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Long-time behavior of a stochastic SIR model ☆

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ABSTRACT

In this paper, we analyze long-time behavior of densities of the distributions of the solution for a stochastic SIR epidemic model. We prove that the densities can converge in L^1 to an invariant density or can converge weakly to a singular measure.

1. Introduction

Epidemics are commonly modeled by using deterministic compartmental models where the population amongst whom the disease is spreading is divided into several classes. Childhood diseases such as measles, whooping cough and rubella et al., are often modeled by SIR models. The following model is one of classic SIR models:

$$\begin{cases} \dot{S}(t) = \Lambda - \beta S(t)I(t) - \mu S(t), \\ \dot{I}(t) = \beta S(t)I(t) - (\mu + \varepsilon + \gamma)I(t), \\ \dot{R}(t) = \gamma I(t) - \mu R(t), \end{cases} \tag{1.1}$$

where the parameter $\Lambda, \beta, \varepsilon, \gamma, \mu$ are positive constants. In model (1.1), $S(t)$ denotes the number of susceptible individuals, $I(t)$ denotes the number of infected individuals and $R(t)$ denotes the number of removed individuals with permanently immunity at time t. The influx of individuals into the susceptibles is given by a constant Λ. The natural death rates are assumed to be equal (denoted by constant μ) and individuals in $I(t)$ suffer an additional death due to disease with rate constant ε; β and γ represent the disease transmission coefficient and the rate of recovery from infection, respectively. The basic reproduction number $R_0 = \beta\Lambda/[\mu(\mu + \varepsilon + \gamma)]$ is the threshold of the system (1.1) for an epidemic to occur. If $R_0 \leqslant 1$, model (1.1) has only the disease-free equilibrium $E_0 = (\Lambda/\mu, 0, 0)$ which is globally asymptotical stable. This means the disease will disappear and the entire population will become susceptible. If $R_0 > 1, E_0$ becomes unstable and there exists a global asymptotically stable endemic equilibrium

$$E^* = \left(\frac{\mu + \varepsilon + \gamma}{\beta}, \frac{\Lambda}{\mu + \varepsilon + \gamma} - \frac{\mu}{\beta}, \frac{\Lambda\gamma}{\mu(\mu + \varepsilon + \gamma)} - \frac{\gamma}{\beta}\right),$$

which implies the disease always remains. For detailed works concerning deterministic SIR model and its extension, the reader may refer to [1–9].

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As is known to us, real life is full of randomness and stochasticity. So it is important whether or not the long-time behavior of the solution for deterministic dynamics system can be changed by stochastic perturbations. So far, various stochastic versions of system (1.1) have been widely studied, see [10–16]. Here we only introduce some of these excellent works. In [10], Tornatore et al. considered that the disease transmission coefficient β was subject to stochastic perturbations in SIR models with or without distributed time delay. They demonstrated that the introduction of stochastic perturbations modified the threshold of system for an epidemic to occur both analytically and numerically. In [14], the asymptotic behavior of global positive solution for the single-group stochastic SIR model

$$\begin{cases} dS(t) = [\Lambda - \beta S(t)I(t) - \mu S(t)]dt + \sigma_1 S(t)dB_1(t), \\ dI(t) = [\beta S(t)I(t) - (\mu + \varepsilon + \gamma)I(t)]dt + \sigma_2 I(t)dB_2(t), \\ dR(t) = [\gamma I(t) - \mu R(t)]dt + \sigma_3 R(t)dB_3(t) \end{cases}$$

has been established by Jiang et al., where $B_i(t), i = 1, 2, 3$ are independent standard Brownian motions. Furthermore, Ji et al. [15] discuss the multi-group stochastic SIR epidemic model. By using appropriate Lyapunov functions, the authors obtained the asymptotic behavior of the solution as follows:

- If $\rho(M_0) \leqslant 1$, the distance between the solution and the disease-free equilibrium is no more than some constant value, which is proportional to the intensity of white noise.
- If $\rho(M_0) > 1$, there is a stationary distribution which is ergodic.

In addition, using the same method as in [15], Yang et al. [16] considered the SIR models with saturated incidence and obtained the existence of stationary distribution which is ergodic.

In this paper, we introduce random noise to model (1.1) as follows:

$$\begin{cases} dS(t) = [\Lambda - \beta S(t)I(t) - \mu S(t)]dt + \sigma_1 S(t)dB(t), \\ dI(t) = [\beta S(t)I(t) - (\mu + \varepsilon + \gamma)I(t)]dt + \sigma_2 I(t)dB(t), \\ dR(t) = [\gamma I(t) - \mu R(t)]dt + \sigma_3 R(t)dB(t). \end{cases} \tag{1.2}$$

Here we assume that the random noise for three populations is correlated, which corresponds to the situation when the same factor (like other epidemic disease, weather and so on) influences $S(t), I(t)$ and $R(t)$. Throughout, we assume $\sigma_i > 0,\ i = 1, 2, 3$. It is obvious that E_0 and E^* are no longer the equilibriums for (1.2). Precisely, there is neither a disease-free equilibrium nor an endemic equilibrium for (1.2).

Since the dynamics of group R has no effect on the disease transmission dynamics, here we only consider

$$\begin{cases} dS(t) = [\Lambda - \beta S(t)I(t) - \mu S(t)]dt + \sigma_1 S(t)dB(t), \\ dI(t) = [\beta S(t)I(t) - (\mu + \varepsilon + \gamma)I(t)]dt + \sigma_2 I(t)dB(t). \end{cases} \tag{1.3}$$

In the proof of the existence and uniqueness of positive solution in [14], the independence of $B_i, i = 1, 2, 3$ plays no essential role. So by using the same method as in [14], the existence and uniqueness of positive solution for system (1.3) hold. Let $u = \ln S$ and $v = \ln I$, then we get

$$\begin{cases} du(t) = [-c_1 + \Lambda e^{-u} - \beta e^{v}]dt + \sigma_1 dB(t), \\ dv(t) = [-c_2 + \beta e^{u}]dt + \sigma_2 dB(t), \end{cases} \tag{1.4}$$

where $c_1 = \mu + \sigma_1^2/2, c_2 = \mu + \varepsilon + \gamma + \sigma_2^2/2$. In order to study the long-time behavior of the solutions of system (1.3), it suffices to concentrate on (1.4).

In this paper, the main aim is to study the existence of a stationary distribution of system (1.4) and its asymptotic stability. Since the Fokker–Planck equation corresponding to system (1.4) is of degenerate type, the approach used in [14,15] to obtain the existence of stationary distribution is invalid for system (1.3) and system (1.4). Our new approach comes from Markov semigroup theory, which was used in [17,18] in order to study long-time behavior of a stochastic prey–predator model. In (1.3), the incidence function is bilinear incidence rate, which is reasonable if the sizes of both S and I do not become two small. But for completeness, we still present sufficient conditions for the disease to extinct exponentially in this paper.

The rest of this paper is organized as follows. In Section 2, we present some auxiliary results concerning Markov semigroups. In Section 3, we formulate the main result and its proofs.

2. Preliminary

As mentioned before, the proof of our result is based on the theory of integral Markov semigroups. We need some auxiliary definitions and results concerning Markov semigroups (see [17,18]). For the convenience of the reader, we present these definitions and results in this section. Let the triple (X, Σ, m) be a σ-finite measure space. Denote by D the subset of the space $L^1 = L^1(X, \Sigma, m)$ which contains all densities, i.e.

$$D = \{f \in L^1 : f \geqslant 0, \|f\| = 1\}.$$