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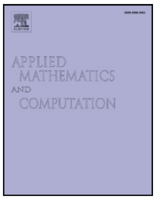

Environmental variability in a stochastic epidemic model

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ABSTRACT

In this paper, we investigate the stochastic dynamics of a simple epidemic model incorporating the mean-reverting Ornstein–Uhlenbeck process analytically and numerically. We define two threshold parameters, the stochastic demographic reproduction number $\mathcal{R}_d^s$ and the stochastic basic reproduction number $\mathcal{R}_0^s$, to utilize in identifying the stochastic extinction and persistence of the disease. We find that the stochastic disease dynamics can be determined by the environment fluctuations which measured by the intensity of volatility and the speed of reversion: the larger intensity of volatility or the smaller speed of reversion can suppress the outbreak of the disease, the smaller intensity of volatility or the the higher speed of reversion can enhance the outbreak of the disease. Furthermore, via numerical simulations, we find that the stochastic model has an endemic stationary distribution which leads to the stochastic persistence of the disease. Our results show that mean-reverting process is a well-established way of introducing stochastic environmental noise into biologically realistic population dynamic models.

1. Introduction

Epidemiological models, which can be identified as the disease transmission in the populations, have been revealed as a powerful tool to analyze the spread and control the infectious diseases qualitatively and quantitatively, as the research results are helpful to predict the developing tendency of infectious diseases, to determine the key factors of the spread of infectious disease and to seek the optimum strategies of preventing and controlling the spread of infectious diseases [1–7].

In recent years, a number of epidemic models have been formulated to describe the impact of environmental noise on the dynamics of infectious diseases [8–15]. And in reality, if the environment is randomly varying, the population is subject to a continuous spectrum of disturbances [8]. The environmental fluctuations may involve the variations of factors such as climate, habitats, health habits, medical quality and so on, which may affect the natural birth rate, death rate and so on [16]. Especially, for human diseases, the nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts [17] and population is subject to a continuous spectrum of disturbances [8]. Hence the variability and randomness of the environment are fed through to the state of the epidemic [18]. In the view of this point, stochastic differential equations (SDE) could be a more appropriate way of modeling disease spreading in many circumstances [10].

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In a stochastic differential equation (SDE) model of a dynamical biological system, environmental variability is often treated by modifying the parameters in the model. And there are two common approaches to modify parameters for a varying environment. The first one assumes that the parameters can be adequately modeled by linear functions of white noise [8,10,13,14,19–27], and the second approach assumes that the parameters satisfy mean-reverting stochastic processes [12,16,28–30]. Allen [16] investigated the relations between these two approaches and showed that the mean-reverting stochastic processes is a practical and biologically realistic way to incorporate the effects of environmental variability in the parameters.

Thanks the insight work of Allen [16], in this paper, we will focus on how environment fluctuations introduced in the natural death rate incorporating the mean-reverting processes affect the extinction and persistence of the disease dynamics. The rest of this article is organized as follows: In Section 2, we derive a deterministic epidemic model (or without noise) and its corresponding stochastic version (or with noise). In Section 3, we show the existence of the global solution. In Section 4, we give the stochastic dynamics in details. In Section 5, we provide some numerical simulations to support our findings. In the last section, Section 6, we provide a brief discussion and the summary of the main results.

2. Model derivations and preliminaries

2.1. The deterministic model

Motivated by Xiao and Chen [31,32], in this subsection, we firstly give the following assumptions to our model:

(H1) Let $S(t)$ and $I(t)$ be the density of the susceptible (S) and infectious (I), respectively, and assume that the total population N is split into a susceptible part S and an infected part I, i.e., $N(t) = S(t) + I(t)$.

(H2) Assume that the maximum per capita birth rate of uninfected hosts is a, the per capita density-dependent reduction in birth rate is c, obviously, $1/c$ is the carrying capacity, and the natural death rate is μ.

(H3) Assume that only susceptible S is capable of reproducing with logistic law, epidemiologically speaking, the infectious I is removed by death (say, its disease-induced death rate is constant α) before having the possibility of reproducing [33]. And assume that the infectious I still contributes with S to population growth toward the carrying capacity, and there is no recovery from the infectious I.

(H4) The disease transmission is assumed to be mass action βSI, where $\beta > 0$ is the transmission coefficient.

Based on the assumptions above, we can obtain the following epidemiological model:

$$\begin{cases} \dfrac{dS}{dt} = aS[1 - c(S+I)] - \beta SI - \mu S, \\ \dfrac{dI}{dt} = \beta SI - (\mu+\alpha)I, \end{cases} \tag{1}$$

It is worthy to note that model (1) is a modified case of Xiao's model [31,32]. Also, model (1) can be seen as a special case $f = 0$ of Ebert's model [34]. In the view of this point, model (1) can be used to investigate the dynamics of the parasites–hosts system.

In the context of epidemic models with variable population size, the view that the qualitative dynamics of such systems are controlled in specific ways by the demographic threshold $\mathcal{R}_d$, the basic reproduction number for the demographic process [35]. For model (1), the demographic reproductive number $\mathcal{R}_d$ is given by

$$\mathcal{R}_d = \frac{a}{\mu}. \tag{2}$$

On the other hand, the basic reproduction number $\mathcal{R}_0$ is often considered as the threshold quantity that determines when an infection can invade and persist in a new host population, which is defined as the expected number of secondary cases produced, in a completely susceptible population, by a typical infected individual during its entire period of infectiousness [36–38]. For model (1), the basic reproduction number is given by

$$\mathcal{R}_0 = \frac{\beta(a-\mu)}{ac(\alpha+\mu)} = \frac{\beta(\mathcal{R}_d - 1)}{c\mathcal{R}_d(\alpha+\mu)}. \tag{3}$$

Then we can obtain following properties of model (1). The proof of this theorem is similar to that in [34] and hence is omitted here.

Theorem 2.1.

(A) *If* $\mathcal{R}_d < 1$, *then for any given initial value* $(S_0, I_0) \in \mathbb{R}^2_+$, *the susceptible* $S(t)$ *and infectious* $I(t)$ *tend to* $(0, 0)$.

(B) *If* $\mathcal{R}_d > 1$ *holds, then*

(B1) *if* $\mathcal{R}_0 \le 1$, *then the disease-free steady state* $\left(\frac{a-\mu}{ac}, 0\right) = \left(\frac{\mathcal{R}_d - 1}{c\mathcal{R}_d}, 0\right)$ *is globally asymptotically stable with respect to initial value* $(S_0, I_0) \in \mathbb{R}^2_+$.

(B2) *if* $\mathcal{R}_0 > 1$, *there exists a unique endemic state*

$$E^* = \left(\frac{\alpha+\mu}{\beta}, \frac{(a-\mu)\beta - ac(\alpha+\mu)}{\beta(ac+\beta)}\right) = \left(\frac{\mathcal{R}_d - 1}{c\mathcal{R}_d\mathcal{R}_0}, \frac{\mu(\mathcal{R}_d - 1)^2(\mathcal{R}_0 - 1)}{c\mathcal{R}_d\mathcal{R}_0(\mathcal{R}_0(\alpha+\mu) + \mu(R_d - 1))}\right),$$