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Research article

A model of HBV infection with intervention strategies: dynamics analysis and numerical simulations

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Abstract: In this paper, we analyze the effect of environment noise on the transmission dynamics of a stochastic hepatitis B virus (HBV) infection model with intervention strategies. By using the Markov semigroups theory, we define the stochastic basic reproduction number and find it can be used to govern disease extinction or persistence. When it is less than one, under a mild extra condition, the stochastic system has a disease-free equilibrium and the disease is predicted to die out with probability one. When it is greater than one, under mild extra conditions, the model admits a stationary distribution which means the persistence of the disease. Thus, we observe that larger intensity of noise (resulting in a smaller stochastic basic reproduction number) can suppress the emergence of hepatitis B outbreak. Numerical simulations are also carried out to investigate the influence of information intervention strategies that may change individual behavior and protect the susceptible from infection. Our analysis shows that the environmental noise can greatly affect the long-term behavior of the system, highlighting the importance of the role of intervention strategies in the control of hepatitis B.

Keywords: stochastic hepatitis B model; Markov semigroups; stochastic basic reproduction number; intervention strategies; extinction and persistence

1. Introduction

Outbreaks of infectious diseases have caused seriously endanger to human life and property safety, which is also a major global health problem. According to the World Health Organization (WHO) report [1]: an estimated 2 billion people worldwide carry hepatitis B virus, about 2 million people suffer from chronic liver infections, more than 780,000 people die every year. How to prevent and control the spread of hepatitis B is one of the hot issues that people care about.

When an infectious disease appears and spreads, one of the measures taken by the disease management department is information intervention [2]. As an important non-drug control measure, intervention strategies (e.g., media coverage, health education) has attracted more and more

attention [3–5]. It plays an important role in helping the government to formulate intervention measures to control diseases and reduce the infection rate of human [6, 7]. For example, during the outbreak of H7N9 influenza in 2013 [8] and the outbreak of cholera virus in Tanzania [9], various information intervention strategies were used. These strategies told people the correct knowledge of disease prevention and greatly reduced the number of contacts per unit time, thus reduced the infection rate. Cui et al. [10, 11] studied the impact of media coverage on the control of infectious diseases and reached the conclusion that media coverage is essential to eradicate the diseases. Therefore, it is of great significance to consider the influence of information intervention in preventing hepatitis B virus (HBV) transmission.

Mathematical models have been showed to be an important tool that helps understand the spread and control of infectious diseases. A large number of mathematical models have been developed to study the dynamics of hepatitis B [12–14]. A mathematical model was developed by Zou et al. [12] to study the transmission dynamics and prevalence of HBV infection in China. They investigated the existence and stability of equilibrium points, sensitivity analysis of the model parameters are also performed. Khan et al. [13] investigated the dynamics of acute and chronic hepatitis B epidemic problem by using a HBV transmission model. They proposed an optimal control strategy to control the spread of hepatitis B. These studies [12–14] used deterministic hepatitis B epidemic models.

For human diseases, due to the unpredictability of human-to-human contacts, the natural growth and spread of epidemic are essentially random [15]. Environmental variations are also important for the development of epidemic [16]. Stochastic epidemic model is more suitable for describing the effect of environmental fluctuations on the dynamics of disease [17–23]. There are very few stochastic hepatitis B epidemic models. Khan et al. [24] discussed the dynamics of disease by proposing a stochastic hepatitis B model with a varying population environment. They investigated the influence of noise intensity on the disease transmission and obtained the sufficient conditions for the extinction and persistence. However, to the best of our knowledge, very few studies, if any, have been done to consider the influence of information intervention into the above-mentioned hepatitis B models [12–14, 24].

In this paper, we studies the dynamics of the stochastic hepatitis B epidemic model incorporating information intervention under environmental noise. By using the Markov semigroups theory, we find that the stochastic basic reproduction number can be used to govern the hepatitis B extinction or persistence. Our innovation points are as follows:

- The effect of information intervention is taken into account the stochastic hepatitis B model.
- By using the Markov semigroups theory, we show the stochastic hepatitis B model admits a stationary distribution.

The hepatitis B model and preliminaries will be introduced in Section 2. In Section 3, we will give our main results. The proofs of the main results in details will be provided in Section 4. In Section 5, some numerical simulations will be conducted to illustrate the influence of environmental noise and information intervention on the hepatitis B dynamics. Finally, we finish the paper with conclusions and future directions in the last section.

2. Model formulation and preliminaries

In this section, we will introduce the stochastic hepatitis B epidemic model incorporating information intervention. It is followed by some preliminaries.

2.1. Model formulation

In [24], Khan et al. proposed the following hepatitis B epidemic model:

$$\begin{cases} dS(t) = [\Lambda - \beta S(t)I(t) - (\mu_0 + \nu)S(t)]dt, \\ dI(t) = [\beta S(t)I(t) - (\mu_0 + \mu_1 + \gamma_1)I(t)]dt, \\ dR(t) = [\gamma_1 I(t) + \nu S(t) - \mu_0 R(t)]dt, \end{cases} \tag{2.1}$$

where $S(t)$, $I(t)$ and $R(t)$ represent susceptible, infective and recovered population, respectively. All parameters are assumed to be positive and the descriptions are listed in Table 1.

Table 1. Parameters used in hepatitis B model (2.1).

Parameter	Parameter description
Λ	The per capita constant birth rate
β	The transmission rate
μ_0	The natural death rate
μ_1	The disease induced death rate
ν	The vaccination rate
γ_1	The constant recovery rate

In this paper, we consider the effect of intervention strategies into the hepatitis B model (2.1). In order to do this, using a function $\beta = \beta_1 - \beta_2 f(I)$ and the function $f(I)$ satisfies the following assumption:

$$(\mathbf{A1})\ \ f(0) = 0,\ f'(I) > 0 \ \text{ and } \ \lim_{I\to\infty} f(I) = 1,$$

where β_1 is the usual contact rate without considering the infectious individuals, and β_2 is the maximum reduced contact rate due to the presence of the infected individuals. Then we obtain the following hepatitis B model with information intervention:

$$\begin{cases} dS(t) = [\Lambda - (\beta_1 - \beta_2 f(I))S(t)I(t) - (\mu_0 + \nu)S(t)]dt, \\ dI(t) = [(\beta_1 - \beta_2 f(I))S(t)I(t) - (\mu_0 + \mu_1 + \gamma_1)I(t)]dt, \\ dR(t) = [\gamma_1 I(t) + \nu S(t) - \mu_0 R(t)]dt. \end{cases} \tag{2.2}$$

To consider the effect of environment noise, we suppose that the contact transmission coefficient β_1 is stochastically perturbed, $\beta_1 \to \beta_1 + \sigma\dot{B}(t)$. The hepatitis B model (2.2) becomes

$$\begin{cases} dS(t) = [\Lambda - (\beta_1 - \beta_2 f(I))S(t)I(t) - (\mu_0 + \nu)S(t)]dt - \sigma S(t)I(t)dB(t), \\ dI(t) = [(\beta_1 - \beta_2 f(I))S(t)I(t) - (\mu_0 + \mu_1 + \gamma_1)I(t)]dt + \sigma S(t)I(t)dB(t), \\ dR(t) = [\gamma_1 I(t) + \nu S(t) - \mu_0 R(t)]dt, \end{cases} \tag{2.3}$$

where $B(t)$ is the standard Brownian motion and σ^2 is the intensity of the noise.

2.2. Preliminaries

In this subsection, we introduce some definitions and results about the Markov semigroup and asymptotic properties [25–31].

2.2.1. Markov semigroup

Let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \text{Prob})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ which meet the general conditions (i.e., it is right continuous and increasing while $\mathcal{F}_0$ contains all P-null sets).

Let $\Sigma = \mathscr{B}(\mathbb{X})$ be the σ-algebra of Borel subsets of $\mathbb{X}$ and m the Lebesgue measure on $(\mathbb{X}, \Sigma)$. $\mathbb{D} = \mathbb{D}(\mathbb{X}, \Sigma, m)$ denotes the subset of the space $L^1 = L^1(\mathbb{X}, \Sigma, m)$ which contains all densities, i.e.,

$$\mathbb{D} = \{g \in L^1 : g \geq 0, \|g\| = 1\},$$

where $\|\cdot\|$ is the norm in L^1. If $P(\mathbb{D}) \subset \mathbb{D}$, then a linear mapping $P : L^1 \to L^1$ is called a Markov operator.

If there exists a measurable function $k : \mathbb{X} \times \mathbb{X} \to [0, \infty)$ such that

$$\int_{\mathbb{X}} k(\mathbf{x}, \mathbf{y}) m(d\mathbf{x}) = 1, \tag{2.4}$$

for almost every $\mathbf{y} \in \mathbb{X}$, then

$$Pg(\mathbf{x}) = \int_{\mathbb{X}} k(\mathbf{x}, \mathbf{y}) g(\mathbf{y}) m(d\mathbf{y})$$

is an integral Markov operator and the function k is called a kernel of the Markov operator P.

A family $\{P(t)\}_{t\geq 0}$ of Markov operators which satisfies conditions:

(a) $P(0) = Id$;

(b) $P(t + s) = P(t)P(s)$ for $s, t \geq 0$;

(c) for every $g \in L^1$ the function $t \mapsto P(t)g$ is continuous.

is called a Markov semigroup.

A Markov semigroup $\{P(t)\}_{t\geq 0}$ is called integral, if for every $t > 0$, the operator $P(t)$ is an integral Markov operator, then there exists a measurable function $k : (0, \infty) \times \mathbb{X} \times \mathbb{X} \to [0, \infty)$ such that

$$P(t)g(\mathbf{x}) = \int_{\mathbb{X}} k(t, \mathbf{x}, \mathbf{y}) g(\mathbf{y}) m(d\mathbf{y})$$

for every density g.

Lemma 2.1. *[25] Let $\{P(t)\}_{t\geq 0}$ be an integral Markov semigroup with a continuous kernel $k(t, \mathbf{x}, \mathbf{y})$ for $t > 0$ which satisfies* (2.4) *for all $y \in \mathbb{X}$. If for every $g \in \mathbb{D}$*

$$\int_0^\infty P(t)g(\mathbf{x})dt > 0,$$

then this semigroup is asymptotically stable or is sweeping with respect to compact sets.

2.2.2. Fokker-Planck equation

For any $A \in \Sigma$, we denote the transition probability function by $\mathscr{P}(t, x, y, z, A)$ for the diffusion process $(S(t), I(t), R(t))$, i.e.

$$\mathscr{P}(t, x, y, z, A) = \text{Prob}\{(S(t), I(t), R(t)) \in A\}$$

with the initial condition $(S(0), I(0), R(0)) = (x, y, z)$. If $(S(t), I(t), R(t))$ is a solution of system (2.3) such that the distribution of $(S(0), I(0), R(0))$ is absolutely continuous with the density $v(x, y, z)$. Then there also exists the density $U(t, x, y, z)$ of $(S(t), I(t), R(t))$ which satisfies the Fokker-Planck equation [30, 31]:

$$\frac{\partial U}{\partial t} = \frac{1}{2}\sigma^2\left(\frac{\partial^2(\varphi U)}{\partial x^2} - 2\frac{\partial^2(\varphi U)}{\partial x \partial y} + \frac{\partial^2(\varphi U)}{\partial y^2}\right) - \frac{\partial(f_1 U)}{\partial x} - \frac{\partial(f_2 U)}{\partial y} - \frac{\partial(f_3 U)}{\partial z}, \tag{2.5}$$

where $\varphi(x, y, z) = x^2y^2$ and

$$\begin{aligned} f_1(x, y, z) &= \Lambda - (\beta_1 - \beta_2 f(y))xy - (\mu_0 + \nu)x, \\ f_2(x, y, z) &= (\beta_1 - \beta_2 f(y))xy - (\mu_0 + \mu_1 + \gamma_1)y, \\ f_3(x, y, z) &= \gamma_1 y + \nu x - \mu_0 z. \end{aligned} \tag{2.6}$$

Let $P(t)V(x, y, z) = U(x, y, z, t)$ for $V \in \mathbb{D}$. Due to the operator $P(t)$ is a contraction on $\mathbb{D}$, it can be extended to a contraction on L^1. Then the operators $\{P(t)\}_{t\geq 0}$ form a Markov semigroup. Denote $\mathscr{A}$ the infinitesimal generator of semigroup $\{P(t)\}_{t\geq 0}$, i.e.

$$\mathscr{A}V = \frac{1}{2}\sigma^2\left(\frac{\partial^2(\varphi V)}{\partial x^2} - 2\frac{\partial^2(\varphi V)}{\partial x \partial y} + \frac{\partial^2(\varphi V)}{\partial y^2}\right) - \frac{\partial(f_1 V)}{\partial x} - \frac{\partial(f_2 V)}{\partial y} - \frac{\partial(f_3 V)}{\partial z}.$$

The adjoint operator of $\mathscr{A}$ is of the form

$$\mathscr{A}^*V = \frac{1}{2}\sigma^2\varphi\left(\frac{\partial^2 V}{\partial x^2} - 2\frac{\partial^2 V}{\partial x \partial y} + \frac{\partial^2 V)}{\partial y^2}\right) + \frac{\partial(f_1 V)}{\partial x} + \frac{\partial(f_2 V)}{\partial y} + \frac{\partial(f_3 V)}{\partial z}.$$

3. Main results

The basic reproduction number $\mathscr{R}_0$ is a threshold which represents how many secondary infections result from the introduction of one infected individual into a population of susceptible [32]. We can calculate the basic reproduction number $\mathscr{R}_0$ for the deterministic hepatitis B model (2.2), given by

$$\mathscr{R}_0 = \frac{\Lambda\beta_1}{(\mu_0 + \nu)(\mu_0 + \mu_1 + \gamma_1)},$$

which can be shown to be a threshold of extinction or persistence of disease for the model (2.2).

In addition, we easily know that there exist two equilibriums for model (2.2): the disease free equilibrium $E_0 = \left(\frac{\Lambda}{\mu_0+\nu}, 0, \frac{\nu\Lambda}{\mu_0(\mu_0+\nu)}\right)$ always exists and the unique endemic equilibrium $E^* = (S^*, I^*, R^*)$ exists whenever $\mathscr{R}_0 > 1$, which is a positive solution of the following system:

$$\begin{cases} \Lambda - (\beta_1 - \beta_2 f(I^*))S^*I^* - (\mu_0 + \nu)S^* = 0, \\ (\beta_1 - \beta_2 f(I^*))S^*I^* - (\mu_0 + \mu_1 + \gamma_1)I^* = 0, \\ \gamma_1 I^* + \nu S^* - \mu_0 R^* = 0, \end{cases}$$

where S^*, I^*, R^* satisfy

$$S^* = \frac{\mu_0 + \mu_1 + \gamma_1}{\beta_1 - \beta_2 f(I^*)}, \quad R^* = \frac{\gamma_1 I^*(\beta_1 - \beta_2 f(I^*)) + \nu(\mu_0 + \mu_1 + \gamma_1)}{\mu_0(\beta_1 - \beta_2 f(I^*))},$$

and

$$\Lambda - (\mu_0 + \mu_1 + \gamma_1)I^* - \frac{(\mu_0 + \nu)(\mu_0 + \mu_1 + \gamma_1)}{\beta_1 - \beta_2 f(I^*)} = 0.$$

Set

$$F(I) := \Lambda - (\mu_0 + \mu_1 + \gamma_1)I - \frac{(\mu_0 + \nu)(\mu_0 + \mu_1 + \gamma_1)}{\beta_1 - \beta_2 f(I)}.$$

Since

$$F(0) = \Lambda - \frac{(\mu_0 + \nu)(\mu_0 + \mu_1 + \gamma_1)}{\beta_1} = \frac{(\mu_0 + \nu)(\mu_0 + \mu_1 + \gamma_1)(\mathscr{R}_0 - 1)}{\beta_1},$$

if $\mathscr{R}_0 > 1$, then $F(0) > 0$. It follows from the assumption $(A1)$ that $F(I)$ is a decreasing function. Therefore, $F(I) = 0$ has a unique positive solution I^* and model (2.2) has a unique endemic equilibrium $E^* = (S^*, I^*, R^*)$ with

$$S^* = \frac{\mu_0 + \mu_1 + \gamma_1}{\beta_1 - \beta_2 f(I^*)}, \quad R^* = \frac{\gamma_1 I^*(\beta_1 - \beta_2 f(I^*)) + \nu(\mu_0 + \mu_1 + \gamma_1)}{\mu_0(\beta_1 - \beta_2 f(I^*))}.$$

Now, we give the results concerning the existence of unique positive solution, extinction and persistence of the disease for the hepatitis B model (2.3). For simplicity, define a bounded set Γ by

$$\Gamma = \left\{(S(t), I(t), R(t)) \in \mathbb{X} : S(t) > 0, I(t) > 0, R(t) > 0, \frac{\Lambda}{\mu_0 + \mu_1} \leq S(t) + I(t) + R(t) \leq \frac{\Lambda}{\mu_0}\right\} \subset \mathbb{X},$$

and denote the stochastic basic reproduction number $\mathscr{R}_s$ for the hepatitis B model (2.3) by

$$\mathscr{R}_s = \frac{\Lambda\beta_1}{(\mu_0 + \nu)\left(\mu_0 + \mu_1 + \gamma_1 + \frac{\sigma^2\Lambda^2}{2(\mu_0+\nu)^2}\right)}.$$

The main results of this paper are given by the following three theorems.

Theorem 3.1. *There exists a unique positive solution $(S(t), I(t), R(t))$ to model (2.3) on $t \geq 0$ for any initial value $(S(0), I(0), R(0)) \in \mathbb{X}$, and the solution will remain in $\mathbb{X}$ with probability one.*

Theorem 3.2. *Let $(S(t), I(t), R(t))$ be the solution of the hepatitis B model (2.3) with initial value $(S(0), I(0), R(0)) \in \Gamma$. If*

$$\mathscr{R}_s < 1, \text{ and } \sigma^2 \leq \frac{\beta_1(\mu_0 + \nu)}{\Lambda}, \tag{3.1}$$

then

$$\limsup_{t\to\infty} \frac{\log I(t)}{t} < 0, \ a.s.$$

namely, $I(t)$ converges to 0 exponentially a.s., and the disease in model (2.3) will die out with probability one. In addition,

$$\lim_{t\to\infty} S(t) = \frac{\Lambda}{\mu_0 + \nu} = S_0, \ \lim_{t\to\infty} R(t) = \frac{\nu\Lambda}{\mu_0(\mu_0 + \nu)} = R_0. \tag{3.2}$$

Theorem 3.3. *For every $t > 0$, the distribution of $(S(t), I(t), R(t))$ has a density $U(t, x, y, z)$. If $\mathscr{R}_s > 1$ and*

$$\begin{aligned}
&\theta_2 < \frac{2\mu_0}{\nu},\ \sigma^2 < \frac{2(\mu_0 + \nu + \frac{1}{2}\theta_2\nu)}{\theta_1 I^*} \min\{1, A_1, A_2\},\\
&A_1 = \frac{2\mu_0 + 2\mu_1 + \gamma_1}{(\mu_0 + \nu + \frac{1}{2}\theta_2\nu)S^{*2} + 2\mu_0 + 2\mu_1 + \gamma_1},\\
&A_2 = \frac{\theta_2(\mu_0 + \frac{1}{2}\nu)}{(\mu_0 + \nu + \frac{1}{2}\theta_2\nu)S^{*2} + \theta_2(\mu_0 + \frac{1}{2}\nu)},
\end{aligned} \tag{3.3}$$

hold, where $\theta_1 = \frac{4\mu_0+2\mu_1+\gamma_1+\nu}{\beta_1-\beta_2 f(I^*)}$, $\theta_2 = \frac{2\mu_0+\mu_1}{\gamma_1}$, then there exists a unique density $U_*(x, y, z)$ which is a stationary solution of model (2.3) *and*

$$\lim_{t\to\infty} \iiint_{\mathbb{X}} |U(t, x, y, z) - U_*(x, y, z)|dxdydz = 0.$$

In addition, we have

$$\Pi \equiv supp\ U_* = \left\{(x, y, z) \in \mathbb{X} : \frac{\Lambda}{\mu_0 + \mu_1} < x + y + z < \frac{\Lambda}{\mu_0}\right\}. \tag{3.4}$$

Remark 3.4. *It follows from Theorem 3.2 that if $\mathscr{R}_s < 1$, the disease in hepatitis B model* (2.3) *will die out. The results of Theorem 3.3 mean that the disease is prevalent if $\mathscr{R}_s > 1$. Therefore, together with Theorem 3.2 and 3.3, we can clearly see that $\mathscr{R}_s$ can be a threshold of disease persistence and extinction. We also conclude that the large random noise can suppress the outbreak of disease.*

4. Proofs of main results

In this section, we analyze the dynamical behavior of the system (2.3) and give the detailed proofs of our main results.

4.1. Proof of Theorem 3.1

The aim of this subsection is to show the existence of unique positive global solution of stochastic model (2.3), namely to prove Theorem 3.1.

Proof. The proof of Theorem 3.1 is similar to that in [4, 24]. Here we omit it.

The following result shows that the solutions of system (2.3) are bounded.

Lemma 4.1. *The unique solution of stochastic hepatitis B epidemic model* (2.3) *on $t \geq 0$ for any initial value $(S(0), I(0), R(0)) \in \mathbb{X}$ will enter Γ and will remain in Γ with probability one.*

Proof. Let the total size of population be $N(t) = S(t) + I(t) + R(t)$. From model (2.3), we have

$$\frac{dN(t)}{dt} = \Lambda - \mu_0 N(t) - \mu_1 I(t).$$

This implies that

$$\Lambda - (\mu_0 + \mu_1)N(t) \leq \frac{dN(t)}{dt} \leq \Lambda - \mu_0 N(t).$$

Letting $t \to \infty$, we get

$$\frac{\Lambda}{\mu_0 + \mu_1} \leq \liminf_{t\to\infty} N(t) \leq \limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu_0}.$$

Then the region

$$\Gamma = \Big\{(S(t), I(t), R(t)) : S(t) > 0, I(t) > 0, R(t) > 0, \frac{\Lambda}{\mu_0 + \mu_1} \leq N(t) \leq \frac{\Lambda}{\mu_0}\Big\}.$$

Therefore, all solution $S(t)$, $I(t)$ and $R(t)$ of model (2.3) are bounded by $\frac{\Lambda}{\mu_0}$. Thus, we get that Γ is the positively invariant bounded set. In conclusion, the trajectories of all solution initiating anywhere of $\mathbb{X}$ will enter Γ and then remain in Γ with probability one.

4.2. Proof of Theorem 3.2

In this subsection, for simplicity, we begin with introducing the following notation and lemma:

$$\langle x(t)\rangle = \frac{1}{t}\int_0^t x(s)ds.$$

Lemma 4.2. *[33] Let $M = \{M(t)\}_{t\geq 0}$ be a real-valued continuous local martingale vanishing at $t = 0$. Then*

$$\lim_{t\to\infty}\langle M, M\rangle_t = \infty \ \ a.s. \Rightarrow \lim_{t\to\infty}\frac{M(t)}{\langle M, M\rangle_t} = 0 \ \ a.s.$$

and

$$\limsup_{t\to\infty}\frac{\langle M, M\rangle_t}{t} < \infty \ \ a.s. \Rightarrow \lim_{t\to\infty}\frac{M(t)}{t} = 0 \ \ a.s.,$$

where $\langle M, M\rangle_t$ denotes the quadratic variation of M.

Next, we investigate the extinction of disease for the stochastic hepatitis B model (2.3), which means to prove Theorem 3.2.

Proof. An integration of system (2.3) yields

$$\begin{cases} \frac{S(t)-S(0)}{t} = \Lambda - (\beta_1 - \beta_2 f(I))\langle S(t)I(t)\rangle - (\mu_0 + \nu)\langle S(t)\rangle - \frac{\sigma}{t}\int_0^t S(s)I(s)dB(s), \\ \frac{I(t)-I(0)}{t} = (\beta_1 - \beta_2 f(I))\langle S(t)I(t)\rangle - (\mu_0 + \mu_1 + \gamma_1)\langle I(t)\rangle + \frac{\sigma}{t}\int_0^t S(s)I(s)dB(s), \\ \frac{R(t)-R(0)}{t} = \gamma_1\langle I(t)\rangle + \nu\langle S(t)\rangle - \mu_0\langle R(t)\rangle. \end{cases} \tag{4.1}$$

According to (4.1), we have

$$\frac{S(t) - S(0)}{t} + \frac{I(t) - I(0)}{t} = \Lambda - (\mu_0 + \nu)\langle S(t)\rangle - (\mu_0 + \mu_1 + \gamma_1)\langle I(t)\rangle. \tag{4.2}$$

We compute that

$$\langle S(t)\rangle = \frac{\Lambda}{\mu_0 + \nu} - \frac{\mu_0 + \mu_1 + \gamma_1}{\mu_0 + \nu}\langle I(t)\rangle + \varphi(t), \tag{4.3}$$

where $\varphi(t)$ is defined by

$$\varphi(t) = -\frac{1}{\mu_0 + \nu}\left[\frac{S(t) - S(0)}{t} + \frac{I(t) - I(0)}{t}\right].$$

Obviously,

$$\lim_{t\to\infty} \varphi(t) = 0.\ a.s.$$

Applying the Itô's formula [16] to system (2.3) leads to

$$d\log I(t) = \left[(\beta_1 - \beta_2 f(I))S(t) - (\mu_0 + \mu_1 + \gamma_1) - \frac{1}{2}\sigma^2 S^2(t)\right]dt + \sigma S(t)dB(t).$$

Integrating it from 0 to t and dividing t on both sides, we obtain

$$\begin{aligned}\frac{\log I(t) - \log I(0)}{t} =& (\beta_1 - \beta_2 f(I))\langle S(t)\rangle - (\mu_0 + \mu_1 + \gamma_1) - \frac{1}{2}\sigma^2\langle S^2(t)\rangle \\ &+ \frac{\sigma}{t}\int_0^t S(s)dB(s) \\ \leq& \beta_1\langle S(t)\rangle - (\mu_0 + \mu_1 + \gamma_1) - \frac{1}{2}\sigma^2\langle S(t)\rangle^2 \\ &+ \frac{\sigma}{t}\int_0^t S(s)dB(s).\end{aligned} \tag{4.4}$$

Substituting (4.3) into (4.4) yields

$$\begin{aligned}\frac{\log I(t) - \log I(0)}{t} \leq& \beta_1\left(\frac{\Lambda}{\mu_0 + \nu} - \frac{\mu_0 + \mu_1 + \gamma_1}{\mu_0 + \nu}\langle I(t)\rangle + \varphi(t)\right) - (\mu_0 + \mu_1 + \gamma_1) \\ &- \frac{1}{2}\sigma^2\left(\frac{\Lambda}{\mu_0 + \nu} - \frac{\mu_0 + \mu_1 + \gamma_1}{\mu_0 + \nu}\langle I(t)\rangle + \varphi(t)\right)^2 + \frac{\sigma}{t}\int_0^t S(s)dB(s) \\ \leq& \frac{\beta_1\Lambda}{\mu_0 + \nu} - \frac{\beta_1(\mu_0 + \mu_1 + \gamma_1)}{\mu_0 + \nu}\langle I(t)\rangle - (\mu_0 + \mu_1 + \gamma_1) \\ &- \frac{1}{2}\frac{\sigma^2\Lambda^2}{(\mu_0 + \nu)^2} + \frac{\sigma^2\Lambda(\mu_0 + \mu_1 + \gamma_1)}{(\mu_0 + \nu)^2}\langle I(t)\rangle + \frac{M(t)}{t} + \psi(t) \\ \leq& -\left(\mu_0 + \mu_1 + \gamma_1 + \frac{1}{2}\frac{\sigma^2\Lambda^2}{(\mu_0 + \nu)^2}\right)(1 - \mathscr{R}_s) \\ &- \left(\frac{\mu_0 + \mu_1 + \gamma_1}{\mu_0 + \nu}\right)\left(\beta_1 - \frac{\sigma^2\Lambda}{\mu_0 + \nu}\right)\langle I(t)\rangle + \frac{M(t)}{t} + \psi(t).\end{aligned} \tag{4.5}$$

where

$$\psi(t) = \beta\varphi(t) - \frac{1}{2}\sigma^2\varphi^2(t) + \frac{\sigma^2(\mu_0 + \mu_1 + \gamma_1)}{\mu_0 + \nu}\langle I(t)\rangle\varphi(t) - \frac{\sigma^2\Lambda\varphi(t)}{\mu_0 + \nu}$$

and $M(t) = \sigma\int_0^t S(s)dB(s)$, which is a local continuous martingale with $M(0) = 0$. Moreover,

$$\limsup_{t\to\infty}\frac{\langle M, M\rangle_t}{t} \leq \frac{\sigma^2\Lambda^2}{\mu_0^2} < \infty\ a.s.$$

By Lemma 4.2 and $\lim_{t\to\infty}\varphi(t) = 0$, we obtain

$$\lim_{t\to\infty}\frac{M(t)}{t} = 0 \ \text{ and } \ \lim_{t\to\infty}\psi(t) = 0\ \ a.s.$$

If the condition (3.1) is satisfied, it follows from (4.5) that

$$\limsup_{t\to\infty}\frac{\log I(t)}{t} \le -\left(\mu_0+\mu_1+\gamma_1+\frac{1}{2}\frac{\sigma^2\Lambda^2}{(\mu_0+\nu)^2}\right)(1-\mathscr{R}_s) - \left(\frac{\mu_0+\mu_1+\gamma_1}{\mu_0+\nu}\right)\left(\beta_1-\frac{\sigma^2\Lambda}{\mu_0+\nu}\right)\langle I(t)\rangle < 0 \ \ a.s.$$

which implies

$$\lim_{t\to\infty} I(t) = 0 \ \ a.s. \tag{4.6}$$

Next, we prove the assertion (3.2). According to model (2.3), we have

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

We also have

$$S(t)+I(t)+R(t) = e^{-\mu_0 t}\left(S(0)+I(0)+R(0)+\int_0^t[\Lambda-\mu_1 I(s)]e^{\mu_0 s}ds\right).$$

Applying L'Hospital's rule and (4.6), we get

$$\begin{aligned}\lim_{t\to\infty}(S(t)+R(t)) &= \lim_{t\to\infty}\left(\frac{S(0)+I(0)+R(0)+\int_0^t[\Lambda-\mu_1 I(s)]e^{\mu_0 s}ds}{e^{\mu_0 t}}-I(t)\right)\\ &= \lim_{t\to\infty}\frac{\Lambda-\mu_1 I(t)}{\mu_0} = \frac{\Lambda}{\mu_0}.\end{aligned} \tag{4.7}$$

Thus, we obtain

$$\lim_{t\to\infty}(S(t)+R(t)) = \frac{\Lambda}{\mu_0} \ \ a.s.$$

According to model (2.3), the first equation with limiting system yields

$$dS(t) = (\Lambda-(\mu_0+\nu)S(t))dt.$$

Then we obtain

$$\lim_{t\to\infty} S(t) = \frac{\Lambda}{\mu_0+\nu} = S_0 \ \ a.s.$$

Therefore, by (4.7), we have

$$\lim_{t\to\infty} R(t) = \frac{\nu\Lambda}{\mu_0(\mu_0+\nu)} = R_0 \ \ a.s.$$

This finishes the proof.

4.3. Proof of Theorem 3.3

The aim of this subsection is to investigate that the solutions of model (2.3) are converging to the endemic dynamics when $\mathscr{R}_s > 1$ under mild extra conditions. We prove Theorem 3.3 about the existence of stationary distribution for the solution of model (2.3), which implies that the disease is persistent.

In order to prove main result, we give the following lemmas and study the asymptotic stability of the Markov semigroups. First, we check that the semigroup has an invariant density.

Lemma 4.3. *For each point $(x_0, y_0, z_0) \in \mathbb{X}$ and $t > 0$, the transition probability function $\mathscr{P}(t, x_0, y_0, z_0, A)$ has a continuous density $k(t, x, y, z; x_0, y_0, z_0)$ with respect to the Lebesgue measure.*

Proof. Let

$$a_0(S, I, R) = \begin{pmatrix} \Lambda - (\beta_1 - \beta_2 f(I))SI - (\mu_0 + \nu)S \\ (\beta_1 - \beta_2 f(I))SI - (\mu_0 + \mu_1 + \gamma_1)I \\ \gamma_1 I + \nu S - \mu_0 R \end{pmatrix} \text{ and } a_1(S, I, R) = \begin{pmatrix} -\sigma SI \\ \sigma SI \\ 0 \end{pmatrix}.$$

Then Lie bracket $[a_0, a_1]$ is given by

$$a_2 = [a_0, a_1] = \begin{pmatrix} -\sigma I(\Lambda - (\mu_0 + \mu_1 + \gamma_1)S + \beta_2 f'(I)S^2 I) \\ \sigma I(\Lambda - (\mu_0 + \nu)S + \beta_2 f'(I)S^2 I) \\ \sigma(\nu - \gamma_1)SI \end{pmatrix}$$

and

$$a_3 = [a_1, a_2] = \begin{pmatrix} -\sigma^2 I(\Lambda I + (\nu - \mu_1 - \gamma_1)S^2 + (\beta_2 f''(I)S - \beta_2 f'(I))S^2 I^2 + \beta_2 f'(I)S^3 I) \\ \sigma^2 I^2(\Lambda + (\nu - \mu_1 - \gamma_1)S + (\beta_2 f''(I)S - \beta_2 f'(I))S^2 I + \beta_2 f'(I)S^3) \\ \sigma^2(\nu - \gamma_1)(S - I)SI \end{pmatrix}.$$

Thus, a_1, a_2, a_3 are linearly independent on $\mathbb{X}$. Then for each $(S, I, R) \in \mathbb{X}$, the vector $a_1(S, I, R)$, $a_2(S, I, R)$, $a_3(S, I, R)$ span the space $\mathbb{X}$. According to the Hörmander theorem on the existence of smooth densities for degenerate diffusion process (see [34], Theorem 4.3), the transition probability function $\mathscr{P}(t, x_0, y_0, z_0, A)$ has a continuous density $k(t, x, y, z; x_0, y_0, z_0)$ and $k \in C^\infty((0, \infty) \times \mathbb{X} \times \mathbb{X})$.

Using the similar method mentioned in [35], we check the positivity of k.

Fix a point $(x_0, y_0, z_0) \in \mathbb{X}$ and a function $\phi \in L^2([0, T]; \mathbb{R})$, then consider the following system:

$$\begin{cases} x_\phi(t) = x_0 + \int_0^t [f_1(x_\phi(s), y_\phi(s), z_\phi(s)) - \sigma\phi x_\phi(s) y_\phi(s)]ds, \\ y_\phi(t) = y_0 + \int_0^t [f_2(x_\phi(s), y_\phi(s), z_\phi(s)) + \sigma\phi x_\phi(s) y_\phi(s)]ds, \\ z_\phi(t) = z_0 + \int_0^t f_3(x_\phi(s), y_\phi(s), z_\phi(s))ds, \end{cases} \tag{4.8}$$

where $f_1(x, y, z)$, $f_2(x, y, z)$, $f_3(x, y, z)$ are defined as (2.6).

Denote $\mathbf{X} = (x, y, z)^T$, $\mathbf{X}_0 = (x_0, y_0, z_0)^T$, let $D_{\mathbf{X}_0;\phi}$ be the Fréchet derivative of the function $h \mapsto \mathbf{X}_{\phi+h}(T)$ from $L^2([0, T]; \mathbb{R})$ to $\mathbb{X}$. Then $k(T, x, y, z; x_0, y_0, z_0) > 0$ for $\mathbf{X} = \mathbf{X}_\phi(T)$ holds, if the derivative $D_{\mathbf{X}_0;\phi}$ has rank 3 for some $\phi \in L^2([0, T]; \mathbb{R})$. Let

$$\Psi(t) = \mathbf{f}'(\mathbf{X}_\phi(t)) + \phi\mathbf{g}'(\mathbf{X}_\phi(t)),$$

where $\mathbf{f}'$, $\mathbf{g}'$ are the Jacobians of

$$f = \begin{pmatrix} f_1(x,y,z) \\ f_2(x,y,z) \\ f_3(x,y,z) \end{pmatrix} \text{ and } g = \begin{pmatrix} -\sigma xy \\ \sigma xy \\ 0 \end{pmatrix},$$

respectively. Let $Q(t,t_0)(0 \le t_0 \le t \le T)$ be a matrix function, and $Q(t_0,t_0) = Id$, $\frac{\partial Q(t,t_0)}{\partial t} = \Psi(t)Q(t,t_0)$, then

$$D_{\mathbf{X}_0;\phi}h = \int_0^T Q(T,s)g(s)h(s)ds.$$

Lemma 4.4. *There exists $T > 0$ such that $k(T,x,y,z;x_0,y_0,z_0) > 0$ for every $(x_0,y_0,z_0) \in \Pi$ and $(x,y,z) \in \Pi$.*

Proof. We consider a continuous control function ϕ and rewrite the system (4.8) as follows:

$$\begin{cases} x'_\phi(t) = f_1(x_\phi(t), y_\phi(t), z_\phi(t)) - \sigma\phi x_\phi(t) y_\phi(t), \\ y'_\phi(t) = f_2(x_\phi(t), y_\phi(t), z_\phi(t)) + \sigma\phi x_\phi(t) y_\phi(t), \\ z'_\phi(t) = f_3(x_\phi(t), y_\phi(t), z_\phi(t)). \end{cases} \tag{4.9}$$

Step 1: Let $\varepsilon \in (0,T)$ and $h(t) = \frac{\chi_{[T-\varepsilon,T]}(t)}{x_\phi(t)y_\phi(t)}$, $t \in [0,T]$, where χ is the characteristic function. Since

$$Q(T,s) = Id + \Psi(T)(s-T) + \frac{1}{2}\Psi^2(T)(s-T)^2 + o((s-T)^2).$$

Then

$$D_{\mathbf{X}_0;\phi}h = \varepsilon\mathbf{v} - \frac{1}{2}\varepsilon^2\Psi(T)\mathbf{v} + \frac{1}{6}\varepsilon^3\Psi^2(T)\mathbf{v} + o(\varepsilon^3),$$

where

$$\mathbf{v} = \begin{pmatrix} -\sigma \\ \sigma \\ 0 \end{pmatrix}.$$

Compute

$$\Psi(T)\mathbf{v} = \sigma \begin{pmatrix} \mu_0 + \nu + (\sigma\phi + \beta_1 - \beta_2 f(y))(y-x) + \beta_2 f'(y)xy \\ -(\mu_0 + \mu_1 + \gamma_1) - (\sigma\phi + \beta_1 - \beta_2 f(y))(y-x) - \beta_2 f'(y)xy \\ \gamma_1 - \nu \end{pmatrix}$$

and

$$\Psi^2(T)\mathbf{v} = \sigma \begin{pmatrix} a_{11} \\ a_{22} \\ a_{33} \end{pmatrix},$$

where

$$\begin{aligned}
a_{11} =& -\beta_2^2 f'^2(y)x^2y^2 + (2(\beta_1 - \beta_2 f(y) + \sigma\phi)(x-y) - (2\mu_0 + \mu_1 + \gamma_1 + \nu))\beta_2 f'(y)xy \\
&+ (\beta_1 - \beta_2 f(y) + \sigma\phi)((2\mu_0 + \mu_1 + \gamma_1 + \nu)x - 2(\mu_0 + \nu)y) \\
&- (\beta_1 - \beta_2 f(y) + \sigma\phi)^2(x-y)^2, \\
a_{22} =& \beta_2^2 f'^2(y)x^2y^2 - 2((\beta_1 - \beta_2 f(y) + \sigma\phi)(x-y) - (\mu_0 + \mu_1 + \gamma_1))\beta_2 f'(y)xy \\
&- (\beta_1 - \beta_2 f(y) + \sigma\phi)(2(\mu_0 + \mu_1 + \gamma_1)x - (2\mu_0 + \mu_1 + \gamma_1 + \nu)y) \\
&+ (\beta_1 - \beta_2 f(y) + \sigma\phi)^2(x-y)^2 + (\mu_0 + \mu_1 + \gamma_1)^2, \\
a_{33} =& -\gamma_1(2\mu_0 + \mu_1 + \gamma_1) + \nu(2\mu_0 + \nu) + (\nu - \gamma_1)(\beta_2 f'(y)xy + (\beta_1 - \beta_2 f(y) + \sigma\phi)(y - x)).
\end{aligned}$$

It is easy to see that $\mathbf{v}$, $\Psi(T)\mathbf{v}$, and $\Psi^2(T)\mathbf{v}$ are linearly independent. Therefore, the rank of $D_{\mathbf{X}_0;\phi}$ is 3.

Step 2: We check that there exist a control function ϕ and $T > 0$ such that $\mathbf{X}_\phi(0) = \mathbf{X}_0$, $\mathbf{X}_\phi(T) = \mathbf{X}$ for any two points $\mathbf{X}_0 \in \Pi$ and $\mathbf{X} \in \Pi$ holds. Let $w_\phi = x_\phi + y_\phi + z_\phi$, then system (4.9) can be replaced by

$$\begin{cases} x'_\phi(t) = g_1(x_\phi(t), w_\phi(t), z_\phi(t)) - \sigma\phi x_\phi(t)(w_\phi(t) - x_\phi(t) - z_\phi(t)), \\ y'_\phi(t) = g_2(x_\phi(t), w_\phi(t), z_\phi(t)), \\ z'_\phi(t) = g_3(x_\phi(t), w_\phi(t), z_\phi(t)), \end{cases} \tag{4.10}$$

where

$$\begin{aligned}
g_1(x, w, z) &= \Lambda - (\beta_1 - \beta_2 f(w - x - z))x(w - x - z) - (\mu_0 + \nu)x, \\
g_2(x, w, z) &= \Lambda + \mu_1(x + z) - (\mu_0 + \mu_1)w, \\
g_3(x, w, z) &= \gamma_1 w - (\gamma_1 - \nu)x - (\gamma_1 + \mu_0)z.
\end{aligned} \tag{4.11}$$

Let

$$\Pi_0 = \left\{(x, w, z) \in \mathbb{X} : 0 < x, z < \frac{\Lambda}{\mu_0}, \frac{\Lambda}{\mu_0 + \mu_1} < w < \frac{\Lambda}{\mu_0} \text{ and } x, z < w\right\}.$$

Now we claim that there exist a control function ϕ and $T > 0$, for any $(x_0, w_0, z_0) \in \Pi_0$ and $(x_1, w_1, z_1) \in \Pi_0$, we have $(x_\phi(0), w_\phi(0), z_\phi(0)) = (x_0, w_0, z_0)$ and $(x_\phi(T), w_\phi(T), z_\phi(T)) = (x_1, w_1, z_1)$.

To construct the function ϕ, first, we find a positive constant T and a differentiable function

$$w_\phi : [0, T] \to \left(\frac{\Lambda}{\mu_0 + \mu_1}, \frac{\Lambda}{\mu_0}\right),$$

such that $w_\phi(0) = w_0$, $w_\phi(T) = w_1$, $w'_\phi(0) = g_2(x_0, w_0, z_0) = w_0^d$, $w'_\phi(T) = g_2(x_1, w_1, z_1) = w_T^d$ and

$$\Lambda - (\mu_0 + \mu_1)w_\phi(t) < w'_\phi(t) < \Lambda - \mu_0 w_\phi(t), \quad t \in [0, T]. \tag{4.12}$$

Next we separate the construction of the function w_ϕ on three subintervals $[0, \varepsilon]$, $[\varepsilon, T-\varepsilon]$ and $[T-\varepsilon, T]$, where $0 < \varepsilon < \frac{T}{2}$. Let

$$\eta = \frac{1}{2}\min\left\{w_0 - \frac{\Lambda}{\mu_0 + \mu_1}, w_1 - \frac{\Lambda}{\mu_0 + \mu_1}, \frac{\Lambda}{\mu_0} - w_0, \frac{\Lambda}{\mu_0} - w_1\right\}.$$

If $w_\phi \in (\frac{\Lambda}{\mu_0+\mu_1} + \eta, \frac{\Lambda}{\mu_0} - \eta)$, then

$$\Lambda - (\mu_0 + \mu_1)w_\phi(t) < -(\mu_0 + \mu_1)\eta < 0, \text{ and } \Lambda - \mu_0 w_\phi(t) > \mu_0\eta > 0 \text{ for } t \in [0, T]. \tag{4.13}$$

Therefore, from (4.13) it follows that we can find a C^2-function w_ϕ: $[0,\varepsilon] \to (\frac{\Lambda}{\mu_0+\mu_1}+\eta, \frac{\Lambda}{\mu_0}-\eta)$ such that

$$w_\phi(0) = w_0, \;\; w'_\phi(0) = w_0^d, \;\; w'_\phi(\varepsilon) = 0,$$

and for all $t \in [0,\varepsilon]$, the differentiable function w_ϕ satisfies (4.12). The same proof works for $t \in [T-\varepsilon, T]$, we also find a C^2-function w_ϕ: $[T-\varepsilon, T] \to (\frac{\Lambda}{\mu_0+\mu_1}+\eta, \frac{\Lambda}{\mu_0}-\eta)$ such that

$$w_\phi(T) = w_1, \;\; w'_\phi(T) = w_T^d, \;\; w'_\phi(T-\varepsilon) = 0,$$

and for all $t \in [T-\varepsilon, T]$, the differentiable function w_ϕ satisfies (4.12). We choose T sufficiently large such that

$$w_\phi : [0,\varepsilon] \cup [T-\varepsilon, T] \to \left(\frac{\Lambda}{\mu_0+\mu_1}+\eta, \frac{\Lambda}{\mu_0}-\eta\right)$$

can be extend to a C^2-function w_ϕ which defined on the whole interval $[0,T]$, then we have

$$\Lambda - (\mu_0+\mu_1)w_\phi(t) < -(\mu_0+\mu_1)\eta < w'_\phi(t) < \mu_0\eta < \Lambda - \mu_0 w_\phi(t), \;\; \text{for } \; t \in [\varepsilon, T-\varepsilon],$$

and the differentiable function w_ϕ satisfies (4.12) on $[0,T]$.

Thus, two C^2-function x_ϕ and z_ϕ can be found to satisfy the second and third equation of (4.10). Finally, there exists a continuous control function ϕ which can be determined from the first equation of (4.10). This completes the proof.

Lemma 4.5. *If $\mathscr{R}_s > 1$, then for every density g and semigroup $\{P(t)\}_{t\geq 0}$,*

$$\lim_{t\to\infty} \iiint_{\Pi} P(t)g(x,y,z)dxdydz = 1.$$

Proof. Let $Z(t) = S(t) + I(t) + R(t)$, then system (2.3) becomes

$$\begin{cases} dS(t) = g_1(S(t), Z(t), R(t))dt - \sigma S(t)(Z(t) - S(t) - R(t))dB(t), \\ dZ(t) = g_2(S(t), Z(t), R(t))dt, \\ dR(t) = g_3(S(t), Z(t), R(t))dt, \end{cases} \tag{4.14}$$

and the functions g_1, g_2 and g_3 are defined in (4.11). For the positive solution $(S(t), I(t), R(t))$ of model (2.3), we have

$$\Lambda - (\mu_0+\mu_1)Z(t) < \frac{dZ(t)}{dt} < \Lambda - \mu_0 Z(t), \;\; t \in (0,+\infty), \;\; a.s. \tag{4.15}$$

Next, we check that for almost each $\omega \in \Omega$, there exists $t_0 = t_0(\omega)$ such that

$$\frac{\Lambda}{\mu_0+\mu_1} < Z(\omega,t) < \frac{\Lambda}{\mu_0}, \;\; \text{for } \; t > t_0.$$

Here we have the following three possible situations.

(**a**) The case $Z(0) \in \left(\frac{\Lambda}{\mu_0+\mu_1}, \frac{\Lambda}{\mu_0}\right)$ is simple to see from (4.15).

(**b**) Consider the case $Z(0) \in \left(0, \frac{\Lambda}{\mu_0+\mu_1}\right)$. Assume our assertion is false, then for $\omega \in \Omega'$, there exists $\Omega' \subset \Omega$ with $\text{Prob}(\Omega') > 0$ such that $Z(\omega,t) \in \left(0, \frac{\Lambda}{\mu_0+\mu_1}\right)$. Obviously, from (4.15) we see $Z(\omega,t)$ is strictly increasing on $[0,\infty]$ for any $\omega \in \Omega'$. Thus, we have

$$\lim_{t\to\infty} Z(\omega,t) = \frac{\Lambda}{\mu_0+\mu_1}, \;\; \omega \in \Omega'.$$

By the second equation of (4.14), we obtain for any $\omega \in \Omega'$

$$Z(t) = e^{-(\mu_0+\mu_1)t} \cdot \left(Z(0) + \int_0^t e^{(\mu_0+\mu_1)s} [\Lambda + \mu_1(S(s) + R(s))] ds \right).$$

Then for $\omega \in \Omega'$, we have

$$\lim_{t\to\infty} S(\omega, t) = \lim_{t\to\infty} R(\omega, t) = 0.$$

Therefore, $\lim_{t\to\infty} I(\omega, t) = \frac{\Lambda}{\mu_0+\mu_1}$, $\omega \in \Omega'$, which implies that

$$\lim_{t\to\infty} \frac{\log I(t) - \log I(0)}{t} = 0, \quad \omega \in \Omega'.$$

Application of the Itô's formula yields

$$d \log I(t) = \left[(\beta_1 - \beta_2 f(I)) S(t) - (\mu_0 + \mu_1 + \gamma_1) - \frac{1}{2}\sigma^2 S^2(t) \right] dt + \sigma S(t) dB(t),$$

and

$$\frac{\log I(t) - \log I(0)}{t} = (\beta_1 - \beta_2 f(I)) \langle S(t) \rangle - (\mu_0 + \mu_1 + \gamma_1) - \frac{1}{2}\sigma^2 \langle S^2(t) \rangle + \frac{\sigma}{t} \int_0^t S(s) dB(s).$$

According to Lemma 4.2, we have

$$\lim_{t\to\infty} \frac{\sigma}{t} \int_0^t S(s) dB(s) = 0.$$

Hence,

$$\begin{aligned} &\lim_{t\to\infty} \frac{\log I(t) - \log I(0)}{t} \\ &= \lim_{t\to\infty} \left((\beta_1 - \beta_2 f(I)) \langle S(t) \rangle - (\mu_0 + \mu_1 + \gamma_1) - \frac{1}{2}\sigma^2 \langle S^2(t) \rangle + \frac{\sigma}{t} \int_0^t S(s) dB(s) \right) \\ &= -(\mu_0 + \mu_1 + \gamma_1). \end{aligned}$$

This leads to the contradiction that $\lim_{t\to\infty} \frac{\log I(t) - \log I(0)}{t} = 0$, then the assertion holds.

(**c**) Consider the case $Z(0) \in \left(\frac{\Lambda}{\mu_0}, +\infty \right)$. The same proof works to the case (**b**), by contradiction, suppose that there exists $\omega \in \Omega'$ with $\text{Prob}(\Omega') > 0$ such that

$$\lim_{t\to\infty} Z(\omega, t) = \frac{\Lambda}{\mu_0}, \quad \omega \in \Omega'.$$

The second and third equation of (4.14) implies that for any $\omega \in \Omega'$

$$\begin{aligned} Z(t) &= e^{-(\mu_0+\mu_1)t} \cdot \left(Z(0) + \int_0^t e^{(\mu_0+\mu_1)s} [\Lambda + \mu_1(S(s) + R(s))] ds \right), \\ R(t) &= e^{-(\mu_0+\gamma_1)t} \cdot \left(R(0) + \int_0^t e^{(\mu_0+\gamma_1)s} [\gamma_1 Z(s) - (\gamma_1 - \nu) S(s)] ds \right). \end{aligned}$$

It follows that

$$\lim_{t\to\infty} S(\omega,t) = \frac{\Lambda}{\mu_0+\nu},\ \lim_{t\to\infty} I(\omega,t) = 0,\ \lim_{t\to\infty} R(\omega,t) = \frac{\nu\Lambda}{\mu_0(\mu_0+\nu)} \text{ for } \omega\in\Omega'.$$

Thus,

$$\begin{aligned}
&\lim_{t\to\infty}\frac{\log I(t)-\log I(0)}{t}\\
=&\lim_{t\to\infty}\left((\beta_1-\beta_2 f(I))\langle S(t)\rangle-(\mu_0+\mu_1+\gamma_1)-\frac{1}{2}\sigma^2\langle S^2(t)\rangle+\frac{\sigma}{t}\int_0^t S(s)dB(s)\right)\\
=&\frac{\beta_1\Lambda}{\mu_0+\nu}-\frac{\sigma^2\Lambda^2}{2(\mu_0+\nu)^2}-(\mu_0+\mu_1+\gamma_1)\\
=&\left(\mu_0+\mu_1+\gamma_1+\frac{\sigma^2\Lambda^2}{2(\mu_0+\nu)^2}\right)(\mathscr{R}_s-1)\\
>&0\ \ a.s.\ \text{ on }\ \Omega'.
\end{aligned}$$

This leads to the contradiction that $\lim_{t\to\infty} I(\omega,t) = 0$ a.s. and the assertion holds.

Remark 4.1. *The results of Lemma 4.4 and Lemma 4.5 mean that if there exists a stationary solution U_* for Fokker-Planck equation* (2.5)*, then supp $U_* = \Pi$.*

Lemma 4.6. *If $\mathscr{R}_s > 1$, then the semigroup $\{P(t)\}_{t\geq 0}$ is asymptotically stable or is sweeping with respect to compact sets.*

Proof. The result of Lemma 4.3 shows that $\{P(t)\}_{t\geq 0}$ is an integral Markov semigroup, which has a continuous kernel $k(t,x,y,z;x_0,y_0,z_0)$ for $t>0$. Thus, the distribution of $(S(t),I(t),R(t))$ has a density $U(x,y,z,t)$ and it satisfies the Fokker-Planck equation (2.5). The Lemma 4.5 implies that certify the restriction of the semigroup $\{P(t)\}_{t\geq 0}$ to the space $L^1(\Pi)$ is sufficient. By Lemma 4.4, we know for every $g\in\mathbb{D}$

$$\int_0^\infty P(t)g dt > 0,\ \text{a.s. on } \Pi.$$

Therefore, in view of Lemma 2.1 that the semigroup $\{P(t)\}_{t\geq 0}$ is asymptotically stable or is sweeping with respect to compact sets. This finishes the proof.

Lemma 4.7. *If $\mathscr{R}_s > 1$ then the semigroup $\{P(t)\}_{t\geq 0}$ is asymptotically stable provided following conditions are satisfied:*

$$\theta_2 < \frac{2\mu_0}{\nu},\ \sigma^2 < \frac{2(\mu_0+\nu+\frac{1}{2}\theta_2\nu)}{\theta_1 I^*}\min\{1,A_1,A_2\}, \tag{4.16}$$

where $\theta_1 = \frac{4\mu_0+2\mu_1+\gamma_1+\nu}{\beta_1-\beta_2 f(I^*)}$, $\theta_2 = \frac{2\mu_0+\mu_1}{\gamma_1}$ and A_1, A_2 are defined in (3.3).

Proof. The Lemma 4.6 implies that the semigroup $\{P(t)\}_{t\geq 0}$ satisfies the Foguel alternative. We construct a nonnegative C^2-function V and a closed set $O\in\Sigma$ such that

$$\sup_{(S,I,R)\in\mathbb{X}\setminus O} \mathscr{A}^*V < 0,$$

where the function V is called Khasminskiĭ function [29]. Since there exists an endemic equilibrium E^* of system (2.2) when $\mathscr{R}_0 > 1$, then we have

$$\Lambda = (\mu_0 + \nu)S^* + (\beta_1 - \beta_2 f(I^*))S^*I^*,$$

$$(\beta_1 - \beta_2 f(I^*))S^*I^* = (\mu_0 + \mu_1 + \gamma_1)I^*,$$

$$\gamma_1 I^* = \mu_0 R^* - \nu S^*.$$

Define a nonnegative C^2-function V by

$$\begin{aligned} V(S,I,R) =& \frac{1}{2}(S - S^* + I - I^* + R - R^*)^2 + \frac{1}{2}(S - S^* + I - I^*)^2 \\ &+ \theta_1\left(I - I^* - I^*\log\frac{I}{I^*}\right) + \frac{\theta_2}{2}(R - R^*)^2 \\ :=& V_1 + V_2 + \theta_1 V_3 + \theta_2 V_4, \end{aligned}$$

where θ_1 and θ_2 are defined in Lemma 4.7. First, we compute

$$\begin{aligned} \mathscr{A}^*V_1 =& (S - S^* + I - I^* + R - R^*)(\Lambda - \mu_0 S - (\mu_0 + \mu_1)I - \mu_0 R) \\ =& (S - S^* + I - I^* + R - R^*)(-\mu_0(S - S^*) - (\mu_0 + \mu_1)(I - I^*) - \mu_0(R - R^*)) \\ =& -\mu_0(S - S^*)^2 - (\mu_0 + \mu_1)(I - I^*)^2 - \mu_0(R - R^*)^2 - (2\mu_0 + \mu_1)(S - S^*)(I - I^*) \\ &- 2\mu_0(S - S^*)(R - R^*) - (2\mu_0 + \mu_1)(I - I^*)(R - R^*). \end{aligned} \tag{4.17}$$

Next, we have

$$\begin{aligned} \mathscr{A}^*V_2 =& (S - S^* + I - I^*)(\Lambda - (\mu_0 + \nu)S - (\mu_0 + \mu_1 + \gamma_1)I) \\ =& (S - S^* + I - I^*)(-(\mu_0 + \nu)(S - S^*) - (\mu_0 + \mu_1 + \gamma_1)(I - I^*)) \\ =& -(\mu_0 + \nu)(S - S^*)^2 - (\mu_0 + \mu_1 + \gamma_1)(I - I^*)^2 \\ &- (2\mu_0 + \mu_1 + \gamma_1 + \nu)(S - S^*)(I - I^*). \end{aligned} \tag{4.18}$$

We calculate

$$\begin{aligned} \mathscr{A}^*V_3 =& (I - I^*)((\beta_1 - \beta_2 f(I))S - (\mu_0 + \mu_1 + \gamma_1)) + \frac{1}{2}I^*\sigma^2 S^2 \\ =& (I - I^*)((\beta_1 - \beta_2 f(I))S - (\beta_1 - \beta_2 f(I^*))S^*) + \frac{1}{2}I^*\sigma^2 S^2 \\ =& -\beta_2 S(f(I) - f(I^*))(I - I^*) + (\beta_1 - \beta_2 f(I^*))(S - S^*)(I - I^*) + \frac{1}{2}I^*\sigma^2 S^2 \\ \le& (\beta_1 - \beta_2 f(I^*))(S - S^*)(I - I^*) + \frac{1}{2}I^*\sigma^2 S^2. \end{aligned} \tag{4.19}$$

At last, for V_4, we have

$$\begin{aligned} \mathscr{A}^*V_4 =& (R - R^*)(\gamma_1 I + \nu S - \mu_0 R) \\ =& (R - R^*)(\nu(S - S^*) + \gamma_1(I - I^*) - \mu_0(R - R^*)) \\ =& -\mu_0(R - R^*)^2 + \nu(S - S^*)(R - R^*) + \gamma_1(I - I^*)(R - R^*). \end{aligned} \tag{4.20}$$

Combining (4.17)–(4.20), we obtain

$$\begin{aligned}
\mathscr{A}^*V =&\mathscr{A}^*V_1 + \mathscr{A}^*V_2 + \theta_1\mathscr{A}^*V_3 + \theta_2\mathscr{A}^*V_4\\
\leq& -\Big(\mu_0 + \nu + \frac{1}{2}\theta_2\nu\Big)(S - S^*)^2 - (2\mu_0 + 2\mu_1 + \gamma_1)(I - I^*)^2\\
&- \theta_2\Big(\mu_0 + \frac{1}{2}\nu\Big)(R - R^*)^2 + \frac{1}{2}\theta_1 I^*\sigma^2 S^2\\
=& -\Big(\mu_0 + \nu + \frac{1}{2}\theta_2\nu - \frac{1}{2}\theta_1 I^*\sigma^2\Big)\Big(S - \frac{2\mu_0 + 2\nu + \theta_2\nu}{2\mu_0 + 2\nu + \theta_2\nu - \theta_1 I^*\sigma^2}S^*\Big)^2\\
&- (2\mu_0 + 2\mu_1 + \gamma_1)(I - I^*)^2 - \theta_2\Big(\mu_0 + \frac{1}{2}\nu\Big)(R - R^*)^2\\
&+ \frac{\theta_1 I^*\Big(\mu_0 + \nu + \frac{1}{2}\theta_2\nu\Big)\sigma^2}{2\mu_0 + 2\nu + \theta_2\nu - \theta_1 I^*\sigma^2}S^{*2}\\
:=& - b_1(S - c_1S^*)^2 - b_2(I - I^*)^2 - b_3(R - R^*)^2 + b_4.
\end{aligned}$$

It follows from the condition (4.16) of Lemma 4.7 that

$$\begin{aligned}
&\frac{\theta_1 I^*\Big(\mu_0 + \nu + \frac{1}{2}\theta_2\nu\Big)\sigma^2}{2\mu_0 + 2\nu + \theta_2\nu - \theta_1 I^*\sigma^2}S^{*2}\\
<&\min\left\{\frac{2\Big(\mu_0 + \nu + \frac{1}{2}\theta_2\nu\Big)^2 S^{*2}}{2\mu_0 + 2\nu + \theta_2\nu - \theta_1 I^*\sigma^2}, 2\mu_0 + 2\mu_1 + \gamma_1, \theta_2\Big(\mu_0 + \frac{1}{2}\nu\Big)\right\}.
\end{aligned}$$

Then the ellipsoid

$$-b_1(S - c_1S^*)^2 - b_2(I - I^*)^2 - b_3(R - R^*)^2 + b_4 = 0$$

lies entirely in $\mathbb{X}$. Thus, there exist a closed set $O \in \Sigma$ which contains this ellipsoid and constant $c > 0$ such that

$$\sup_{(S,I,R)\in\mathbb{X}\setminus O} \mathscr{A}^*V \leq -c < 0.$$

Remark 4.2. *Together with Lemma 4.6 and Lemma 4.7, we get Theorem 3.3.*

5. Numerical simulations

In this section, we give some numerical simulations to illustrate the effectiveness of our analytical results. Choosing the function $f(I) = \frac{I}{H+I}$ (as in [3, 11]), which satisfy the assumption (**A1**) clearly. Using the Milstein method for stochastic differential equations [36], we consider the following

discretization for system (2.3):

$$\begin{cases} S(k+1) = S(k) + \Big(\Lambda - \Big(\beta_1 - \dfrac{\beta_2 I(k)}{H + I(k)}\Big)S(k)I(k) - (\mu_0 + \nu)S(k)\Big)\Delta t \\ \qquad\qquad - \sigma S(k)I(k)\sqrt{\Delta t}\xi(k) - \dfrac{\sigma^2}{2}S(k)I(k)(\xi^2(k) - 1)\Delta t, \\ I(k+1) = I(k) + \Big(\Big(\beta_1 - \dfrac{\beta_2 I(k)}{H + I(k)}\Big)S(k)I(k) - (\mu_0 + \mu_1 + \gamma_1)I(k)\Big)\Delta t \\ \qquad\qquad + \sigma S(k)I(k)\sqrt{\Delta t}\xi(k) + \dfrac{\sigma^2}{2}S(k)I(k)(\xi^2(k) - 1)\Delta t, \\ R(k+1) = R(k) + (\gamma_1 I(k) + \nu S(k) - \mu_0 R(k))\Delta t, \end{cases}$$

where $\xi(k), k = (1, 2, ..., n)$ are independent Gaussian random variables $N(0, 1)$. We take the parameter values in system (2.3) as in Table 2. The initial value of population size is $S(0) = 0.9, I(0) = 0.8, R(0) = 0.6$ [24]. For system (2.2), with parameter values of Table 2, easy to calculate that the basic reproduction number $\mathscr{R}_0 = \frac{\Lambda\beta_1}{(\mu_0+\nu)(\mu_0+\mu_1+\gamma_1)} = 3.7500 > 1$. Thus, for any initial values $(S(0), I(0), R(0))$, there exist a unique endemic equilibrium $E^* = (0.9273, 0.2347, 3.7207)$ which is globally stable and a disease-free equilibrium $E_0 = (1.2500, 0, 3.7500)$. For a clear comparison with the path of stochastic hepatitis B model (2.3), we show the path of $S(t)$, $I(t)$, $R(t)$ for deterministic model (2.1) in Figure 1.

Table 2. Parameters values of numerical experiments for system (2.3).

Parameter	Parameter description	Value	The source of data
Λ	Birth rate	0.5	[24]
β_1	Transmission rate	0.6	[24]
β_2	Maximum reduced contact rate	0.3	Assumed
μ_0	Natural death rate	0.1	[24]
μ_1	Disease induced death rate	0.05	Assumed
ν	Vaccination rate	0.3	[24]
γ_1	Recovery rate	0.4	[24]
H		10	[11].

5.1. Stochastic endemic dynamics

Choosing the intensity of noise $\sigma = 0.1$, then $\mathscr{R}_s = 1.3445 > 1$, $\sigma^2 = 0.0100 < 0.5595$ and the condition $\theta_2 = 0.6250 < 0.6667$ hold. It follows from Theorem 3.3 that the disease will persistent and we give the simulation result in Figure 2(a). Compared with Figure 1, the solution of system (2.3) in Figure 2(a) shows small fluctuations. We increase the intensity of noise σ to 0.3 ($\mathscr{R}_s = 1.2091 > 1$, $\sigma^2 = 0.0900 < 0.5595$) and 0.5 ($\mathscr{R}_s = 1.0063 > 1$, $\sigma^2 = 0.2500 < 0.5595$), respectively. We find that the fluctuations become stronger with the increase of the noise intensity and the simulation results are showed in Figure 2(b)–(c).

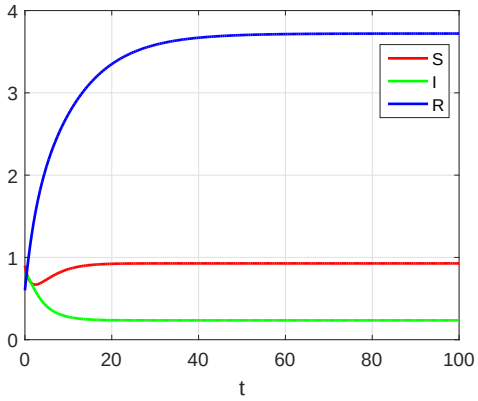
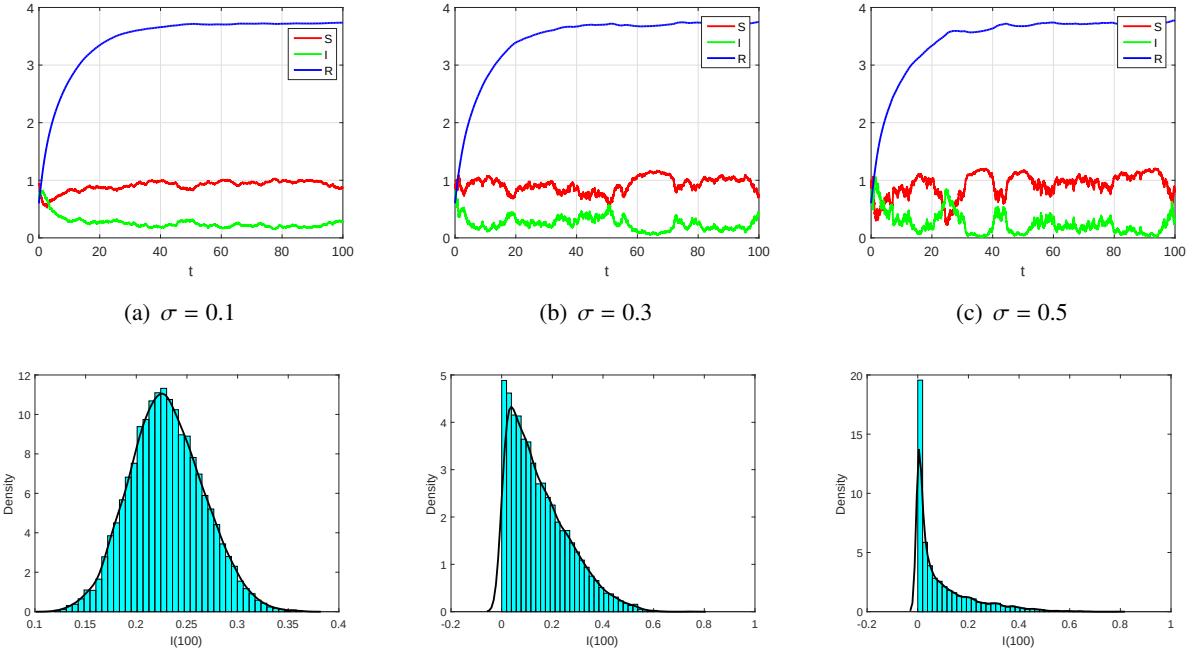


Figure 1. The paths of $S(t)$, $I(t)$ and $R(t)$ for deterministic model (2.2) with initial $(S(0), I(0), R(0)) = (0.9, 0.8, 0.6)$.



(a) $\sigma = 0.1$ (b) $\sigma = 0.3$ (c) $\sigma = 0.5$

(d) $\sigma = 0.1$ (e) $\sigma = 0.3$ (f) $\sigma = 0.5$

Figure 2. The path of $S(t)$, $I(t)$ and $R(t)$ for model (2.3) and the histogram of the probability density function of $I(100)$ with initial $(S(0), I(0), R(0)) = (0.9, 0.8, 0.6)$ under different noise intensities $\sigma = 0.1$, $\sigma = 0.3$ and $\sigma = 0.5$, respectively.

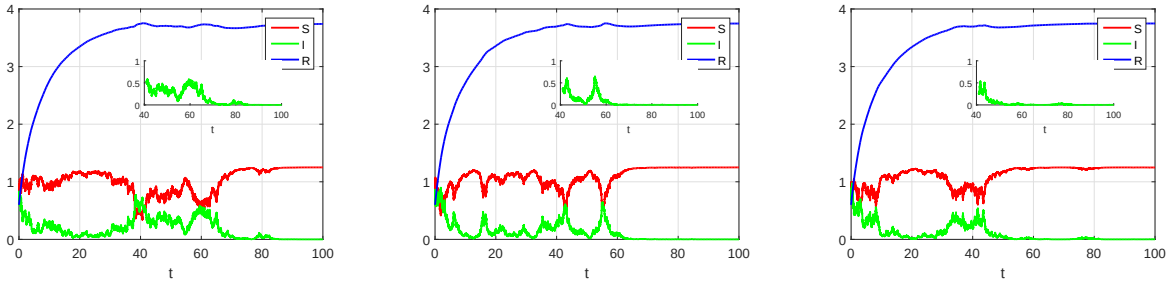
Moreover, the histograms of the probability density function for $I(t)$ are obtained from 10,000 simulation runs for three different noise intensities at $t = 100$, which is displayed in Figure 2(d)–(f). From Figure 2(d)–(f), we can see that the skewness of the distribution for $I(100)$ is changing with the increase of the noise intensity σ. More precisely, the distribution is close to a standard distribution when $\sigma = 0.1$ (see Figure 2(d)). But if increase σ to 0.3 and 0.5, respectively, the distributions are positively skewed (see Figure 2(e)–(f)).

5.2. *Stochastic disease-free dynamics.*

We note that in this paper, the model (2.3) is a continuous time and continuous state space model, the values of $I(t)$ are non-zero quantities increase to 16th decimal during the numerical experiments. Therefore, we assume that 10,000 individuals are deemed to be 1 unit populations approximately, and assume that the disease is regarded as extinction when the value of $I(t)$ less than 0.0001 [4].

To learn the stochastic disease-free dynamics of model (2.3), we choose $\sigma = 0.52$, then $\mathscr{R}_s = 0.9852 < 1$ and $\sigma^2 = 0.2704 < \frac{\beta_1(\mu_0+\nu)}{\Lambda} = 0.4800$. According to Theorem 3.2, the disease goes extinct exponentially almost surely, which is illustrated by Figure 3(a). We increase σ to 0.54 ($\mathscr{R}_s = 0.9642 < 1$, $\sigma^2 = 0.2916 < 0.4800$) and to 0.56 ($\mathscr{R}_s = 0.9434 < 1$, $\sigma^2 = 0.3136 < 0.4800$), respectively. We find that the disease goes to extinction with probability one, as shown in Figure 3(b)-(c).

Furthermore, we conduct 10,000 numerical simulation runs and calculate the mean extinction time of $I(t)$. We obtain that the mean extinction time for the three different noise intensities σ (i.e., 0.52, 0.54, 0.56) is 82.5642, 76.9118, 69.5481, respectively. Then we conclude that the mean extinction time of disease decreases with the increase of noise intensity σ.



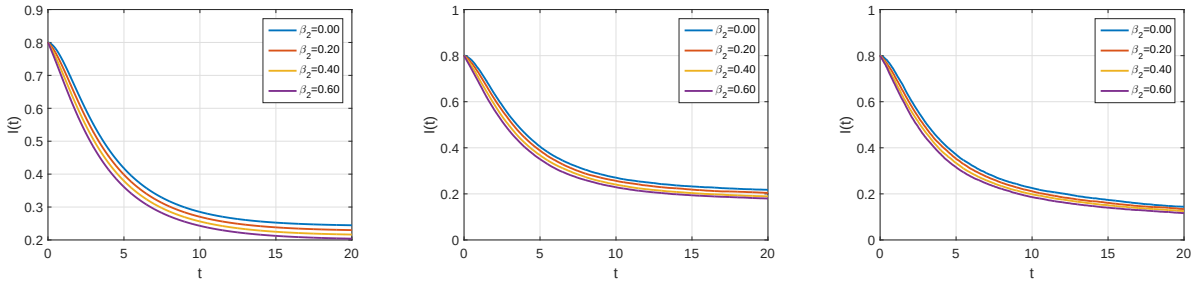
(a) $\sigma = 0.52$ (b) $\sigma = 0.54$ (c) $\sigma = 0.56$

Figure 3. The path of $S(t)$, $I(t)$ and $R(t)$ for model (2.3) with initial $(S(0), I(0), R(0)) = (0.9, 0.8, 0.6)$ under different noise intensities $\sigma = 0.52$, $\sigma = 0.54$ and $\sigma = 0.56$, respectively.

5.3. *Effect of information intervention*

In the following, we show the influence of information intervention. Therefore, we mainly discuss the effect of different values of β_2 on infected population. First, we show the $I(t)$ for deterministic model (2.2) under different values of β_2 ($\beta_2 = 0, 0.2, 0.4, 0.6$) in Figure 4(a). It can be seen from Figure 4(a) that β_2 has great influence on $I(t)$. The number of the infected population decreases with the increase of the β_2.

We next choose noise intensity $\sigma = 0.25$, then make 10,000 numerical simulation runs and calculate mean value. From Figure 4(b), it can be seen that the increase β_2 can reduce the value of $I(t)$. Further, we fix β_2 and compare Figure 4(a) with 4(b), we find that noise intensity σ can also reduce the number of the infected population. If we increase σ to 0.5, then we have a similar conclusion and the simulation result is shown in Figure 4(c). This simulation shows that the information intervention can help reduce the number of the disease outbreak.



(a) $\sigma = 0$ (b) $\sigma = 0.25$ (c) $\sigma = 0.50$

Figure 4. The path of $I(t)$ for model (2.3) with initial $(S(0), I(0), R(0)) = (0.9, 0.8, 0.6)$ under different noise intensities $\sigma = 0.00$, $\sigma = 0.25$ and $\sigma = 0.50$, respectively. Each sub-figure is obtained by the averaging of 10,000 simulation runs.

6. Concluding remarks

Outbreaks of infectious diseases have caused substantial deaths, social and economic losses in the whole world. As a result, a large number of prevention strategies, including information policies, such as media coverage, health education, psychological suggestion, etc. have been used to help to understand the transmission and control of infectious diseases. Environmental noise, an important component in real world, also has greatly affect on the development of infectious diseases. In this paper, we investigated the long term behavior of the stochastic model for the transmission dynamic of hepatitis B with varying population environment. Our major results are as follows:

(i) Using the Markov semigroups theory, we investigated the dynamics of the stochastic hepatitis B model with information intervention perturbed by environment noises. Our results showed that the dynamics of the stochastic hepatitis B model can be governed by the stochastic basic reproduction number $\mathscr{R}_s$. There exists a disease-free equilibrium and the disease is predicted to die out with probability one when $\mathscr{R}_s < 1$ under mild extra condition. If $\mathscr{R}_s > 1$ under mild extra conditions, the stochastic model has an endemic equilibrium and the disease would persist.

(ii) We showed that environmental noises can play an important role in the long-term dynamics. If the intensity of noises is small enough to imply that $\mathscr{R}_s > 1$, which means the disease will prevail and there exists a stationary distribution for the stochastic model. If the intensity of noises is large (leading to $\mathscr{R}_s < 1$), then the disease would be eradicated. Thus, large environmental noises are able to suppress the emergence of the disease outbreak. We further evaluated the influence of information intervention. It leads to the changes in human behavior, which reduces the effective contact rates of susceptible people and provide (temporary) protection from infection. We found that large intensity of information intervention (β_2) can lead to the decrease of $I(t)$ (see Figure 4). Therefore, information intervention can also reduce the number of infected population and suppress the outbreak of hepatitis B.

The environmental white noise considered in this paper is a continuous random process. However, population systems may have sudden impact of various factors, such as volcanoes, toxic pollutants, earthquakes, abrupt climate change, etc. Stochastic models with Brownian motion cannot describe these phenomena. It is worth studying epidemic model with a discontinuous random process (Lévy

noise, Markov noise) by using Markov semigroups theory. On the other hand, we considered the role of information intervention in the control of hepatitis B. We found that information intervention can actually mitigate the spread of hepatitis B and reduce the number of infected population. But we note that these information intervention policies and vaccination often require more costs. How to minimize the infected populations and the costs of these control strategies by considering the optimal control problem? We will study this issue later.

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Conflict of interest

The authors declare there is no conflict of interest.

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