A stochastic model for the transmission dynamics of hepatitis B virus

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\textbf{ABSTRACT}
In this paper, we formulate a stochastic model for hepatitis B virus transmission with the effect of fluctuation environment. We divide the total population into four different compartments, namely, the susceptible individuals in which the disease transmission rate is distributed by white noise, the acutely infected individuals in which the same perturbation occur, the chronically infected individuals and the recovered individuals. We use the stochastic Lyapunov function theory to construct a suitable stochastic Lyapunov function for the existence of positive solution. We also then establish the sufficient conditions for the hepatitis B extinction and the hepatitis B persistence. At the end numerical simulation is carried out by using the stochastic Runge–Kutta method to support our analytical findings.

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\section{1. Introduction}
The environmental variation has a critical influence on the infectious diseases, which is the second leading cause of death in the world [3,14,16]. Biological phenomenon are always effected due to the environmental noise. West Nile virus, Dengue, Malaria and hepatitis B etc., are some of the best examples, for which the nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts and population is subject to a continuous spectrum of disturbances [1]. The contagious disease of hepatitis B causes inflammation of liver results from hepatitis B virus infection in which the epidemics growth and spread are random due to the unpredictability of person-to-person contacts [2]. Together with these realistic patterns, theoretical tactics for taking environmental variation in the epidemiological models deliver a useful framework for discovering the role of environment in population ecology.

Mathematical modelling is an influential tool to describe the dynamical behaviour of various infectious diseases in the real world problem. Several mathematicians and ecologists have developed different epidemic models to realize and control the spread of transmissible diseases in the community. In the past two decades the field of mathematical modelling has been used frequently for the study of communication of different types of infectious diseases (see e.g. [13,15,17,20]). There are two types of epidemic models viz deterministic...
epidemic models and the stochastic epidemic models. But for modelling the biological phenomena stochastic differential equation is more reasonable than deterministic [4]. Stochastic models produce more valuable output than deterministic models as by running a stochastic model several times, we can build up a distribution of the predicted outcomes, e.g. the number of infected classes at time \(t\), whilst a deterministic model will just give a single predicted value [6–8,12,21,22]. Many epidemic models have been discovered for the description of viral dynamic of hepatitis B, which are mostly using the deterministic approach (see for detail [5,9,11,18,23]). Recently Khan et al. [10] presented a stochastic model for the transmission dynamic of hepatitis B.

In the resent study, we proposed a stochastic epidemic model with a varying population environment by extending the work of Khan et al. [10]. Keeping in view the characteristic of hepatitis B, we divided the total population in four different compartments, the susceptible population in which the transmission rate is distributed by white noise, the infected individuals are further divided into two stages, namely the acute, the chronic hepatitis B individuals and the recovered population. For the existence and uniqueness of positive solution to the proposed stochastic hepatitis B model, we use the Lyapunov function theory and construct a suitable stochastic Lyapunov function. We then discuss the hepatitis B extinction and the hepatitis B persistence and derive the sufficient condition for the hepatitis B. Moreover, we carry out the numerical simulation by using the stochastic Runge–Kutta method to support our analytical findings.

2. Stochastic hepatitis B model

In this section, we present the stochastic epidemic model for the transmission dynamic of hepatitis B with varying population environment. We place the following assumption on the model:

\((X_1)\). The total population \(N(t)\) at time \(t\) is divided into four compartments: the susceptible individuals, \(S(t)\), the acutely infected with hepatitis B individuals, \(A(t)\), the chronically infected with hepatitis B individuals, \(C(t)\) and the recovered individuals, \(R(t)\), i.e. \(N(t) = S(t) + A(t) + C(t) + R(t)\) varies with time \(t\).

\((X_2)\). All parameters and state variables of the proposed model are non-negative.

\((X_3)\). The contact of susceptible individuals with acutely and chronically infected hepatitis B individuals primarily leads to acutely infected class.

\((X_4)\). For the effect of randomly fluctuating environment taking, \(\beta_i \rightarrow \beta_i + \eta_i \dot{B}_i(t)\) for \(i = 1,2\), where \(B_i(t)\) is the standard Brownian motion with the property \(B_i(0) = 0\) and with the intensity of white noise \(\eta_i^2 > 0\).

The assumption \((X_1)\)–\((X_4)\) leads to the stochastic hepatitis B epidemic model, which is represented by the following system of four stochastic differential equations:

\[
\begin{align*}
\text{d}S(t) &= \left[ \Lambda - \sum_{i=1}^{2} \beta_i S(t) I_i(t) - (\mu_0 + \nu)S(t) \right] \text{d}t - \sum_{i=1}^{2} \eta_i S(t) I_i(t) \text{d}B(t), \\
\text{d}I_1(t) &= \left[ \sum_{i=1}^{2} \beta_i S(t) I_i(t) - (\mu_0 + \gamma + \gamma_1)I_1(t) \right] \text{d}t + \sum_{i=1}^{2} \eta_i S(t) I_i(t) \text{d}B(t),
\end{align*}
\]
\[ \text{d}I_2(t) = [\gamma_1 I_1(t) - (\mu_0 + \mu_1 + \gamma_2)I_2(t)] \text{d}t, \]
\[ \text{d}R(t) = [\gamma_1 I_1(t) + \gamma_2 I_2(t) + vS(t) - \mu_0 R(t)] \text{d}t, \]  

where \( \Lambda \) represents the per capita constant birth rate. \( \beta_i \) for \( i = 1,2 \) represent the transmission rate of hepatitis B. The natural and disease mortality rates are denoted by \( \mu_1 \) and \( \mu_0 \) respectively. \( v \) represents the vaccination rate of hepatitis B. \( \gamma \) represents the moving rate of acutely infected individuals to chronic stage. \( \gamma_1 \) and \( \gamma_2 \) are respectively described the constant recovery rate of acutely and chronically infected hepatitis B individuals.

### 3. Preliminaries

Let \((\Omega, F, P)\) be a complete probability space with a filtration \(\{F_t\}_{t \geq 0}\) satisfying the usual conditions (i.e. it is increasing and right continuous while \(F_0\) contains all \(P\)-null sets), \(B_i(t)\) (\(i = 1, 2, 3, 4\)) are defined on this complete probability space, we also let \(R^n_+ = \{x \in R^n : x_i > 0, 1 < i \leq n\}\). Consider the \(n\)-dimensional stochastic differential equation

\[ \text{d}x(t) = h(x(t), t) \text{d}t + g(x(t), t) \text{d}B(t) \quad \text{for } t = t_0, \]  

with initial value \(x(0) = x_0 \in R^n\) and \(B(t)\) represents an \(n\)-dimensional standard Brownian motion on the complete probability space \((\Omega, F, \{F_t\}_{t \geq 0}, P)\). The differential operator \(L\) associated with Equation (2) is defined by

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

If \(L\) acts on a function \(V \in C^{2,1}(R^n \times [t_0, \infty]; R_+)\), then

\[ LV(x, t) = V_t(x, t) + V_x(x, t)h(x, t) + \frac{1}{2} \text{trace} \left[ g^T(x, t) V_{xx} (x, t) g(x, t) \right], \]  

where

\[ V_t = \frac{\partial V}{\partial t}, \quad V_x = \left( \frac{\partial V}{\partial x_1}, \frac{\partial V}{\partial x_2}, \ldots \right), \quad V_{xx} = \left( \frac{\partial^2 V}{\partial x_i \partial x_j} \right)_{d \times d}. \]  

Using the Itô formula, if \(x(t) \in R^n\), then

\[ \text{d}V(x, t) = LV(x(t), t) \text{d}t + V_x(x(t), t) g(x(t), t) \text{d}B(t). \]  

### 4. Existence and uniqueness

In this section, we discuss the solution of the stochastic hepatitis B model (1).

**Theorem 4.1:** The solution \((S(t), I_1(t), I_2(t), R(t))\) of the proposed stochastic hepatitis B epidemic model (1) is unique on \(t \geq 0\) for any initial value \((S(0), I_1(0), I_2(0), R(0)) \in R^n_+\), and the solution will remain in \(R^n_+\) with probability one, namely, \((S(t), I_1(t), I_2(t), R(t)) \in R^n_+\) for all \(t \geq 0\) a.s. (almost surely).
The solution of Equation (10) yields $\tau_c = \infty$ a.s., then sufficiently large, so that $S(0)$, $I_1(0)$, $I_2(0)$ and $R(0)$ all lie within the interval $[1/(k_0), k_0]$. For each integer $k \geq k_0$, define the stopping time

$$
\tau_k = \left\{ t \in [0, \tau_c) : \min\{S(t), I_1(t), I_2(t), R(t)\} \leq \frac{1}{k} \text{ or } \max\{S(t), I_1(t), I_2(t), R(t)\}\right\}. 
$$

Proof: The co-efficient of the equation is locally Lipschitz continuous for any given initial size of population $(S(0), I_1(0), I_2(0), R(0)) \in R_+^4$, there is a unique local solution $(S(t), I_1(t), I_2(t), R(t))$ on $t \in [0, \tau_c)$, where $\tau_c$ is the explosion time (for detail see references [4,5]). To show that this solution is global, we prove that $\tau_c = \infty$ a.s. Let $k_0 \geq 0$ be sufficiently large, so that $S(0)$, $I_1(0)$, $I_2(0)$ and $R(0)$ all lie within the interval $[1/(k_0), k_0]$. Hence there is an integer $k_1 \geq k_0$, such that

$$
P(\tau_{k_1} \leq \tau_c) > \epsilon. \tag{8}
$$

Hence there is an integer $k_1 \geq k_0$, such that

$$
P(\tau_{k_1} \leq \tau_c) \geq \epsilon \quad \text{for all } k \geq k_1. \tag{9}
$$

Let $N(t) = S(t) + I_1(t) + I_2(t) + R(t)$, then for $t \leq \tau_c$, we can see that

$$
dN(t) = \left(\Lambda - \mu_0 N(t) - \mu_1 I_2(t)\right) dt \leq \left(\Lambda - \mu_0 N(t)\right) dt. \tag{10}
$$

The solution of Equation (10) yields

$$
N(t) \leq \begin{cases}
\frac{\Lambda}{\mu_0} & \text{if } N(0) \leq \frac{\Lambda}{\mu_0}, \\
N(0) & \text{if } N(0) > \frac{\Lambda}{\mu_0},
\end{cases} \tag{11}
$$

Now, we define a $C^2$-function $F : R_+^4 \to R_+$, such that

$$
F(S, I_1, I_2, R) = S + I_1 + I_2 + R - 3 - \left(\log S + \log I_1 + \log I_2 + \log R\right). \tag{12}
$$

Clearly the function $F$ is non-negative, which can be seen from $u - 1 - \log u \geq 0$, for all $u > 0$. Let $k \geq k_0$ and $T \geq 0$ be arbitrary. Applying the Ito formula on Equation (12), we get

$$
dF(S, I_1, I_2, R) = \left(1 - \frac{1}{S}\right) dS + \frac{1}{2S^2} (dS)^2 + \left(1 - \frac{1}{I_1}\right) dI_1 + \left(1 - \frac{1}{I_2}\right) dI_2
$$

$$
+ \frac{1}{2} \sum_{i=1}^{2} \frac{1}{I_i^2} (dI_i)^2 + \left(1 - \frac{1}{R}\right) dR,
$$

$$
= LF(S, I_1, I_2, R) dt + \sum_{i=1}^{2} \eta_i (I_i - S) dB_i(t), \tag{13}
$$
where $LF : \mathbb{R}_+^4 \to \mathbb{R}_+$ is defined by the following equation:

$$
LF(S, I_1, I_2, R) = \left(1 - \frac{1}{S}\right) \left(\Lambda - \frac{1}{2} \sum_{i=1}^{2} \beta_i S I_i - (\mu_0 + v) S\right) + \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 I_i^2
$$

$$
+ \left(1 - \frac{1}{I_1}\right) \left(\frac{1}{2} \sum_{i=1}^{2} \beta_i S I_i - (\mu_0 + \gamma + \gamma_1) I_1\right) + \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 S^2
$$

$$
+ \left(1 - \frac{1}{I_2}\right) (\gamma I_1 - (\mu_0 + \mu_1 + \gamma_2) I_2)
$$

$$
+ \left(1 - \frac{1}{R}\right) (\gamma_1 I_1 + \gamma_2 I_2 + v S - \mu_0 R),
$$

$$
= \Lambda + 4\mu_0 + v + \gamma + \gamma_1 + \mu_1 + \gamma_2 + \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 (S^2 + I_i^2) + \frac{1}{2} \sum_{i=1}^{2} \beta_i I_i
$$

$$
- \mu_0 N - \frac{\Lambda}{S} - \frac{1}{2} \sum_{i=1}^{2} \beta_i S I_i - \mu_1 I_2 - \gamma \frac{I_1}{I_2} - \gamma_1 \frac{I_1}{R} - \gamma_2 \frac{I_2}{R} - v \frac{S}{R},
$$

$$
\leq \Lambda + 4\mu_0 + v + \gamma + \gamma_1 + \mu_1 + \gamma_2 + \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 (S^2 + I_i^2) + \frac{1}{2} \sum_{i=1}^{2} \beta_i I_i,
$$

$$
\leq \Lambda + 4\mu_0 + v + \gamma + \gamma_1 + \mu_1 + \gamma_2 + \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 (S^2 + I_i^2 + R^2)
$$

$$
+ \frac{1}{2} \sum_{i=1}^{2} \beta_i (S + I_i + R),
$$

$$
\leq \Lambda + 4\mu_0 + v + \gamma + \gamma_1 + \mu_1 + \gamma_2 + \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 M^2 + \frac{1}{2} \sum_{i=1}^{2} \beta_i M := K.
$$

(14)

Therefore

$$
E [F(S(\tau_k \wedge T), I_1(\tau_k \wedge T), I_2(\tau_k \wedge T), R(\tau_k \wedge T))] \leq F(S(0), I_1(0), I_2(0), R(0))
$$

$$
+ E \left[ \int_0^{\tau_k \wedge T} K \, dt \right],
$$

$$
\leq F(S(0), I_1(0), I_2(0), R(0)) + KT.
$$

(15)

Setting $\Omega_k = \tau_k \leq T$ for $k \geq k_1$ and by Equation (9), $P(\Omega_k) \geq \epsilon$. Note that for every $\omega \in \Omega_k$, there exist at least one $S(\tau_k, \omega), I_1(\tau_k, \omega), I_2(\tau_k, \omega)$ that equal $k$ or $1/k$, and hence $F(S(\tau_k), I_1(\tau_k), I_2(\tau_k), R(\tau_k))$ is no less then $k - 1 - \log k$ or $(1/k) - 1 + \log k$.

Consequently

$$
F(S(\tau_k), I_1(\tau_k), I_2(\tau_k), R(\tau_k)) \geq E(k - 1 - \log k) \wedge \left(\frac{1}{k} - 1 + \log k\right).
$$

(16)
It is then follows from Equations (9) and (15) that

\[ F(S(0), I_1(0), I_2(0), R(0)) + KT \geq E \left[ 1_{\Omega_{\omega}} F(S(\tau_k), I_1(\tau_k), I_2(\tau_k), R(\tau_k)) \right] \]

\[ \geq e \left[ (k - 1 - \log k) \land \left( \frac{1}{k} - 1 + \log k \right) \right], \quad (17) \]

where \( 1_{\Omega_{\omega}} \) is the indicator function of \( \Omega \). Letting \( k \to \infty \) leads to the contradiction \( \infty > F(S(0), I_1(0), I_2(0), R(0)) + MT = \infty \), which implies that \( \tau_\infty = \infty \) a.s.

**Theorem 4.2:** For any initial value \((S(0), I_1(0), I_2(0), R(0)) \in R_+^4\), the solution of the proposed model (1) will remain in \( R_+^4 \) with probability one namely \((S(t), I_1(t), I_2(t), R(t)) \in R_+^4\) for all \( t \geq 0 \) a.s. (almost surely).

**Proof:** Let \( I \subset [0, +\infty) \) and assume that the solution of the proposed stochastic hepatitis B epidemic model (1) exist in \( I \), then for all \( t \in I \), solution of the first equation of system (1) becomes

\[ S(t) = e^{-(\mu_0 + \gamma)t} - \int_0^t \left( \sum_{i=1}^2 \beta_i I_i(u) + 1/2 \sum_{i=1}^2 \gamma_i^2 I_i^2(u) \right) du - \sum_{i=1}^2 \int_0^t \gamma_i I_i(u) dB_i(u) \]

\[ \times \left[ S(0) + \Lambda \int_0^t e^{(\mu_0 + \gamma)u} \left( \sum_{i=1}^2 \beta_i I_i(u) + 1/2 \sum_{i=1}^2 \gamma_i I_i^2(u) \right) dv + \sum_{i=1}^2 \int_0^u \gamma_i I_i(v) dB_i(v) \right] \]

\[ > 0. \quad (18) \]

Next, we investigate the solution of the second equation of system (1), which looks like

\[ I_1(t) = e^{-(\mu_0 + \gamma_1) t} - \int_0^t \left( \sum_{i=1}^2 \beta_i I_i(u) - 1/2 \sum_{i=1}^2 \gamma_i^2 S^2(u) \right) du + \sum_{i=1}^2 \int_0^t \gamma_i S(u) dB_i(u) \]

\[ \times \left[ I_1(0) + \int_0^t \sum_{i=1}^2 \beta_i S(u) e^{(\mu_0 + \gamma_1) s - \int_0^s (\sum_{i=1}^2 \beta_i S(u) + 1/2 \sum_{i=1}^2 \gamma_i^2 S^2(u)) du - \sum_{i=1}^2 \int_0^u \gamma_i S(u) dB_i(u) \right] \]

\[ ds \geq 0. \quad (19) \]

Similarly it can be shown that \( I_2(t) \geq 0 \) and \( R(t) > 0 \). Hence \((S(t), I_1(t), I_2(t), R(t)) \in R_+^4\), for all \( t \geq 0 \).

**Remark 4.1:** It is clear from Theorems 4.1 and 4.2 that for any initial value \((S(0), I_1(0), I_2(0), R(0)) \in R_+^4\), there is a unique global solution \((S(t), I_1(t), I_2(t), R(t)) \in R_+^4\) a.s., of model (1). Hence

\[ d(S + I_1 + I_2 + R) \leq (\Lambda - \mu_0(S + I_1 + I_2 + R)). \quad (20) \]

Solution of Equation (20) yields

\[ (S(t) + I_1(t) + I_2(t) + R(t)) \leq \frac{\Lambda}{\mu_0} + e^{-\mu_0 t} \left( S(0) + I_1(0) + I_2(0) + R(0) - \frac{\Lambda}{\mu_0} \right) \]

\[ (\Lambda - \mu_0(S(t) + I_1(t) + I_2(t) + R(t))). \quad (21) \]
If \( S(0) + I_1(0) + I_2(0) + R(0) \leq \Lambda / \mu_0 \), then \( S(t) + I_1(t) + I_2(t) + R(t) \leq \Lambda / \mu_0 \), a.s., then the region

\[
\Omega^* = \left\{ (S, I_1, I_2, R) : S > 0, I_1 \geq 0, I_2 \geq 0, R > 0, S + I_1 + I_2 + R \leq \frac{\Lambda}{\mu_0} \right\}
\]

is a positively invariant set of system (1).

### 5. Extinction and persistence

In this section, we derive the condition for the extinction and persistence of the hepatitis B. For convenience, we introduce the following notation and definition. Let

\[
\langle x(t) \rangle = \frac{1}{t} \int_0^t x(r) dr.
\]

The parameter \( R_0 \) is defined to be the basic reproduction number \( R_0 \) for the proposed stochastic hepatitis B model (1), which is given by the following equation:

\[
R_0 = R_1 + R_2,
\]

where

\[
R_1 = \sum_{i=1}^{2} \left( \frac{\beta_i \Lambda}{(\mu_0 + v) \left( \mu_0 + \gamma + \gamma_1 + \frac{\eta_1^2 \Lambda^2}{2(\mu_0 + v)^2} \right)} \right),
\]

\[
R_2 = \sum_{i=1}^{2} \left( \frac{\beta_i \Lambda}{(\mu_0 + v) \left( \mu_0 + \mu_1 + \gamma_2 + \frac{\eta_2^2 \Lambda^2}{2(\mu_0 + v)^2} \right)} \right).
\]

**Lemma 5.1 (Strong law of large number, [19]):** Let \( M = \{M_t\}_{t \geq 0} \) be a real valued continuous local martingale vanishing at \( t = 0 \), then

\[
\lim_{t \to \infty} \langle M, M \rangle_t = \infty \quad \text{a.s. implies that} \quad \lim_{t \to \infty} \frac{M_t}{\langle M, M \rangle_t} = 0 \quad \text{a.s. and also,}
\]

\[
\lim_{t \to \infty} \sup_{t} \frac{\langle M, M \rangle_t}{t} < 0 \quad \text{a.s. implies that} \quad \lim_{t \to \infty} \frac{M_t}{t} = 0 \quad \text{a.s.}
\]

**Definition 5.1 ([19]):** The proposed stochastic hepatitis B model (1) is said to be persistent in mean, if

\[
\lim_{t \to \infty} \inf_{t} \int_0^t I_1(r) dr > 0 \quad \text{a.s.} \quad \lim_{t \to \infty} \inf_{t} \int_0^t I_2(r) dr > 0 \quad \text{a.s.}
\]

**Lemma 5.2 ([19]):** Let \( f \in C([0, \infty) \times \Omega_1([0, \infty]) \) and \( F(t) \in C([0, \infty) \times \Omega, R) \). If there exist positive constants \( \lambda_0, \lambda \) and \( T \), such that

\[
\log f(t) \leq \lambda t - \lambda_0 \int_0^t f(s) ds + F(t) \quad \text{a.s. for all} \quad t \geq T \quad \text{and}
\]

\[
\lim_{t \to \infty} \frac{F(t)}{t} = 0 \quad \text{a.s. then} \quad \lim_{t \to \infty} \sup_{t} \frac{1}{t} \int_0^t f(s) ds \leq \frac{\lambda}{\lambda_0} \quad \text{a.s.}
\]
5.1. Extinction

For the extinction of the disease, we have the following result.

**Theorem 5.1:** Let \((S(t), I_1(t), I_2(t), R(t))\) be the solution of the proposed hepatitis B model (1) with initial value \((S(0), I_1(0), I_2(0), R(0)) \in \Omega^*\). If

\((a) R_1 < 1 \text{ and } \sum_{i=1}^{2} \beta_i (\mu_0 + v) > \sum_{i=1}^{2} \eta_i i, \text{ then}

\[
\lim_{t \to \infty} \left( \frac{\log I_1(t)}{t} \right) < 0 \text{ and } \lim_{t \to \infty} \left( \frac{\log I_2(t)}{t} \right) < 0,
\]

a.s. (namely \(I_1(t) \to 0 \text{ and } I_2(t) \to 0\) exponentially a.s., i.e. the hepatitis B infection dies out with probability one). In addition

\[
\lim_{t \to \infty} S(t) = \frac{\Lambda}{\mu_0 + v},
\]

\[
\lim_{t \to \infty} I_1(t) = 0, \quad \lim_{t \to \infty} I_2(t) = 0, \quad \lim_{t \to \infty} R(t) = \frac{v \Lambda}{\mu_0 (\mu_0 + v)}.
\]

**Proof:** The integration of the proposed stochastic hepatitis B model (1) leads to the following system of equations:

\[
\frac{S(t) - S(0)}{t} = \Lambda - \sum_{i=1}^{2} \beta_i \langle S(t)I_i(t) \rangle - (\mu_0 + v) \langle S(t) \rangle - \sum_{i=1}^{2} \eta_i t \int_{0}^{t} S(r)I_i(r) \, dB_i(r),
\]

\[
\frac{I_1(t) - I_1(0)}{t} = \sum_{i=1}^{2} \beta_i \langle S(t)I_i(t) \rangle - (\mu_0 + \gamma + \gamma_1) \langle I_1(t) \rangle + \sum_{i=1}^{2} \eta_i t \int_{0}^{t} S(r)I_i(r) \, dB_i(r),
\]

\[
\frac{I_2(t) - I_2(0)}{t} = \gamma \langle I_1(t) \rangle - (\mu_0 + \mu_1 + \gamma_2) \langle I_2(t) \rangle,
\]

\[
\frac{R(t) - R(0)}{t} = \gamma_1 \langle I_1(t) \rangle + \gamma_2 \langle I_2(t) \rangle + v \langle S(t) \rangle - \mu_0 \langle R(t) \rangle.
\]

We compute from Equation (28) that

\[
\frac{S(t) - S(0)}{t} + \frac{I_1(t) - I_1(0)}{t} + \frac{I_2(t) - I_2(0)}{t} = \Lambda - (\mu_0 + v) \langle S(t) \rangle - (\mu_0 + \gamma) \langle I_1(t) \rangle - (\mu_0 + \mu_1 + \gamma_2) \langle I_2(t) \rangle,
\]

\[
\langle S(t) \rangle = \frac{\Lambda}{\mu_0 + v} - \frac{(\mu_0 + \gamma)}{(\mu_0 + v)} \langle I_1(t) \rangle - \frac{(\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)} \langle I_2(t) \rangle + \phi_1(t),
\]
where \( \phi_1(t) \) is defined by the following equation:

\[
\phi_1(t) = - \frac{1}{\mu_0 + v} \left[ \frac{S(t) - S(0)}{t} + \frac{I_1(t) - I_1(0)}{t} + \frac{I_2(t) - I_2(0)}{t} \right].
\]

Obviously \( \phi_1(t) \to 0 \) as \( t \to \infty \). Applying the Ito formula to the second equation of model (1), which yields

\[
d\log I_1(t) = \left[ \sum_{i=1}^{2} \beta_i S(t) - (\mu_0 + \gamma + \gamma_1) - \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 S^2(t) \right] dt + \sum_{i=1}^{2} \eta_i S(t) dB_i(t).
\]

Integrating Equation (30) from 0 to \( t \) and dividing by \( t \), which leads to the following equation:

\[
\frac{\log I_1(t) - \log I_1(0)}{t} = \sum_{i=1}^{2} \beta_i \langle S(t) \rangle - (\mu_0 + \gamma + \gamma_1) - \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 \langle S^2(t) \rangle
\]

\[
+ \sum_{i=1}^{2} \eta_i \int_0^t S(r) dB_i(r),
\]

\[
\leq \sum_{i=1}^{2} \beta_i \langle S(t) \rangle - (\mu_0 + \gamma + \gamma_1) - \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 \langle S(t) \rangle^2
\]

\[
+ \sum_{i=1}^{2} \eta_i \int_0^t S(r) dB_i(r).
\]

Substituting the last equation of Equation (29) in the last inequality of Equation (31) and by the use of \( M_i(t) = \eta_i \int_0^t S(r) dB_i(r) \) for \( i = 1, 2 \), which is a local continuous martingale and \( M_i(0) = 0 \), yields

\[
\frac{\log I_1(t) - \log I_1(0)}{t} \leq \sum_{i=1}^{2} \beta_i \left( \frac{\Lambda}{(\mu_0 + v)} - \frac{\mu_0 + \gamma}{(\mu_0 + v)} \langle I_1(t) \rangle - \frac{\mu_0 + \mu_1 + \gamma_2}{(\mu_0 + v)} \langle I_2(t) \rangle + \phi_1(t) \right)
\]

\[
- \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 \left( \frac{\Lambda}{(\mu_0 + v)} - \frac{\mu_0 + \gamma}{(\mu_0 + v)} \langle I_1(t) \rangle - \frac{\mu_0 + \mu_1 + \gamma_2}{(\mu_0 + v)} \langle I_2(t) \rangle \right)
\]

\[
+ \phi_1(t) \right)^2 - (\mu_0 + \gamma + \gamma_1) + \frac{1}{t} \sum_{i=1}^{2} M_i(t),
\]

\[
\leq \sum_{i=1}^{2} \frac{\beta_i \Lambda}{(\mu_0 + v)} - \left( \mu_0 + \gamma + \gamma_1 + \frac{1}{2} \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{(\mu_0 + v)^2} \right)
\]

\[
- \sum_{i=1}^{2} \left( \frac{\beta_i (\mu_0 + \gamma_1)}{(\mu_0 + v)} - \frac{\eta_i^2 \Lambda (\mu_0 + \gamma_1)}{(\mu_0 + v)^2} \right) \langle I_1(t) \rangle.
\]
\[- \sum_{i=1}^{2} \left( \frac{\beta_i(\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)} - \frac{\eta_i^2 \Lambda(\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)^2} \right) \langle I_2(t) \rangle \]

\[+ \Phi_1(t) + \frac{1}{t} \sum_{i=1}^{2} M_i(t), \]

\[= \left( \mu_0 + \gamma + \gamma_1 + \frac{1}{2} \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda}{(\mu_0 + v)^2} \right) (1 - R_1) \]

\[- \sum_{i=1}^{2} \left( \frac{\beta_i(\mu_0 + \gamma_1)}{(\mu_0 + v)} - \frac{\eta_i^2 \Lambda(\mu_0 + \gamma_1)}{(\mu_0 + v)^2} \right) \langle I_1(t) \rangle \]

\[- \sum_{i=1}^{2} \left( \frac{\beta_i(\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)} - \frac{\eta_i^2 \Lambda(\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)^2} \right) \langle I_2(t) \rangle \]

\[+ \Phi_1(t) + \frac{1}{t} \sum_{i=1}^{2} M_i(t), \quad (32) \]

where

\[\Phi_1(t) = \sum_{i=1}^{2} \beta_i \phi(t) - \phi^2(t) - \sum_{i=1}^{2} \eta_i^2 \frac{\Lambda}{\mu_0 + v} \langle I_2(t) \rangle \phi_1(t) \]

\[+ \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda \phi(t)}{(\mu_0 + v)} + \sum_{i=1}^{2} \frac{\eta_i^2 (\mu_0 + \gamma) \phi_1(t)}{(\mu_0 + v)} \langle I_1(t) \rangle. \quad (33)\]

Moreover

\[\lim_{t \to \infty} \sup \sum_{i=1}^{2} \frac{(M_i \Lambda_1)_t}{t} \leq \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{(\mu_0 + v)^2} < \infty \quad \text{a.s.} \quad (34)\]

By Lemma 5.1 and the use of \(\phi_1(t) = 0\) as \(t \to \infty\), we obtain

\[\lim_{t \to \infty} \sup \sum_{i=1}^{2} \frac{M_i(t)}{t} = 0 \quad \text{and} \quad \lim_{t \to \infty} \Phi_1(t) = 0 \quad \text{a.s.} \quad (35)\]

If the condition (a) is satisfied, it follows from the last inequality of Equation (32) and taking the limit superior of both sides, then

\[\lim_{t \to \infty} \sup \frac{\log I_1(t)}{t} \leq - \left( \mu_0 + \gamma + \gamma_1 + \frac{1}{2} \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{(\mu_0 + v)^2} \right) (1 - R_1) \]

\[- \sum_{i=1}^{2} \left( \frac{\beta_i(\mu_0 + \gamma_1)}{(\mu_0 + v)} - \frac{\eta_i^2 \Lambda(\mu_0 + \gamma_1)}{(\mu_0 + v)^2} \right) \langle I_1(t) \rangle \]

\[- \sum_{i=1}^{2} \left( \frac{\beta_i(\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)} - \frac{\eta_i^2 \Lambda(\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)^2} \right) \langle I_2(t) \rangle. \quad (36)\]
Equation (36) implies that
\[
\lim_{t \to \infty} I_1(t) = 0.
\]
Similarly it can be shown that
\[
\lim_{t \to \infty} I_2(t) = 0.
\]
Next, we prove the assertion (27), so from the proposed stochastic hepatitis B model (1), we have
\[
\frac{d}{dt}(S + I_1 + I_2 + R) = \left(\Lambda - \mu_0(S + I_1 + I_2 + R) - \mu_1 I_2\right) dt. \hspace{1cm} (37)
\]
The solution of Equation (37) yields that
\[
S(t) + I_1(t) + I_2(t) + R(t) = e^{-\mu_0 t} \left(S(0) + I_1(0) + I_2(0) + R(0) + \int_0^t (\Lambda - \mu_1 I_2(s)) e^{\mu_0 s} ds\right). \hspace{1cm} (38)
\]
Applying the L'Hospital rule together with the application of Equation (37), we obtain
\[
\lim_{t \to \infty} (S(t) + R(t)) = \frac{\Lambda}{\mu_0 + v} \text{ a.s.} \hspace{1cm} (39)
\]
According to the model (1), the first equation with limiting system becomes
\[
\frac{dS(t)}{dt} = (\Lambda - (\mu_0 + v)S(t)) dt.
\]
So, we obtain
\[
\lim_{t \to \infty} S(t) = \frac{\Lambda}{\mu_0 + v} \text{ a.s.}
\]
Similarly, we obtain
\[
\lim_{t \to \infty} R(t) = \frac{v \Lambda}{\mu_0(\mu_0 + v)} \text{ a.s.}
\]
5.2. Persistence

For the persistence of the disease, we have the following result.

**Theorem 5.2:** If

(a) \( R_1 > 1 \) and \( \beta_1 (\mu_0 + v) > \eta_1^2 \Lambda \), then

for any initial value \((S(0), I_1(0), I_2(0), R(0)) \in \Omega^*\), the solution \((S(t), I_1(t), I_2(t), R(t))\) of the proposed model (1) has the following property:

\[
I_1^* \leq \lim_{t \to \infty} \inf \langle I_1(t) \rangle \leq \lim_{t \to \infty} \sup \langle I_1(t) \rangle \leq I_1^* \quad \text{a.s.,}
\]

where

\[
I_1^* = \frac{\left( \mu_0 + v \right)^2 \left( \mu_0 + \gamma + \gamma_1 + \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{(\mu_0 + v)^2} \right) (R_1 - 1)}{(\mu_0 + \gamma_1) \left( \sum_{i=1}^{2} \beta_i (\mu_0 + v) - \eta_i^2 \Lambda \right)},
\]

\[
I_1^* = \frac{\left( \mu_0 + v \right) \left( \mu_0 + \gamma + \gamma_1 + \frac{1}{2} \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{\beta_i (\mu_0 + \gamma + \gamma_1)} \right) (R_1 - 1)}{\sum_{i=1}^{2} \beta_i (\mu_0 + \gamma + \gamma_1)}.
\]

**Proof:** Again, we compute from Equation (29) that

\[
\frac{S(t) - S(0)}{t} + \frac{I_1(t) - I_1(0)}{t} = \Lambda - (\mu_0 + v) \langle S(t) \rangle - (\mu_0 + \gamma + \gamma_1) \langle I_1(t) \rangle,
\]

\[
\langle S(t) \rangle = \frac{\Lambda}{\mu_0 + v} - \frac{(\mu_0 + \gamma + \gamma_1)}{(\mu_0 + v)} \langle I_1(t) \rangle + \phi_2(t),
\]

where \( \phi_2(t) \) is defined by the following equation:

\[
\phi_2(t) = -\frac{1}{\mu_0 + v} \left[ \frac{S(t) - S(0)}{t} + \frac{I_1(t) - I_1(0)}{t} \right].
\]

Using Equation (41) in Equation (31), we obtain

\[
\frac{\log I_1(t) - \log I_1(0)}{t} \leq \left( \mu_0 + \gamma + \gamma_1 + \frac{1}{2} \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{(\mu_0 + v)^2} \right) (R_1 - 1)
\]

\[
- \sum_{i=1}^{2} \left( \frac{\beta_i (\mu_0 + \gamma_1)}{(\mu_0 + v)} - \frac{\eta_i^2 \Lambda (\mu_0 + \gamma_1)}{(\mu_0 + v)^2} \right) \langle I_1(t) \rangle
\]

\[
- \sum_{i=1}^{2} \left( \frac{\beta_i (\mu_0 + \mu_1 + \gamma_2)}{(\mu_0 + v)} \right)
\]

\[
+ \Phi_2(t) + \frac{1}{t} \sum_{i=1}^{2} M_i(t),
\]

\[\text{(40)}\]
Using the second equation of Equation (41) in Equation (45), we get

\[
\log I_1(t) - \log I_1(0) \geq \left( \mu_0 + \gamma + \gamma_1 + \frac{1}{2} \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{(\mu_0 + v)^2} \right) (R_1 - 1) - \sum_{i=1}^{2} \beta_i (\mu_0 + \gamma + \gamma_1) (\mu_0 + v) I_1(t) + \phi_2(t) + \frac{1}{t} \sum_{i=1}^{2} M_i(t).
\]

The inequalities (42) can be re-written as

\[
\langle I_1(t) \rangle \leq \frac{(\mu_0 + v)^2 \left( \mu_0 + \gamma + \gamma_1 + \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{2(\mu_0 + v)^2} \right) (R_1 - 1)}{(\mu_0 + \gamma_1) \left( \sum_{i=1}^{2} (\beta_i (\mu_0 + v) - \eta_i^2 \Lambda) \right)} + \sum_{i=1}^{2} \frac{M_i(t)}{t} + \frac{\log I_1(0)}{t} - \frac{\log I_1(t)}{t} + \Phi_2(t).
\]

For \( R_1 > 1, \beta_i (\mu_0 + v) > \eta_i^2 \Lambda \) together with Lemma 5.2 and by the use of Equation (34), then taking the supremum, we get

\[
\lim_{t \to \infty} \sup \langle I_1(t) \rangle \leq \frac{(\mu_0 + v)^2 \left( \mu_0 + \gamma + \gamma_1 + \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{2(\mu_0 + v)^2} \right) (R_1 - 1)}{(\mu_0 + \gamma_1) \left( \sum_{i=1}^{2} (\beta_i (\mu_0 + v) - \eta_i^2 \Lambda) \right)} = I_1^*.
\]

Now on the other hand

\[
\frac{\log I_1(t) - \log I_1(0)}{t} = \sum_{i=1}^{2} \beta_i \langle S(t) \rangle - (\mu_0 + \gamma + \gamma_1) - \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 \langle S^2(t) \rangle + \frac{2}{t} \int_0^t S(r) dB_i(r),
\]

\[
\geq \sum_{i=1}^{2} \beta_i \langle S(t) \rangle - (\mu_0 + \gamma + \gamma_1) - \frac{1}{2} \sum_{i=1}^{2} \eta_i^2 \frac{\Lambda^2}{(\mu_0 + v)^2} + \frac{2}{t} \int_0^t S(r) dB_i(r).
\]
The inequality (46) with the application of Equation (35) and then taking the limit inferior of both side leads to the following equation:

$$\lim_{t \to \infty} \inf \langle I_1(t) \rangle \geq \frac{(\mu_0 + v) \left( \mu_0 + \gamma + \gamma_1 + \frac{1}{2} \sum_{i=1}^{2} \frac{\eta_i^2 \Lambda^2}{\beta_i (\mu_0 + \gamma + \gamma_1)} \right)}{\sum_{i=1}^{2} \beta_i (\mu_0 + \gamma + \gamma_1)} (R_1 - 1) = I_{1*}. \quad (47)$$

Thus from Equations (44) and (47), we have the assertion (40), i.e.

$$I_{1*} \leq \lim_{t \to \infty} \inf \langle I_1(t) \rangle \leq \lim_{t \to \infty} \sup \langle I_1(t) \rangle \leq I_1^* \quad \text{a.s.}$$
Figure 2. The plot represents the simulation of the proposed model (1) for parameter values $\Lambda = 0.5$, $\beta_1 = 0.6$, $\beta_2 = 0.5$, $\mu_0 = 0.1$, $v = 0.3$, $\eta_1 = 0.5$, $\eta_2 = 0.6$, $\gamma = 0.1$, $\gamma_1 = 0.4$, $\mu_1 = 0.02$. Using the stochastic Runge–Kutta method with step size $\Delta t = 0.01$ and initial values $(S(0), I_1(0), I_2(0), R(0)) = (0.9, 0.4, 0.2, 0.1)$: (a) susceptible population, (b) acutely infected population, (c) chronically infected population.

6. Numerical simulation

In this section, the stochastic Runge–Kutta method is used to find the numerical simulation of the proposed stochastic hepatitis B epidemic model (1) for the verification of our analytical findings. Clearly, we observe the influence of noise intensity on the hepatitis B transmission. We perform simulation of the model, see Figures 1 and 2. For the verification of the hepatitis B extinction, i.e. Theorem 5.1, we assume the parameter values as $\Lambda = 0.5$, $\beta_1 = 0.6$, $\beta_2 = 0.5$, $\mu_0 = 0.1$, $v = 0.4$, $\gamma = 0.1$, $\gamma_1 = 0.4$, $\gamma_2 = 0.3$, $\mu_1 = 0.02$, $\eta_1 = 0.9$, $\eta_2 = 0.8$. Moreover, the time interval is taken to be $0---50$ unit with step size 0.01. It is easy to ensure the condition (a) i.e. $R_1 = 0.8301886792 < 1$, $\sum_{i=1}^{2} \beta_i (\mu_0 + v) = 0.55 > 0.18 = \sum_{i=1}^{2} \eta_i^2 \Lambda$ and therefore, the acutely infected with hepatitis B and chronically infected with hepatitis B population exponentially tends to zero with probability
one as shown in Figure 1(b,c). This indicates the extinction of the hepatitis B. Similarly, in the case of persistence the parameter values chosen are \( \Lambda = 0.5, \beta_1 = 0.6, \beta_2 = 0.5, \mu_0 = 0.1, v = 0.3, \mu_1 = 0.02, \gamma = 0.1, \gamma_1 = 0.4, \gamma_2 = 0.3, \eta_1 = 0.5 \) and \( \eta_1 = 0.6 \), which ensure the condition for the hepatitis B persistent, i.e. Theorem 5.2 holds. In other words, we have \( R_1 = 1.277213353 > 1, \beta_1(\mu_0 + v) = 0.24 > \eta_1^2 \Lambda = 0.0625 \). Thus the model (1) maintains the persistence (see Figure 2).

7. Conclusion

Real world problems are not deterministic but including stochastic effect. In this paper, we formulated the stochastic epidemic model for the hepatitis B virus transmission and studied the dynamic with varying population environment. We then showed the existence of positive solution and established a suitable stochastic Lyapunov function by using the concept of Lyapunov functions theory. We also discussed the extinction as well as the persistence and derived the sufficient condition for the hepatitis B extinction and the hepatitis B persistence. The conditions of disease extinction and disease persistence are in the form of expressions involving the system parameters and intensities of noise terms. Clearly, we observed the influence of noise intensity on the hepatitis B transmission. The extinction of the hepatitis B infected individuals increasing with the increasing noise strength on the susceptible population. Similarly the disease persisting decreases with the increasing noise strength. Finally, we performed the numerical simulation for supporting our analytical findings. Our work shows that stochastic epidemic models give another option to model the phenomena of viral dynamic, which give us a more realistic way.

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