Chaos, Solitons and Fractals 108 (2018) 123–128

Contents lists available at ScienceDirect

Chaos, Solitons and Fractals

Nonlinear Science, and Nonequilibrium and Complex Phenomena

journal homepage: www.elsevier.com/locate/chaos

The extinction and persistence of the stochastic hepatitis B epidemic model

Tahir Khan[a,*], Amir Khan[b], Gul Zaman[a]

[a] Department of Mathematics, University of Malakand Chakdara, Dir(L), Pakhtunkhwa, Pakistan
[b] Department of Statistics and Mathematics, University of Swat, Pakhtunkhwa, Pakistan

ARTICLE INFO

Article history:
Received 22 September 2017
Revised 22 December 2017
Accepted 25 January 2018

Keywords:
Stochastic hepatitis B epidemic model
Extinction
Persistence
Numerical simulation

ABSTRACT

We discuss the dynamic of a stochastic hepatitis B epidemic model. A stochastic hepatitis B model is formulated with a varying population environment for a long term behavior. The proposed model consists of three classes, namely the susceptible individuals in which the transmission rate is distributed by white noise, the infected individuals in which the same perturbation occurs and the recovered individuals. We derive sufficient conditions for the extinction and the persistence. Finally, we carry out the numerical simulations to support our analytical results.

1. Introduction

In the real world problem biological phenomenon are always affected by the environmental noise. The environmental variations have a critical influence on the development of an epidemic [1,2]. For human disease, the nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts and population is subject to a continuous spectrum of disturbances [3,4]. So the variability and randomness of the environment is fed through to the state of the epidemic. The contagious disease of hepatitis B causes inflammation of liver results from hepatitis B virus infection is one of the best example in which the epidemics growth and spread are random due to the unpredictability of person-to-person contacts [5].

Mathematical modeling is a powerful tool to describe the dynamical behavior of various diseases in the real world. A number of mathematicians and ecologists have developed diverse epidemic models to realize and control the spread of transmissible diseases in the community. In the past two decades the field of mathematical modeling has been widely used to study the transmission of variety of infectious diseases (see e.g., [6–11]). There are two types of epidemic models viz the deterministic epidemic model and the stochastic epidemic model. Mathematical modeling of a biological phenomena the stochastic differential equation models are more suitable than the deterministic one [12], because it can provide an additional degree of realism in comparison to their deterministic counterparts. Stochastic models produce more valuable output as compared to the deterministic ones because running a stochastic model several times, we can build up a distribution of the predicted outcomes, e.g., the number of infected classes at time t. On the other hand, a deterministic model will just give a single predicted value [13–17]. Many deterministic epidemic models have been developed for the description of viral dynamic of hepatitis B, (see for detail [18–23]).

In this paper, we propose a stochastic epidemic model for the transmission dynamics of hepatitis B virus with a varying population environment for a long term behavior. We categorize the total population into three different classes. The first class is the susceptible individuals in which the transmission rate is distributed by white noise. The second class includes the infected individuals in which the same perturbation occurs. The third class consists of the recovered individuals. We discuss the disease extinction, the disease persistence and derive sufficient conditions for them. We perform numerical simulation using the stochastic Runge–Kutta method to support our analytical results.

2. Background and the model formulation

In this section, we present the stochastic hepatitis B epidemic: Susceptible-infected-recovered model with varying population environment in the long term behavior. We impose the following assumptions on the model:

(A_1). The total population $N(t)$ at time t is subdivided into three different compartments: The susceptible individuals $S(t)$, the hep-

* Corresponding author.
E-mail addresses: tkhan87@uom.edu.pk (T. Khan), gzaman@uom.edu.pk (G. Zaman).

https://doi.org/10.1016/j.chaos.2018.01.036
0960-0779/

atitis B infected individuals $I(t)$ and the recovered individuals $R(t)$, i.e., $N(t)=S(t)+I(t)+R(t)$, varies with time t [24].

(A_2). All parameters and state variables of the proposed model are non-negative.

(A_3). The hepatitis B vaccine provides indefinite protection. Thus the susceptible population goes to the recovered population after getting successful vaccination.

(A_4). For the effect of randomly fluctuating environment, that fluctuations in the environment will manifest themselves mainly as fluctuations in the hepatitis B transmission parameter β, i.e., $\beta \to \beta+\eta\dot{B}(t)$, where $B(t)$ is standard Brownian motion with the property $B(0)=0$ and with the intensity of white noise $\eta^2>0$.

The assumptions (A_1)–(A_4) lead to the following stochastic epidemic model is consisting of three differential equations:

$$\begin{aligned} dS(t) &= \Big(\Lambda-\beta S(t)I(t)-(\mu_0+\nu)S(t)\Big)dt-\eta S(t)I(t)dB(t),\\ dI(t) &= \Big(\beta S(t)I(t)-(\mu_0+\mu_1+\gamma_1)I(t)\Big)dt+\eta S(t)I(t)dB(t),\\ dR(t) &= \Big(\gamma_1 I(t)+\nu S(t)-\mu_0 R(t)\Big)dt. \end{aligned} \tag{1}$$

In the above model (1), Λ represents the per capita constant birth rate. μ_0 and μ_1 respectively represent the natural death rate and the disease induced death rate. The vaccination rate is denoted by ν, while γ_1 represents the constant recovery rate for hepatitis B infected individuals.

3. Existence and uniqueness

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

Theorem 1. *For an initial value $(S(0),I(0),R(0))\in R^3_+$, the solution $(S(t), I(t), R(t))$ of the proposed stochastic hepatitis B epidemic model (1) is unique, for $t\geq 0$. Moreover, the solution remains in R^3_+ with probability 1, i.e., $(S(t),I(t),R(t))\in R^3_+$ for all $t\geq 0$ a.s (almost surely).*

Proof. It is clear that the coefficients of the equations of the model are locally lipschitz continuous for any given initial size of population $(S(0),I(0),R(0))\in R^3_+$. It follows that there is a unique local solution $(S(t), I(t), R(t))$ on $t\in[0,\tau_e)$, where τ_e is the explosion time (for detail see the reference [12,19]). To show that this solution is global, we prove that $\tau_e=\infty$ a.s. Let $k_0\geq 0$ be sufficiently large, so that $S(0)$, $I(0)$ and $R(0)$ all lie within the interval $[\frac{1}{k_0},k_0]$. For each integer $k\geq k_0$, define the stopping time

$$\begin{aligned} \tau_k &= \{t\in[0,\tau_e): min\{S(t),I(t),R(t)\}\\ &\leq \frac{1}{k} \text{ or } max\{S(t),I(t),R(t)\}\Big\}. \end{aligned}\tag{2}$$

In this paper, we set $inf\phi=\infty$, where ϕ denotes the empty set. According to the definition, τ_k increases as $k\to\infty$. Set $\tau_\infty=\lim_{k\to\infty}$ with $\tau_\infty\leq\tau_e$ a.s. If we can show that $\tau_\infty=\infty$ a.s., then $\tau_e=\infty$ and $(S(t),I(t),R(t))\in R^3_+$ a.s. for all $t\geq 0$. In other words to complete the proof, we need to show that $\tau_e=\infty$ a.s. If this statement is false, then there exists a pair of constants $T>0$ and $\epsilon\in(0,1)$, such that

$$P\{\tau_\infty\leq T\}>\epsilon. \tag{3}$$

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

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

Let $N(t)=S(t)+I(t)+R(t)$, then for $t\leq\tau_k$, one may observe that

$$dN(t)=\Big(\Lambda-\mu_0N(t)-\mu_1I(t)\Big)dt\leq\Big(\Lambda-\mu_0N(t)\Big)dt. \tag{4}$$

Solving Eq. (4), we arrive at

$$N(t)\leq\begin{cases}\frac{\Lambda}{\mu_0}, & \text{if } N(0)\leq\frac{\Lambda}{\mu_0},\\ N(0), & \text{if } N(0)>\frac{\Lambda}{\mu_0},\end{cases} := M. \tag{5}$$

Now, we define a C^2-function $H:R^3_+\to R_+$, such that

$$H(S,I,R)=S+I+R-3-(\log S+\log I+\log R). \tag{6}$$

Clearly the function H is non-negative, which can be seen from $y-1-\log y\geq 0$, for all $y>0$. Let $k\geq k_0$ and $T>0$ be arbitrary. The application of *Itô* formula to Eq. (6) yields

$$\begin{aligned} dH(S,I,R) &= \Big(1-\frac{1}{S}\Big)dS+\frac{1}{2S^2}(dS)^2\\ &\quad+\Big(1-\frac{1}{I}\Big)dI+\frac{1}{2I^2}(dI)^2+\Big(1-\frac{1}{R}\Big),\\ &= LH(S,I,R)dt+\eta(I-S)dB(t). \end{aligned}\tag{7}$$

In Eq. (7), $LH:R^3_+\to R_+$ is defined by the following equation

$$\begin{aligned} LH(S,I,R) &= \Big(1-\frac{1}{S}\Big)\Big(\Lambda-\beta S(t)I(t)-(\mu_0+\nu)S(t)\Big)+\frac{1}{2}\eta^2I^2\\ &\quad+\Big(1-\frac{1}{I}\Big)\Big(\beta S(t)I(t)-(\mu_0+\mu_1+\gamma_1)I\Big)+\frac{1}{2}\eta^2S^2\\ &\quad+\Big(1-\frac{1}{R}\Big)\Big(\gamma_1I+\nu S(t)-\mu_0R(t)\Big)\Big),\\ &= \Lambda-(\mu_0+\nu)S-\frac{\Lambda}{S}+\beta I+(\mu_0+\nu)+\frac{1}{2}\eta^2I^2\\ &\quad-(\mu_0+\mu_1+\gamma_1)I-\beta S+(\mu_0+\mu_1+\gamma_1)+\frac{1}{2}\eta^2S^2\\ &\quad+\gamma_1I+\nu S-\mu_0R-\gamma_1\frac{I}{R}-\nu\frac{S}{R}+\mu_0,\\ &\leq \Lambda+\beta I+\mu_0+\nu+\frac{1}{2}\eta^2(S^2+I^2)+\mu_0+\mu_1+\gamma_1\\ &\quad+\gamma_1I+\nu S+\mu_0,\\ &\leq \Lambda+3\mu_0+\nu+\eta^2M^2+(\beta+\gamma_1+\nu)M+\mu_0+\mu_1\\ &\quad+\gamma_1+\mu_0:=K. \end{aligned}\tag{8}$$

Consequently

$$\begin{aligned} &E\Big[H(S(\tau_k\wedge T),I(\tau_k\wedge T),R(\tau_k\wedge T))\Big]\\ &\leq H(S(0),I(0),R(0))+E\Big[\int_0^{\tau_k\wedge T}Kdt\Big],\\ &\leq H(S(0),I(0),R(0))+KT. \end{aligned}\tag{9}$$

Setting $\Omega_k=\tau_k\leq T$ for $k\geq k_1$. As a result, Eq. (3) reads $P(\Omega_k)\geq\epsilon$. Note that for every $\omega\in\Omega_k$, there exists at least one $S(\tau_k,\omega)$, $I(\tau_k,\omega)$, $R(\tau_k,\omega)$ that equal k or $\frac{1}{k}$, and hence $H(S(\tau_k), I(\tau_k), R(\tau_k))$ is not less than $k-1-\log k$ or $\frac{1}{k}-1+\log k$. Thus we have

$$H(S(\tau_k),I(\tau_k),R(\tau_k))\geq E\Big(k-1-\log k\Big)\wedge\Big(\frac{1}{k}-1+\log k\Big). \tag{10}$$

It then follows from Eqs. (3) and (9), that

$$\begin{aligned} H(S(0),I(0),R(0))+KT &\geq E\Big[1_{\Omega(\omega)}H\Big(S(\tau_k),I(\tau_k,R(\tau_k))\Big)\Big],\\ &\geq \epsilon\Big[(k-1-\log k)\wedge\Big(\frac{1}{k}-1+\log k\Big)\Big], \end{aligned}\tag{11}$$

where $1_{\Omega(\omega)}$ is the indicator function of Ω. Letting $k\to\infty$ leads to the contradiction $\infty>H\big(S(0),I(0),R(0)\big)+MT=\infty$, which implies that $\tau_\infty=\infty$ a.s. □

Remark 1. It is clear from Theorem 1 that for any initial value $(S(0),I(0),R(0))\in R^3_+$, there is a unique global solution $\big(S(t),I(t),R(t)\big)\in R^3_+$ almost surely of the model (1). Hence

$$dN(t)\leq\Big(\Lambda-\mu_0N(t)\Big). \tag{12}$$