



Impact of information intervention on stochastic hepatitis B model and its variable-order fractional network

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Abstract This paper aims at analyzing the dynamical behavior of a SIR hepatitis B epidemic stochastic model via a novel approach by incorporating the effect of information interventions and random perturbations. Initially, we demonstrate the positivity and global existence of the solutions. Afterward, we derive the stochastic threshold parameter \mathbf{R}_s , followed by the fact that this number concludes the transmission of hepatitis B from the population. By increasing the intensity of noise, we get \mathbf{R}_s less than one, inferring that ultimately hepatitis B will lapse. While decreasing the intensity of noise to a sufficient level, we have $\mathbf{R}_s > 1$. For the case $\mathbf{R}_s > 1$, adequate results for the presence of stationary distribution are achieved, showing the prevalence of hepatitis B. The present study also involves the derivation of the necessary conditions for the persistence of the epidemic. Finally, the main theoretical solutions are plotted through simulations. Discussion on theoretical and numerical results shows that utilizing random perturbations and information interventions have a pronounced impact on the syndrome's dynamics. Furthermore, since most communities interact with each other, and the disease spread rate is affected by this factor, a new variable-order fractional network of the stochastic hepatitis B model is offered. Subsequently, this study will provide a robust theoretical basis for comprehending worldwide SIR stochastic and variable-order fractional network-related case studies.

1 Introduction

Human history reveals that infectious diseases are the real source of threat and adversely affect an individual's health and socioeconomic status. Referring to the H7N9 bird flu (the first novel sub-type of influenza virus) originated from Anhui and Shanghai in 2013 [1, 2]. The H7N9 symptoms of the disease include high fever, particularly at the initial days of the infection. The disease continues to spread, and till now, the disease caused thousand of infections and hundreds of deaths. We also know that the recent COVID-19 outbreak (announced pandemic by the WHO) which almost affected the entire world and caused millions of infections and more than 1.17M deaths [2]. With the occurrence of infectious diseases, people from all sectors start thinking of treating and preventing the disease at the earliest. Government and policymakers are spending massive budgets on the treatment and control of such infectious diseases. To make mathematical or other strategies to

control the disease [3–5], so the very first intervention of the health officials is awareness about the disease [6, 7]. With the help of high awareness and health education through media coverage, the disease may be prevented or at least one can delay its occurrence [8, 9]. For example, during the H7N9 flu, media played an active role in reporting infections, deaths, symptoms, control measures, and many other features of the disease on daily basis [10]. In the present COVID-19, media are playing more active role and at every movement, people are aware of the disease' status both in and outside of the country [11]. Researchers suggest that the majority of the infectious disease could be significantly controlled and the infection rate may be drastically reduced with the help of information coverage. There are many diseases which could be cured by imposing both pharmaceutical and non-pharmaceutical intervention strategies [30]. Spread of some diseases can be controlled with psychotherapy, because majority of infections are directly connected with an individual mind [13]. It is observed that psychological counseling in a positive sense will

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generate positive emotions which is important in resetting health activities and controlling epidemics [13, 14]. In conclusion, we can argue that information interventions like health educations, psychological therapy, and media coverage may be considered.

It is interesting that good psychological advice can offer positive feelings to produce a negative impact of negative feelings caused by the perception of a negative emotion on the disease [13, 14, 30]. Infectious disease control and the development of healthy lives are ultimately boosted with the immediate effect of information [15]. Therefore, the interference of information (media reporting, wellbeing Psychological intervention, education) as a type of non-drug treatment is really relevant [9].

The mathematical formulation is a valuable method for explaining the dynamics of different outbreaks. A variety of ecologists, as well as mathematicians, have investigated the spread of mutual transmissible diseases with the help of different models describing the epidemics for understanding and monitoring. It is very commonly used to figure out the transmission as well as optimization of altered infectious diseases by models in term of various mood, i.e., deterministic epidemic models [20–22], fractional epidemic models [16–19], stochastic epidemic models [23–25], and age-structure models [26, 27]. Fractional epidemic models [28–30]. In the case of infectious disease modeling, the more appropriating one as the stochastic modeling [31–34], due to it offer an extra degree of realistic comparable to its deterministic model compartments. It is also investigated that the stochastic-based models provide more output rather than deterministic ones as we may construct the division of the predicted results by running a stochastic model many times, while deterministic model, on the other hand, will give only one predicted value [35–40]. It has also been taken with keen interest that many studies are investigated by considering the intervention with the help of the information in the infectious diseases modeling. For example, Joshi [41] considered SIR as a model for infectious diseases based on intervention with information and found that information could decrease infection rates. In addition, the influence of information on vaccination has been studied by Buonomo et al. [42], with the help of SEIR model. Similarly, another study has been performed to investigate the effects of information as well as to forecast the control of disease [43]. More recently, the extinction as well as the persistence analysis of an epidemic model for hepatitis B have been performed by Khan and their co-authors [44]. However, to the best of our knowledge, no one incorporates HBV epidemic model with information intervention as well as variable-order fractional network. We formulate a new stochastic system in mathematical form to discuss the hepatitis B dynamics under the effect of information with random perturbations. We study the qualitative analysis of the global results and show that the solution satisfied the properties of uniqueness. We also perform the extinction analysis to obtain the conditions for extinction. Moreover, the analysis of persistence will be also investigated. Then, we will study the stationary

distribution for the proposed model and then simulate the model to verify the analytical findings.

Also, in the current study, a new variable fractional-order network is proposed for modeling of hepatitis B. By studying the fractional order [45–56] or variable fractional-order network, we aim to consider the impact of the interaction between different communities on the disease rate in different societies. The relationships and travels between two countries, for instance, will definitely affect the rate of diseases in both of them. Hence, in this study, a network model is proposed. Also, to take advantage of fractional calculus, the model on the network is supposed to be fractional, which is the generalized form of integer model. In brief, by proposing this fractional-order network model, we hope to obtain more accurate results.

Organization of the paper: Sect. 2 is devoted to the formulation with biological feasibility. We show that the model preserves the properties of existence and uniqueness in Sect. 3, during the extinction with sufficient condition of stationary distribution as well as for persistence shown in Sects. 4, 6 and 5, respectively. Finally, the model results have been verified with the help of numerical simulations in Sect. 7. In Sect. 8, we show variable-order fractional network for the proposed model. The work has been finished in Sect. 9 with a brief conclusion.

2 Model formulation

Many authors and researchers have investigated by taking data about intervention into sequence infectious endemic systems like SIR, TB, HBV, and SEIR [41–43], and they realized that availability of data can minimize the level of infected cases. In our manuscript, we proposed an SIR epidemic model corresponding to hepatitis B along with the effect of stochastic and information intervention to investigate the dynamics of hepatitis B virus disease. Our newly formulated SIR hepatitis B virus model consists of susceptible, infected, recovered, as well as information intervention compartments. The following is our proposed SIR hepatitis B epidemic model:

$$\begin{aligned}\frac{dS(t)}{dt} &= \Lambda - \beta S(t)I(t) - \mu_2 m Z(t)S(t) - (v + \mu_0)S(t), \\ \frac{dI(t)}{dt} &= \beta I(t)S(t) - (\mu_0 + \gamma_1 + \mu_1)I(t), \\ \frac{dR(t)}{dt} &= \gamma_1 I(t) + \mu_2 m Z(t)S(t) + vS(t) - \mu_0 R(t), \\ \frac{dZ(t)}{dt} &= \frac{dI(t)}{(1 + hI(t))} - aZ(t),\end{aligned}\tag{1}$$

where $S(t)$, $I(t)$, and $R(t)$ symbolized by susceptible class, infected class, and recovered class, and $Z(t)$ represents the density of data availability in the of infected classes in the society. While, the model included parameters are summarized in the following list and assumed parameters will be positive:

- Λ : Per capita new born susceptible individuals.
- μ_0 : Natural death rate.
- μ_1 : HBV disease death rate.
- β : The infected rate of susceptible.
- v : Vaccination rate of HBV.
- μ_2 : The intensity response rate.
- γ_1 : The rate of recovering.
- m : Intervention information rate through which the behaviors of some individuals may changed their.
- d : Represents information rate.
- h : The constant of “Saturation”
- a : The decreasing rate of information.

We know the “basic reproduction number” \mathcal{R}_0 of model (1) is $\mathcal{R}_0 = \frac{\beta\Lambda}{(\mu_0 + \gamma_1 + \mu_1)(v + \mu_0)}$. The model (1) has two equilibrium points.

Disease-free equilibrium: If $1 < \mathcal{R}_0$, then the model (1) will have their free equilibrium point (DEE) is denoted by $E_1 = (S_1, I_1, R_1, Z_1) = (\frac{\Lambda}{v + \mu_0}, 0, \frac{v\Lambda}{\mu_0(v + \mu_0)}, 0)$, and the E_1 is asymptotically and globally stable.

Endemic equilibrium: If $1 < \mathcal{R}_0$, then the system (1) exists an endemic equilibrium (EE) which is denoted by $E_2 = (S_2, I_2, R_2, Z_2)$, and the E_2 is globally asymptotically stable.

Many researchers have defined and provide various random fluctuations for different diseases [57–59]. Among them is the well-known approach of fluctuating parameter in the considered system around an average value because of the consistent oscillation in the different environments [60, 60, 61]. It is clear that system (1) has the properties of a deterministic situation of modeling. Really, safe white noising will has a much more affect on infected population [23, 33, 34, 69], and the scholars pointed out that stochastic noising will bring changes in the basic number of reproduction for such type of disease. For the effect of environment noise, we may consider that the environment affect on different individuals is studied by the perturbations due to stochastic and it is related by proportionality to every compartment S, I, R . We used the standard Brownian motions is symbolized by $B_1(t), B_2(t)$ and $B_3(t)$ along with the intensities η_1, η_2 and η_3 . Obviously, the motion due to Brownian will obey the fundamental postulates of $B_1(0) = B_2(0) = B_3(0) = 0$. Then, we can write the the type of deterministic model (1) in stochastic style is given as

$$\begin{aligned}
 dS(t) &= [\Lambda - \mu_2 m Z(t) S(t) - \beta S(t) I(t) - (v + \mu_0) S(t)] dt \\
 &\quad + \eta_1 S(t) dB_1(t), \\
 dI(t) &= [\beta I(t) S(t) - (\mu_0 + \gamma_1 + \mu_1) I(t)] dt \\
 &\quad + \eta_2 I(t) dB_2(t), \\
 dR(t) &= [\gamma_1 I(t) + \mu_2 m Z(t) S(t) + v S(t) - \mu_0 R(t)] dt \\
 &\quad + \eta_3 R(t) dB_3(t), \\
 dZ(t) &= \left[\frac{dI(t)}{(1 + hI(t))} - aZ(t) \right] dt.
 \end{aligned} \tag{2}$$

However, considering the influence of information intervention and white noise with variable-order fractional network on system (1), we have made an attempt to raise and unfold the reasoning behind the following major issues: A1:

- A_1 : To address the impact of environmental noise on the HBV transmission.
- A_2 : To address which role does information intervention play in the HBV transmission of the disease.
- A_3 : To find the necessary requirement for the existence of stationary distribution.
- A_3 : In similarly way, what conditions are required for the variable-order fractional network.

3 Qualitative analysis of positive solution

In the given part, we study the the properties of obtain solution of the problem (2). Before discussion, we introduce some basic concept related to our main study. For this let $(\Omega, \{F_t\}_{t \geq 0}, P)$ i.e., completely probabilistic space which may satisfy the condition of $X(t) = (S(t), I(t), R(t), Z(t)), |X(t)| = (S^2(t) + I^2(t) + R^2(t) + Z^2(t))^{\frac{1}{2}}$, and $\mathbb{R}_+^b = \{x \in \mathbb{R}^b : \chi_i > 0, i = 1, 2, \dots, b\}$, where $\{F_t\}_{t \geq 0}$ is the filtration. We also consider a differential equation due to stochastic with b dimensions as

$$dx(t) = f(t, x(t))dt + g(t, x(t))dB(t) \quad t \geq t_0, \tag{3}$$

with $x(t(0)) = x_0$, where $x(0) \in \mathbb{R}^d$ and $B(t)$ is the standard m -dimensional Brownian motion. The linear operator \mathcal{L} for the above equation is defined by

$$\begin{aligned}
 \mathcal{L} &= \frac{\partial}{\partial t} + \sum_{i=1}^d f_i(x, t) \frac{\partial}{\partial x_i} \\
 &\quad + \frac{1}{2} \sum_{i,j=1}^d [g^T(x, t)g(x, t)]_{ij} \frac{\partial^2}{\partial x_i \partial x_j}.
 \end{aligned}$$

Similarly, if \mathcal{L} applied on V , and $V \in C^{2,1}(\mathbb{R}^d \times [t_0, \infty); \mathbb{R}_+)$, then

$$\mathcal{L}V = V_t + V_x f + \frac{1}{2} \text{trace} [g^T V_{xx} g].$$

Moreover

$$\begin{aligned}
 V_t &= \frac{\partial V}{\partial t}, \quad V_x = \left(\frac{\partial V}{\partial x_1}, \dots, \frac{\partial V}{\partial x_d} \right)^T, \\
 V_{xx} &= \left(\frac{\partial^2 V}{\partial x_i \partial x_j} \right)_{d \times d}.
 \end{aligned}$$

By generalized formula of Itô, it can be written as

$$dV = \mathcal{L}V dt + V_x g dB(t).$$

Now, with the help of above basic concepts, we are going to discuss the main result related to the existence and uniqueness analysis. To get these result, we have a bounded set Γ as given under

$$\Gamma = \left\{ (S, I, R, Z) \in R_+^4 : \frac{\Lambda}{\mu_0 + \mu_1} \leq S + I + R \leq \frac{\Lambda}{\mu_0}, 0 \leq Z \leq \frac{d\Lambda}{a(\mu_0 + h\Lambda)} \right\}. \tag{4}$$

Theorem 1 *The solution of system (2), i.e., $(Z(t), R(t), I(t), S(t))$ will be one for any $0 \leq t$ and for initial value $(Z(0), R(0), I(0), S(0),) \in R_+^4$, it will remain in R_+^4 with probability 1, namely, $(S(t), I(t), R(t), Z(t)) \in R_+^4 \forall 0 \leq t$.*

Proof Clearly, the coefficient are continuous locally lipschitz for $(Z(0), R(0), I(0), S(0))$ in R_+^4 , which show that the solution (Z, R, I, S) is unique and local in the interval $[0, \tau_e)$ at any t , where the explosion time is symbolized by τ_e (see for more detail [23,31,63]). If we can prove $\tau_e = \infty$ implies that the output is of global form. Take k_0 is a constant and assume that is non-negative, and be a large number, so that $Z(0), R(0), I(0)$ and $S(0)$ lies in $[k_0, \frac{1}{k_0}]$. Then, define $k_0 \leq k$ and

$$\tau_k = \{t \in [0, \tau_e) : \min\{Z(t), R(t), I(t), S(t)\} \leq \frac{1}{k} \text{ or } \max\{Z(t), R(t), I(t), S(t) \geq 0\}\}. \tag{5}$$

Now, define $\inf \Phi = \infty$, as Φ is the empty set. Here, τ_k is increasing as k tends to ∞ . Assume that τ_∞ is the limiting value, i.e., $\lim_{k \rightarrow \infty} \tau_k = \tau_\infty \leq \tau_e$ a.s. Here, it is enough to show that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$, which prove that $(S(t), I(t), R(t), Z(0)) \in R_+^4$ a.s., for every $t \geq 0$. We verify that $\tau_e = \infty$ a.s. to alternately to achieve the remaining derivation. If this not holds, then there will be two constants $T > 0$ lies and $\epsilon \in (0, 1)$, like

$$P\{\tau_\infty \leq T\} > \epsilon. \tag{6}$$

Therefore, integer $k_0 \leq k_1$ will be there as

$$P\{\tau_k \leq T\} \geq \epsilon, \text{ for all } k \geq k_1.$$

Define the following Lyapunov function:

$$H = \left(-C + S - C \frac{\log S}{C}\right) + (I - 1 - \log I) + (R - 1 - \log R) + (Z - 1 - \log Z). \tag{7}$$

Applying *Itô* formula to H , and C will be determined later

$$dH(S, I, R, Z) = \left[\left(1 - \frac{C}{S}\right) \left(\Lambda - \beta SI - \mu_2 m Z S - (\mu_0 + v) S\right) + \frac{1}{2} C \eta_1^2 \right] dt + \left(1 - \frac{C}{S}\right) \eta_1 S dB_1(t)$$

$$+ \left[\left(1 - \frac{1}{I}\right) \left(\beta SI - (\mu_0 + \gamma_1 + \mu_1) I\right) + \frac{1}{2} \eta_2^2 \right] dt + \left(1 - \frac{1}{I}\right) \eta_2 I dB_2(t) + \left[\left(1 - \frac{1}{R}\right) \left(\gamma_1 I + \mu_2 m Z S + v S - \mu_0 R\right) + \frac{1}{2} \eta_3^2 \right] dt + \left(1 - \frac{1}{R}\right) \eta_3 R dB_3(t) + \left[\left(1 - \frac{1}{Z}\right) \left(\frac{dI}{1 + hI} - aZ\right) \right] dt, = LH(S, I, R, Z) dt + \eta_1 (S - C) dB_1(t) + \eta_2 (I - 1) dB_2(t) + \eta_3 (R - 1) dB_3(t). \tag{8}$$

In Eq. (8), we define the $LH : R_+^4 \rightarrow R_+$ by the assertion

$$\begin{aligned} LH &= \left(1 - \frac{C}{S}\right) \left(\Lambda - \beta SI - \mu_2 m Z S - (\mu_0 + v) S\right) + \left(1 - \frac{1}{I}\right) \left(\beta SI - (\mu_0 + \gamma_1 + \mu_1) I\right) + \left(1 - \frac{1}{R}\right) \left(\gamma_1 I + \mu_2 m Z S + v S - \mu_0 R\right) + \left(1 - \frac{1}{Z}\right) \left(\frac{dI}{1 + hI} - aZ\right) + \frac{1}{2} C \eta_1^2 + \frac{1}{2} \eta_2^2 + \frac{1}{2} \eta_3^2, \\ &= \Lambda - \mu_2 m Z S - (v + \mu_0) S - \frac{C\Lambda}{S} + C\mu_2 m Z + C\beta I + C(v + \mu_0) - (\mu_0 + \gamma_1 + \mu_1) I - S\beta + (\mu_0 + \gamma_1 + \mu_1) + \gamma_1 I + Sv - R\mu_0 + \mu_2 m Z S - \gamma_1 \frac{I}{R} - \frac{vS}{R} + \mu_0 - \frac{\mu_2 m Z S}{R} + \frac{dI}{1 + hI} - aZ - \frac{dI}{(1 + hI)Z} + a + \frac{C\eta_1^2 + \eta_2^2 + \eta_3^2}{2} \\ &\leq \Lambda + C\beta I - (\mu_0 + \mu_1) I + C\mu_2 m Z + C(\mu_0 + v) + (\mu_0 + \gamma_1 + \mu_1) + \mu_0 + \frac{dI}{1 + hI} + a + \frac{C\eta_1^2 + \eta_2^2 + \eta_3^2}{2} \\ &\leq \Lambda + (C\beta - (\mu_0 + \mu_1)) I + \frac{C\mu_2 m d\Lambda}{a(\mu_0 + h\Lambda)} + C(\mu_0 + v) + 2\mu_0 + \gamma_1 + \frac{d}{h} + a + \frac{C\eta_1^2 + \eta_2^2 + \eta_3^2}{2} := K. \end{aligned} \tag{9}$$

Here, $C = \frac{\mu_0 + \mu_1}{\beta}$, such that $C\beta - (\mu_0 + \mu_1) = 0$

$$LH(S, I, R, Z) \leq \Lambda + \frac{C\mu_2 m d\Lambda}{a(\mu_0 + h\Lambda)}$$

$$\begin{aligned}
 &+C(\mu_0 + v) + 2\mu_0 + \gamma_1 + \frac{d}{h} \\
 &+ a + \frac{\frac{\mu_0 + \mu_1}{\beta} \eta_1^2 + \eta_2^2 + \eta_3^2}{2} := K. \tag{10}
 \end{aligned}$$

Here, K is a positive constant, so accordingly

$$\begin{aligned}
 &E \left[H(S, R, I)(\tau_k \wedge T) \right] \\
 &\leq H(Z(0), R(0), I(0), S(0)) \\
 &\quad + E \left[\int_0^{\tau_k \wedge T} K dt \right], \tag{11} \\
 &\leq H(Z(0), R(0), I(0), S(0)) + TK.
 \end{aligned}$$

Putting $\Omega_k = \tau_k \leq T$, $k \geq k_1$. Also, from Eq. (6), it could be noted that $P(\Omega_k) \geq \epsilon$. Consider $\omega \in \Omega_k$, at least one $S(\tau_k, \omega)$, $I(\tau_k, \omega)$, $R(\tau_k, \omega)$, $Z(\tau_k, \omega)$ exists, which must be equal of k or $\frac{1}{k}$. Therefore, $H(Z(\tau_k), R(\tau_k), I(\tau_k), S(\tau_k))$ is greater than $-1 + k - \log k$ or $\log + \frac{1}{k} + (-1)k$. Moreover

$$H(Z, R, I, S) \geq E(k - 1 - \log) \wedge \left(-1 + \log k + \frac{1}{k} \right). \tag{12}$$

Following Eqs. (6) and (11), we obtain:

$$\begin{aligned}
 &H(Z(0), R(0), I(0), S(0)) + TK \\
 &\geq E \left[1_{\Omega(\omega)} H(Z(\tau_k), R(\tau_k), I(\tau_k), S(\tau_k)) \right] \tag{13} \\
 &\geq \epsilon \left[(-1 + k - \log k) \wedge \left(-1 + \log k + \frac{1}{k} \right) \right].
 \end{aligned}$$

Here, the function $I_{\Omega(\omega)}$ is called the indicating mapping of Ω . Assuming $k \rightarrow \infty$ implies the results contradiction, i.e., TM, which means that $\tau_\infty = \infty$ a.s. \square

Lemma 1 *Let any of the starting values $(S(0), I(0), R(0), Z(0)) \in R_+^4$, have one solution of system (2) on $0 \leq t$ will in Γ having probability of 1.*

Proof We know that $N(t) = S(t) + I(t) + R(t)$, so we can obtain

$$\begin{aligned}
 dN(t) &= (\Lambda - \mu_0 N(t) - \mu_1 I(t)) dt + \eta_1 B_1(t) \\
 &\quad + \eta_2 B_2(t) + \eta_3 B_3(t), \tag{14} \\
 &\leq (\Lambda - \mu_0 N(t)) dt + \eta_1 B_1(t) \\
 &\quad + \eta_2 B_2(t) + \eta_3 B_3(t).
 \end{aligned}$$

Integrating Eq. (14) from $0 - t$. Thus, we implies that $\frac{\Lambda}{\mu_0 + \mu_1} \leq \frac{dN(t)}{dt} \leq \Lambda - \mu_0 N(t)$; then, we have

$$\begin{aligned}
 \frac{\Lambda}{\mu_0 + \mu_1} &\leq \liminf_{t \rightarrow \infty} N(t) \\
 &\leq \limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu_0}. \tag{15}
 \end{aligned}$$

It can be seen from the values of S , I , and R that the proposed model (2) is bounded by $\frac{\Lambda}{\mu_0}$. Last equation of system (2) implies that $I \leq \frac{\Lambda}{\mu_0}$; we get $\limsup_{t \rightarrow \infty} Z \leq \frac{d\Lambda}{a(\mu_0 + h\Lambda)}$. Therefore, we get Γ is the invariant, positive, and having bounds. After this, all the solution curves lying any where of R_+^4 will lie in Γ and having full probabilistic. \square

4 Extinction of the disease

The current section is devoted to discuss the extinction, and therefore, first, we define the basic reproductive parameter which is denoted by R_s for stochastic model as stated by Eq. (2) and define by

$$R_s = \frac{\beta \Lambda}{\mu_0 \left(\mu_0 + \gamma_1 + \mu_1 + \frac{\eta_2^2}{2} \right)}.$$

Theorem 2 *Let (Z_t, R_t, I_t, S_t) is the root of model (2) with $(Z_0, R_0, I_0, S_0) \in R_+^4$, if $R_s < 1$, After this, the root of stochastic hepatitis B system (2) satisfies*

$$\begin{aligned}
 \lim_{t \rightarrow \infty} \langle S(t) \rangle &= \frac{\Lambda}{\mu_0 + v} \quad a.s., \\
 \limsup_{t \rightarrow \infty} \frac{\log I_t}{t} &< 0 \quad a.s., \\
 \lim_{t \rightarrow \infty} \langle R(t) \rangle &= \frac{v\Lambda}{\mu_0(\mu_0 + v)} \quad a.s., \\
 \lim_{t \rightarrow \infty} \langle Z(t) \rangle &= 0 \quad a.s., \tag{16}
 \end{aligned}$$

namely the disease extinct with probability one.

Proof By directly integrating the proposed system (2), we could obtain the below system of equations

$$\begin{aligned}
 \frac{S(t) - S(0)}{t} &= \Lambda - \beta \langle S(t)I(t) \rangle - \mu_2 m \langle S(t)Z(t) \rangle \\
 &\quad - (\mu_0 + v) \langle S(t) \rangle + \frac{\eta_1}{t} \int_0^t S dB_1(s), \\
 \frac{I(t) - I(0)}{t} &= \beta \langle S(t)I(t) \rangle - (\mu_0 + \mu_1 + \gamma_1) \langle I(t) \rangle \\
 &\quad + \frac{\eta_2}{t} \int_0^t I dB_2(s), \tag{17} \\
 \frac{R(t) - R(0)}{t} &= \mu_1 \langle I(t) \rangle + \mu_2 m \langle S(t)Z(t) \rangle - \mu_0 \langle R(t) \rangle \\
 &\quad + v \langle S(t) \rangle + \frac{\eta_3}{t} \int_0^t R dB_3(s).
 \end{aligned}$$

One can calculate by Eq. (17), such that

$$\begin{aligned} & \frac{R(t) - R(0)}{t} + \frac{S(t) - S(0)}{t} + \frac{I(t) - I(0)}{t} \\ &= -(\mu_0 + \gamma_1)\langle I(t) \rangle + \Lambda - \mu_0\langle S(t) \rangle \\ & \quad - \mu_0\langle R(t) \rangle + \frac{\eta_1}{t} \int_0^t SdB_1(s) \\ & \quad + \frac{\eta_2}{t} \int_0^t IdB_2(s) + \frac{\eta_3}{t} \int_0^t RdB_3(s). \end{aligned} \tag{18}$$

Calculation leads to

$$\langle S(t) \rangle = \frac{\Lambda}{\mu_0} - \frac{\mu_0 + \gamma_1}{\mu_0} \langle I(t) \rangle - \langle R(t) \rangle + \phi(t), \tag{19}$$

where

$$\begin{aligned} \Phi(t) = & \left[-\frac{R(t) - R(0)}{t} - \frac{S(t) - S(0)}{t} - \frac{I(t) - I(0)}{t} \right. \\ & + \frac{\eta_1}{t} \int_0^t SdB_1(s) + \frac{\eta_2}{t} \int_0^t IdB_2(s) \\ & \left. + \frac{\eta_3}{t} \int_0^t RdB_3(s) \right] \frac{1}{\mu_0}. \end{aligned} \tag{20}$$

Utilizing *I*tô formula to the second equation of system (2), then

$$\begin{aligned} d\log I(t) = & 7 \left[\beta S(t) - (\mu_0 + \gamma_1 + \mu_1) - \frac{1}{2} \eta_2^2 \right] dt \\ & + \eta_2 dB_2(t). \end{aligned} \tag{21}$$

The integration leads to the following:

$$\begin{aligned} \frac{\log I_t - \log I_0}{t} = & \frac{1}{t} \int_0^t \left[\beta S - (\mu_0 + \mu_1 + \gamma_1) \right. \\ & \left. - \frac{1}{2} \eta_2^2 \right] ds + \frac{1}{t} \int_0^t \eta_2 dB_2(s). \end{aligned} \tag{22}$$

Following [64], i.e., the large number theorem and local martingales gives

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \int_0^t \eta_i dB_i(s) = 0, \tag{23}$$

Where $i = \{1, 2, 3, \dots\}$, and obviously, $\phi(t) = 0$ if we use Eq. (23) and $t \rightarrow \infty$

$$\begin{aligned} & \left[\beta S - (\mu_0 + \mu_1 + \gamma_1) - \frac{1}{2} \eta_2^2 \right] \\ & \leq \beta \left(\frac{\Lambda}{\mu_0} - \frac{\mu_0 + \mu_1}{\mu_0} \langle I(t) \rangle - \langle R(t) \rangle + \phi(t) \right) \\ & \quad - (\mu_1 + \mu_0 + \gamma_1) - \frac{1}{2} \eta_2^2 \\ & \leq \left(\mu_1 + \mu_0 + \gamma_1 + \frac{\eta_2^2}{2} \right) (\mathbf{R}_s - 1). \end{aligned} \tag{24}$$

Therefore we obtain

$$\limsup_{t \rightarrow \infty} \frac{\log I_t}{t} \leq \left(\mu_1 + \mu_0 + \gamma_1 + \frac{\eta_2^2}{2} \right) (\mathbf{R}_s - 1) < 0. \tag{25}$$

Denote $\Omega_1 = \{\omega \in \Omega : \limsup_{t \rightarrow \infty} I(\omega, t) = 0\}$. In the view of Eq. (25), we have

$$P(\omega_1) = 1; \tag{26}$$

this implies that at any non-negative ϵ_1 ; there is $T_1 = T_1(\omega, \epsilon_1)$, so $I(t) < \epsilon_1$, a.s, for $T_1 > t$, then

$$\begin{aligned} dZ(\omega, t) = & \left[\frac{dI(\omega, t)}{I + hI(\omega, t)} - aZ(\omega, t) \right] dt \\ & \leq [d\epsilon_1 - aZ(\omega, t)] dt \text{ for } \omega \in \Omega_1, t \leq T_1. \end{aligned} \tag{27}$$

Comparison of Theorem [65] gives

$$\begin{aligned} Z(\omega, t) \leq & e^{-at} (Z(T_1) + \int_{T_1}^t d\epsilon_1 \cdot e^{-as} ds) \\ & \leq Z(T_1) e^{-dt} + \frac{d\epsilon_1}{a} \text{ for } \omega \in \Omega_1, t \leq T_1. \end{aligned} \tag{28}$$

Then

$$\limsup_{t \rightarrow \infty} Z(\omega, t) \leq \frac{d\epsilon_1}{a} \text{ for } \omega \in \Omega_1, t \leq T_1. \tag{29}$$

As ϵ_1 is arbitrary, which implies $0 \leq \limsup_{t \rightarrow \infty} Z$. Also, $\limsup_{t \rightarrow \infty} Z(\omega, t) \geq 0$. Because of this

$$\lim_{t \rightarrow \infty} \langle Z(\omega, t) \rangle = 0. \tag{30}$$

Let $\Omega_2 = \omega \in \Omega : \limsup_{t \rightarrow \infty} Z(\omega, t) = 0 \subset \Omega_1$, and then, for $\epsilon_2 \leq 0$, there is a constant $T_2 = T_2(\omega, t) \leq T_1$ exists and so $Z(t) < \epsilon_2$, a.s. for $t > T_2$.

Now, put Eqs. (23), (25), and (30) in Eq. (19), we can obtain

$$\lim_{t \rightarrow \infty} \langle R(t) + S(t) \rangle = \frac{\Lambda}{\mu_0} \text{ s.a.} \tag{31}$$

From first equation of model Eq. (17), we have

$$\begin{aligned} \frac{S(t) - S(0)}{t} = & \Lambda - \beta \langle S(t)I(t) \rangle - \mu_2 m \langle S(t)Z(t) \rangle \\ & - (\mu_0 + v) \langle S(t) \rangle + \frac{\eta_1}{t} \int_0^t SdB_1(s), \end{aligned} \tag{32}$$

which implies that

$$\langle S(t) \rangle = \frac{\Lambda}{(\mu_0 + v)} - \frac{\beta \langle S(t)I(t) \rangle}{(\mu_0 + v)}$$

$$\frac{\mu_2 m \langle S(t)Z(t) \rangle}{(\mu_0 + v)} - \frac{S(t) - S(0)}{t} + \frac{\eta_1}{t} \int_0^t S dB_1(s); \tag{33}$$

consequently, Eqs. (23), (25), (30), and (20), we can get

$$\lim_{t \rightarrow \infty} \langle S(t) \rangle = \frac{\Lambda}{(\mu_0 + v)}, \quad \text{s.a.}$$

Now, we have

$$\lim_{t \rightarrow \infty} \langle R(t) \rangle = \frac{v\Lambda}{\mu_0(\mu_0 + v)} \quad \text{s.a.}$$

The last equation is completion of our proof. □

5 Persistence of the stochastic hepatitis B model

Now, here, we are going to present the condition for the existence of solution for system (2), while the main work of the on-going part is defined by the below result and some well-known axioms.

Lemma 2 (Strong Law) [34, 35] *Let $M = \{M\}_{0 \leq t}$ be continuous and real valued along with local martingale which vanish as $t \rightarrow 0$, and then*

$$\lim_{t \rightarrow \infty} \langle M, M \rangle_t = \infty, \text{ a.s.}, \Rightarrow \lim_{t \rightarrow \infty} \frac{M_t}{\langle M, M \rangle_t} = 0, \text{ a.s.} \tag{34}$$

$$\limsup_{t \rightarrow \infty} \frac{\langle M, M \rangle_t}{t} < \infty, \text{ a.s.}, \Rightarrow \lim_{t \rightarrow \infty} \frac{M_t}{t} = 0, \text{ a.s.}$$

Lemma 3 [34] *Let $(S(t), I(t), R(t), Z(t))$ be the solution of system (2) with $(S(0), I(0), R(0), Z(0)) \in \mathbb{R}_+^4$, and then, $\limsup_{t \rightarrow \infty} (S(t) + I(t) + R(t) + Z(t)) < \infty$, a.s. Moreover*

$$\lim_{t \rightarrow \infty} \frac{S(t)}{t} = 0, \quad \lim_{t \rightarrow \infty} \frac{I(t)}{t} = 0, \quad \lim_{t \rightarrow \infty} \frac{R(t)}{t} = 0, \quad \lim_{t \rightarrow \infty} \frac{Z(t)}{t} = 0 \quad \text{a.s.}, \tag{35}$$

$$\lim_{t \rightarrow \infty} \frac{\ln S(t)}{t} = 0, \quad \lim_{t \rightarrow \infty} \frac{\ln I(t)}{t} = 0, \quad \lim_{t \rightarrow \infty} \frac{\ln R(t)}{t} = 0, \quad \lim_{t \rightarrow \infty} \frac{\ln Z(t)}{t} = 0 \quad \text{a.s.}, \tag{36}$$

and

$$\begin{aligned} \lim_{t \rightarrow \infty} \frac{\int_0^t S(u) dB_1(u)}{t} &= 0, & \lim_{t \rightarrow \infty} \frac{\int_0^t I(u) dB_2(u)}{t} &= 0, \\ \lim_{t \rightarrow \infty} \frac{\int_0^t R(u) dB_3(u)}{t} &= 0, & \lim_{t \rightarrow \infty} \frac{\int_0^t Z(u)}{t} &= 0, \quad \text{a.s.} \end{aligned} \tag{37}$$

Then, the solution of (2)

$$\begin{aligned} \limsup_{t \rightarrow \infty} (S(t) + I(t) + R(t)) &= \frac{\Lambda}{\mu_0}, \\ \limsup_{t \rightarrow \infty} Z(t) &= \frac{d}{ah} \quad \text{a.s.} \end{aligned} \tag{38}$$

Definition 1 [69] The proposed model (2) is said to be persistent, if

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \int_0^t I(r) dr > 0 \quad \text{a.s.} \tag{39}$$

Theorem 3 *If $\mathbf{R}_0^s = \frac{\beta\Lambda}{(\mu_0 + v + \frac{\mu_3 md}{ah} + \frac{\eta_1^2}{2})(\mu_0 + \mu_1 + \gamma_1 + \frac{\eta_2^2}{2})}$ then for any initial value $(S(0), I(0), R(0), Z(0)) \in \mathbb{R}_+^4$, the disease $I(t)$ has the axiom*

$$\liminf_{t \rightarrow \infty} \langle I(t) \rangle \geq \frac{2\Lambda\sqrt{\mathbf{R}_0^s - 1}}{c_1\beta} \quad \text{a.s.}, \tag{40}$$

where $c_1 = \frac{\Lambda}{(\mu_0 + v + \frac{\mu_3 md}{ah} + \frac{\eta_1^2}{2})}$. Then, we can say that the disease will prevail if $\mathbf{R}_0^s > 1$.

Proof Set

$$V_1 = -c_1 \ln S - c_2 \ln I, \tag{41}$$

where c_1 and c_2 are constants and we will find later. Applying Itô formula, so we have

$$\begin{aligned} dV_1 &= \mathcal{L}V_1 - c_1\eta_1 dB_1(t) - c_2\eta_2 dB_2(t), \tag{42} \\ \mathcal{L}V_1 &= c_1\mathcal{L}(-\ln S) + c_2\mathcal{L}(-\ln I), \\ &= -c_1 \frac{\Lambda}{S} + c_1\beta I + c_1\mu_3 mZ + c_1(\mu_0 + v) \\ &\quad + c_1 \frac{1}{2} \eta_1^2 - c_2\beta S + c_2(\mu_0 + \mu_1 + \gamma_1) \\ &\quad + c_2 \frac{1}{2} \eta_2^2, \\ &\leq -c_1 \frac{\Lambda}{S} - c_2\beta S + c_1(\mu_0 + v + \frac{\mu_3 md}{ah}) \\ &\quad + \frac{1}{2} \eta_1^2 + c_2(\mu_0 + \mu_1 + \gamma_1) \\ &\quad + \frac{1}{2} \eta_2^2 + c_1\beta I, \\ &\leq -2\sqrt{c_1 c_2 \Lambda \beta} + c_1(\mu_0 + v + \frac{\mu_3 md}{ah}) \\ &\quad + \frac{1}{2} \eta_1^2 + c_2(\mu_0 + \mu_1 + \gamma_1) \\ &\quad + \frac{1}{2} \eta_2^2 + c_1\beta I. \end{aligned} \tag{43}$$

Let

$$\begin{aligned}
 c_1 &= \frac{\Lambda}{\left(\mu_0 + v + \frac{\mu_3 m d}{ah} + \frac{1}{2}\eta_1^2\right)}, \\
 c_2 &= \frac{\Lambda}{\left(\mu_0 + \mu_1 + \gamma_1 + \frac{1}{2}\eta_2^2\right)}, \\
 \mathcal{L}V_1 &\leq -2\sqrt{\frac{\Lambda^2\beta\Lambda}{\left(\mu_0 + v + \frac{\mu_3 m d}{ah} + \frac{1}{2}\eta_1^2\right)\left(\mu_0 + \mu_1 + \gamma_1 + \frac{1}{2}\eta_2^2\right)}} \\
 &\quad + 2\Lambda + c_1\beta I, \\
 &= -2\Lambda\left(\sqrt{\frac{\beta\Lambda}{\left(\mu_0 + v + \frac{\mu_3 m d}{ah} + \frac{1}{2}\eta_1^2\right)\left(\mu_0 + \mu_1 + \gamma_1 + \frac{1}{2}\eta_2^2\right)}} - 1\right) \\
 &\quad + c_1\beta I, \\
 &= -2\Lambda\left(\sqrt{\mathbf{R}_0^s} - 1\right) + c_1\beta I.
 \end{aligned} \tag{44}$$

Substituting Eqs. (45) into (41), and then integrating both side of the stochastic hepatitis B model (2)

$$\begin{aligned}
 &\frac{V_1(S(t), I(t)) - V_1(S(0), I(0))}{t} \\
 &\leq -2\Lambda\left(\sqrt{\mathbf{R}_0^s} - 1\right) + c_1\beta\langle I \rangle - \frac{c_1\eta_1^2 B_1(t)}{t} \\
 &\quad - \frac{c_2\eta_2^2 B_2(t)}{t} \\
 &= -2\Lambda\left(\sqrt{\mathbf{R}_0^s} - 1\right) + c_1\beta\langle I \rangle + \Psi(t),
 \end{aligned} \tag{46}$$

where $\Psi(t) = -\frac{c_1\eta_1^2 B_1(t)}{t} - \frac{c_2\eta_2^2 B_2(t)}{t}$. From strong law as stated in Lemma (2), we arrive

$$\lim_{t \rightarrow \infty} \Psi(t) = 0. \tag{47}$$

From Eq. (46), we have

$$\begin{aligned}
 \langle I(t) \rangle &\geq \frac{2\Lambda(\sqrt{\mathbf{R}_0^s} - 1)}{c_1\beta} - \frac{1}{c_1\beta}\Psi(t) \\
 &\quad + \frac{1}{c_1\beta}\left(\frac{V_1(S(t), I(t)) - V_1(S(0), I(0))}{t}\right).
 \end{aligned} \tag{48}$$

According to Lemma (3) and Eq. (47), and the limit superior of Eq. (5), we have

$$\liminf_{t \rightarrow \infty} \langle I(t) \rangle \geq \frac{2\Lambda(\sqrt{\mathbf{R}_0^s} - 1)}{c_1\beta} \text{ a.s.} \tag{49}$$

This finishes the proof of Theorem (3). \square

6 Stationary distribution and ergodicity of the stochastic hepatitis B model

We follow Hasminskii et al. [66] and will prove the ergodicity, and stationary distribution for the model (2)

solution. Let $X(t)$ be Markov process and regular time and homogeneous over R^d

$$dX(t) = bXdt + \sum_{r=1}^k \sigma_r(X)dB_r(t). \tag{50}$$

The diffusion array is define as follows:

$$A(x) = (a_{ij}(x)), \quad a_{ij}(t) = \sum_{r=1}^k \sigma_r^i(t)\sigma_r^j(x).$$

Lemma 4 *The Markov process $X(t)$ has ergodic stationary distribution $\Pi(\cdot)$ and unique a bounded open domain $U \subset R^d$ with regular boundary U exists and holds the following [67, 68]:*

1. *There is a positive number ϖ will positive, such that $\sum_{i,j=1}^d a_{ij}(x)\zeta_1\zeta_2 \geq \varpi|\zeta|^2, x \in U, \zeta \in R^d$.*
2. *Positive function C^2 and mapping V exist: LV is negative $\forall x \in R^d \setminus U$. Moreover, assume $\rho(\cdot)$ be a function which is integrable w.r.t $\pi(\cdot)$, and then, for every $x \in R^d \setminus U$*

$$P\left\{\lim_{T \rightarrow \infty} \frac{1}{T} \int_0^T \rho(X(t))dt = \int_{R^d} \rho(x)\pi(dx)\right\} = 1.$$

We present the theorem stated below for the required stationary process as well as ergodicity.

Theorem 4 *For the proposed system (2) with $(S_0, I_0, R_0, Z_0) \in R_+^4$, if $\mathbf{R}_s > 1$, then the stationary distribution $\pi(\cdot)$ exists, and is ergodic.*

Proof We assume α_1 and α_2 be numbers and are sufficiently large, such that

$$U = \left\{ (x_1, x_2, x_3, x_4) \in \Gamma \left. \begin{aligned} &\frac{1}{\alpha_1} \leq x_1, x_2, x_3 \leq \frac{\Lambda}{\mu_0} \\ &-\frac{1}{\alpha_1}, \frac{1}{\alpha_2} \leq x_4 \leq \frac{d\Lambda}{a(\mu_0 + h\Lambda)} - \frac{1}{\alpha_2} \end{aligned} \right\}. \tag{51}$$

It is also to be noted that system (2) can also be written as

$$\begin{aligned}
 d \begin{bmatrix} S \\ I \\ R \end{bmatrix} &= \begin{bmatrix} \Lambda - \beta S(t)I(t) - \mu_2 m Z(t)S(t) - (\mu_0 + v)S(t) \\ \beta S(t)I(t) - I(t)(\gamma_1 + \mu_1 + \mu_0) \\ \gamma_1 I(t) + \mu_2 m Z(t)S(t) + vS(t) - \mu_0 R(t) \\ \frac{dI(t)}{(1+hI(t))} - aZ(t) \end{bmatrix} dt \\
 &\quad + \begin{bmatrix} \eta_1 S(t)dB_1(t) \\ \eta_2 I(t)dB_2(t) \\ \eta_3 R(t)dB_3(t) \\ 0 \end{bmatrix}.
 \end{aligned}$$

Consequently, the diffusion matrix for the associated problem (2) is as

$$B = \begin{bmatrix} \eta_1^2 S^2 & 0 & 0 & 0 \\ 0 & \eta_2^2 I^2 & 0 & 0 \\ 0 & 0 & \eta_3^2 R^2 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

As U is subset of R_+^4 while $\xi \in R_+^4 \setminus \{(\xi_1, \xi_2, \xi_3, \xi_4) \in R_+^4 : \xi_1 = \xi_2 = \xi_3 = \xi_4\}$, then a positive number C exists as

$$\begin{aligned} & \sum_{i,j=1}^4 a_{ij}(S, I, R, Z)\xi_i\xi_j \\ &= \eta_1^2 S^2 \xi_1^2 + \eta_2^2 I^2 \xi_2^2 + \eta_3^2 R^2 \xi_3^2 + Z^2 \xi_4^2 \geq C \end{aligned} \tag{52}$$

Then, condition (1) of Lemma (4) holds. Let $V_1(I)$, $V_2(S, I)$ and $V_3(S, R, Z)$ be the function for $(S, I, R, Z) \in R_+^4$ and defined by

$$\begin{aligned} V_1(I) &= \frac{1}{q} I^{-q}, \\ V_2(S, I) &= \frac{1}{q} I^{-q} \left(\frac{\Lambda}{\mu_0} - S \right), \\ V_3(S, R, Z) &= \frac{1}{S} + R + \frac{1}{Z}, \end{aligned} \tag{53}$$

where q is positive constant and we will determine it later. Defining a function like

$$\begin{aligned} V(S, I, R, Z) &= V_1(I) + V_2(S, I) + V_3(S, R, Z), \\ V(S, I, R, Z) &= \frac{1}{q} I^{-q} + \frac{1}{q} I^{-q} \left(\frac{\Lambda}{\mu_0} - S \right) \\ &+ \left(\frac{1}{Z} + \frac{1}{S} + R \right). \end{aligned} \tag{54}$$

By the expression $It\hat{o}$ and the application of model (2), we obtain

$$\begin{aligned} LV_1(I) &= -I^{-q} (S\beta - (\mu_0 + \mu_1 + \gamma_1)) - \frac{1}{2}(q+1)\eta_2^2 I^{-q} \\ &= I^{-q} \left(- \left(\mu_0 + \mu_1 + \gamma_1 + \frac{\eta_2^2}{2} \right) (\mathbf{R}_s - 1) \right. \\ &\quad \left. - \frac{q}{2}\eta_2^2 \right) + I^{-q}\beta \left(\frac{\Lambda}{\mu_0} - S \right). \end{aligned} \tag{55}$$

Then, we compute $LV_2(S, I)$

$$\begin{aligned} LV_2(S, I) &= I^{-q} \left(\frac{\Lambda}{\mu_0} - S \right) \left((\mu_0 + \mu_1 + \gamma_1) - S\beta \right. \\ &\quad \left. + \frac{1}{2}(q+1)\eta_2^2 \right) - \frac{1}{q} I^{-q} \\ &\quad (\Lambda - \beta SI - \mu S\mu_2 m Z S + \mu_0 R) \\ &\leq I^{-q} \left(\frac{\Lambda}{\mu_0 + v} - S \right) \left(\mu_0 + \mu_1 + \gamma_1 - \frac{\mu_0}{q} \right. \\ &\quad \left. + \frac{1}{2}(q+1)\eta_2^2 \right) + \frac{1}{q} \mu_2 m Z S I^{-q} + \frac{\beta \Lambda}{q\mu_0} I^{-q}. \end{aligned} \tag{56}$$

For $V_3(S, R, Z)$, it implies that

$$\begin{aligned} V_3(S, R, Z) &= -\frac{\Lambda}{S^2} + \frac{\beta I}{S} + \frac{\mu_0 + v}{S} + \frac{\mu_2 m Z}{S} - \frac{\eta_1^2}{S} \\ &\quad + \gamma_1 I(t) + \mu_2 m Z(t) S(t) + v S(t) \\ &\quad - \mu_0 R(t) \frac{dI}{(I+hI)Z^2} + \frac{a}{Z} \\ &\leq -\frac{\Lambda}{S^2} + \left[\frac{\beta \Lambda}{\mu_0} + \mu_0 + \frac{\mu_1 m d \Lambda}{a_\mu (\mu + h \Lambda)} \right. \\ &\quad \left. - \frac{\mu_2^2 d^2 \Lambda^2}{a^2 (\mu_0 + h \Lambda)^2 - \frac{e t a_1^2}{2}} - \frac{\eta_1^2}{2} \right] \frac{1}{S} \\ &\quad + \frac{\gamma_1 \Lambda}{\mu_0} + \frac{\mu_2 m d \Lambda^2}{a \mu_0 (\mu_0 + h \Lambda)} \\ &\quad + \frac{d \mu_0 I}{(\mu_0 + h \Lambda) Z^2} \frac{d}{Z}. \end{aligned} \tag{57}$$

Combining (55), (56), and (57), we have

$$\begin{aligned} LV &\leq I^{-q} \left(-(\mu_0 + \mu_1 + \gamma_1 + \frac{\eta_2^2}{2})(\mathbf{R}_s - 1) - \frac{q}{2}\eta_2^2 \right) \\ &\quad + I^{-q} \left(\frac{\Lambda}{\mu_0 + v} - S \right) (\mu_0 + \beta + \mu_1 + \gamma_1 - \frac{\mu_0}{q}) \\ &\quad + \frac{1}{2}(q+1)\eta_2^2 + \frac{1}{q} \mu_2 m Z S I^{-q} + \frac{\beta \Lambda}{q\mu_0} I^{-q} \\ &\quad - \frac{\Lambda}{S^2} + \left[\frac{\beta \Lambda}{\mu_0} + \mu_0 + \frac{\mu_1 m d \Lambda}{a_\mu (\mu + h \Lambda)} \right. \\ &\quad \left. + \frac{\mu_2^2 d^2 \Lambda^2}{a^2 (\mu_0 + h \Lambda)^2} - \frac{\eta_1^2}{2} \right] \frac{1}{S} \\ &\quad + \frac{\gamma_1 \Lambda}{\mu_0} + \frac{\mu_2 m d \Lambda^2}{a \mu_0 (\mu_0 + h \Lambda)} + \frac{d \mu_0 I}{(\mu_0 + h \Lambda) Z^2}. \end{aligned} \tag{58}$$

Since $\mathbf{R}_s \geq 0$, and choose sufficiently small, such that

$$\begin{aligned} & -(\mu_0 + \mu_1 + \gamma_1 + \frac{\eta_2^2}{2})(\mathbf{R}_s - 1) - \frac{q}{2}\eta_2^2 < 0 \\ & \mu_0 + \mu_1 + \gamma_1 + \beta - \frac{\mu_0}{q} - \frac{1}{2}(1+q)\eta_2^2 < 0. \end{aligned}$$

Likewise, $\frac{\Lambda}{\mu_0 + \mu_1} \leq S + I + R \leq Z \leq \frac{d\Lambda}{a(\mu + h\Lambda)}$, then for $(S, I, R, Z) \in \Gamma U$ either $S \leq \frac{1}{\alpha_1} I \leq \frac{1}{\alpha_1} R \leq \frac{1}{\alpha_1} Z$ or $Z \leq \frac{1}{\alpha_2}$. It is simple from Eq. (58), as for α_1 or α_2 , sufficiently large $LV \leq -1$ for $(S, I, R, Z) \in \Gamma \setminus U$. Therefore, Lemma (4) holds and the model (2) admits an ergodic invariant distribution $\Pi(\cdot)$, which is unique. Hence, (S_t, I_t, R_t, Z_t) is ergodic and

$$P \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \chi(S_s, I_s, R_s, Z_s) \in \Gamma ds = \int_{R^4} \chi \Gamma^\pi(dx) \right\} = 1, \tag{59}$$

where $\chi \Gamma$ is the characteristic function of Γ . □

7 Numerical simulation of the stochastic hepatitis B model

Now, in this part, we are presenting some approximate solution by simulation to understand about the theoretical results. For this, we have applied the stochastic Runge–Kutta method to obtain the following discretization transformation of system (2):

$$\begin{aligned} S_{i+1} &= S_i + \left[\Lambda - \mu_2 m Z_i S_i - \beta S_i I_i - (\mu_0 + v) S_i \right] \Delta t \\ &\quad + \eta_1 S_i \sqrt{\Delta t} \zeta_{1,i} + \frac{\eta_1^2}{2} S_i (\zeta_{1,i}^2 - 1) \Delta t, \\ I_{i+1} &= I_i + \left[\beta S_i I_i - (\mu_0 + \gamma_1 + \mu_1) I_i \right] \Delta t \\ &\quad + \eta_2 I_i \sqrt{\Delta t} \zeta_{2,i} + \frac{\eta_2^2}{2} I_i (\zeta_{2,i}^2 - 1) \Delta t, \end{aligned} \tag{60}$$

$$\begin{aligned} R_{i+1} &= R_i + \left[(\gamma_1 I_i + \mu_2 m Z_i S_i + v S_i - \mu_0 R_i) \right] \Delta t \\ &\quad + \eta_3 R_i \sqrt{\Delta t} \zeta_{3,i} + \frac{\eta_3^2}{2} R_i (\zeta_{3,i} - 1) \Delta t, \\ Z_{i+1} &= Z_i + \left[\frac{dI_i}{(1 + hI_i)} - a Z_i \right] \Delta t. \end{aligned}$$

We perform approximate solution by simulation techniques of the considered hepatitis B stochastic disease system (2) and (1). For this propose, we use the well-known “stochastic Runge-Kutta method”. This analysis verify our analytical findings of the influence of environmental noise and information intervention. It has been found that white noise and information intervention have a big effects on the disease. Now, we are going to assume the numerical value of the parameters with biological feasibility to verify the extinction and the diseases free equilibrium E_1 result are as, see Fig. 1. Here, we take the noise $\eta_1 = 0.25, \eta_2 = 0.12, \eta_3 = 0.525$, and the parameters have the assigned values given as $\Lambda = 5.5, \beta = 0.015, \mu_0 = 0.4, \gamma_1 = 0.31, \mu_1 = 0.4, m = .02, \mu_2 = 0.1, v = 0.04, h = 0.4, d = 0.4$, and $a = 0.9$, whereas the initial population sizes will be considered as aforementioned. The result of Theorem 2 shows the behavior of the proposed equation (2) and at the equilibrium point E_1 before infection is stochastically asymptotically stable. Figure 1a and b shows that the stochastic system (2) and the deterministic system (1), respectively, have the same behavior. Both the model’s solutions are converging to the disease-free equilibrium point E_1 . This shows that the disease will extinct, i.e., the infection cases will approaches to zero, while there will be always susceptible population.

In a similar fashion, we assume the following parameter value and the intensity of white noise to show the permanence or stationary distribution and endemic equilibrium E_2 , i.e., $\Lambda = 5.5, \beta = 0.015, \mu_0 = 0.2, \gamma_1 = 0.1, \mu_1 = 0.004, m = 0.2, \mu_2 = 0.2, v = 0.04, h = 0.4$,

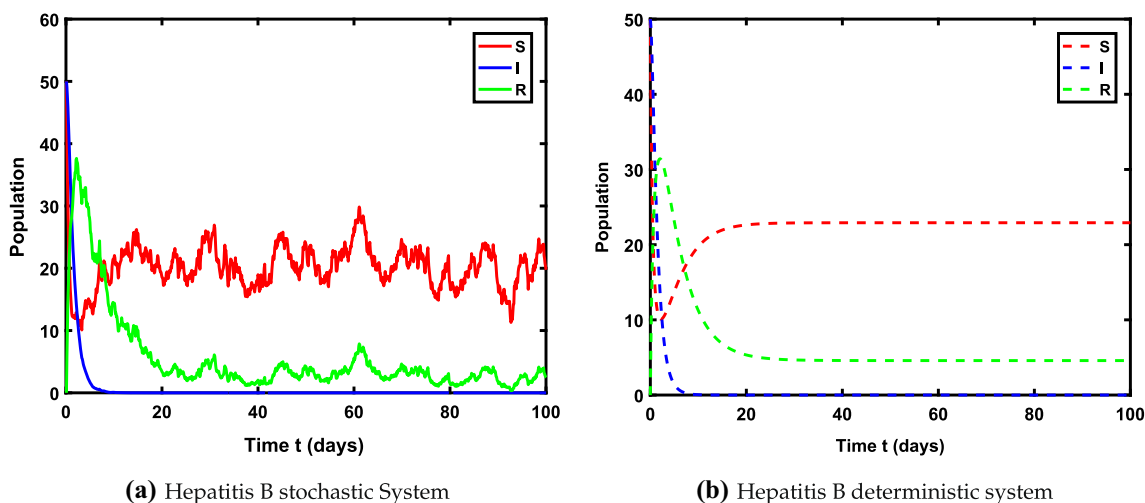


Fig. 1 The trajectories show the dynamics of $S(t), I(t)$, and $R(t)$: **a** represents the stochastic model (2) and **b** represents its corresponding deterministic model

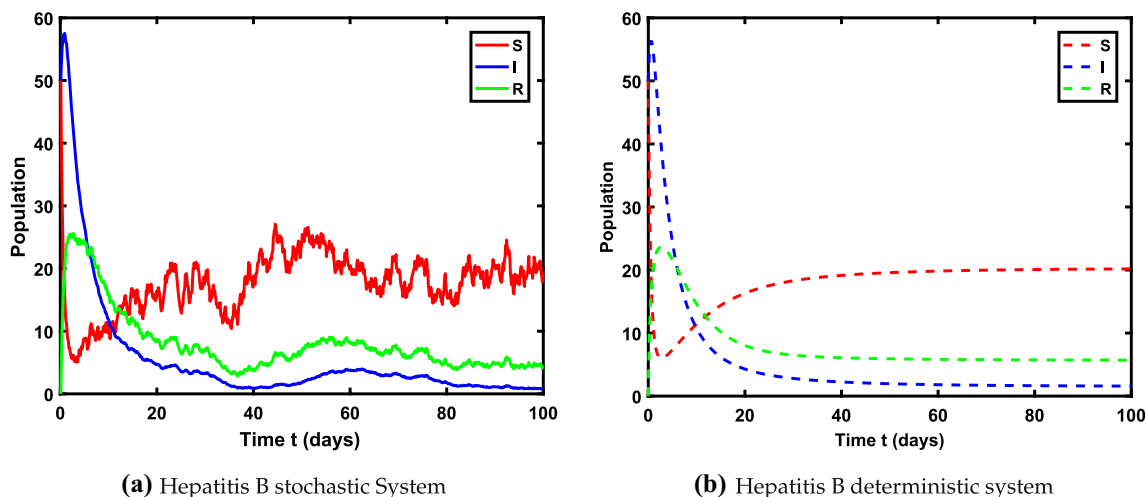


Fig. 2 The trajectories shows the dynamics of $S(t)$, $I(t)$, and $R(t)$: **a** represents the stochastic model (2) and **b** represents its corresponding deterministic model

$d = 0.2$ and $a = 0.9$, and the noise intensities are $\eta_1 = 0.25$, $\eta_2 = 0.12$, and $\eta_3 = 0.25$, while the initial population sizes will be taken as above. The Theorem 3 axioms are satisfied. Therefore, the stochastic system (2) solutions moves up and down for a more time through the feasible unique disease equilibrium E_2 of the idealistic system (1), and the mean motion through the disease equilibrium point E_2 are low because of the small noise waves that the disease come back will exist. Models of both types tend to the disease equilibrium point E_2 (shown in Fig. 2a and b). Figure 2 shows the comparison between Eq. (1) and Eq. (2). Once again the three curves in Fig. 3a shows the dynamical behavior of susceptible, infected, and recovered population, which shows that the model maintains the permanence.

Furthermore, for the stationary distribution, to display the effect of the noise intensity on the stationary distribution, we have run the numerical simulation 1000 times and obtain the average extinction time of $S(t)$, $I(t)$, and $R(t)$ at $t = 351$, and their distributions are exhibited in Fig. 3 (see for more details Fig. 3a–c). Then, the conditions of Theorem 4 are satisfied.

8 Variable-order fractional network

Herein, we aim to answer this question: How can we model the impact of the disease rate in different societies. In other words, we want to embed the interaction between different communities in the modeling of the disease. For instance, the relationships and travels between two countries will definitely affect the rate of diseases in both of them. To the best of our knowledge, up to now, no study has investigated this matter in the modeling of hepatitis B. We proposed to consider the model of the disease as neurons affected by each other in a network. Also, the fractions calculus, which is the generalized form of integer one and provides more

accurate results, is considered for the disease modeling. To construct a network of stochastic hepatitis B model, we couple N numbers of these models through the nearest-neighbor method. It is supposed that the interaction between neurons is through the susceptible components, i.e., the coupling is made on the variable S . Consequently, the equations of the network is described as

$$\begin{aligned} \frac{d^{q(t)} S_i}{dt^{q(t)}} &= \Lambda - \beta S_i(t) I_i(t) - \mu_2 m Z_i(t) S_i(t) \\ &\quad - (v + \mu_0) S_i(t) \\ &\quad + \frac{l}{2p} \sum_{j=i-p}^{i+p} (S_j - S_i) \\ \frac{d^{q(t)} I_i}{dt^{q(t)}} &= \beta I_i(t) S_i(t) - (\mu_0 + \gamma_1 + \mu_1) I_i(t) \\ \frac{d^{q(t)} R_i}{dt^{q(t)}} &= \gamma_1 I_i + \mu_2 m Z_i(t) S_i(t) + v S_i(t) - \mu_0 R_i(t) \\ \frac{d^{q(t)} Z_i}{dt^{q(t)}} &= \frac{dI_i(t)}{(1 + hI_i(t))} - a Z_i(t), \end{aligned} \tag{61}$$

where $q(t)$ indicates the time-varying fractional-order derivative. l is the coupling strength, P is the number of nearest neighbors on each side, and N denotes the number of systems in the network. Also, similar to (2), using the standard Brownian motions, i.e., $B_1(t)$, $B_2(t)$, and $B_3(t)$ along with the intensities h_1 , h_2 and h_3 , we can propose the type of deterministic model (1) in stochastic style as follows:

$$\begin{aligned} d^{q(t)} S_i &= [\Lambda - \beta S_i(t) I_i(t) - \mu_2 m Z_i(t) S_i(t) \\ &\quad - (v + \mu_0) S_i(t) + \frac{l}{2p} \sum_{j=i-p}^{i+p} (S_j - S_i)] dt^{q(t)} \\ &\quad + \eta_1 S_i(t) dB_1(t) \end{aligned}$$

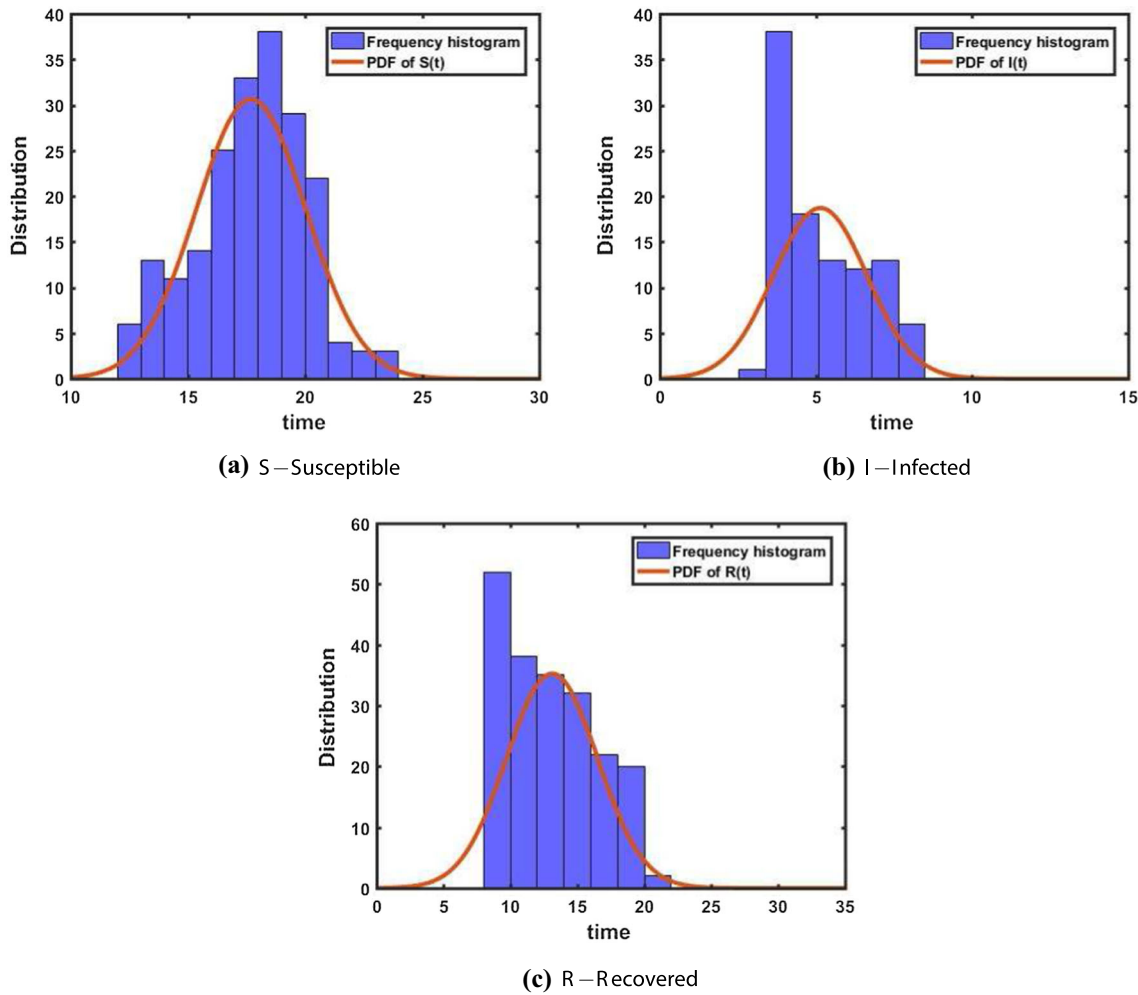


Fig. 3 Frequency histograms for $S(t)$, $I(t)$, and $R(t)$ under the noise intensities $\eta_1 = 0.25$, $\eta_2 = 0.12$, and $\eta_3 = 0.25$ of model (2)

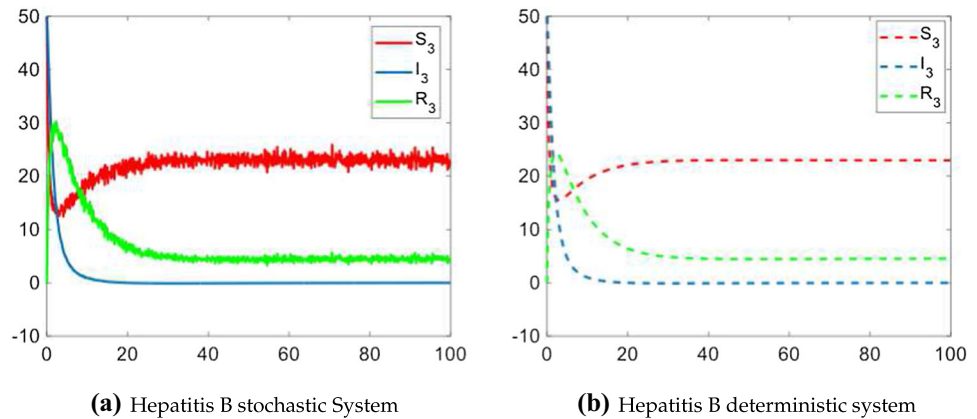
$$\begin{aligned} \frac{d^{q(t)} I_i}{dt^{q(t)}} &= [\beta I_i(t) S_i(t) - (\mu_0 + \gamma_1 + \mu_1) I_i(t)] dt^{q(t)} \\ &\quad + \eta_2 I_i(t) dB_2(t) \\ \frac{d^{q(t)} R_i}{dt^{q(t)}} &= [\gamma_1 I_i + \mu_2 m Z_i(t) S_i(t) + v S_i(t) - \mu_0 R_i(t)] dt^{q(t)} \\ &\quad + \eta_3 R_i(t) dB_3(t) \\ \frac{d^{q(t)} Z_i}{dt^{q(t)}} &= \left[\frac{dI_i(t)}{(1 + hI_i(t))} - a Z_i(t) \right] dt^{q(t)}. \end{aligned} \tag{62}$$

Figure 4 shows the time history of the variable fractional-order network of hepatitis B model. The time-varying fractional derivative is considered as $q(t) = 0.9 + 0.05 \tan^{-1} \frac{T(i)}{20}$. For this simulation, the parameters of the model are the same as the other simulation. As it is shown in this figure, although the parameters are the same as the previous model, the results are completely different. This confirms the importance of two factors: Fig. 4a the effects of fractional derivative and Fig. 4b the impacts of other communities, in the modeling of the system.

9 Conclusion

We investigated the dynamics of hepatitis B via a stochastic SIR model by incorporating environmental noise and information intervention. We found that white noise and information intervention have considerable effects on the disease. From the dynamics, we noted that it plays a crucial role in the disease spreading and prevalence. We particularly proved the existence analysis and uniqueness analysis and showed that the proposed problem is well posed. We also defined the stochastic reproductive number by \mathbf{R}_s for the problem (2) to discuss the analysis of extinction, persistence, and stationary distribution. On behalf of threshold quantity \mathbf{R}_s , we proved that the hepatitis B could be eradicated if the value of \mathbf{R}_s is less than unity, see Theorem (2), and this may reduce the maximum value of infected cases population (see Fig. 1). Moreover, for the stochastic model (2), we have obtained sufficient conditions for the disease prevails of a unique stationary distribution; see Theorem (4). Therefore, the information intervention may reduce the maximum value of infected cases. At the end, all the theoretical findings are justified with

Fig. 4 The trajectories show the dynamics of neuron (62) in the proposed network: **a** represents the stochastic model and **b** represents its corresponding deterministic model



the help of simulation analysis via the stochastic Runge-Kutta method. Then, as a new approach, a variable-order fractional network of the stochastic hepatitis B model is introduced to consider the effects of different societies on each other. It was supposed that populations are jointed through the nearest-neighbor method. In a feature work, we will investigate the optimal control of the proposed variable-order network.

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