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# Persistence and Extinction for Stochastic HBV Epidemic Model with Treatment Cure Rate

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ABSTRACT: With the current struggles of the world nowadays with several epidemics, modeling the dynamics of disease outbreaks has become much more important than any time before. In this context, the present paper aims at studying a stochastic hepatitis B virus epidemic model with treatment cure rate. Our model consists of three epidemic compartments describing the interaction between the susceptible, the infected and the recovered individuals; an SIR model where the infected individuals transmit the infection to the susceptible ones with a transmission rate perturbed by white noise. Our paper begins by establishing that our hepatitis B stochastic model has unique global solution. It moves then to giving sufficient conditions for the stochastic extinction and persistence of the hepatitis B disease. Finally, our paper provides some numerical results to support the analytical study, showing numerically that the treatment cure rate facilitates the extinction of the hepatitis B disease among the population.

Key Words: HBV epidemic model, extinction, persistence, numerical simulation.

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## 1. Introduction

It is well-known that hepatitis B virus (HBV) is one of the major health problems in the world nowadays. It is a viral infection that can cause both chronic and acute disease by attacking the liver. According to World Health Organization (WHO), 296 million people had chronic HBV infection in 2019. The WHO reported also that 1.5 million new infections are registered every year, and an estimated 820000 people lost their lives due to the disease in 2019, mostly due to cirrhosis and hepatocellular carcinoma [1]. In order to reduce loss of lives and the high public health cost on society caused by the disease, mathematical modeling has become an important tool to understand the disease and control its spread.

At the beginning of the  $20^{th}$  century, Kermack and McKendrich [2] developed one of the earliest mathematical models that studied the interaction between susceptible and infected individuals; this model was known as the SI model. Since then, modeling the dynamics of epidemic disease outbreaks has been the central focus of many researchers, especially in recent years. These studies include, for example, the new epidemic coronavirus disease (COVID-19) [3,4], human immunodeficiency virus (HIV) [5,6], Hepatitis C Virus (HCV) [7,8], HBV [9,10,11] and many others. In the case of HBV, which is the main focus of this paper, which can be prevented by safe and available vaccines providing protection of

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the population; in other words, the susceptible can be recovered after receiving an effective vaccination. Hence, the models developed to study HBV infectious disease generally include three types of individuals, namely the susceptible S, the infected I and the recovered R individuals. In order to have a wide view on the HBV infection it will be more accurate to take into account three compartmental model instead of two [12,13,14,15].

The role of the incidence rates in epidemic models is so crucial to study the dynamical behavior of the disease. In fact, these rates can be categorized into two main types, namely the bilinear incidence rate and the saturated incidence rate. In order to describe the HBV disease transmission coefficient, many authors have used the bilinear incidence rate [12,13,14] while others have used a more generalized rate, namely the saturated incidence rate [9,15]. All the afore-mentioned papers present deterministic models for the HBV disease since they have ignored other dimensions, including the existence of randomness of natural transmissions. In the real world, in fact, all the biological phenomena are generally subject to environmental noises. Infections are no exception; they are always affected, as part of natural phenomena, by environmental randomness. That is, random environmental fluctuation manifests itself in the transmission coefficient, the birth and death rates, and other parameters in the system [16,17].

Many researchers have recently modeled randomness of the HBV infection by perturbing the infection rate via introducing white noise [18,19,20,21,22,23]. On the other hand, other researchers have described the environment fluctuation using Levy noise in the HBV model [24,25]. Recently, Khan et. al [26] has modeled the HBV infection by an SIR stochastic epidemic model. In order to build their model, they have imposed on the model the assumption that the HBV vaccine gives indefinite protection, and so the individuals of the susceptible class can transit to the class of recovered individuals after taking a successful vaccination. The authors have supposed that the infected individuals transmit the infection to the susceptible ones with a rate noted  $\beta$ , the latter is perturbed by white noise in order to present the random fluctuation environment, i.e.,  $\beta \to \beta + \eta \dot{B}(t)$ , with B(t) is standard Brownian motion and the intensity of the white noise is denoted by  $\eta$ . Actually, they have studied the disease extinction and disease persistence, also they have derived sufficient condition for them. Their HBV stochastic epidemic model is represented as follows

$$\begin{cases} dS = (\lambda - \beta S(t)I(t) - (\delta_0 + \mu) S(t)) dt - \eta SIdB(t), \\ dI = (\beta S(t)I(t) - (\delta_0 + \delta_1 + \gamma) I(t)) dt + \eta SIdB(t), \\ dR = (\gamma I(t) + \mu S(t) - \delta_0 R(t)) dt. \end{cases}$$

Where  $\lambda$  represents the constant birth rate,  $\delta_0$  represents the natural death rate of all the population,  $\delta_1$  represents the death rate as a result of the disease,  $\mu$  represents the vaccination rate, while  $\gamma_1$  represents the constant recovery rate for the infected individuals.

Since our contribution is motivated by the above work, we will assume that after a period of time an infected individual can become susceptible again with a certain rate, namely the treatment cure rate  $\alpha$ . The schematic representation of the HBV disease with treatment cure rate is represented in Fig. 1. In fact, this cure rate is taken into account in many diseases modeled by susceptible-infected-susceptible (SIS) [27,28] and others modeled by SIR epidemic model [29,30]. Our main motivation will be to add the effect of the treatment cure rate on the dynamics of the HBV disease. Our proposed model is presented as follows

$$\begin{cases}
dS(t) = (\lambda - \beta S(t)I(t) - (\delta_0 + \mu) S(t) + \alpha I) dt - \eta S(t)I(t)dB(t), \\
dI(t) = (\beta S(t)I(t) - (\delta_0 + \delta_1 + \gamma + \alpha) I(t)) dt + \eta S(t)I(t)dB(t), \\
dR(t) = (\gamma I(t) + \mu S(t) - \delta_0 R(t)) dt.
\end{cases} (1.1)$$

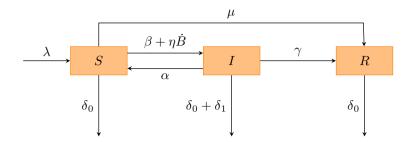


Figure 1: Schematic representation of the mathematical model (1.1).

In this paper, we will study mainly the above stochastic HBV model (1.1) by establishing its well-posedness and giving the conditions of the extinction and the persistence in mean of the HBV disease. The following section presents the equilibria and the well-posedness of the problem. We will show in Section 3 the stochastic analysis of the HBV model, followed in Section 4 by giving some numerical simulations. The last section is a conclusion part of the present work.

## 2. Analytical results

In this section, we present the equilibria of the HBV model and we establish the well-posedness of the problem. Then, we show that the solution of the HBV model exists and they are unique and non-negative.

# 2.1. The well-posedness of the problem

In order to show that the solution (S(t), I(t), R(t)) of the stochastic HBV model (1.1) exists and they are unique and non-negative, we will present some preliminaries

Throughout this paper, we define a complete probability space  $(\Omega, F, P)$  with a filtration  $\{\mathscr{F}_t\}_t$  satisfying the usual conditions (i.e. it is continuous and  $\mathscr{F}_0$  contains all P-null sets), and the standard Brownian motion  $t \to B(t)$  is defined on this complete probability space. Consider the infinitesimal operator L associated with the following 3-dimensional stochastic differential equation

$$dY = u(t, Y(t))dt + v(t, Y(t))dB(t), \tag{2.1}$$

with  $Y = (y_1, y_2, y_3)$ ,  $u(t, Y(t)) : \mathbb{R}_+ \times \mathbb{R}^3 \to \mathbb{R}^3$ ,  $v(t, Y(t)) : \mathbb{R}_+ \times \mathbb{R}^3 \to \mathbb{R}^3$  indicating the drift and the diffusion parts of the Eq. (2.1) respectively (see [31]), we define L as follows

$$L = \frac{\partial}{\partial t} + \sum_{i=1}^{3} u_i(t, Y) \frac{\partial}{\partial Y_i} + \frac{1}{2} \sum_{i,j=1}^{3} \left[ v^{\mathrm{T}}(t, Y) v(t, Y) \right]_{ij} \left( \frac{\partial^2}{\partial y_i \partial y_j} \right).$$

Let  $W \in C^{1,2}(\mathbb{R}_+, \mathbb{R}_+ \times \mathbb{R}^2)$ , if L acts on W, then

$$LW(t,Y) = W_t(t,Y) + W_y(t,Y) a(t,Y) + \frac{1}{2} \operatorname{trace}[b^T W_{yy}b],$$

where

$$W_t = \frac{\partial W}{\partial t}, \quad W_y = \left(\frac{\partial W}{\partial y_1}, \frac{\partial W}{\partial y_2}, \frac{\partial W}{\partial y_3}\right), \quad W_{yy} = \left(\frac{\partial^2 W}{\partial y_i \partial y_j}\right)_{3 \times 3}.$$

Itô formula [31], gives

$$dW(t,Y) = LW(t,Y) dt + W_{y}(t,Y) b(t,Y) dB(t).$$
(2.2)

The main result of this subsection is given as follows

**Theorem 2.1.** For any initial value  $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$ , the solution of our proposed model (1.1) is unique for all  $t \geq 0$ . Besides, the solution remains in  $\mathbb{R}^3_+$  with probability 1, i.e.,  $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$  for all  $t \geq 0$  almost surely (a.s).

*Proof.* We remark that for any initial value  $(S(0), I(0), R(0)) \in \mathbb{R}^3_+$ , the drift and the diffusion coefficient corresponding to our model (1.1) are locally Lipschitz, then there exists a unique local solution on  $[0,\tau_e)$ , where  $\tau_e$  is the explosion time. Now, we prove that  $\tau_e = \infty$  a.s. Let  $p_0 \ge 0$  be an integer sufficiently large, so that  $S(0), I(0), R(0) \in [\frac{1}{p_0}, p_0]$ . For each integer  $p \geq p_0$ , define the stopping time

$$\tau_p = \inf \left\{ t \in [0,\tau_e) : \min \left\{ S(t), I(t), R(t) \right\} \leq \frac{1}{p} \text{ or } \max \left\{ S(t), I(t), R(t) \right\} \geq p \right\}.$$

In the sequel of the paper, we set inf  $\phi = \infty$ , where  $\phi$  denotes the empty set. In fact it is obvious that  $\tau_p$  increases as  $p \to \infty$ . Set  $\tau_\infty = \lim_{k \to \infty} \tau_k$  with  $\tau_\infty \le \tau_e$  a.s. To finish the proof if we can show that  $\tau_{\infty} = \infty$  a.s. then  $\tau_e = \infty$  and consequently,  $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$  a.s. for all  $t \geq 0$ . If  $\tau_e \neq \infty$ , as a result there exists a pair of constant T > 0 and another constant  $\epsilon \in (0,1)$ , such that

$$P\left\{\tau_{\infty} \le T\right\} > \epsilon.$$

Consequently there is an integer  $p_1 \geq p_0$ , such that

$$P\{\tau_p \le T\} \ge \epsilon$$
, for all  $p \ge p_1$ . (2.3)

Let N(t) = S(t) + I(t) + R(t), where N denoted the total of population then for  $t \leq \tau_p$ , we observe

$$dN(t) = (\lambda - \delta_0 N(t) - \delta_1 I(t)) dt \le (\lambda - \delta_0 N(t)) dt.$$
(2.4)

Solving equation (2.4), we get

$$N(t) \le \frac{\lambda}{\delta_0} + \left(N(0) - \frac{\lambda}{\delta_0}\right) e^{-\delta_0 t}.$$

In consequence

$$N(t) \le \begin{cases} \frac{\lambda}{\delta_0}, & \text{if } N(0) \le \frac{\lambda}{\delta_0}, \\ N(0), & \text{if } N(0) > \frac{\lambda}{\delta_0}. \end{cases}$$

That is to say,

$$N(t) \le \frac{\lambda}{\delta_0} + \left(N(0) - \frac{\lambda}{\delta_0}\right) e^{-\delta_0 t} \le M_0 \quad \text{a.s., for all} \quad t \in [0, \tau_p],$$
(2.5)

where  $M_0 = \max \left\{ N(0), \frac{\lambda}{\delta_0} \right\}$ . Now, we define a  $C^2$ -function  $F : \mathbb{R}^3_+ \to \mathbb{R}_+$ , such that

$$(S, I, R) \to S + I + R - 3 - (\log S + \log I + \log R).$$

Clearly for all x > 0, we can see that  $x - 1 - \log x \ge 0$ . So H is non-negative. Let  $p \ge p_0$  and T > 0 be arbitrary. The application of Itô formula to the function F gives

$$dF(S, I, R) = \left(1 - \frac{1}{S}\right) dS + \frac{1}{2S^2} (dS)^2 + \left(1 - \frac{1}{I}\right) dI + \frac{1}{2I^2} (dI)^2 + \left(1 - \frac{1}{R}\right) dR.$$

Then we find

$$dF(S, I, R) = LF(S, I, R)dt + \eta(I - S)dB(t), \tag{2.6}$$

where  $LF: \mathbb{R}^3_+ \to \mathbb{R}_+$  is given as follows

$$LF(S, I, R) = \left(1 - \frac{1}{S}\right) (\lambda - \beta SI - (\delta_0 + \mu) S + \alpha I) + \frac{1}{2} \eta^2 I^2$$

$$+ \left(1 - \frac{1}{I}\right) (\beta SI - (\delta_0 + \delta_1 + \gamma + \alpha) I) + \frac{1}{2} \eta^2 S^2$$

$$+ \left(1 - \frac{1}{R}\right) (\gamma I + \mu S - \delta_0 R),$$

$$= \lambda - (\delta_0 + \mu) S + \alpha I - \frac{\lambda}{S} + \beta I + (\delta_0 + \mu) - \alpha \frac{I}{S} + \frac{1}{2} \eta^2 I^2$$

$$- (\delta_0 + \delta_1 + \gamma + \alpha) I - \beta S + (\delta_0 + \delta_1 + \gamma + \alpha) + \frac{1}{2} \eta^2 S^2$$

$$+ \gamma I + \mu S - \delta_0 R - \gamma \frac{I}{R} - \mu \frac{S}{R} + \delta_0,$$

$$\leq \lambda + \beta I + \delta_0 + \mu + \frac{1}{2} \eta^2 (S^2 + I^2) + \delta_0 + \alpha + \delta_1 + \gamma + \gamma I + \mu S + \delta_0,$$

$$\leq \lambda + 3\delta_0 + \mu + \eta^2 M^2 + (\beta + \gamma + \mu) M + \delta_1 + \gamma + \alpha := K.$$

The integration of Eq. (2.6) from 0 to  $\tau_p$  leads to the following equation

$$E[H(S(\tau_{p} \wedge T), I(\tau_{p} \wedge T), R(\tau_{p} \wedge T))]$$

$$\leq H(S(0), I(0), R(0)) + E\left[\int_{0}^{\tau_{p} \wedge T} K dt\right]$$

$$\leq H(S(0), I(0), R(0)) + KT. \tag{2.7}$$

Let define,  $\Omega_p = \{ \tau_p \leq T \}$  for  $p \geq p_1$  the equation (2.3) become  $P(\Omega_p) \geq \epsilon$ . Keep in mind that for every  $\omega \in \Omega_p$ , there exists at least one  $S(\tau_p, \omega)$ ,  $I(\tau_p, \omega)$ ,  $R(\tau_p, \omega)$  that equal to p or  $\frac{1}{p}$ . Accordingly

$$H\left(S\left(\tau_{p}\right),I\left(\tau_{p}\right),R\left(\tau_{p}\right)\right)\geq p-1-\log p \quad \text{or} \quad H\left(S\left(\tau_{p}\right),I\left(\tau_{p}\right),R\left(\tau_{p}\right)\right)\geq 1/p-1+\log p.$$

So

$$H\left(S\left(\tau_{p}\right), I\left(\tau_{p}\right), R\left(\tau_{p}\right)\right) \ge E\left(\left(p - 1 - \log p\right) \wedge \left(\frac{1}{p} - 1 + \log p\right)\right).$$
 (2.8)

From equations (2.7) and (2.8), we find

$$\begin{split} H(S(0),I(0),R(0)) + KT &\geq E\left[1_{\Omega_n} H\left(S\left(\tau_p\right),I\left(\tau_p\right),R\left(\tau_p\right)\right)\right] \\ &\geq \epsilon \left\lceil \left(p-1-\log p\right) \wedge \left(\frac{1}{p}-1+\log p\right)\right\rceil, \end{split}$$

Where  $1_{\Omega_p}$  is the indicator function of  $\Omega_p$ . Letting  $p \to \infty$  leads to the contradiction

$$\infty > H(S(0), I(0), R(0)) + KT = \infty,$$

hence  $\tau_{\infty} = \infty$  a.s.

Finally, the HBV model (1.1) has a unique global solution  $(S(t), I(t), R(t)) \in \mathbb{R}^3_+$ .

**Remark 2.2.** As a result of the inequality (2.5), if  $S(0) + I(0) + R(0) \le \frac{\lambda}{\delta_0}$ , then  $N(t) \le \frac{\lambda}{\delta_0}$  a.s., for all  $t \ge 0$ .

Therefore, the region

$$\Delta^* = \left\{ (S, I, R) \in \mathbb{R}^3_+ : S + I + R \leqslant \frac{\lambda}{\delta_0} \right\}. \tag{2.9}$$

is almost surely positively invariant by the system (1.1).

The dynamics of our model will be studied in this region. That is to say, we assume that  $(S(0), I(0), R(0)) \in \Delta^*$ .

## 2.2. The equilibria

The HBV epidemic model has a unique disease-free equilibrium (DFE)

$$E_f = (S_f, I_f, R_f) = \left(\frac{\lambda}{\delta_0 + \mu}, 0, \frac{\mu \lambda}{\delta_0(\delta_0 + \mu)}\right).$$

The equation associated with infection is

$$\frac{dI}{dt} = \beta S(t)I(t) - (\delta_0 + \delta_1 + \gamma + \alpha)I(t) = \mathcal{F}(t) - \mathcal{V}(t),$$

where,  $\mathfrak{F}(t) = \beta S(t)I(t)$  and  $\mathfrak{V}(t) = (\delta_0 + \delta_1 + \gamma + \alpha)I(t)$ .

By the next generation matrix approach [32], the formula that provides us the basic reproduction number is  $\mathcal{R}_0 = \rho\left(-FV^{-1}\right)$ , which  $\rho(A)$  is explicitly the spectral radius of the matrix A. In our case,  $F = \frac{\partial \mathcal{F}}{\partial I}\big|_{E_f} = \frac{\beta \lambda}{\delta_0 + \mu}$  and  $V = \frac{\partial \mathcal{V}}{\partial I}\big|_{E_f} = \delta_0 + \delta_1 + \gamma + \alpha$ .

Then, the basic reproduction number for the corresponding deterministic version of our model (1.1) is defined by

$$\mathcal{R}_{0} = \frac{\beta \lambda}{\left(\delta_{0} + \mu\right)\left(\delta_{0} + \delta_{1} + \gamma + \alpha\right)}.$$

This threshold describes the average number of the new HBV infected individuals produced by one HBV infected individual in the studied susceptible population. Also, when  $\mathcal{R}_0 \geq 1$ , we define another steady state associated to the HBV model, namely the endemic equilibrium  $E_d = (S_d, I_d, R_d)$  with

$$S_{d} = \frac{\delta_{0} + \delta_{1} + \gamma + \alpha}{\beta}, \qquad I_{d} = \frac{\left(\delta_{0} + \mu\right)\left(\delta_{0} + \delta_{1} + \gamma + \alpha\right)}{\beta\left(\delta_{0} + \delta_{1} + \gamma\right)} \left(\Re_{0} - 1\right),$$

$$R_{d} = \frac{\mu\left(\delta_{0}^{2} + \delta_{1}^{2} + \gamma^{2} + \alpha\delta_{0} + \alpha\delta_{1} + \alpha\gamma + \delta_{0}\gamma + \delta_{1}\gamma + 2\delta_{0}\delta_{1}\right)}{\delta_{0}\beta\left(\delta_{0} + \delta_{1} + \gamma\right)} + \frac{\gamma\left(\delta_{0} + \mu\right)\left(\delta_{0} + \delta_{1} + \gamma + \alpha\right)}{\delta_{0}\beta\left(\delta_{0} + \delta_{1} + \gamma\right)} (\Re_{0} - 1).$$

# 3. Stochastic analysis of the model

The extinction and the persistence of the HBV disease will be investigated, for this end we introduce the following definition

$$\langle I(t) \rangle = \frac{1}{t} \int_0^t I(r) dr.$$

**Lemma 3.1.** (Strong law of large number [33]): Let  $M = \{M\}_{t \geq 0}$  be a real valued continuous local martingale vanishing at t=0, then

- $\lim_{t\to\infty} \langle M, M \rangle_t = \infty$ , a.s.,  $\Rightarrow \lim_{t\to\infty} \frac{M_t}{\langle M, M \rangle_t} = 0$  almost surely.
- $\lim_{t\to\infty} \sup \frac{\langle M,M\rangle_t}{t} < \infty$ , a.s.,  $\Rightarrow \lim_{t\to\infty} \sup \frac{M_t}{t} = 0$ , almost surely.

**Definition 3.2.** ([33]): The proposed model is supposed to be persistent in mean, if

$$\lim_{t \to \infty} \inf \int_0^t I(r)dr > 0 \quad almost \ surely.$$

**Lemma 3.3.** ([34]): Let  $g \in C[[0,\infty) \times \Omega, (0,\infty)]$  and  $G(t) \in C([0,\infty) \times \Omega, \mathbf{R})$ . If there exist positive constants  $\lambda_0$ ,  $\lambda$  and T, such that

- $\log g(t) \le \lambda t \lambda_0 \int_0^t g(s)ds + G(t)$  almost surely for all  $t \ge T$ ,
- $\lim_{t\to\infty} \frac{G(t)}{t} = 0$  almost surely.

Then,

$$\lim_{t \to \infty} \sup \frac{1}{t} \int_0^t g(s) ds \le \frac{\lambda}{\lambda_0} \qquad almost \ surely.$$

### 3.1. The stochastic disease extinction

Now, We look into the conditions that could lead to the extinction of the studied disease. Firstly, we define the basic reproduction number for the stochastic HBV model (1.1) as follows

$$\mathcal{R}_s = \frac{\beta \lambda}{(\delta_0 + \mu) \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{\eta^2 \lambda^2}{2(\delta_0 + \mu)^2}\right)}.$$

**Theorem 3.4.** Let (S(t), I(t), R(t)) be the solution of the HBV model (1.1) with initial value  $(S(0), I(0), R(0)) \in \Delta^*$ .

If the two conditions  $\Re_s < 1$  and  $\beta(\delta_0 + \mu) > \eta^2 \lambda$  are verified then  $\lim_{t \to \infty} \left(\frac{\log I(t)}{t}\right) < 0$  a.s.

*Proof.* The integration of each equation in the proposed system (1.1) gives

$$\frac{S(t) - S(0)}{t} = \frac{1}{t} \int_{0}^{t} \left\{ (\lambda - \beta S(r)I(r) - (\delta_{0} + \mu) S(r) + \alpha I(r)) dr - \eta S(r)I(r)dB(r) \right\} 
= \lambda - \beta \langle S(t)I(t) \rangle - (\delta_{0} + \mu) \langle S(t) \rangle + \alpha \langle I(t) \rangle - \frac{\eta}{t} \int_{0}^{t} S(r)I(r)dB(r), 
\frac{I(t) - I(0)}{t} = \frac{1}{t} \int_{0}^{t} \left\{ (\beta S(r)I(r) - (\delta_{0} + \delta_{1} + \gamma + \alpha) I(r)) dr + \eta S(r)I(r)dB(r) \right\} 
= \beta \langle S(t)I(t) \rangle - (\delta_{0} + \delta_{1} + \gamma + \alpha) \langle I(t) \rangle + \frac{\eta}{t} \int_{0}^{t} S(r)I(r)dB(r), 
\frac{R(t) - R(0)}{t} = \frac{1}{t} \int_{0}^{t} \left\{ \gamma I(r) + \mu S(r) - \delta_{0}R(r) \right\} dr 
= \gamma \langle I(t) \rangle + \mu \langle S(t) \rangle - \delta_{0} \langle R(t) \rangle.$$
(3.1)

If we add the two first equations of (3.1) together we get

$$\frac{S(t) - S(0)}{t} + \frac{I(t) - I(0)}{t} = \lambda - (\delta_0 + \mu) \langle S(t) \rangle - (\delta_0 + \delta_1 + \gamma) \langle I(t) \rangle.$$

Hence

$$\langle S(t) \rangle = \frac{\lambda}{\delta_0 + \mu} - \frac{\delta_0 + \delta_1 + \gamma}{\delta_0 + \mu} \langle I(t) \rangle + \zeta(t), \tag{3.2}$$

Where  $\zeta$  is defined by

$$\zeta(t) = -\frac{1}{\delta_0 + \mu} \left[ \frac{S(t) - S(0)}{t} + \frac{I(t) - I(0)}{t} \right].$$

Clearly when  $t \to \infty$  then  $\zeta(t)$  converge towards 0. We apply the itô formula to  $\log I(t)$ , we get

$$d\log I(t) = \left[ \beta S(t) - (\delta_0 + \delta_1 + \alpha + \gamma) - \frac{1}{2} \eta^2 S^2(t) \right] dt + \eta S(t) dB(t). \tag{3.3}$$

We integre the equation (3.3) from 0 to t and and then we divide by t, we find

$$\frac{\log I(t) - \log I(0)}{t} = \beta \langle S(t) \rangle - (\delta_0 + \delta_1 + \gamma + \alpha) - \frac{1}{2} \eta^2 \langle S^2(t) \rangle 
+ \frac{\eta}{t} \int_0^t S(r) dB(r) 
\leq \beta \langle S(t) \rangle - (\delta_0 + \delta_1 + \gamma + \alpha) - \frac{1}{2} \eta^2 \langle S(t) \rangle^2 
+ \frac{\eta}{t} \int_0^t S(r) dB(r).$$
(3.4)

Substituting the Eq. (3.2) in the Eq. (3.4) and considering the local continuous martingale M(t) $\eta \int_0^t S(r)dB(r)$  with M(0)=0, we achieve

$$\frac{\log I(t) - \log I(0)}{t} \leq \beta \left( \frac{\lambda}{\delta_0 + \mu} - \frac{\delta_0 + \delta_1 + \gamma}{\delta_0 + \mu} \langle I(t) \rangle + \zeta(t) \right) \\
- (\delta_0 + \delta_1 + \gamma + \alpha) \\
- \frac{1}{2} \eta^2 \left( \frac{\lambda}{\delta_0 + \mu} - \frac{\delta_0 + \delta_1 + \gamma}{\delta_0 + \mu} \langle I(t) \rangle + \zeta(t) \right)^2 \\
+ \frac{\eta}{t} \int_0^t S(r) dB(r), \\
\leq \frac{\beta \lambda}{\delta_0 + \mu} - \frac{\beta (\delta_0 + \delta_1 + \gamma)}{\delta_0 + \mu} \langle I(t) \rangle \\
- (\delta_0 + \delta_1 + \gamma + \alpha) \\
- \frac{1}{2} \frac{\eta^2 \lambda^2}{(\delta_0 + \mu)^2} + \frac{\eta^2 \lambda (\delta_0 + \delta_1 + \gamma)}{(\delta_0 + \mu)^2} \langle I(t) \rangle \\
+ \frac{M(t)}{t} + \phi(t),$$

Finally

$$\frac{\log I(t) - \log I(0)}{t} \le -\left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{(\delta_0 + \mu)^2}\right) (1 - \Re_s) 
-\left(\frac{\delta_0 + \delta_1 + \gamma}{\delta_0 + \mu}\right) \left(\beta - \frac{\eta^2 \lambda}{\delta_0 + \mu}\right) \langle I(t) \rangle 
+ \frac{M(t)}{t} + \phi(t).$$
(3.5)

Where

$$\phi(t) = \beta \zeta(t) - \frac{1}{2} \eta^2 \zeta^2(t) + \frac{\eta^2 (\delta_0 + \delta_1 + \gamma)}{\delta_0 + \mu} \langle I(t) \rangle \zeta(t)$$
$$- \frac{\lambda \eta^2 \zeta(t)}{\delta_0 + \mu} - \frac{\eta^2}{2} \frac{(\delta_0 + \delta_1 + \gamma)^2}{(\delta_0 + \mu)^2} \langle I(t) \rangle^2.$$

Besides  $\lim_{t\to\infty}\sup\frac{\langle M(t),M(t)\rangle}{t}\leq \frac{\eta^2\lambda^2}{\delta_0+\mu}<\infty$ , by Lemma 1 we can conclude  $\lim_{t\to\infty}\sup\frac{M(t)}{t}=0$ . Also  $\lim_{t\to\infty}\zeta(t)=0$  as a result  $\lim_{t\to\infty}\phi(t)=0$ . If the two conditions  $\mathcal{R}_s<1$  and  $\beta\left(\delta_0+\mu\right)>\eta^2\lambda$  are satisfied, then Eq. (3.5) becomes

$$\lim_{t \to \infty} \sup \frac{\log I(t)}{t} \le -\left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda}{\delta_0 + \mu}\right) (1 - \mathcal{R}_s) - \left(\frac{\delta_0 + \delta_1 + \gamma}{\delta_0 + \mu}\right) \left(\beta - \frac{\eta^2 \lambda}{\delta_0 + \mu}\right) \langle I(t) \rangle < 0 \quad \text{a.s.}$$
(3.6)

Corollary 3.5. If  $\lim_{t\to\infty} \frac{\log I(t)}{t} < 0$  a.s., then I(t) converge exponentially towards 0 a.s., that is to say the HBV infection dies out with probability one.

In addition  $\lim_{t\to\infty} S(t) = \frac{\lambda}{\delta_0 + \mu}$  a.s.,  $\lim_{t\to\infty} I(t) = 0$  a.s.,  $\lim_{t\to\infty} R(t) = \frac{\mu\lambda}{\delta_0(\delta_0 + \mu)}$  a.s.

*Proof.* Obviously if  $\lim_{t\to\infty} \frac{\log I(t)}{t} < 0$  then

$$\lim_{t \to \infty} I(t) = 0 \quad \text{a.s.} \tag{3.7}$$

The sum of all the equations of the model (1.1), gives

$$d(S(t) + I(t) + R(t)) = (\lambda - \delta_0(S(t) + I(t) + R(t)) - \delta_1 I(t)) dt.$$
(3.8)

The solution of the Eq. (3.8) becomes

$$S(t) + I(t) + R(t) = e^{-\delta_0 t} \left( S(0) + I(0) + R(0) + \int_0^t (\lambda - \delta_1 I(s)) e^{\delta_0 s} ds \right).$$

Using the L'Hospital rule and Eq. (3.7) we find

$$\lim_{t \to \infty} (S(t) + R(t)) = \lim_{t \to \infty} \left( \frac{S(0) + I(0) + R(0) + \int_0^t (\lambda - \delta_1 I(s)) e^{\delta_0 s} ds}{e^{\delta_0 t}} - I(t) \right) = \frac{\lambda}{\delta_0}.$$

Thus, we get

$$\lim_{t \to \infty} (S(t) + R(t)) = \frac{\lambda}{\delta_0} \quad \text{a.s.}$$
 (3.9)

The first equation of the proposed system (1.1) with limiting system becomes

$$dS(t) = (\lambda - (\delta_0 + \mu) S(t) + \alpha I(t)) dt. \tag{3.10}$$

The solution of the Eq. (3.10) becomes

$$S(t) = e^{-(\delta_0 + \mu)t} \left( S(0) + \int_0^t (\lambda + \alpha I(s)) e^{(\delta_0 + \mu)s} ds \right).$$

Using the L'Hospital rule and Eq. (3.7) we find

$$\lim_{t \to \infty} S(t) = \lim_{t \to \infty} \left( \frac{S(0) + \int_0^t (\lambda + \alpha I(s)) e^{(\delta_0 + \mu)s} ds}{e^{(\delta_0 + \mu)t}} \right) = \frac{\lambda}{\delta_0 + \mu}.$$

From Equation (3.9) we obtain

$$\lim_{t\to\infty}R(t)=\frac{\mu\lambda}{\delta_0\left(\delta_0+\mu\right)}\qquad\text{a.s.}$$

## 3.2. The stochastic disease persistence

We search the conditions that could lead to the persistence in mean of the studied HBV disease.

**Theorem 3.6.** If  $\Re_s > 1$  and  $\beta(\delta_0 + \mu) > \eta^2 \lambda$ , then for any initial value  $(S(0), I(0), R(0)) \in \Delta^*$ , the solution (S(t), I(t), R(t)) of the proposed model (1.1) has the following property

$$I_2 \le \lim_{t \to \infty} \inf(I(t)) \le \lim_{t \to \infty} \sup \langle I(t) \rangle \le I_1 \quad a.s.,$$

where

$$I_{1} = \frac{\left(\delta_{0} + \mu\right)^{2} \left(\delta_{0} + \delta_{1} + \gamma + \alpha + \frac{\eta^{2} \lambda^{2}}{2(\delta_{0} + \mu)^{2}}\right) \left(\Re_{s} - 1\right)}{\left(\delta_{0} + \delta_{1} + \gamma\right) \left(\beta \left(\delta_{0} + \mu\right) - \eta^{2} \lambda\right)},$$

$$I_{2} = \frac{\left(\delta_{0} + \mu\right) \left(\delta_{0} + \delta_{1} + \gamma + \alpha + \frac{\eta^{2} \lambda^{2}}{2(\delta_{0} + \mu)^{2}}\right) \left(\Re_{s} - 1\right)}{\beta \left(\delta_{0} + \delta_{1} + \gamma\right)}.$$

*Proof.* According to the last inequality of the Eq. (3.5)

$$\frac{\log I(t) - \log I(0)}{t} \le \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{(\delta_0 + \mu)^2}\right) (\mathcal{R}_s - 1) 
- \left(\frac{\delta_0 + \delta_1 + \gamma}{\delta_0 + \mu}\right) \left(\beta - \frac{\eta^2 \lambda}{\delta_0 + \mu}\right) \langle I(t) \rangle 
+ \frac{M(t)}{t} + \phi(t).$$

From the last inequality we can conclude

$$\frac{\log I(t)}{t} \le \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{(\delta_0 + \mu)^2}\right) (\mathcal{R}_s - 1)$$

$$- \left(\frac{\delta_0 + \delta_1 + \gamma}{\delta_0 + \mu}\right) \left(\beta - \frac{\eta^2 \lambda}{\delta_0 + \mu}\right) \langle I(t) \rangle$$

$$+ \frac{M(t)}{t} + \phi(t) + \frac{\log I(0)}{t}.$$
(3.11)

The inequality (3.11) can be re-written as

$$\langle I(t) \rangle \leq \frac{(\delta_0 + \mu)^2 \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{(\delta_0 + \mu)^2}\right)}{(\delta_0 + \delta_1 + \gamma) \left(\beta \left(\delta_0 + \mu\right) - \eta^2 \lambda\right)} \left(\mathcal{R}_s - 1\right) + \frac{\left(\delta_0 + \mu\right)^2}{\left(\delta_0 + \delta_1 + \gamma\right) \left(\beta \left(\delta_0 + \mu\right) - \eta^2 \lambda\right)} \times \left[\frac{M(t)}{t} + \phi(t) + \frac{\log I(0)}{t} - \frac{\log I(t)}{t}\right].$$

Firstly we suppose that  $\Re_s > 1$  and  $\beta\left(\delta_0 + \mu\right) > \eta^2 \lambda$  and we have  $\lim_{t \to \infty} \phi(t) = 0$ . On the other hand, since  $\lim_{t \to \infty} \sup \frac{\langle M(t), M(t) \rangle}{t} < \infty$  then by Lemma 1  $\lim_{t \to \infty} \sup \frac{M(t)}{t} = 0$ . Furthermore, the Eq. (3.11) implies that the function I verify the conditions of the Lemma 2 then we get

$$\lim_{t \to \infty} \sup \langle I(t) \rangle \le \frac{\left(\delta_0 + \mu\right)^2 \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{\left(\delta_0 + \mu\right)^2}\right)}{\left(\delta_0 + \delta_1 + \gamma\right) \left(\beta \left(\delta_0 + \mu\right) - \eta^2 \lambda\right)} \left(\mathcal{R}_s - 1\right) = I_1. \tag{3.12}$$

On the other hand, substituting Eq. (3.2) in the Eq. (3.4) we obtain

$$\frac{\log I(t) - \log I(0)}{t} = \frac{\beta \lambda}{\delta_0 + \mu} - \frac{\beta \left(\delta_0 + \delta_1 + \gamma\right)}{\delta_0 + \mu} \langle I(t) \rangle + \beta \phi(t) 
- \frac{1}{2} \eta^2 \left\langle S^2(t) \right\rangle - \left(\delta_0 + \delta_1 + \gamma + \alpha\right) + \frac{M(t)}{t}, 
\geq \frac{\beta \lambda}{\delta_0 + \mu} - \frac{\beta \left(\delta_0 + \delta_1 + \gamma\right)}{\delta_0 + \mu} \langle I(t) \rangle + \beta \phi(t) 
- \frac{1}{2} \frac{\eta^2 \lambda^2}{\delta_0^2} - \left(\delta_0 + \delta_1 + \gamma + \alpha\right) + \frac{M(t)}{t}, 
\geq \frac{\beta \lambda}{\delta_0 + \mu} - \frac{\beta \left(\delta_0 + \delta_1 + \gamma\right)}{\delta_0 + \mu} \langle I(t) \rangle + \beta \phi(t) 
- \frac{1}{2} \frac{\eta^2 \lambda^2}{\left(\delta_0 + \mu\right)^2} - \left(\delta_0 + \delta_1 + \gamma + \alpha\right) + \frac{M(t)}{t} 
\geq - \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{\left(\delta_0 + \mu\right)^2}\right) (1 - \Re_s) 
- \frac{\beta \left(\delta_0 + \delta_1 + \gamma\right)}{\delta_0 + \mu} \langle I(t) \rangle + \beta \phi(t) + \frac{M(t)}{t}.$$

Finally

$$\frac{\log I(t) - \log I(0)}{t} \ge \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{(\delta_0 + \mu)^2}\right) (\mathcal{R}_s - 1)$$

$$- \frac{\beta (\delta_0 + \delta_1 + \gamma)}{(\delta_0 + \mu)} \langle I(t) \rangle + \beta \phi(t) + \frac{M(t)}{t}.$$
(3.13)

From Eq. (3.13) we obtain

$$\begin{split} \langle I(t) \rangle & \geq \frac{\left(\delta_0 + \mu\right) \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{\left(\delta_0 + \mu\right)^2}\right)}{\beta \left(\delta_0 + \delta_1 + \gamma\right)} \left( \mathcal{R}_s - 1 \right) \\ & + \frac{\left(\delta_0 + \mu\right)}{\beta \left(\delta_0 + \delta_1 + \gamma\right)} \left[ \frac{M(t)}{t} + \phi(t) + \frac{\log I(0)}{t} - \frac{\log I(t)}{t} \right]. \end{split}$$

Note that we have  $\log(I(t)) \leq \log\left(\frac{\lambda}{\delta_0}\right)$ , then this inequality becomes

$$\langle I(t) \rangle \ge \frac{\left(\delta_0 + \mu\right) \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{\left(\delta_0 + \mu\right)^2}\right)}{\beta \left(\delta_0 + \delta_1 + \gamma\right)} \left(\mathcal{R}_s - 1\right) + \frac{\left(\delta_0 + \mu\right)}{\beta \left(\delta_0 + \delta_1 + \gamma\right)} \left[\frac{M(t)}{t} + \phi(t) + \frac{\log I(0)}{t} - \frac{\log \left(\lambda/\delta_0\right)}{t}\right]. \tag{3.14}$$

Firstly we suppose that  $\Re_s > 1$ , and we have  $\lim_{t \to \infty} \phi(t) = 0$ . Furthermore, since  $\lim_{t \to \infty} \sup \frac{\langle M(t), M(t) \rangle}{t} < \infty$  then by Lemma 1  $\lim_{t \to \infty} \sup \frac{M(t)}{t} = 0$ . Taking the limit inferior of both side of the Eq. (3.14) we get

$$\lim_{t \to \infty} \inf \langle I(t) \rangle \ge \frac{\left(\delta_0 + \mu\right) \left(\delta_0 + \delta_1 + \gamma + \alpha + \frac{1}{2} \frac{\eta^2 \lambda^2}{(\delta_0 + \mu)^2}\right)}{\beta \left(\delta_0 + \delta_1 + \gamma\right)} \left(\Re_s - 1\right) = I_2. \tag{3.15}$$

Therefore, by Eqs. (3.12) and (3.15) we have

$$I_2 \le \lim_{t \to \infty} \inf(I(t)) \le \lim_{t \to \infty} \sup \langle I(t) \rangle \le I_1$$
 a.s.

# 4. Numerical results

The illustration of the theoretical findings results by numerical simulations is shown in this section. In our numerical simulations, we will solve the system (1.1) with taking into account the parameter values from the Table 1.

Table 1: The used parameters in numerical simulation.

Notation	Parameter description	Fig. 2	Fig. 3	Fig. 4	Fig. 5
λ	Birth rate	0.5	0.6	0.6	0.7
$\beta$	Transmission rate	0.6	0.7	0.7	0.9
$\delta_0$	Natural death rate	0.1	0.1	0.1	0.1
${\delta}_1$	Disease induced death rate	0.2	0.2	0.2	0.2
$\mu$	Vaccination rate	0.4	0.2	0.2	0.4
$\gamma$	Recovery rate	0.4	0.4	0.4	0.4
$\eta$	Amount of environmental white noise	0.6	0.25	0.6	0.1
$\alpha$	The treatment cure rate	0.1	0.2	0.2	

Fig. 2 shows the evolution of the susceptible, infected and recovered individuals during a certain period of time. For the values listed in Table 1, we have  $\mathcal{R}_0 = 0.75 < 1$ ,  $\mathcal{R}_s = 0.6122 < 1$  and  $\beta(\delta_0 + \mu) = 0.3 > \eta^2 \lambda = 0.18$ . It can be seen from Fig. 2a and Fig. 2b that the curves converge towards the disease-free equilibrium  $E_f = (1,0,4)$ . In other terms, the two curves which describe the infected individuals corresponding to the deterministic and the stochastic models converge both toward zero, which means the extinction of the HBV infection which is consistent with the theoretical results.

Fig. 3 depicts the dynamics of system (1.1) during the period of observation. For this illustration, we consider the parameter values stated in Table 1, we get  $\mathcal{R}_0 = 1.5556 > 1$ ,  $\mathcal{R}_s = 1.3659 > 1$  and we have  $\beta(\delta_0 + \mu) = 0.21 > \eta^2 \lambda = 0.038$ . Indeed, the curves in Fig. 3a and Fig. 3b converge towards the disease equilibrium  $E_d = (1.2857, 0.3061, 3.3061)$ . We can conclude also that the function which describes the infected individuals corresponding to the stochastic model obeys  $0.2296 \le \liminf_{t \to \infty} \langle I(t) \rangle \le \lim \sup_{t \to \infty} \langle I(t) \rangle \le 0.2795$ , which means the persistence of the HBV infection which is in good agreement with the theoretical finding.

Fig. 4 shows the behavior of the HBV infection after the period of observation. More explicitly the plots in Fig. 4a and Fig. 4b converge respectively towards  $E_f = (2,0,4)$  and  $E_d = (1.2857, 0.3061, 3.3061)$ . In this case we get,  $\mathcal{R}_0 = 1.5556 > 1$  and  $\mathcal{R}_s = 0.8642 < 1$ . Strong random fluctuations in our system (1.1) driven by standard Brownian motions accelerate the extinction of the infection. As a result, we can deduce that with the same parameters, the stochastic model may indicate the extinction of the HBV disease, whereas the deterministic model predicts that the disease will persist over time.

Fig. 5 illustrates the effect of the treatment cure rate on the HBV dynamical behavior. More clearly, Fig. 5a and Fig. 5b correspond respectively to the susceptible and infected individuals for both the HBV stochastic model (1.1) and its corresponding deterministic version. The numerical simulation is presented for four different values of  $\alpha$ , which are  $\alpha=0.2,\ 0.4,\ 0.6$ , 0.8 with the value of other parameters are listed in Table 1. The corresponding  $\mathcal{R}_0$  are  $\mathcal{R}_0=1.4000,\ 1.1455,\ 0.9692,\ 0.8400$  while the corresponding  $\mathcal{R}_s$  are  $\mathcal{R}_s=1.3849,\ 1.1353,\ 0.9620,\ 0.8345$ . Note that the values of  $\mathcal{R}_0$  and  $\mathcal{R}_s$  in descending order, for the two first values of both  $\mathcal{R}_0$  and  $\mathcal{R}_s$  are greater than unity, while the two last values are less than one. First, note that an increase in  $\alpha$  results in a decrease in both  $\mathcal{R}_0$  and  $\mathcal{R}_s$ . Besides, with an increase in the level of  $\alpha$ , we remark a corresponding increase in the level of the susceptible individuals while a corresponding decrease in the level of the infected individuals. For both deterministic and stochastic models, the parameter  $\alpha$  influences the behavior of the epidemic, i.e. for small values of  $\alpha$  we note the persistence of the HBV studied model while large values of  $\alpha$  results in the extinction of the HBV studied model.

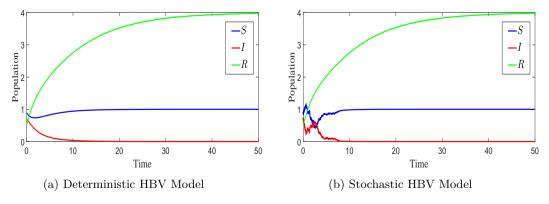


Figure 2: The dynamics of the disease showing the extinction of the HBV disease.

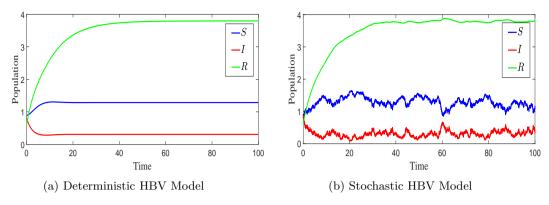


Figure 3: The dynamics of the disease showing the persistence of the HBV disease.

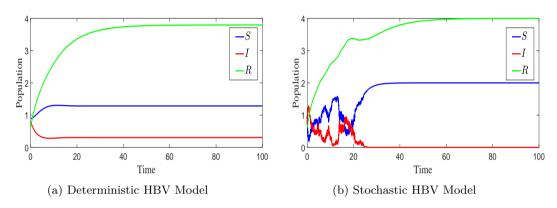


Figure 4: The dynamics of the disease showing the extinction of the stochastic HBV model and the persistence for its corresponding deterministic one.

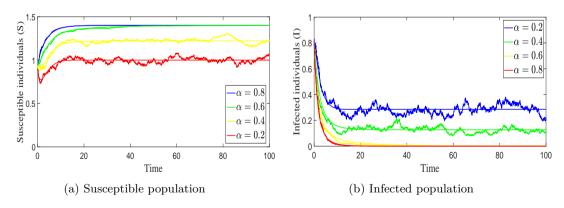


Figure 5: The dynamics of the disease showing the effect of the treatment cure rate.

# 5. Conclusion

In this paper, we have formulated a stochastic HBV epidemic model; an SIR model where the infected individuals transmit the infection to the susceptible ones with an transmission rate perturbed by white noise. The well-posedness of the problem is proved and we have given the conditions of the disease extinction as well as the conditions of its persistence. These conditions depend on system parameters

and also the intensity of the white noise. Moreover, it is obvious that the extinction of the HBV disease increases with an increase in the noise intensity, with an increasing in the value of the treatment cure rate, or with an increasing in both. Similarly, the persistence of the disease decreases with an increasing in the noise intensity, with an increasing in the value of the treatment cure rate, or with an increasing in both. We have performed numerical simulations to guarantee the analytical results. It seems that in order to modelize an epidemic disease in a realistic way, we have to add environment noise. That is why a stochastic epidemic model is more adequate than deterministic one. As future perspective of this present work, one can study the same problem under the effect of stochastic Lévy jump process.

## References

- 1. Organization W. H. Hepatitis b factsheet. Available at: https://www.who.int/news-room/fact-sheets/detail/hepatitis-b.
- Kermack, W. O., McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. Proc. R. Soc. Lond. 115(772): 700-721.
- 3. Khoshnaw, S. H., Salih, R. H., Sulaimany, S. (2020). Mathematical modelling for coronavirus disease (COVID-19) in predicting future behaviours and sensitivity analysis. *Math. Model. Nat. Phenom.* 15(33).
- Khyar, O., Allali, K. (2020). Dynamic Analysis of SLIR Model Describing the Effectiveness of Quarantine Against the Spread of COVID-19. International Symposium on Mathematical and Computational Biology. Springer Cham, pp. 243-259.
- Huo, H. F., Chen, R., Wang, X. Y. (2016). Modelling and stability of HIV/AIDS epidemic model with treatment. Appl. Math. 40(13-14): 6550-6559.
- Djordjevic, J., Silva, C. J., Torres, D. F. (2018). A stochastic SICA epidemic model for HIV transmission. Appl Math Lett. 84: 168-175.
- 7. Pitcher, A. B., Borquez, A., Skaathun, B., Martin, N. K. (2019). Mathematical modeling of hepatitis c virus (HCV) prevention among people who inject drugs: A review of the literature and insights for elimination strategies. *J. Theor. Biol.* 481: 194-201.
- 8. Cui, J. A., Zhao, S., Guo, S., Bai, Y., Wang, X., Chen, T. (2020). Global dynamics of an epidemiological model with acute and chronic HCV infections. *Appl Math Lett.* 103: 106203.
- 9. Khan, T., Ullah, Z., Ali, N., Zaman, G. (2019). Modeling and control of the hepatitis B virus spreading using an epidemic model. *Chaos Solitons Fractals*, 124: 1-9.
- Kamyad, A. V., Akbari, R., Heydari, A. A., Heydari, A. (2014). Mathematical modeling of transmission dynamics and optimal control of vaccination and treatment for hepatitis B virus. Comput Math Methods Med. 2014.
- 11. Zhang, T., Wang, K., Zhang, X. (2015). Modeling and analyzing the transmission dynamics of HBV epidemic in Xinjiang, China. *PLoS One.* 10(9): e0138765.
- 12. Khan, T., Zaman, G., Chohan, M. I. (2017). The transmission dynamic and optimal control of acute and chronic hepatitis B. J. Biol. Dyn. 11(1): 172-189.
- 13. Mehmood, M., Hamid, M., Ashraf, S., Tian, Z. (2021). Galerkin time discretization for transmission dynamics of HBV with non-linear saturated incidence rate. *Appl. Math.* 410(126481).
- Letsa-Agbozo, J.K., Kumah, M.S., Buabasah, D.Y. (2016). Sir model of hepatitis B disease in the North Tongu district. Int. j. appl. Res. 2: 229-234.
- 15. Macías-Díaz, J. E., Ahmed, N., Rafiq, M. (2019). Analysis and nonstandard numerical design of a discrete three-dimensional hepatitis B epidemic model. *Mathematics*. 7(12): 1157.
- Øksendal, B. (2003). Stochastic Differential Equations. Stochastic Differential Equations. Springer Berlin Heidelberg, pp. 65-84.
- 17. Truscott, J. E., Gilligan, C. A. (2003). Response of a deterministic epidemiological system to a stochastically varying environment. *PNAS*. 100(15): 9067-9072.
- 18. Din, A., Li, Y., Yusuf, A. (2021). Delayed hepatitis B epidemic model with stochastic analysis. *Chaos Solitons Fractals*. 146: 110839.
- 19. Khan, T., Jung, I. H., Zaman, G. (2019). A stochastic model for the transmission dynamics of hepatitis B virus. J. Biol. Dyn. 13(1): 328-344.
- Liu, P., Din, A., Huang, L., Yusuf, A. (2021). Stochastic optimal control analysis for the hepatitis B epidemic model. Results Phys. 104372.
- Din, A., Khan, A., Baleanu, D. (2020). Stationary distribution and extinction of stochastic coronavirus (COVID-19) epidemic model. Chaos Solitons Fractals. 139: 110036.
- 22. Khan, A., Hussain, G., Yusuf, A., Usman, A. H. (2021). A hepatitis stochastic epidemic model with acute and chronic stages. Adv. Differ. Equ. 2021(1): 1-10.

- 23. Liya, L. I. U., Jiang, D., Hayat, T., Ahmad, B. (2018). Dynamics of a hepatitis B model with saturated incidence. *Acta Math. Sci.* 38(6): 1731-1750.
- 24. Kiouach, D., Sabbar, Y. (2020). Ergodic stationary distribution of a stochastic hepatitis B epidemic model with interval-valued parameters and compensated poisson process. *Comput Math Methods Med.* 2020.
- Boukanjime, B., El Fatini, M. (2019). A stochastic Hepatitis B epidemic model driven by Lévy noise. Phys. A: Stat. Mech. 521: 796-806.
- 26. Khan, T., Khan, A., and Zaman, G. (2018). The extinction and persistence of the stochastic hepatitis B epidemic model. Chaos Solitons Fractals. 108: 123-128.
- 27. Khan, T., Khan, A., Zaman, G. (2018). The extinction and persistence of the stochastic hepatitis B epidemic model. Chaos Solitons Fractals. 108: 123-128.
- 28. Gray, A., Greenhalgh, D., Hu, L., Mao, X., Pan, J. (2011). A stochastic differential equation SIS epidemic model. SIAM J Appl Math. 71(3): 876-902.
- 29. Zhang, X., Jiang, D., Hayat, T., Ahmad, B. (2017). Dynamics of a stochastic SIS model with double epidemic diseases driven by Lévy jumps. *Phys. A: Stat. Mech.* 471: 767-777.
- 30. Song, Y., Miao, A., Zhang, T., Wang, X., Liu, J. (2018). Extinction and persistence of a stochastic SIRS epidemic model with saturated incidence rate and transfer from infectious to susceptible. Adv Differ Equ. 2018(1): 1-11.
- 31. Lahrouz, A., Settati, A., El Fatini, M., Pettersson, R., Taki, R. (2020). Probability analysis of a perturbed epidemic system with relapse and cure. *Int. J. Comput. Methods.* 17(03): 1850140.
- 32. Øksendal, B., Sulem, A. (2005). Stochastic Control of jump diffusions. Springer Berlin Heidelberg, pp. 39-58.
- 33. Heesterbeek, J. A. P. (1990). On the definition and the computation of the basic reproduction ratio R0 in models for infectious diseases in heterogeneous populations. *J. Math. Biol.* 28: 365-382.
- 34. Zhao, Y., Jiang, D., O'Regan, D. (2013). The extinction and persistence of the stochastic SIS epidemic model with vaccination. *Phys. A: Stat. Mech.* 392(20): 4916-4927.

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