(t), I(t), R(t))$ on $t \geq 0$. The solution is still in \mathbb{R}_+^3 with probability 1, namely, $(S(t), I(t), R(t)) \in \mathbb{R}_+^3$ on $t \geq 0$ a.s.

Proof. See Appendix A.

Corollary 4.1 The set Δ is positively invariant; in other words, if $(S(0), I(0), R(0)) \in \Delta$, then $\mathbb{P}((S(t), I(t), R(t)) \in \Delta) = 1, \forall t \geq 0$.

4.2. Moment exponential stability

Lemma 4.1 [26] If there is the function $V(t, x) \in C^{1,2}(\mathbb{R} \times \mathbb{R}^n)$ so that

$$K_1|x|^p \leq V(t, x) \leq K_2|x|^p, LV(t, x) \leq -K_3|x|^p, t \geq 0, p > 0, K_i > 0, i = 1, 2, 3, \quad (4.6)$$

then the equilibrium point of the Eq (4.1) is p th moment exponentially stable. This implies that the number of infected people goes extinct at an exponential rate. When $p = 2$, it usually means that it is exponentially stable at the mean square, and global asymptotically stable at the equilibrium point $x = 0$.

Lemma 4.2 [27] Let $p \geq 2, \varepsilon, x, y > 0$, then the following two inequalities are true

$$x^{p-1}y \leq \frac{(p-1)\varepsilon}{p}x^p + \frac{1}{p\varepsilon^{p-1}}y^p, \quad x^{p-2}y^2 \leq \frac{(p-2)\varepsilon}{p}x^p + \frac{2}{p\varepsilon^{(p-2)/2}}y^p. \quad (4.7)$$

Theorem 4.2 Let $p \geq 2$. If $R_0^d < 1$ and

$$\sigma^2 < \frac{2[(\mu_0 + \mu_1 + \gamma)(\mu_0 + a_1\Lambda) - \beta\Lambda](\mu_0 + a_1\Lambda)}{(p-1)\Lambda^2}, \quad (4.8)$$

then E^0 is p th moment exponentially stable in Δ .

Proof. See Appendix B.

Corollary 4.2 If $R_0^d < 1$ and

$$\sigma^2 < \frac{2[(\mu_0 + \mu_1 + \gamma)(\mu_0 + a_1\Lambda) - \beta\Lambda](\mu_0 + a_1\Lambda)}{\Lambda^2}, \quad (4.9)$$

then E^0 is globally asymptotically stable in Δ .

4.3. Extinction of the disease

In studying Hepatitis B model, we are interested in when the disease becomes extinct and when it becomes persistent in the population. This section establishes sufficient conditions for disease elimination in model (2.2). First, we need to give the basic symbols and lemmas associated with this problem. For the integrable function $x(t)$ defined on $(0, \infty)$, is denoted as $\langle x(t) \rangle = \frac{1}{t} \int_0^t x(s) ds$. The threshold R_0^s of model (2.2) is as follows

$$R_0^s = \frac{\frac{\beta\Lambda}{\mu_0+a_1\Lambda} - \frac{\sigma^2\Lambda^2}{2(\mu_0+a_1\Lambda)^2}}{\mu_0 + \mu_1 + \gamma} = \frac{\mu_0 + \nu + a_1\Lambda}{\mu_0 + a_1\Lambda} R_0^d - \frac{\sigma^2\Lambda^2}{2(\mu_0 + a_1\Lambda)^2(\mu_0 + \mu_1 + \gamma)}.$$

Theorem 4.3 Let $(S(t), I(t), R(t))$ be the solution of model (2.2) with $(S(0), I(0), R(0)) \in \Delta$. Suppose (i) $\sigma^2 > \frac{\beta^2}{2(\mu_0+\mu_1+\gamma)}$; or (ii) $R_0^s < 1$, $\sigma^2 \leq \frac{2\beta(\mu_0+a_1\Lambda)}{\Lambda}$. Then

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \leq \frac{\beta^2}{2\sigma^2} - (\mu_0 + \mu_1 + \gamma) < 0 \text{ a.s.} \quad \text{if (i) holds,} \quad (4.10)$$

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \leq (R_0^s - 1)(\mu_0 + \mu_1 + \gamma) < 0 \text{ a.s.} \quad \text{if (ii) holds.} \quad (4.11)$$

In other words, $I(t)$ approaches zero at an exponential rate a.s., namely, extinction is a certainty. Proof. See Appendix C.

4.4. Persistence

The conditions that allow the disease to persist are discussed in this section. Now, let's start with the relevant knowledge.

Definition 4.2 If $\liminf_{t \rightarrow \infty} \langle I(t) \rangle > 0$ a.s., then the disease is persistence in the mean.

Lemma 4.3 [28] Let $F \in C([0, \infty) \times \Omega, \mathbb{R})$, $f \in C([0, \infty) \times \Omega, (0, \infty))$ such that $\lim_{t \rightarrow \infty} \frac{F(t)}{t} = 0$ a.s. Assume there are $\lambda_0, \lambda > 0$ so that for all $t \geq 0$,

$$\ln f(t) \geq \lambda t - \lambda_0 \int_0^t f(\tau) d\tau + F(t) \quad \text{a.s.}$$

Thus,

$$\liminf_{t \rightarrow \infty} \langle f(t) \rangle \geq \frac{\lambda}{\lambda_0} \quad \text{a.s.}$$

Theorem 4.4 If $R_0^s > 1$, then $I(t)$ is persistence in the mean, i.e.,

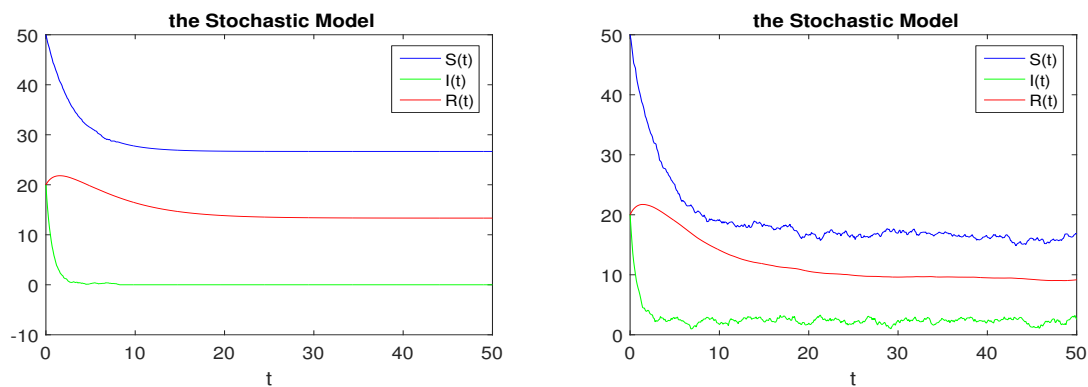
$$\liminf_{t \rightarrow \infty} \langle I(t) \rangle \geq \frac{(\mu_0 + a_1\Lambda)(\mu_0 + \mu_1 + \gamma)(R_0^s - 1)}{\beta \left[\mu_0 + \mu_1 + \Lambda \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) \right]} > 0 \quad \text{a.s.}$$

Proof. See Appendix D.

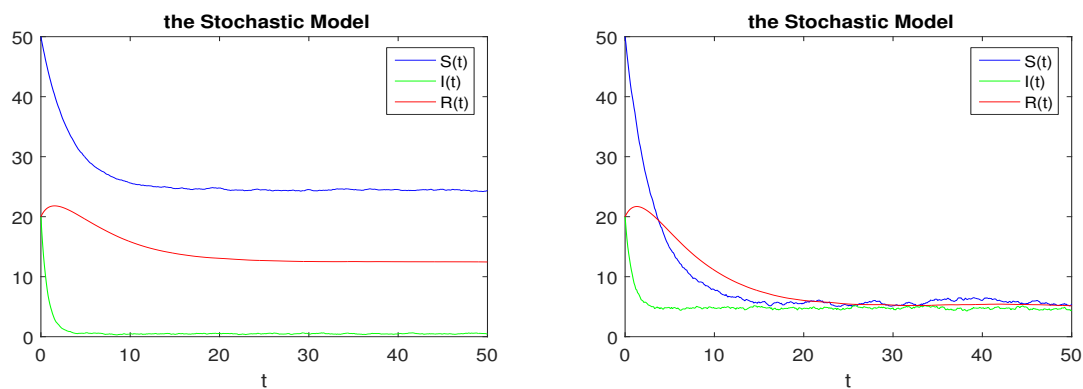
4.5. Numerical simulation

Example 4.1 In model (2.2), let $\beta = 0.01$, $\sigma = 0.4$, $S(0) = 50$, $I(0) = 20$, $R(0) = 20$. For other parameter values, see Example 3.1. Through calculation, we can get $\sigma^2 = 0.16 < \frac{2[(\mu_0 + \mu_1 + \gamma)(\mu_0 + a_1 \Lambda) - \beta \Lambda](\mu_0 + a_1 \Lambda)}{\Lambda^2} = 0.38125$, $R_0^d = 0.56 < 1$. Then the conditions of Corollary 4.2 are verified. Figure 2(a) illustrates that E^0 is globally asymptotically stable, which verifies Corollary 4.2.

Example 4.2 In model (2.2), let $\beta = 0.4$, $\sigma = 0.8$. For other parameter values, see Example 3.1. Through calculation, we can get $\sigma^2 = 0.64 > \frac{\beta^2}{2(\mu_0 + \mu_1 + \gamma)} \doteq 0.6154$. Then the condition (i) of Theorem 4.3 is verified. By Theorem 4.3, one has $\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \leq \frac{\beta^2}{2\sigma^2} - (\mu_0 + \mu_1 + \gamma) = -0.005 < 0$ a.s. As a result, $I(t)$ approaches 0 at an exponential rate with probability 1. In other words, the disease has disappeared. As a confirmation of our findings, we present simulations based on the Euler-Maruyama (EM) method, as shown in Figure 2(b).



(a). Simulations of $(S(t), I(t), R(t))$, when $\beta = 0.01$, $\sigma = 0.4$. (b). Simulations of $(S(t), I(t), R(t))$, when $\beta = 0.4$, $\sigma = 0.8$.



(c). Simulations of $(S(t), I(t), R(t))$, when $\beta = 0.1$, $\sigma = 0.15$. (d). Simulations of $(S(t), I(t), R(t))$, when $\beta = 0.8$, $\sigma = 0.4$.

Figure 2. Simulations of $(S(t), I(t), R(t))$ for HBV stochastic model (2.2).

Example 4.3 In model (2.2), let $\beta = 0.1$, $\sigma = 0.15$. For other parameter values, see Example 3.1. Through calculation, we can get $R_0^s \doteq 0.615 < 1$, $\sigma^2 = 0.0225 \leq \frac{2\beta(\mu_0 + a_1 \Lambda)}{\Lambda} = 0.025$. Then the condition (ii) of Theorem 4.3 is verified. By Theorem 4.3, one has $\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \leq (R_0^s - 1)(\mu_0 + \mu_1 + \gamma) \doteq -0.05005 < 0$ a.s. As a result, $I(t)$ approaches 0 at an exponential rate with probability 1. In other words, the disease has disappeared. The simulation result is shown in Figure 2(c).

Example 4.4 In model (2.2), let $\beta = 0.8$, $\sigma = 0.4$. For other parameter values, see Example 3.1. Through calculation, we can get $R_0^s \doteq 9.8 > 1$. Then Theorem 4.4 is verified. By Theorem 4.4, one has

$$\liminf_{t \rightarrow \infty} \langle I(t) \rangle \geq \frac{(\mu_0 + a_1 \Lambda)(\mu_0 + \mu_1 + \gamma)(R_0^s - 1)}{\beta \left[\mu_0 + \mu_1 + \Lambda \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) \right]} \doteq 0.003697 > 0 \quad \text{a.s.}$$

Thus, the disease persists. The simulation result is shown in Figure 2(d).

5. Optimal control analysis

In order to control the spread of HBV, this section adopts the optimal control theory [29–31]. Our aim is to seek an effective control strategy to reduce HBV infection in the population. In order to reduce Hepatitis B infection rates and to promote vaccination rates, three control variables are used, for instance, $u_1(t)$, $u_2(t)$ and $u_3(t)$. The specific explanation is as below:

1) $u_1(t)$ is the isolation rate. Through this control variable, infected persons are isolated to avoid contact between infected persons and susceptible persons;

2) $u_2(t)$ represents the cure rate. Through this control variable, the number of patients can be decreased by using effective drugs to treat the infected persons;

3) $u_3(t)$ represents vaccination rate. The spread of Hepatitis B can be reduced through vaccination.

5.1. Optimal control of Hepatitis B model (2.1)

To design a control strategy to eliminate Hepatitis B, we will consider the optimal strategy of deterministic model (2.1). The control strategy is achieved by minimizing the target function as below

$$J(u_1, u_2, u_3) = \int_0^T \left[\omega_1 I(t) + \frac{1}{2} (\omega_2 u_1^2(t) + \omega_3 u_2^2(t) + \omega_4 u_3^2(t)) \right] dt, \quad (5.1)$$

subjugate to the control model

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{\beta S I}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \nu + u_3(t)) S, \\ \frac{dI}{dt} &= \frac{\beta S I}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \mu_1 + \gamma + u_2(t) + u_3(t)) I, \\ \frac{dR}{dt} &= (\gamma + u_2(t) + u_3(t)) I + (\nu + u_3(t)) S - \mu_0 R, \end{aligned} \quad (5.2)$$

with

$$S(0) \geq 0, I(0) \geq 0, R(0) \geq 0. \quad (5.3)$$

In Eq (5.1), $\omega_1, \omega_2, \omega_3, \omega_4 > 0$. In the target function, ω_1 represents the weight constant of Hepatitis B infection $I(t)$. ω_2, ω_3 and ω_4 represent the weight constants of quarantine of infected persons and susceptible persons, infected persons' treatment and vaccination, respectively. $\frac{1}{2}\omega_2 u_1^2(t)$, $\frac{1}{2}\omega_3 u_2^2(t)$ and $\frac{1}{2}\omega_4 u_3^2(t)$ represent costs related to segregation, treatment and vaccine inoculation, respectively. The objective of this section is to seek u_1^*, u_2^*, u_3^* so that

$$J(u_1^*, u_2^*, u_3^*) = \min \{ J(u_1, u_2, u_3), u_1, u_2, u_3 \in U \} \quad (5.4)$$

subordinate to problems (5.2) and (5.3), where

$$U := \{ (u_1, u_2, u_3) | 0 \leq u_i \leq 1, u_i(t) \text{ is Lebesgue measurable on } [0, T], i = 1, 2, 3 \}. \quad (5.5)$$

5.1.1. Existence of solution

As a result of this part, it will be demonstrated that the control problems (5.2) and (5.3) have a solution. Let

$$\frac{dX}{dt} = L_1 X + L_2(X), \quad (5.6)$$

where

$$X = \begin{pmatrix} S(t) \\ I(t) \\ R(t) \end{pmatrix},$$

$$L_1 = \begin{pmatrix} -(\mu_0 + \nu + u_3(t)) & 0 & 0 \\ 0 & -(\mu_0 + \mu_1 + \gamma + u_2(t) + u_3(t)) & 0 \\ \nu + u_3(t) & \gamma + u_2(t) + u_3(t) & -\mu_0 \end{pmatrix}, \quad (5.7)$$

$$L_2(X) = \begin{pmatrix} \Lambda - \frac{\beta S I}{f(S,I)} (1 - u_1(t)) \\ \frac{\beta S I}{f(S,I)} (1 - u_1(t)) \\ 0 \end{pmatrix}.$$

It is obvious that Eq (5.6) is a nonlinear system with bounded coefficients. Let

$$F(X) = L_1 X + L_3(X), \quad (5.8)$$

which satisfies

$$\begin{aligned} |L_3(X_1) - L_3(X_2)| &\leq k_1 |S_1(t) - S_2(t)| + k_2 |I_1(t) - I_2(t)| + k_3 |R_1(t) - R_2(t)| \\ &\leq K_1 (|S_1(t) - S_2(t)| + |I_1(t) - I_2(t)| + |R_1(t) - R_2(t)|), \end{aligned} \quad (5.9)$$

where $K_1 = \max \{k_i\}, i = 1, 2, 3$ is not affected by state parameters in system (5.2). And we could write it the same way for this

$$|F(X_1) - F(X_2)| \leq K_2 |X_1 - X_2|, \quad (5.10)$$

where $K_2 = \max \{K_1, \|L_1\|\} < \infty$, which implies that F is continuous and uniformly Lipschitz. It goes without saying that state variables and control variables can't be negative. Thus, the solution of model (5.2) exists. As a next step, we will determine the control variables for minimizing the objective function.

Theorem 5.1 There is an optimal control $u^* = (u_1^*, u_2^*, u_3^*) \in U$, so that

$$J(u_1^*, u_2^*, u_3^*) = \min J(u_1, u_2, u_3) \quad (5.11)$$

subject to systems (5.2) and (5.3).

Proof. Our method of demonstrating optimal control is the one proposed in [31, 32]. Because the state parameters and the control variables are both positive. Therefore, in the minimization problem, the necessary convexity in $u_1(t)$, $u_2(t)$ and $u_3(t)$ of the objective functional defined in Eq (5.1) is satisfied. As defined, $u_1, u_2, u_3 \in U$ are specified as enclosed and convex variables. In order for optimal control to exist, the optimal system (5.2) must have a bound, which ensures its compactness. In addition, the function is composed of control variables and state variables, which shows the convexity of the objective function. Therefore, the problem under consideration satisfies all assumptions, so (u_1^*, u_2^*, u_3^*) exists.

5.1.2. Optimality conditions

Define $u = (u_1, u_2, u_3)$, $x = (S, I, R)$. Then the definition of Lagrangian L is as below:

$$L(x, u) = \omega_1 I(t) + \frac{1}{2} (\omega_2 u_1^2(t) + \omega_3 u_2^2(t) + \omega_4 u_3^2(t)). \quad (5.12)$$

And the related Hamiltonian H is defined as below:

$$H(x, u, \lambda) = \lambda g(x, u) + L(x, u), \quad (5.13)$$

where

$$g(x, u) = (g_1(x, u), g_2(x, u), g_3(x, u)), \quad \lambda = (\lambda_1, \lambda_2, \lambda_3),$$

with

$$\begin{aligned} g_1(x, u) &= \Lambda - \frac{\beta S I}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \nu + u_3(t)) S, \\ g_2(x, u) &= \frac{\beta S I}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \mu_1 + \gamma + u_2(t) + u_3(t)) I, \\ g_3(x, u) &= (\gamma + u_2(t) + u_3(t)) I + (\nu + u_3(t)) S - \mu_0 R. \end{aligned} \quad (5.14)$$

Secondly, the main research tool for optimal solution of control problem is the standard Pontryagin maximum principle. Suppose that (x^*, u^*) is the optimal solution of (5.1)–(5.3), then there is one nontrivial vector function λ , so that

$$\begin{cases} \frac{dx^*(t)}{dt} = \frac{\partial H}{\partial \lambda}(x^*, u^*, \lambda), \\ 0 = \frac{\partial H}{\partial u}(x^*, u^*, \lambda), \\ \frac{d\lambda(t)}{dt} = -\frac{\partial H}{\partial x}(x^*, u^*, \lambda), \end{cases} \quad (5.15)$$

with

$$H(x^*, u^*, \lambda) = \max_{u \in [0,1]} H(x^*(t), u^*(t), \lambda(t)), \quad (5.16)$$

and the transversal condition

$$\lambda(T) = 0. \quad (5.17)$$

Theorem 5.2 For the optimal control problems (5.1) and (5.2), S^* , I^* and R^* are the optimal state solutions of the optimal control variables (u_1^*, u_2^*, u_3^*) . Then there are adjoint variables $\lambda_1(t)$, $\lambda_2(t)$ and $\lambda_3(t)$, so that

$$\begin{aligned} \lambda'_1(t) &= \frac{(\lambda_1(t) - \lambda_2(t))\beta I^* (1 + a_2 I^*) (1 - u_1^*(t))}{f^2(S^*, I^*)} \\ &\quad + (\lambda_1(t) - \lambda_3(t)) u_3^*(t) + \lambda_1(t) (\mu_0 + \nu) - \lambda_3(t) \nu, \\ \lambda'_2(t) &= -\omega_1 + \frac{(\lambda_1(t) - \lambda_2(t))\beta S^* (1 + a_1 S^*) (1 - u_1^*(t))}{f^2(S^*, I^*)} \\ &\quad + (\lambda_2(t) - \lambda_3(t)) (u_2^*(t) + u_3^*(t)) \\ &\quad + \lambda_2(t) (\mu_0 + \mu_1 + \gamma) - \lambda_3(t) \gamma, \\ \lambda'_3(t) &= \lambda_3(t) \mu_0 \end{aligned} \quad (5.18)$$

with transversality conditions

$$\lambda_i(T) = 0, \quad i = 1, 2, 3. \quad (5.19)$$

And the specific forms of $u_1^*(t)$, $u_2^*(t)$ and $u_3^*(t)$ are as below

$$u_1^*(t) = \max \left\{ \min \left\{ \frac{(\lambda_2(t) - \lambda_1(t))\beta S^* I^*}{f(S^*, I^*) \omega_2}, 1 \right\}, 0 \right\}, \quad (5.20)$$

$$u_2^*(t) = \max \left\{ \min \left\{ \frac{(\lambda_2(t) - \lambda_3(t)) I^*}{\omega_3}, 1 \right\}, 0 \right\}, \quad (5.21)$$

$$u_3^*(t) = \max \left\{ \min \left\{ \frac{(\lambda_1(t) - \lambda_3(t)) S^* + (\lambda_2(t) - \lambda_3(t)) I^*}{\omega_4}, 1 \right\}, 0 \right\}. \quad (5.22)$$

Proof. Taking the partial derivatives of $S(t)$, $I(t)$, $R(t)$ in (5.13) yield the adjoint variables (5.18). In addition, to calculate u_1^* , u_2^* , u_3^* , we take partial derivatives of u_1 , u_2 , u_3 in (5.13). Then we obtain the optimal control variables (5.20)–(5.22).

By giving the equation of the adjoint variables (5.18) and its related conditions (5.3) and (5.19) and the optimal control parameters (5.20)–(5.22), control variables and state variables of the control problem are solved. The next step is to investigate the control theory of stochastic systems when the same control measures are applied.

5.2. Optimal control for HBV stochastic model (2.2)

As mentioned in Section 5.1, in this section, we study the stochastic optimal control of model (2.2), and the following stochastic control system can be obtained

$$\begin{aligned} dS &= \left(\Lambda - \frac{\beta SI}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \nu + u_3(t)) S \right) dt - \frac{\sigma SI}{f(S, I)} dB(t), \\ dI &= \left(\frac{\beta SI}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \mu_1 + \gamma + u_2(t) + u_3(t)) I \right) dt + \frac{\sigma SI}{f(S, I)} dB(t), \\ dR &= [(\gamma + u_2(t) + u_3(t)) I + (\nu + u_3(t)) S - \mu_0 R] dt \end{aligned} \quad (5.23)$$

with

$$S(0) \geq 0, I(0) \geq 0, R(0) \geq 0. \quad (5.24)$$

For easy reading, the vectors are given as below:

$$\begin{aligned} x(t) &= (S(t), I(t), R(t))', \quad u(t) = (u_1(t), u_2(t), u_3(t))', \quad g(x(t)) = (g_1(t), g_2(t), g_3(t))' \\ f(x(t), u(t)) &= (f_1(x, u), f_2(x, u), f_3(x, u))', \end{aligned} \quad (5.25)$$

and

$$dx(t) = f(x(t), u(t)) dt + g(x(t)) dB(t) \quad (5.26)$$

with $x(0) = (S(0), I(0), R(0))' = x_0$, where

$$\begin{aligned} f_1(x(t), u(t)) &= \Lambda - \frac{\beta SI}{f(S, I)}(1 - u_1(t)) - (\mu_0 + \nu + u_3(t))S, \\ f_2(x(t), u(t)) &= \frac{\beta SI}{f(S, I)}(1 - u_1(t)) - (\mu_0 + \mu_1 + \gamma + u_2(t) + u_3(t))I, \\ f_3(x(t), u(t)) &= (\gamma + u_2(t) + u_3(t))I + (\nu + u_3(t))S - \mu_0 R, \\ g_1(t) &= -\frac{\sigma SI}{f(S, I)}, g_2(t) = \frac{\sigma SI}{f(S, I)}, g_3(t) = 0. \end{aligned} \quad (5.27)$$

The quadratic function is considered as below:

$$J(x, u) = \frac{1}{2}E \left\{ \int_0^{t_f} \left[C_1 I + \frac{1}{2} (C_2 u_1^2 + C_3 u_2^2 + C_4 u_3^2) \right] dt + \frac{C_5}{2} S^2 + \frac{C_6}{2} I^2 + \frac{C_7}{2} R^2 \right\}, \quad (5.28)$$

where $C_i > 0$, $i = \overline{1, 7}$. The objective of this section is to obtain the control vector $u^*(t) = (u_1^*(t), u_2^*(t), u_3^*(t))$ so that

$$J(u) \geq J(u^*), \quad \forall u \in U, \quad (5.29)$$

where the specific form of U is as below:

$$U = \{u_i(t) : u_i(t) \in [0, u_i^{\max}], \forall u_i \in L^2[0, t_f], t \in (0, t_f], i = 1, 2, 3\}, \quad (5.30)$$

where $u_i^{\max} > 0$. The next step is to apply the stochastic maximum principle to define the Hamiltonian $H(x, u, p, q)$, which has the following form:

$$H(x, u, p, q) = -l(x, u) + \langle g(x), q \rangle + \langle f(x, u), p \rangle, \quad (5.31)$$

where $q = (q_1, q_2, q_3)'$, $p = (p_1, p_2, p_3)'$ are two different adjoint vectors, and $\langle \cdot, \cdot \rangle$ denotes Euclidean inner product space. As a result of applying the maximum principle, one has

$$dx^*(t) = \frac{\partial H(x^*, u^*, p, q)}{\partial p} dt + g(x^*(t)) dB(t), \quad (5.32)$$

$$dp^*(t) = -\frac{\partial H(x^*, u^*, p, q)}{\partial x} dt + q(t) dB(t), \quad (5.33)$$

$$H_m(x^*, u^*, p, q) = \min_{u \in U} H_m(x^*, u^*, p, q). \quad (5.34)$$

In this case, $x^*(t)$ is one optimal path of $x(t)$. It is observed that, at both initial and terminal conditions of Eqs (5.32) and (5.33) are

$$x^*(0) = x_0, \quad (5.35)$$

$$p(t_f) = -\frac{\partial h(x^*(t_f))}{\partial x}. \quad (5.36)$$

As shown in Eq (5.34), the optimal control $x^*(t)$ is an operator of $q(t)$, $p(t)$ and $x^*(t)$. Thus, it means that

$$u^*(t) = \Phi(x^*, p, q), \quad (5.37)$$

In this case, Φ can be computed by (5.34). Therefore, Equations (5.32) and (5.33) are written as the following equations

$$dx^*(t) = \frac{\partial H(x^*, u^*, p, q)}{\partial p} dt + g(x^*(t)) dB(t), \quad (5.38)$$

$$dp(t) = -\frac{\partial H(x^*, u^*, p, q)}{\partial x} dt + q(t) dB(t). \quad (5.39)$$

So,

$$\begin{aligned} H = & C_1 I + \frac{1}{2} (C_2 u_1^2 + C_3 u_2^2 + C_4 u_3^2) + \frac{C_5}{2} S^2 + \frac{C_6}{2} I^2 + \frac{C_7}{2} R^2 \\ & + p_1 \left[\Lambda - \frac{\beta S I}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \nu + u_3(t)) S \right] \\ & + p_2 \left[\frac{\beta S I}{f(S, I)} (1 - u_1(t)) - (\mu_0 + \mu_1 + \gamma + u_2(t) + u_3(t)) I \right] \\ & + p_3 [(\gamma + u_2(t) + u_3(t)) I + (\nu + u_3(t)) S - \mu_0 R] - \frac{\sigma S I}{f(S, I)} q_1 + \frac{\sigma S I}{f(S, I)} q_2. \end{aligned} \quad (5.40)$$

According to random maximum principle,

$$dp^*(t) = -\frac{\partial H(x^*, u^*, p, q)}{\partial x} dt + q(t) dB(t). \quad (5.41)$$

We have

$$\begin{aligned} p'_1(t) = & \frac{(p_1(t) - p_2(t)) \beta I^* (1 + a_2 I^*) (1 - u_1^*(t))}{f^2(S^*, I^*)} + (p_1(t) - p_3(t)) u_3^*(t) \\ & + p_1(t) (\mu_0 + \nu) - p_3(t) \nu + \frac{\sigma I^* (1 + a_2 I^*)}{f^2(S^*, I^*)} (q_1 - q_2), \\ p'_2(t) = & -C_1 + \frac{(p_1(t) - p_2(t)) \beta S^* (1 + a_1 S^*) (1 - u_1^*(t))}{f^2(S^*, I^*)} \\ & + (p_2(t) - p_3(t)) (u_2^*(t) + u_3^*(t)) + p_2(t) (\mu_0 + \mu_1 + \gamma) \\ & - p_3(t) \gamma + \frac{\sigma S^* (1 + a_1 S^*)}{f^2(S^*, I^*)} (q_1 - q_2), \\ p'_3(t) = & p_3(t) \mu_0. \end{aligned} \quad (5.42)$$

An auxiliary initial conditions and end conditions are granted as below

$$S^*(0) = \tilde{S}, I^*(0) = \tilde{I}, R^*(0) = \tilde{R}, p(t_f) = -\frac{\partial h(x^*(t_f))}{\partial x}, \quad (5.43)$$

$$h(S, I, R) = \frac{k_1}{2} S^2 + \frac{k_2}{2} I^2 + \frac{k_3}{2} R^2. \quad (5.44)$$

In this case, $p_1(t_f) = -k_1 S$, $p_2(t_f) = -k_2 I$, $p_3(t_f) = -k_3 R$. In the Hamiltonian equation, by taking the derivatives of u_1, u_2, u_3 , we can figure out that

$$u_1^*(t) = \max \left\{ \min \left\{ \frac{(p_2(t) - p_1(t)) \beta S^* I^*}{f(S^*, I^*) C_2}, 1 \right\}, 0 \right\}, \quad (5.45)$$

$$u_2^*(t) = \max \left\{ \min \left\{ \frac{(p_2(t) - p_3(t)) I^*}{C_3}, 1 \right\}, 0 \right\}, \quad (5.46)$$

$$u_3^*(t) = \max \left\{ \min \left\{ \frac{(p_1(t) - p_3(t)) S^* + (p_2(t) - p_3(t)) I^*}{C_4}, 1 \right\}, 0 \right\}. \quad (5.47)$$

In control theory, the desired objective is achieved by adjusting control variables. By substituting the control parameters into the system, the optimal goal of the power system can be obtained. The limits of control can be set according to Eq (5.27). We will establish the objective function by referring to the method of Eq (5.28). As a result of objective functions, there is a direct relationship between optimality and optimality. So special attention should be paid when selecting the objective function. Whenever the control objective function has multiple factors, the more important items should be given weight. Before applying the Pontryagin Maximum Rules [33], the existence and compactness of an optimal control are tested. The optimal control makes the goal function reach a maximum or minimum value at a certain point. Differential equations can be optimized to Hamiltonian at a certain point. Hamiltonian is defined as below:

Hamiltonian = (integrand of the goal functional) + (adjoint)(RHS of Differential system).

Optimal control involves finding the necessary point u^* to maximize the Hamiltonian equation. We are able to obtain the adjoint system (5.43) by taking the derivative of H w.r.t. with respect to the state variable and substituting it with the final condition.

6. Numerical simulations

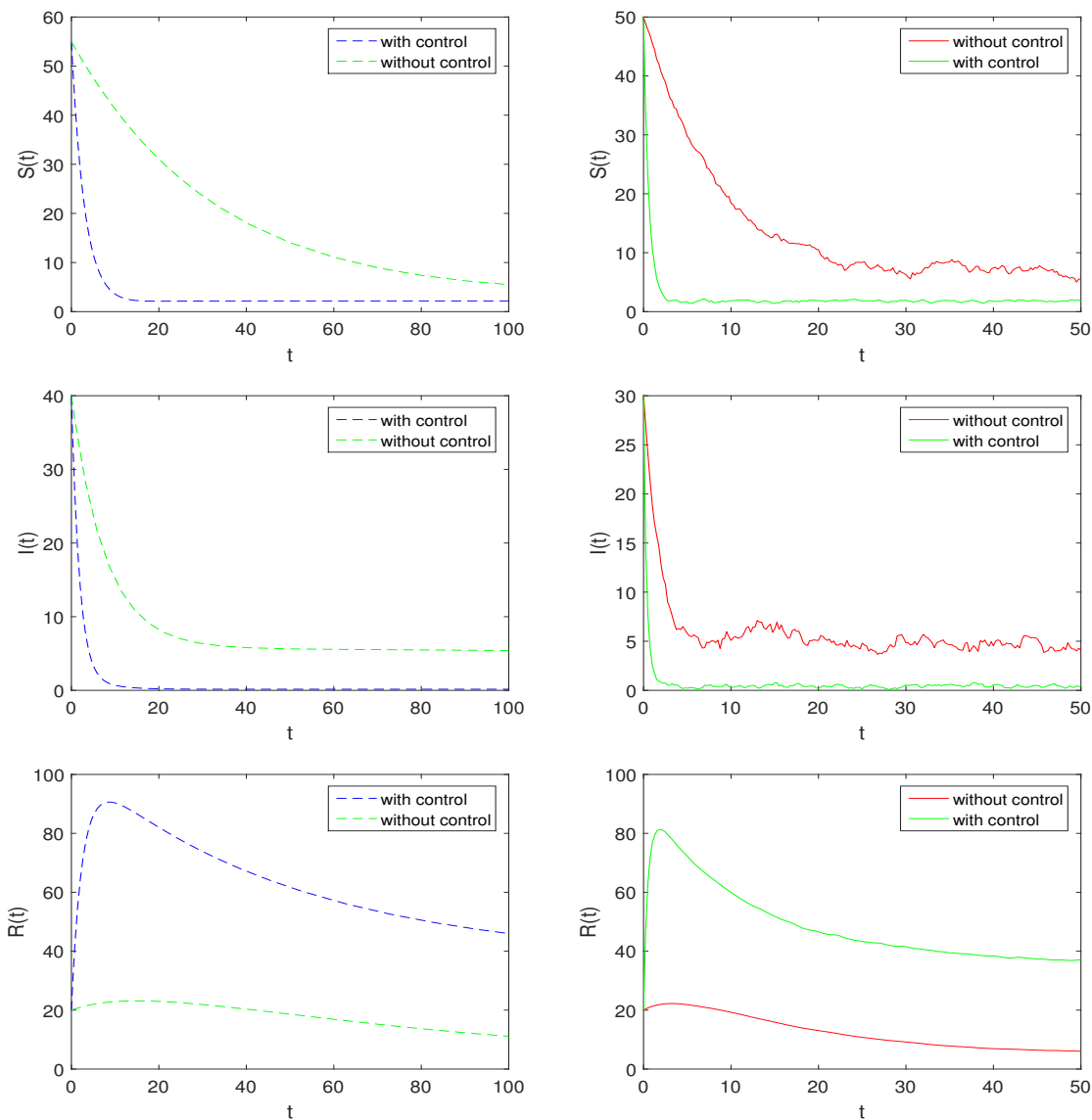
Our analysis results are supported by approximate simulations of Hepatitis B models (2.1) and (2.2), respectively. Simulations can be performed from qualitative aspects. To test the rationality of the results, we use the stochastic Runge-Kutta method is adopted to simulate model (2.2), and the calculation model is obtained as below:

$$\begin{aligned} S_{k+1} &= S_k + \left(\Lambda - \frac{\beta S_k I_k}{f(S_k, I_k)} - (\mu_0 + \nu) S_k \right) \Delta t - \frac{\sigma S_k I_k}{f(S_k, I_k)} \sqrt{\Delta t} \xi_k + \frac{\sigma^2 S_k I_k}{2f(S_k, I_k)} (\xi_k^2 - 1) \Delta t, \\ I_{k+1} &= I_k + \left(\frac{\beta S_k I_k}{f(S_k, I_k)} - (\mu_0 + \mu_1 + \gamma) I_k \right) \Delta t + \frac{\sigma S_k I_k}{f(S_k, I_k)} \sqrt{\Delta t} \xi_k + \frac{\sigma^2 S_k I_k}{2f(S_k, I_k)} (\xi_k^2 - 1) \Delta t, \\ R_{k+1} &= R_k + (\gamma I_k + \nu S_k - \mu_0 R_k) \Delta t, \end{aligned} \quad (6.1)$$

where $\sigma > 0$ is the white noise value, $\xi_k (k = \overline{1, n})$ is a standalone Gaussian stochastic variable with $N(0, 1)$ and the step length $\Delta t > 0$.

Next, the qualitative characteristics of deterministic and stochastic optimal controls are modeled. Firstly, Runge-Kutta iterative technique is used to simulate the deterministic model. The time interval is set as $[0, 100]$ in the positive direction, and the state system (5.2) and transverse condition (5.19) are solved by the prescribed method. Then the adjoint equation (5.18) with the same time interval is simulated by Runge-Kutta iterative method in the backward direction supported by transverse condition (5.19). Note that the values of other parameters except β are shown in Example 3.1. The results are

shown in Figure 3(a). Figure 3(a) shows the dynamic curves of susceptible population, infected person and recovered person in HBV deterministic model (2.1) with and without optimal control value. A clear difference can be observed between the two conditions with and without control. The simulation results show that with the implementation of control measures, the number of the infected and susceptible population tend to decrease, while the number of recovered patients tend to increase.



(a). Control system (5.2) with $\beta = 0.5$.

(b). Control system (5.23) with $\beta = 0.8$, $\sigma = 0.9$.

Figure 3. Simulations of $(S(t), I(t), R(t))$ with and without controls for the deterministic and stochastic HBV models.

Next, we simulate optimal control techniques for stochastic models. The stochastic Runge-Kutta iterative technique is applied to simulate the optimal control system. Considering the transverse condition, the optimal control strategy is realized by approximating the state and adjoint model. First, we apply the stochastic Runge-Kutta iterative method to calculate the state system (5.42). Secondly, under the transverse condition (5.43), the corresponding adjoint equation (5.42) of the system is ob-

tained by using the reverse technique and the iterative technique of the state equation. The control is then modified by applying the convex combination of the control and the values from the characterizations (5.45)–(5.47). The algorithm is repeated over and over again, and the iteration is performed until the difference between the values obtained in two successive iterations is very small. Note that the values of other parameters except β , σ are shown in Example 3.1. The results are shown in Figure 3(b). Figure 3(b) respectively shows the dynamic curves of susceptible population, infected persons and recovered persons in the stochastic model (2.2) with and without optimal control values. Under the optimal control parameter values, the dynamic behavior before and after the optimal control is significantly different. The images show that through isolation, treatment and vaccination, the number of susceptible and infected persons is decreasing, but the number of recovered people is increasing.

7. Conclusions

This paper investigates the transmission dynamics of HBV. The optimal control strategy is developed to control the transmission of HBV in the population. To this end, we first establish new HBV models with general incidence rate. We calculate the basic reproduction number, equilibrium points of deterministic Hepatitis B model to study the local asymptotic stability under certain conditions. Secondly, we calculate the random threshold. The random Lyapunov function theory is applied to verify that the model has one unique global positive solution. The extinction, persistence and stability of stochastic Hepatitis B model are given. These conditions are expressed as expressions containing the stochastic system parameters and the intensity of the noise term. It is clear that noise intensity has an important effect on disease transmission. To control the transmission of HBV, optimal control strategies are used to eliminate the transmission of HBV. In order to reduce Hepatitis B infection rates and to promote vaccination rates, three control variables are used, for instance, isolation of patients, treatment of patients, and vaccine inoculation. Runge-Kutta method is used for numerical simulations to support the theoretical results. It can be found that when the white noise is stronger, the extinction rate of the disease is higher. Disease is more persistent when white noise is lower in intensity. Virus dynamics-based stochastic epidemic models perform better in our study. A broad range of biomedical applications can be made from this theory, as it provides a solid foundation for studying similar diseases. An infection dynamics model based on stochastic delayed infection can, for example, be considered for studying the effects of incubation periods. Additionally, our research can be applied to analyze other epidemics, such as COVID-19, tuberculosis, HIV and so on.

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Conflict of interest

The authors declare there is no conflict of interest.

References

1. T. Khan, G. Zaman, M. I. Chohan, The transmission dynamic and optimal control of acute and chronic Hepatitis B, *J. Biol. Dyn.*, **11** (2017), 172–189. <https://doi.org/10.1080/17513758.2016.1256441>
2. A. Din, Y. J. Li, Q. Liu, Viral dynamics and control of Hepatitis B virus (HBV) using an epidemic model, *Alex. Eng. J.*, **59** (2020), 667–679. <https://doi.org/10.1016/j.aej.2020.01.034>
3. T. Khan, Z. Ullah, Z. Ali, G. Zaman, Modeling and control of the Hepatitis B virus spreading using an epidemic model, *Chaos, Solitons Fractals*, **124** (2019), 1–9. <https://doi.org/10.1016/j.chaos.2019.04.033>
4. P. T. Karaji, N. Nyamoradi, Analysis of a fractional SIR model with general incidence function, *Appl. Math. Lett.*, **108** (2020), 106499. <https://doi.org/10.1016/j.aml.2020.106499>
5. S. M. Simelane, P. G. Dlamini, A fractional order differential equation model for Hepatitis B virus with saturated incidence, *Results Phys.*, **24** (2021), 104114. <https://doi.org/10.1016/j.rinp.2021.104114>
6. F. Huang, J. L. Li, Exponential ultimate boundedness and stability of stochastic differential equations with impulsive, *Asian J. Control*, **25** (2023), 88–100. <https://doi.org/10.1002/asjc.2786>
7. F. Huang, J. L. Li, Exponential ultimate boundedness and stability of impulsive stochastic functional differential equations, *Int. J. Control*, **96** (2023), 568–576. <https://doi.org/10.1080/00207179.2021.2005259>
8. T. Khan, A. Khan, G. Zaman, The extinction and persistence of the stochastic Hepatitis B epidemic model, *Chaos, Solitons Fractals*, **108** (2018), 123–128. <https://doi.org/10.1016/j.chaos.2018.01.036>
9. A. Din, Y. J. Li, T. Khan, K. Anwar, G. Zaman, Stochastic dynamics of Hepatitis B epidemics, *Results Phys.*, **20** (2021), 103730. <https://doi.org/10.1016/j.rinp.2020.103730>
10. P. J. Liu, A. Din, L. F. Huang, A. Yusuf, Stochastic optimal control analysis for the Hepatitis B epidemic model, *Results Phys.*, **26** (2021), 104372. <https://doi.org/10.1016/j.rinp.2021.104372>
11. A. Din, Y. J. Li, Stationary distribution extinction and optimal control for the stochastic Hepatitis B epidemic model with partial immunity, *Phys. Scr.*, **96** (2021), 74005. <https://doi.org/10.1088/1402-4896/abfacc>
12. B. Wu, J. W. Jia, Asymptotic behavior of a stochastic delayed model for chronic Hepatitis B infection, *Complexity*, **2020** (2020), 1875475. <https://doi.org/10.1155/2020/1875475>
13. A. Din, Y. J. Li, A. Yusuf, Delayed Hepatitis B epidemic model with stochastic analysis, *Chaos, Solitons Fractals*, **146** (2021), 110839. <https://doi.org/10.1016/j.chaos.2021.110839>
14. A. Din, Y. J. Li, Stochastic optimal analysis for the Hepatitis B epidemic model with Markovian switching, *Math. Meth. Appl. Sci.*, **2022** (2022), 1–26. <https://doi.org/10.1002/mma.8218>
15. A. Gray, D. Greenhalgh, L. Hu, X. Mao, J. Pan, A stochastic differential equation SIS epidemic model, *SIAM J. Appl. Math.*, **71** (2011), 876–902. <https://doi.org/10.1137/10081856X>
16. I. A. Baba, E. Hincal, Global stability analysis of two-strain epidemic model with bilinear and non-monotone incidence rates, *Eur. Phys. J. Plus*, **132** (2017), 208. <https://doi.org/10.1140/epjp/i2017-11476-x>

17. J. J. Wang, J. Z. Zhang, Z. Jin, Analysis of an SIR model with bilinear incidence rate, *Nonlinear Anal. Real World Appl.*, **11** (2010), 2390–2402. <https://doi.org/10.1016/j.nonrwa.2009.07.012>
18. T. T. Xue, X. L. Fan, J. Zhu, A class of deterministic and stochastic fractional epidemic models with vaccination, *Comput. Math. Methods Med.*, **2022** (2022), 1–22. <https://doi.org/10.1155/2022/1797258>
19. Q. Liu, D. Q. Jiang, N. Z. Shi, Threshold behavior in a stochastic SIQR epidemic model with standard incidence and regime switching, *Appl. Math. Comput.*, **316** (2018), 310–325. <https://doi.org/10.1016/j.amc.2017.08.042>
20. T. T. Xue, X. L. Fan, Z. G. Chang, Dynamics of a stochastic SIRS epidemic model with standard incidence and vaccination, *Math. Biosci. Eng.*, **19** (2022), 10618–10636. <https://doi.org/10.3934/mbe.2022496>
21. Q. S. Yang, D. Q. Jiang, N. Z. Shi, C. Y. Ji, The ergodicity and extinction of stochastically perturbed SIR and SEIR epidemic models with saturated incidence, *J. Math. Anal. Appl.*, **388** (2012), 248–271. <https://doi.org/10.1016/j.jmaa.2011.11.072>
22. Y. N. Zhao, D. Q. Jiang, The threshold of a stochastic SIRS epidemic model with saturated incidence, *Appl. Math. Lett.*, **34** (2014), 90–93. <https://doi.org/10.1016/j.aml.2013.11.002>
23. J. R. Beddington, Mutual interference between parasites or predators and its effect on searching efficiency, *J. Anim. Ecol.*, **44** (1975), 331–340.
24. P. Crowley, E. Martin, Functional responses and interference within and between year classes of a dragonfly population. *J. N. Am. Benthol. Soc.*, **8** (1989), 211–221.
25. X. Mao, *Stochastic Differential Equations and Applications*, Horwood, Chichester, 2007.
26. V. N. Afanas'ev, V. B. Kolmanovskii, V. R. Nosov, *Mathematical Theory of Control Systems Design*, Springer, Dordrecht, The Netherlands, 1996.
27. D. Kiouach, Y. Sabbar, Stability and threshold of a stochastic SIRS epidemic model with vertical transmission and transfer from infectious to susceptible individuals, *Discrete Dyn. Nat. Soc.*, **2018** (2018), 7570296. <https://doi.org/10.1155/2018/7570296>
28. C. Y. Ji, D. Q. Jiang, Threshold behavior of a stochastic SIR model, *Appl. Math. Model.*, **38** (2014), 5067–5079. <https://doi.org/10.1016/j.apm.2014.03.037>
29. G. Zaman, Y. H. Kang, I. H. Jung, Optimal treatment of an SIR epidemic model with time delay, *Biosystems*, **98** (2009), 43–50. <https://doi.org/10.1016/j.biosystems.2009.05.006>
30. M. T. Xia, L. Bottcher, T. Chou, Controlling epidemics through optimal allocation of test kits and vaccine doses across networks, *IEEE Trans. Network Sci. Eng.*, **9** (2022), 1422–1436. <https://doi.org/10.1109/TNSE.2022.3144624>
31. G. Zaman, Y. H. Kang, I. H. Jung, Stability analysis and optimal vaccination of an SIR epidemic model, *Biosystems*, **93** (2008), 240–249. <https://doi.org/10.1016/j.biosystems.2008.05.004>
32. A. V. Kamyad, R. Akbari, A. A. Heydari, A. Heydari, Mathematical modeling of transmission dynamics and optimal control of vaccination and treatment for Hepatitis B virus, *Comput. Math. Methods Med.*, **2014** (2014), 475451. <https://doi.org/10.1155/2014/475451>
33. L. S. Pontryagin, V. G. Boltyanskii, R. V. Gamkrelize, E. F. Mishchenko, *The Mathematical Theory of Optimal Processes*, Wiley, New York, 1962.

Appendix A Proof of Theorem 4.1.

Let $(S(0), I(0), R(0)) \in \Delta$, $N(t) = S(t) + I(t) + R(t)$. By model (2.2), one has

$$dN(t) = [\Lambda - \mu_0 N(t) - \mu_1 I(t)] dt. \quad (7.1)$$

Then,

$$dN(\tau) < [\Lambda - \mu_0 N(\tau)] d\tau, \quad \forall 0 \leq \tau \leq t \text{ a.s.} \quad (7.2)$$

By integration, we get

$$N(\tau) < \frac{\Lambda}{\mu_0} + \left(N(0) - \frac{\Lambda}{\mu_0} \right) e^{-\mu_0 \tau}, \quad \forall 0 \leq \tau \leq t \text{ a.s.} \quad (7.3)$$

So $N(\tau) < \frac{\Lambda}{\mu_0}$,

$$S(\tau), I(\tau), R(\tau) \in \left(0, \frac{\Lambda}{\mu_0} \right), \quad \forall 0 \leq \tau \leq t \text{ a.s.} \quad (7.4)$$

There is no doubt that model (2.2) meets the local Lipschitz condition. For $(S(0), I(0), R(0)) \in \mathbb{R}_+^3$, in this model (2.2), there is a unique local solution $(S(t), I(t), R(t))$, $t \in [0, \tau_e)$. In this case, τ_e refers to the duration of the explosion. In order to prove that $\tau_e = \infty$ a.s., we must do the following. Make $k_0 > 0$ large enough so that $(S(0), I(0), R(0)) > k_0$. For each integer $k \leq k_0$, the stopping time is defined as below

$$\begin{aligned} \tau_k &= \inf \{ t \in [0, \tau_e) : S(t) \leq k \text{ or } I(t) \leq k \text{ or } R(t) \leq k \}, \\ \tau_0 &= \lim_{k \rightarrow 0} \tau_k = \inf \{ t \in [0, \tau_e) : S(t) \leq 0 \text{ or } I(t) \leq 0 \text{ or } R(t) \leq 0 \}. \end{aligned}$$

A C^2 -function $V : \mathbb{R}_+^3 \rightarrow \mathbb{R}_+$ is defined as below

$$V(S, I, R) = -\ln \left(\frac{S}{\frac{\Lambda}{\mu_0}} \right) - \ln \left(\frac{I}{\frac{\Lambda}{\mu_0}} \right) - \ln \left(\frac{R}{\frac{\Lambda}{\mu_0}} \right) = -\ln S I R + 3 \ln \frac{\Lambda}{\mu_0}.$$

By applying the formula Itô, we can get

$$\begin{aligned} dV(S, I, R) &= \left[-\frac{\Lambda}{S(\tau)} + \frac{\beta I(\tau)}{f(S(\tau), I(\tau))} + (\mu_0 + \nu) + \frac{\sigma^2 I^2(\tau)}{2f^2(S(\tau), I(\tau))} \right] d\tau \\ &\quad + \left[-\frac{\beta S(\tau)}{f(S(\tau), I(\tau))} + (\mu_0 + \mu_1 + \gamma) + \frac{\sigma^2 S^2(\tau)}{2f^2(S(\tau), I(\tau))} \right] d\tau \\ &\quad + \left[-\gamma \frac{I(\tau)}{R(\tau)} - \nu \frac{S(\tau)}{R(\tau)} + \mu_0 \right] d\tau + \frac{\sigma(I(\tau) - S(\tau))}{f(S(\tau), I(\tau))} dB(\tau) \\ &\leq \left[3\mu_0 + \mu_1 + \gamma + \nu + \frac{\beta I(\tau)}{f(S(\tau), I(\tau))} + \frac{\sigma^2 (S^2(\tau) + I^2(\tau))}{2f^2(S(\tau), I(\tau))} \right] d\tau \\ &\quad + \frac{\sigma(I(\tau) - S(\tau))}{f(S(\tau), I(\tau))} dB(\tau), \quad \tau \in [0, t \wedge \tau_k]. \end{aligned} \quad (7.5)$$

For $\tau \in [0, t \wedge \tau_k]$, one has

$$\begin{aligned} \frac{S(\tau)}{f(S(\tau), I(\tau))} &\leq \frac{S(\tau)}{1 + a_1 S(\tau)} \leq \frac{\frac{\Lambda}{\mu_0}}{1 + a_1 \frac{\Lambda}{\mu_0}} \leq \frac{\Lambda}{\mu_0 + a_1 \Lambda}, \\ \frac{I(\tau)}{f(S(\tau), I(\tau))} &\leq \frac{I(\tau)}{1 + a_1 S(\tau)} \leq \frac{\frac{\Lambda}{\mu_0}}{1 + a_1 \frac{\Lambda}{\mu_0}} \leq \frac{\Lambda}{\mu_0 + a_1 \Lambda}. \end{aligned} \quad (7.6)$$

Thus,

$$dV(S, I, R) \leq Kd\tau + \frac{\sigma(I(\tau) - S(\tau))}{f(S(\tau), I(\tau))} dB(\tau) \text{ a.s.}, \quad (7.7)$$

where $K = 3\mu_0 + \mu_1 + \gamma + \nu + \frac{\beta\Lambda}{\mu_0 + a_1\Lambda} + \frac{\sigma^2\Lambda^2}{(\mu_0 + a_1\Lambda)^2}$. Integrate the above inequality from 0 to $\tau_k \wedge t$, and then taking the expectation, according to the properties of Brownian motion, we get

$$\begin{aligned} \mathbb{E}V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t)) &\leq V(S(0), I(0), R(0)) + \mathbb{E} \int_0^{\tau_k \wedge t} Kdt \\ &\leq V(S(0), I(0), R(0)) + Kt < \infty. \end{aligned}$$

Because $V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t)) > 0$, so

$$\begin{aligned} \mathbb{E}V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t)) &= \mathbb{E}[1_{\{\tau_k \leq t\}} V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t))] \\ &+ \mathbb{E}[1_{\{\tau_k > t\}} V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t))] \geq \mathbb{E}[1_{\{\tau_k \leq t\}} V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t))]. \end{aligned}$$

For τ_k , some component of $S(\tau_k), I(\tau_k), R(\tau_k)$ is equal to k . Thus, $V(S(\tau_k), I(\tau_k), R(\tau_k)) \geq -\ln\left(\frac{k\mu_0}{\Lambda}\right)$. So,

$$\begin{aligned} \mathbb{E}V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t)) &\geq \mathbb{E}[1_{\{\tau_k \leq t\}} V(S(\tau_k \wedge t), I(\tau_k \wedge t), R(\tau_k \wedge t))] \\ &\geq -\ln\left(\frac{k\mu_0}{\Lambda}\right) \mathbb{P}(\tau_k \leq t). \end{aligned} \quad (7.8)$$

By (7.8), we obtain

$$\mathbb{P}(\tau_k \leq t) \leq -\frac{V(S(0), I(0), R(0)) + Kt}{\ln\left(\frac{k\mu_0}{\Lambda}\right)}. \quad (7.9)$$

Extending k to 0, one has $\mathbb{P}(\tau_0 \leq t) = 0, t > 0$. Therefore, $\mathbb{P}(\tau_0 = \infty) = 1$. Consequently, $\tau_0 = \tau_e = \infty$ a.s.

Appendix B Proof of Theorem 4.2.

Let $p \geq 2$. The Lyapunov function is considered as below

$$V = \tau_1 \left(\frac{\Lambda}{\mu_0 + \nu} - S \right)^p + \frac{I^p}{p}, \quad (7.10)$$

Here $\tau_1 > 0$ will be determined later. The first inequality in (4.6) is easily proved to be true. Then,

$$\begin{aligned} LV &= -p\tau_1\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^p (\mu_0 + \nu) + \left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^{p-1} \frac{p\tau_1\beta SI}{f(S, I)} \\ &\quad + \left[\frac{\beta S}{f(S, I)} - (\mu_0 + \mu_1 + \gamma)\right] I^p + \frac{\tau_1 p(p-1)\sigma^2 S^2 I^2}{2f^2(S, I)} \\ &\quad \times \left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^{p-2} + \frac{(p-1)\sigma^2 S^2 I^p}{2f^2(S, I)}. \end{aligned} \quad (7.11)$$

In Δ , we get

$$\begin{aligned} LV &\leq -p\tau_1\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^p (\mu_0 + \nu) + I\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^{p-1} \cdot \frac{p\tau_1\beta\Lambda}{\mu_0 + a_1\Lambda} \\ &\quad - \left[(\mu_0 + \mu_1 + \gamma) - \frac{\beta\Lambda}{\mu_0 + a_1\Lambda} - \frac{(p-1)\sigma^2\Lambda^2}{2(\mu_0 + a_1\Lambda)^2}\right] I^p \\ &\quad + \frac{\tau_1 p(p-1)\sigma^2\Lambda^2}{2(\mu_0 + a_1\Lambda)^2} \cdot I^2\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^{p-2} \end{aligned} \quad (7.12)$$

According to Lemma 4.2, we can get

$$\begin{aligned} I\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^{p-1} &\leq \frac{(p-1)\varepsilon}{p}\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^p + \frac{1}{p\varepsilon^{p-1}}I^p, \\ I^2\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^{p-2} &\leq \frac{(p-2)\varepsilon}{p}\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^p + \frac{2}{p\varepsilon^{(p-2)/2}}I^p. \end{aligned} \quad (7.13)$$

Then

$$\begin{aligned} LV &\leq \left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^p \times \left[-p\tau_1(\mu_0 + \nu) + \frac{\beta\Lambda\tau_1\varepsilon(p-1)}{\mu_0 + a_1\Lambda} + \frac{(p-2)(p-1)\varepsilon\tau_1\sigma^2\Lambda^2}{2(\mu_0 + a_1\Lambda)^2}\right] \\ &\quad + I^p \times \left[\frac{\tau_1\beta\Lambda}{(\mu_0 + a_1\Lambda)\varepsilon^{p-1}} + \frac{\tau_1(p-1)\sigma^2\Lambda^2}{(\mu_0 + a_1\Lambda)^2\varepsilon^{(p-2)/2}}\right] \\ &\quad + I^p \times \left[-(\mu_0 + \mu_1 + \gamma) + \frac{\beta\Lambda}{\mu_0 + a_1\Lambda} + \frac{(p-1)\sigma^2\Lambda^2}{2(\mu_0 + a_1\Lambda)^2}\right]. \end{aligned} \quad (7.14)$$

Select ε small enough to make the coefficient of $\left(\frac{\Lambda}{\mu_0 + \nu} - S\right)^p$ negative. By (4.8), we have $-(\mu_0 + \mu_1 + \gamma) + \frac{\beta\Lambda}{\mu_0 + a_1\Lambda} + \frac{(p-1)\sigma^2\Lambda^2}{2(\mu_0 + a_1\Lambda)^2} < 0$. When $-(\mu_0 + \mu_1 + \gamma) + \frac{\beta\Lambda}{\mu_0 + a_1\Lambda} + \frac{(p-1)\sigma^2\Lambda^2}{2(\mu_0 + a_1\Lambda)^2} < 0$, select τ_1 is positive, so that the coefficient of I^p is negative.

Appendix C Proof of Theorem 4.3.

Applying Itô formula for the second equation of model (2.2), one has

$$d \ln I(t) = \left[\frac{\beta S}{f(S, I)} - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 S^2}{2f^2(S, I)} \right] dt + \frac{\sigma S}{f(S, I)} dB(t). \quad (7.15)$$

Integrating the above equation from 0 to t , and dividing both sides by t at the same time, then we get

$$\ln I(t) = \ln I(0) + \int_0^t \left[\frac{\beta S(\tau)}{f(S(\tau), I(\tau))} - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 S^2(\tau)}{2f^2(S(\tau), I(\tau))} \right] d\tau + M(t), \quad (7.16)$$

where $M(t) := \int_0^t \frac{\sigma S(\tau)}{f(S(\tau), I(\tau))} dB(\tau)$. Then

$$\frac{\ln I(t)}{t} = \frac{\ln I(0)}{t} + \frac{1}{t} \int_0^t \left[-\frac{\sigma^2}{2} \left(\frac{S(\tau)}{f(S(\tau), I(\tau))} - \frac{\beta}{\sigma^2} \right)^2 - (\mu_0 + \mu_1 + \gamma) + \frac{\beta^2}{2\sigma^2} \right] d\tau + \frac{M(t)}{t}. \quad (7.17)$$

The following formula can be obtained from the martingale theorem of large numbers

$$\limsup_{t \rightarrow \infty} \frac{M(t)}{t} = 0 \quad \text{a.s.} \quad (7.18)$$

If condition (i) is met, then by (7.17), (7.18), we have

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \leq \frac{\beta^2}{2\sigma^2} - (\mu_0 + \mu_1 + \gamma) < 0 \quad \text{a.s.} \quad (7.19)$$

From the definition of $f(S, I)$, we get

$$\frac{S}{f(S, I)} \leq \frac{S}{1 + a_1 S} \leq \frac{\Lambda}{\mu_0 + a_1 \Lambda}. \quad (7.20)$$

If condition (ii) is met, then by (7.20), one has

$$\begin{aligned} \frac{\ln I(t)}{t} &= \frac{\ln I(0)}{t} + \frac{1}{t} \int_0^t \left[\frac{\beta S(\tau)}{f(S(\tau), I(\tau))} - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 S^2(\tau)}{2f^2(S(\tau), I(\tau))} \right] d\tau + \frac{M(t)}{t} \\ &\leq \frac{\ln I(0)}{t} + \frac{1}{t} \int_0^t \left[\frac{\beta S(\tau)}{1 + a_1 S(\tau)} - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 S^2(\tau)}{2(1 + a_1 S(\tau))^2} \right] d\tau + \frac{M(t)}{t} \\ &\leq \frac{\ln I(0)}{t} + \frac{\beta \Lambda}{\mu_0 + a_1 \Lambda} - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 \Lambda^2}{2(\mu_0 + a_1 \Lambda)^2} + \frac{M(t)}{t} \\ &\leq (R_0^s - 1)(\mu_0 + \mu_1 + \gamma) + \frac{\ln I(0)}{t} + \frac{M(t)}{t}. \end{aligned} \quad (7.21)$$

If condition (ii) is met, then by (7.21), one has

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \leq (R_0^s - 1)(\mu_0 + \mu_1 + \gamma) < 0 \quad \text{a.s.} \quad (7.22)$$

The above inequality indicates that

$$\lim_{t \rightarrow \infty} I(t) = 0 \quad \text{a.s.} \quad (7.23)$$

Appendix D Proof of Theorem 4.4.

By Corollary 4.1, we have $S(t) < \frac{\Lambda}{\mu_0}$ a.s. Then,

$$\begin{aligned} \frac{\beta S}{f(S, I)} &= \frac{\beta \Lambda}{\mu_0 + a_1 \Lambda} - \frac{\beta \mu_0}{\mu_0 + a_1 \Lambda} \cdot \frac{\frac{\Lambda}{\mu_0} - S}{f(S, I)} - \frac{\beta \Lambda a_2 I}{(\mu_0 + a_1 \Lambda) f(S, I)} - \frac{\beta \Lambda a_3 S I}{(\mu_0 + a_1 \Lambda) f(S, I)} \\ &\geq \frac{\beta \Lambda}{\mu_0 + a_1 \Lambda} - \frac{\beta \mu_0}{\mu_0 + a_1 \Lambda} \cdot \left(\frac{\Lambda}{\mu_0} - S \right) - \frac{\beta \Lambda}{(\mu_0 + a_1 \Lambda)} \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) I \\ &= \frac{\beta \mu_0 S}{\mu_0 + a_1 \Lambda} - \frac{\beta \Lambda}{(\mu_0 + a_1 \Lambda)} \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) I. \end{aligned} \quad (7.24)$$

By model (2.2), one has

$$d(S + I + R) = [\Lambda - \mu_0(S + I + R) - \mu_1 I] dt. \quad (7.25)$$

Integrating the above equation from 0 to t , and dividing both sides by t at the same time, then

$$\begin{aligned} &\frac{S(t) + I(t) + R(t) - (S(0) + I(0) + R(0))}{t} \\ &= \Lambda - \mu_0 \langle S(t) \rangle - (\mu_0 + \mu_1) \langle I(t) \rangle - \mu_0 \langle R(t) \rangle. \end{aligned} \quad (7.26)$$

Then

$$\langle S(t) \rangle = \frac{\Lambda}{\mu_0} - \frac{\mu_0 + \mu_1}{\mu_0} \langle I(t) \rangle - \langle R(t) \rangle - \varphi(t), \quad (7.27)$$

here $\varphi(t) = [S(t) + I(t) + R(t) - (S(0) + I(0) + R(0))]/(\mu_0 t)$. In this situation,

$$\lim_{t \rightarrow \infty} \varphi(t) = 0 \text{ a.s.} \quad (7.28)$$

Applying Itô formula for the second equation of model (2.2) and combining with (7.20), (7.24), we get

$$\begin{aligned} d \ln I(t) &= \left[\frac{\beta S}{f(S, I)} - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 S^2}{2f^2(S, I)} \right] dt + \frac{\sigma S}{f(S, I)} dB(t) \\ &\geq \left[\frac{\beta \mu_0 S}{\mu_0 + a_1 \Lambda} - \frac{\beta \Lambda}{\mu_0 + a_1 \Lambda} \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) I \right. \\ &\quad \left. - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 \Lambda^2}{2(\mu_0 + a_1 \Lambda)^2} \right] dt + \frac{\sigma S}{f(S, I)} dB(t). \end{aligned} \quad (7.29)$$

Integrating the above equation from 0 to t , and combining with (7.27), then

$$\begin{aligned} \ln I(t) &\geq \ln I(0) + \frac{\beta \mu_0 t}{\mu_0 + a_1 \Lambda} \left(\frac{\Lambda}{\mu_0} - \frac{\mu_0 + \mu_1}{\mu_0} \langle I(t) \rangle - \langle R(t) \rangle - \varphi(t) \right) - \frac{\beta \Lambda}{\mu_0 + a_1 \Lambda} \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) \\ &\quad \times \int_0^t I(\tau) d\tau - \left[(\mu_0 + \mu_1 + \gamma) + \frac{\sigma^2 \Lambda^2}{2(\mu_0 + a_1 \Lambda)^2} \right] \cdot t + \int_0^t \frac{\sigma S(\tau)}{f(S(\tau), I(\tau))} dB(\tau) \\ &\geq \left[\frac{\beta \Lambda}{\mu_0 + a_1 \Lambda} - (\mu_0 + \mu_1 + \gamma) - \frac{\sigma^2 \Lambda^2}{2(\mu_0 + a_1 \Lambda)^2} \right] \cdot t - \frac{\beta}{\mu_0 + a_1 \Lambda} \left[\mu_0 + \mu_1 + \Lambda \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) \right] \\ &\quad \times \int_0^t I(\tau) d\tau - \frac{\beta \mu_0}{\mu_0 + a_1 \Lambda} (\langle R(t) \rangle + \varphi(t)) \cdot t + \int_0^t \frac{\sigma S(\tau)}{f(S(\tau), I(\tau))} dB(\tau) + \ln I(0) \\ &\geq (\mu_0 + \mu_1 + \gamma) (R_0^s - 1) \cdot t - \frac{\beta}{\mu_0 + a_1 \Lambda} \left[\mu_0 + \mu_1 + \Lambda \left(a_2 + a_3 \frac{\Lambda}{\mu_0} \right) \right] \times \int_0^t I(\tau) d\tau + \Phi(t), \end{aligned} \quad (7.30)$$

here $\Phi(t) = \ln I(t) + \int_0^t \frac{\sigma S(\tau)}{f(S(\tau), I(\tau))} dB(\tau) - \frac{\beta \mu_0}{\mu_0 + a_1 \Lambda} (\langle R(t) \rangle + \varphi(t)) t$. By combining the martingale theorem of large numbers with (7.28), we can get

$$\lim_{t \rightarrow \infty} \frac{\Phi(t)}{t} = 0 \text{ a.s.} \quad (7.31)$$

Lemma 4.3 implies that

$$\liminf_{t \rightarrow \infty} \langle I(t) \rangle \geq \frac{(\mu_0 + \mu_1 + \gamma)(\mu_0 + a_1 \Lambda)(R_0^s - 1)}{\beta [\mu_0 + \mu_1 + \Lambda(a_2 + a_3 \frac{\Lambda}{\mu_0})]} > 0 \text{ a.s.} \quad (7.32)$$



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