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A stochastic SIRS epidemic model with non-monotone incidence rate under regime-switching

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Abstract

In this paper, we propose and discuss a stochastic SIRS epidemic model with non-monotone incidence rate under regime-switching. First of all, we show that there is a unique positive solution, which is a prerequisite for analyzing the long-term behavior of the stochastic model. Then, a threshold dynamic determined by the basic reproduction number $\mathcal{R}_0^s$ is established: the disease can be eradicated almost surely if $\mathcal{R}_0^s < 1$ and under mild extra conditions, whereas if $\mathcal{R}_0^s > 1$, the densities of the distributions of the solution can converge in L^1 to an invariant density by using the Markov semigroups theory. Finally, based on realistic parameters obtained from previous literatures, numerical simulations have been performed to verify our analytical results.

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1. Introduction

Mathematical model is an important tool to better understand epidemiological patterns and disease control. Since Kermack and McKendrick [1] published their insights on the underlying mechanisms of transmission and control of infectious diseases, various epidemic models (for example, deterministic models [2–4], spatially explicit models [5–7], models on complex network [8–10], models based on real data [11–13] and stochastic models [15,16,22]) have been extensively established and investigated, which have led to great progress in the prevention and control of diseases. In the study of infectious disease dynamics, incidence

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Fig. 1.1. The green line, red line and blue line represent the incidence βI , $\frac{\beta I}{1+\alpha I^2}$ and $\frac{\beta I}{1+\alpha I}$, respectively. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

rate is an important indicator that describes the average number of new cases per unit time. It is traditionally assumed that the incidence rate of disease transmission is bilinear, i.e., $g(I)S = \beta IS$, which appears quite unrealistic if the number of infective individuals is large enough. In practice, many infectious diseases also exhibit multiple peaks during the outbreaks. For these reasons, there is a great interest in investigating epidemic models with nonlinear incidence rates. For example, Liu et al. [3] proposed a nonlinear saturated incidence rate $g(I)S = \frac{\beta I^h S}{1+\alpha I^h}$ to model the effect of behavioral changes, where $\beta, I, \alpha, h > 0$. The saturated incidence rate $g(I)S = \frac{\beta IS}{1+\alpha I}$ (a special case of $\frac{\beta I^h S}{1+\alpha I^h}$) is introduced into the epidemic model by Capasso and Serio [18]. This incidence rate is more realistic than the bilinear rate βIS , as it considers the behavioral change and crowding effect of the infective individuals and also prevents unboundedness of the contact rate (see Fig. 1.1).

It is well known that the aggressive control measures and policies (such as border screening, mask wearing, quarantine, isolation, etc.), play an important role in administering efficient interventions which control disease spread and hopefully eliminate epidemic diseases. For instance, during the outbreak of severe acute respiratory syndrome (SARS) in 2003 [4] and the outbreak of H1N1 influenza pandemic in 2009 [17], these two diseases have been well controlled by taking these intervention actions. Xiao and Ruan [19] in 2007 introduced a non-monotone incidence function $g(I)S = \frac{\beta IS}{1+\alpha I^2}$ ($l = 1, h = 2$) to understand the influence which was intervened in the spread of diseases by government. Then they established the following SIRS epidemic model:

$$\begin{cases} dS_t = (\Lambda - \mu S_t - \frac{\beta S_t I_t}{1+\alpha I_t^2} + \theta R_t)dt, \\ dI_t = (\frac{\beta S_t I_t}{1+\alpha I_t^2} - (\mu + \gamma + \delta)I_t)dt, \\ dR_t = (\gamma I_t - (\mu + \theta)R_t)dt, \end{cases} \quad (1.1)$$

where S_t, I_t, R_t are the number of susceptible (S), infectious (I) and recovered (R) at time t , respectively. Λ is the constant input of new members into the population per unit time, μ is the natural death rate of the population, γ is the natural recovery rate of the infective individuals, δ is the disease-caused mortality rate, θ is the rate at which recovered individuals lose immunity and return to the susceptible class. The incidence $\frac{\beta I}{1+\alpha I^2}$ increases when I is small and decreases when I is large (see Fig. 1.1), which explains the phenomenon where the rate of contacting between infected I and susceptible S decreases after government intervention. βI measures the

infection force of the disease, β is the proportionality constant, α is the parameter measuring the inhibitory effect. In addition, all parameters of model (1.1) are assumed to be positive constants.

In the real world, 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. Gard [21] showed that the population dynamics is inevitably affected by environmental white noise which is an important component in an ecosystem. May [14] revealed the fact that due to environmental noise the parameters involved in the system exhibit random fluctuation to a greater or lesser extent. Cai et al. [15] found the presence of even a tiny amount of white noises can suppress disease outbreak. Li et al. [16] pointed out that the presence of white noise is capable of supporting the irregular recurrence of influenza epidemic, and the average level of $I(t)$ always decreases with the increasing noise intensity. In addition, Allen [39] pointed out that, one of the most important differences between deterministic and stochastic models is that deterministic models predict an outcome with absolute certainty, whereas stochastic models provide only the probability of an outcome. It also has been shown that some stochastic epidemic models can provide an additional degree of realism in comparison with their deterministic system. Therefore, it is important to investigate the effect of white noise in the environment on population dynamics. Recently, Cai et al. [22] investigated the stochastic dynamics of a simple epidemic model which incorporates the mean-reverting Ornstein–Uhlenbeck process. They found that the stochastic epidemic dynamics can be determined by the environment fluctuations which measured by the intensity of volatility and the speed of reversion. However, they only roughly provided numerical simulations for stationary distribution, but did not prove that the existence of stationary distribution when $R_0^s > 1$. Liu et al. [23] studied a stochastic multigroup SIQR epidemic model with standard incidence rates. They gave the existence of a stationary distribution, but unfortunately they did not give the ergodic analyzing of their model, as the diffusion matrix is not satisfaction the uniform ellipticity condition. In particular, Cai et al. [15] assumed that the environmental white noise mainly influence the disease transmission rate β of the populations. That is $\beta \rightarrow \beta + \sigma \frac{dB_t}{dt}$, where σ denotes the intensity of white noise and $\frac{dB_t}{dt}$ is a Gaussian white noise, i.e., B_t is a standard Brownian motion. By replacing βdt with $\beta dt + \sigma dB(t)$ in the deterministic SIRS model (1.1), they obtained the following stochastic SIRS model

$$\begin{cases} dS_t = \left(\Lambda - \mu S_t - \frac{\beta S_t I_t}{1 + \alpha I_t^2} + \theta R_t \right) dt - \frac{\sigma S_t I_t}{1 + \alpha I_t^2} dB_t, \\ dI_t = \left(\frac{\beta S_t I_t}{1 + \alpha I_t^2} - (\mu + \gamma + \delta) I_t \right) dt + \frac{\sigma S_t I_t}{1 + \alpha I_t^2} dB_t, \\ dR_t = (\gamma I_t - (\mu + \theta) R_t) dt. \end{cases} \quad (1.2)$$

When studying epidemic models, it is naturally important to know whether the models tend to a disease-free state or the disease will remain permanently. For the deterministic model (1.1), a threshold dynamic determined by the basic reproduction number $\hat{R}_0$ is established: if $\hat{R}_0 < 1$, the disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$ is globally asymptotically stable in the feasible region, whereas if $\hat{R}_0 > 1$, then there has a unique endemic equilibrium $E^* = (S^*, I^*, R^*)$ which is globally asymptotically stable (see [19]). For the stochastic model (1.2), the basic reproduction number is defined by

$$\bar{R} = \frac{\frac{\Lambda\beta}{\mu} - \frac{\Lambda^2\sigma^2}{2\mu^2}}{\mu + \gamma + \delta}, \quad (1.3)$$

using the Markov semigroup theory, they obtained that if $\bar{R} < 1$, under mild extra conditions, the disease dies out in probability; whereas if $\bar{R} > 1$, under mild extra conditions, the densities of the distributions of the solution can converge in L^1 to an invariant density (see Section 5.1 in [15]). Additionally, we also recommend readers to refer to Feng et al. [24,25], Lin et al. [26], Wang et al. [27], Cai et al. [28], Liu et al. [29], Li et al. [30], Yang et al. [31] and the references therein.

On the other hand, the epidemic dynamics are usually affected by a random switching in the external environments. For instance, seasonal variations in temperature, rainfall and resource availability are ubiquitous and can exert strong pressures on disease dynamics (see refs. [32–35,37]). Hemmes et al. [32] showed that influenza virus survives (IVS) better at low temperature and relative humidity (RH) with little dependence on absolute humidity (AH). Arundel et al. [33] studied that RH can affect the incidence of respiratory infections and allergies and they found that the survival or infectivity of viruses is minimized by exposure to relative humidities between 40% and 70%. Sobsey and Meschke [34] reviewed that generally viruses with higher lipid tend to be more persistent at lower RH, while viruses with lesser or no lipid content are more stable at higher relative humidities. Shaman and Kohn [35] used the influenza virus data to explore the effects of absolute humidity (AH) on influenza virus transmission (IVT) and influenza virus survival (IVS) and found that the relationship between AH and IVS is strongly nonlinear, and there is the appearance of a similar nonlinear relationship between AH and IVT. Rogers and Randolph [37] pointed out that weather influences the survival and reproduction rates of the vectors and rates of development, survival and reproduction of pathogens within vectors. Therefore, it is important to investigate the effect of random switching in the external environments on disease dynamics. We also recommend readers to refer to Minhaz Ud-Dean [36], Semenza and Menne [38] and the references therein for learning more information about the effect of temperature, humidity and weather factors on transmission efficiency of disease.

However, the white noise cannot describe the phenomena that the population may suffer sudden catastrophic shocks or alien species invasion in nature [40]. The telegraph noise (namely, color noise) can be illustrated as a switching between two or more regimes of environment, which differ by factors such as nutrition or as rain falls [41]. Frequently, the switching among different environments is memoryless and the waiting time for the next switch is exponentially distributed [42,44–53]. Thus we can model the regime switching by a continuous-time Markov chain with values in a finite state space, which drives the changes of the main parameters of epidemic models with state switchings of the Markov chain. In particular, Li et al. [53] studied the spread dynamics of a stochastic SIRS epidemic model with nonlinear incidence and Markovian switching. They gave the threshold value of the disease persistence and extinction and then gave the ergodic analyzing of their model. Regrettably, they neglected the effect of the white noise for the transmission of the disease. Liu et al. [42] explored the dynamical behavior of a stochastic SIQR epidemic model with standard incidence which is perturbed by both white and telegraph noises. They only gave the threshold between persistence and extinction of the disease, but did not give the ergodic analyzing of their model.

Inspired by the above facts, in present paper, we will firstly incorporate telegraph noise into stochastic model (1.2) to get a new model, then we provide the threshold value of the disease persistence and extinction in this model, which finally proves the existence of a unique ergodic stationary distribution of the new model. Here, we model the random by

a continuous-time Markov chain $\{r(t), t \geq 0\}$ in a finite state space $\mathbb{S} = \{1, 2, \dots, N\}$ with generator $\Gamma = (\gamma_{ij})_{N \times N}$ given by

$$\mathbb{P}\{r(t + \Delta t) = j | r(t) = i\} = \begin{cases} \gamma_{ij}\Delta t + o(\Delta t) & \text{if } i \neq j, \\ 1 + \gamma_{ii}\Delta t + o(\Delta t) & \text{if } i = j, \end{cases}$$

where $\gamma_{ij} \geq 0$ for $i, j = 1, 2, \dots, N$ with $i \neq j$ and $\gamma_{ii} = -\sum_{j \neq i} \gamma_{ij}$ for each $i = 1, 2, \dots, N$. Assume Markov chain $r(t)$ is irreducible and independent of Brownian motion. Therefore, there exists a unique stationary distribution $\pi = \{\pi_1, \pi_2, \dots, \pi_N\}$ of $r(t)$ which satisfies $\pi \Gamma = 0$, $\sum_{i=1}^N \pi_i = 1$ and $\pi_i > 0, \forall i \in \mathbb{S}$. Then we can further obtain the following model with regime switching:

$$\begin{cases} dS_t = (\Lambda_{r(t)} - \mu_{r(t)}S_t - \frac{\beta_{r(t)}S_t I_t}{1 + \alpha_{r(t)}I_t^2} + \theta_{r(t)}R_t)dt - \frac{\sigma_{r(t)}S_t I_t}{1 + \alpha_{r(t)}I_t^2}dB_t, \\ dI_t = \left(\frac{\beta_{r(t)}S_t I_t}{1 + \alpha_{r(t)}I_t^2} - (\mu_{r(t)} + \gamma_{r(t)} + \delta_{r(t)})I_t \right)dt + \frac{\sigma_{r(t)}S_t I_t}{1 + \alpha_{r(t)}I_t^2}dB_t, \\ dR_t = (\gamma_{r(t)}I_t - (\mu_{r(t)} + \theta_{r(t)})R_t)dt, \end{cases} \quad (1.4)$$

where $\Lambda_i, \mu_i, \beta_i, \alpha_i, \theta_i, \sigma_i, \gamma_i$ and δ_i are all positive constants for any $i \in \mathbb{S}$.

This article formulates a stochastic SIRS epidemic model with regime switching and the infectious forces under intervention strategies. Our main idea is to investigate the effect of the white and telegraph noises on the spread dynamics of the disease in the host population based on realistic parameters obtained from previous literatures. The rest of this article is organized as follows: In Section 2, we present the main results of system (1.4). In Sections 3 and 4, we present the detailed proof of the theoretical results. In Section 5, we perform numerical simulations to verify our analytical results based on realistic parameter values which are mainly taken from the work in [61]. Finally, we provide a brief discussion and summary of main results. As the proof of our result is based on the theory of integral Markov semigroups, for the convenience of the reader, in the appendix we present some auxiliary results concerning Markov semigroups.

Throughout this paper, unless otherwise specified, we let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, P)$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t \geq 0}$ satisfying the usual conditions (i.e., it is right continuous and $\mathcal{F}_0$ contains all P-null sets).

2. Main results

To investigate the dynamical behavior of stochastic model (1.4), the first concerning thing is whether the solution is global and positive.

Theorem 2.1. *For any given initial value $(S_0, I_0, R_0, r(0)) \in \Delta \times \mathbb{S}$, there is a unique solution (S_t, I_t, R_t) exists on $t \geq 0$ and the solution will remain in $\mathbb{R}_+^3$ with probability 1, where $\mathbb{R}_+^3 := \{(x, y, z) \in \mathbb{R}^3 : x > 0, y > 0, z > 0\}$ and*

$$\Delta = \left\{ (S, I, R) \in \mathbb{R}_+^3 : \min \left\{ \frac{\Lambda_i}{\mu_i + \delta_i} \right\} < S + I + R < \max \left\{ \frac{\Lambda_i}{\mu_i} \right\}, i \in \mathbb{S} \right\}. \quad (2.1)$$

Remark 2.1. There exists a unique positive solution (S_t, I_t, R_t) of model (1.4) on $t \geq 0$ with any given initial value $(S_0, I_0, R_0, r(0)) \in \Delta \times \mathbb{S}$, and the solution will remain in Δ with probability 1.

In studying epidemic models, the most interesting and important issues are usually to establish the threshold condition for the extinction and persistence of the disease. Frequently, the

threshold behavior for many epidemic models are defined by the basic reproduction number R_0 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 [20]. It is a common case (including models (1.1) and (1.2)) that a disease dies out if R_0 is less than unity and the disease is established in the population if R_0 is greater than unity. Naturally, we wish to examine the dynamic behavior of model (1.4) and then we may ask what is the corresponding basic reproduction number $\mathcal{R}_0^s$? First of all, we define the basic reproduction number for the SDE model (1.4) as follows:

$$\mathcal{R}_0^s := \frac{\sum_{i=1}^N \pi_i \left(\frac{\Delta_i \beta_i}{\mu_i} - \frac{\Lambda_i^2 \sigma_i^2}{2\mu_i^2} \right)}{\sum_{i=1}^N \pi_i (\mu_i + \gamma_i + \delta_i)}. \quad (2.2)$$

Theorem 2.2. Let (S_t, I_t, R_t) be a solution of the model (1.4) for any given initial value $(S_0, I_0, R_0, r(0)) \in \Delta \times \mathbb{S}$. If

$$\begin{aligned} \sigma_i^2 &> \frac{\beta_i \mu_i}{\Lambda_i} \text{ for any } i \in \mathbb{S} \text{ and } \sum_{i=1}^N \pi_i \left(\frac{\beta_i^2}{2\sigma_i^2} - \mu_i - \gamma_i - \delta_i \right) < 0 \text{ or} \\ \sigma_i^2 &< \frac{\beta_i \mu_i}{\Lambda_i} \text{ for any } i \in \mathbb{S} \text{ and } \mathcal{R}_0^s < 1, \end{aligned} \quad (2.3)$$

then the disease I_t tends to zero exponentially.

Since stationary distribution of stochastic system means that all the individuals can be coexistent and persistent in the long term, many authors proved the existence of a unique ergodic stationary distribution (see [28,29,46,50,51] and the references cited therein). It is worthy to note that in those literatures the proofs depend heavily on the uniform ellipticity condition. However, in this paper the diffusion matrix of model (1.4) is given by

$$A_i = \sigma_i^2 \begin{pmatrix} \frac{S^2 I^2}{(1+\alpha_i I^2)^2} & \frac{-S^2 I^2}{(1+\alpha_i I^2)^2} & 0 \\ \frac{-S^2 I^2}{(1+\alpha_i I^2)^2} & \frac{S^2 I^2}{(1+\alpha_i I^2)^2} & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad i \in \mathbb{S}.$$

Obviously, A_i does not satisfy the uniform ellipticity condition for it is degenerate. Hence, in this paper, we employ Markov semigroup theory to obtain a stable stationary distribution.

Theorem 2.3. Let (S_t, I_t, R_t) be a solution of system (1.4) with initial value $(S_0, I_0, R_0, r(0)) \in \Delta \times \mathbb{S}$. Then for every $t > 0$ the distribution of $(S_t, I_t, R_t, r(t))$ has a density $u(t, S, I, R, i)$. If $\mathcal{R}_0^s > 1$, then there exists a unique density $u_*(S, I, R, i)$ such that

$$\lim_{t \rightarrow \infty} \sum_{i=1}^N \iiint_{\Delta} |u(t, S, I, R, i) - u_*(S, I, R, i)| dS dI dR = 0.$$

Remark 2.2. Theorem 2.3 suggests that if $\mathcal{R}_0^s > 1$ is satisfied, then the stochastic model (1.4) has the ergodic property where the positive solution converges to the unique stationary distribution. This reveals the persistence of the disease almost everywhere under certain conditions.

Remark 2.3. From Theorems 2.2 and 2.3, one can find that the value of $\mathcal{R}_0^s$ mainly determines the persistence or extinction of the disease. If $\sigma_i^2 < \frac{\beta_i \mu_i}{\Lambda_i}$ for any $i \in \mathbb{S}$ and $\mathcal{R}_0^s < 1$, then the

disease will be extinct, while if $\mathcal{R}_0^s > 1$, then there is a unique ergodic stationary distribution for system (1.4). Therefore, $\mathcal{R}_0^s$ is the threshold of system (1.4) for the epidemic occurs or not.

Remark 2.4. In Theorems 2.1, 2.2 and 2.3, we extend a part of the results of Cai et al. [15] (Please see 5.1 Section). From Eqs. (1.3) and (2.2), one can find that the basic reproduction number $\mathcal{R}_0^s$ is also an extension of $\bar{R}$. In particular, in the case $N = 1$, then $\mathcal{R}_0^s$ becomes to $\bar{R}$.

Remark 2.5. The proof of Theorem 2.1 is very similar to Theorem 4.3 of Cai et al. [15] and is omitted. Hence, in the rest of this paper, we are devoted to the proof of Theorems 2.2 and 2.3.

3. The Proof of Theorem 2.2

In this section, we prove Theorem 2.2 about the extinction of disease for the stochastic model (1.4).

Proof. By the generalized Itô's formula, one can see that

$$\begin{aligned} d \ln I_t &= \left(\frac{\beta_{r(t)} S_t}{1 + \alpha_{r(t)} I_t^2} - (\mu_{r(t)} + \gamma_{r(t)} + \delta_{r(t)}) - \frac{\sigma_{r(t)}^2 S_t^2}{2(1 + \alpha_{r(t)} I_t^2)^2} \right) dt + \frac{\sigma_{r(t)} S_t}{1 + \alpha_{r(t)} I_t^2} dB_t, \\ &= \varphi \left(\frac{S_t}{1 + \alpha_{r(t)} I_t^2} \right) dt + \frac{\sigma_{r(t)} S_t}{1 + \alpha_{r(t)} I_t^2} dB_t \end{aligned} \quad (3.1)$$

where $\varphi(x_t) = \beta_{r(t)} x_t - (\mu_{r(t)} + \gamma_{r(t)} + \delta_{r(t)}) - \frac{\sigma_{r(t)}^2 x_t^2}{2}$. Since

$$\begin{aligned} \varphi \left(\frac{S_\tau}{1 + \alpha_{r(\tau)} I_\tau^2} \right) &= \frac{\beta_{r(\tau)} S_\tau}{1 + \alpha_{r(\tau)} I_\tau^2} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}) - \frac{\sigma_{r(\tau)}^2 S_\tau^2}{2(1 + \alpha_{r(\tau)} I_\tau^2)^2} \\ &= \frac{-\sigma_{r(\tau)}^2}{2} \left(\frac{S}{1 + \alpha_{r(\tau)} I^2} - \frac{\beta_{r(\tau)}}{\sigma_{r(\tau)}^2} \right)^2 + \frac{\beta_{r(\tau)}^2}{2\sigma_{r(\tau)}^2} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}) \\ &\leq \frac{\beta_{r(\tau)}^2}{2\sigma_{r(\tau)}^2} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}). \end{aligned}$$

Substituting this inequality into Eq. (3.1), one can see that

$$d \ln I_t \leq \left(\frac{\beta_{r(\tau)}^2}{2\sigma_{r(\tau)}^2} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}) \right) dt + \frac{\sigma_{r(t)} S_t}{1 + \alpha_{r(t)} I_t^2} dB_t,$$

Integrating both sides from 0 to t and dividing by t , one can see that

$$\frac{\ln I_t - \ln I_0}{t} \leq \frac{\int_0^t \left(\frac{\beta_{r(\tau)}^2}{2\sigma_{r(\tau)}^2} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}) \right) d\tau}{t} + \frac{G_t}{t}, \quad (3.2)$$

where $G_t = \int_0^t \frac{\sigma_{r(\tau)} S_\tau}{1 + \alpha_{r(\tau)} I_\tau^2} dB_\tau$. Note that G_t is a local martingales, whose quadratic variation is $\langle G_t, G_t \rangle_t = \int_0^t \frac{\sigma_{r(\tau)}^2 S_\tau^2}{(1 + \alpha_{r(\tau)} I_\tau^2)^2} d\tau \leq \frac{\Lambda_t^2 \sigma_t^2}{\mu_t^2} t$. Making use of the strong law of large numbers for local martingales leads to $\lim_{t \rightarrow \infty} \frac{G_t}{t} = 0$ a.s.. On the other hand, under the first condition of

Eq. (2.3) and taking the limit, it is obtained from the Birkhoff Ergodic theorem that

$$\begin{aligned} \lim_{t \rightarrow \infty} \frac{\ln I_t}{t} &\leq \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \frac{\beta_{r(\tau)}^2}{2\sigma_{r(\tau)}^2} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}) d\tau \\ &= \sum_{i=1}^N \pi_i \left(\frac{\beta_i^2}{2\sigma_i^2} - \mu_i - \gamma_i - \delta_i \right) < 0 \text{ a.s..} \end{aligned} \quad (3.3)$$

Now we consider the case that $\sigma_i^2 < \frac{\beta_i \mu_i}{\Lambda_i}$, that is $\frac{\Lambda_i}{\mu_i} < \frac{\beta_i}{\sigma_i^2}$. Noting that $\varphi(x)$ is monotone increasing for $x \in [0, \frac{\beta_i}{\sigma_i^2}]$ yields

$$\varphi\left(\frac{S_\tau}{1 + \alpha_{r(\tau)} I_\tau^2}\right) \leq \varphi(S_\tau) \leq \varphi\left(\frac{\Lambda_{r(\tau)}}{\mu_{r(\tau)}}\right) = \frac{\Lambda_{r(\tau)} \beta_{r(\tau)}}{\mu_{r(\tau)}} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}) - \frac{\Lambda_{r(\tau)}^2 \sigma_{r(\tau)}^2}{2\mu_{r(\tau)}^2} \quad (3.4)$$

By the similar arguments, we can conclude that

$$\begin{aligned} \lim_{t \rightarrow \infty} \frac{\ln I_t}{t} &\leq \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \frac{\Lambda_{r(\tau)} \beta_{r(\tau)}}{\mu_{r(\tau)}} - (\mu_{r(\tau)} + \gamma_{r(\tau)} + \delta_{r(\tau)}) - \frac{\Lambda_{r(\tau)}^2 \sigma_{r(\tau)}^2}{2\mu_{r(\tau)}^2} d\tau \\ &= \sum_{i=1}^N \pi_i \left(\frac{\Lambda_i \beta_i}{\mu_i} - (\mu_i + \gamma_i + \delta_i) - \frac{\Lambda_i^2 \sigma_i^2}{2\mu_i^2} \right) \\ &= \sum_{i=1}^N \pi_i (\mu_i + \gamma_i + \delta_i) \left(\frac{\sum_{i=1}^N \pi_i \left(\frac{\Lambda_i \beta_i}{\mu_i} - \frac{\Lambda_i^2 \sigma_i^2}{2\mu_i^2} \right)}{\sum_{i=1}^N \pi_i (\mu_i + \gamma_i + \delta_i)} - 1 \right) \\ &< 0 \text{ a.s..} \end{aligned} \quad (3.5)$$

In view of Eqs. (3.3) and (3.5), we get

$$\lim_{t \rightarrow \infty} I_t = 0 \text{ a.s..} \quad (3.6)$$

The proof is complete. $\square$

4. The Proof of Theorem 2.3

In this section, we will adopt Lemmas A.1 and A.2 (see the Appendix A) to prove Theorem 2.3. To begin with, we establish an partially integral Markov semigroup connected with model (1.4).

Throughout this section, if B is a vector or matrix, we use B' to denote its transpose.

Let $(S_t^{(i)}, I_t^{(i)}, R_t^{(i)})$ ($i \in \mathbb{S}$) be a solution of system

$$\begin{cases} dS_t^{(i)} = \left(\Lambda_i - \mu_i S_t^{(i)} - \frac{\beta_i S_t^{(i)} I_t^{(i)}}{1 + \alpha_i (I_t^{(i)})^2} + \theta_i R_t^{(i)} \right) dt - \frac{\sigma_i S_t^{(i)} I_t^{(i)}}{1 + \alpha_i (I_t^{(i)})^2} dB_t, \\ dI_t^{(i)} = \left(\frac{\beta_i S_t^{(i)} I_t^{(i)}}{1 + \alpha_i (I_t^{(i)})^2} - (\mu_i + \gamma_i) I_t^{(i)} \right) dt + \frac{\sigma_i S_t^{(i)} I_t^{(i)}}{1 + \alpha_i (I_t^{(i)})^2} dB_t, \\ dR_t^{(i)} = \left(\gamma_i I_t^{(i)} - (\mu_i + \theta_i) R_t^{(i)} \right) dt, \end{cases} \quad (4.1)$$

which is studied in [15].

Denote by $\mathcal{A}_i$ the differential operators

$$\mathcal{A}_i g = \frac{1}{2} \left(\frac{\partial}{\partial S} - \frac{\partial}{\partial I} \right)^2 \left[\frac{\sigma_i^2 S^2 I^2}{(1 + \alpha_i I^2)^2} \right] - \frac{\partial(h_i^1 g)}{\partial S} - \frac{\partial(h_i^2 g)}{\partial I} - \frac{\partial(h_i^3 g)}{\partial R}, \quad g \in L^1(\mathbb{X}, \Sigma, m),$$

where $\mathbb{X}$ is the state space of the SDE model (4.1), Σ is the σ -algebra of Borel subsets of $\mathbb{X}$, m is the Lebesgue measure on $(\mathbb{X}, \Sigma)$ and

$$h_i^1(S_t, I_t, R_t) = \Lambda_i - \mu_i S_t - \frac{\beta_i S_t I_t}{1 + \alpha_i I_t^2} + \theta_i R_t,$$

$$h_i^2(S_t, I_t, R_t) = \frac{\beta_i S_t I_t}{1 + \alpha_i I_t^2} - (\mu_i + \gamma_i + \delta_i) I_t,$$

$$h_i^3(S_t, I_t, R_t) = \gamma_i I_t - (\mu_i + \theta_i) R_t.$$

Next, we show that, for any $i \in \mathbb{S}$, the operator $\mathcal{A}_i$ generates an integral Markov semigroup $\{\mathcal{T}_i(t)\}_{t \geq 0}$ on the space $L^1(\mathbb{X}, \Sigma, m)$ and

$$\int_0^\infty \mathcal{T}_i(t) g dt > 0 \text{ a.e. on } \mathbb{X}.$$

Lemma 4.1 [15]. *For each point (S_0, I_0, R_0, i) with $i \in \mathbb{S}$ and $t > 0$, the transition probability function $\mathcal{T}_i(t, S_0, I_0, R_0, A_i)$ has a continuous density $k_i(t, S_t^{(i)}, I_t^{(i)}, R_t^{(i)}; S_0, I_0, R_0)$ with respect to the Lebesgue measure, where A_i is the diffusion matrix of system (4.1).*

Remark 4.1. By virtue of Lemma 4.1, it follows that $\{\mathcal{T}_i(t)\}_{t \geq 0}$ is an integral Markov semigroup with a continuous kernel $k_i(t, S_t^{(i)}, I_t^{(i)}, R_t^{(i)}; S_0, I_0, R_0)$ for $t > 0$.

Lemma 4.2 [15]. *For each $(S_0, I_0, R_0, i) \in \Delta \times \mathbb{S}$ and $(S_t^{(i)}, I_t^{(i)}, R_t^{(i)}, i) \in \Delta \times \mathbb{S}$, there exists $T > 0$ such that $k_i(T, S_t^{(i)}, I_t^{(i)}, R_t^{(i)}; S_0, I_0, R_0) > 0$, where Δ is defined as in Eq. (2.1).*

Remark 4.2. According to Lemmas 4.1 and 4.2, for every $g \in \mathbb{D}$ and $i \in \mathbb{S}$, we have

$$\int_0^\infty \mathcal{T}_i(t) g dt > 0 \text{ a.s. on } \Delta,$$

where Δ and $\mathbb{D}$ are given in Eqs. (2.1) and (A.1) (see the Appendix A), respectively.

Remark 4.3. From Remark 4.1 and Lemma 4.2, it follows immediately that semigroup $\{\mathcal{T}_i(t)\}_{t \geq 0}$ is partially integral.

Let (S_t, I_t, R_t) be the unique solution of system (1.4) with $(S_0, I_0, R_0, r(0)) \in \mathbb{X} \times \mathbb{S}$, then $(S_t, I_t, R_t, r(t))$ constitutes a Markov process on $\mathbb{X} \times \mathbb{S}$. In view of Section 5 in [57], for $t > 0$ the distribution of the process $(S_t, I_t, R_t, r(t))$ is absolutely continuous, denote by $u = (u_1, u_2, \dots, u_N)$ (where $u_i := u(t, x, y, z, i)$) the density distribution of the process $(S_t, I_t, R_t, r(t))$. Then the vector u satisfies the following master equation:

$$\frac{\partial u}{\partial t} = \Gamma' u + \mathcal{A} u, \quad (4.2)$$

where $\mathcal{A} u = (\mathcal{A}_1 u_1, \mathcal{A}_2 u_2, \dots, \mathcal{A}_N u_N)'$. By virtue of Remark 4.1, for any $i \in \mathbb{S}$ the operator $\mathcal{A}_i$ generates an integral semigroup $\{\mathcal{T}_i(t)\}_{t \geq 0}$ on the space $L^1(\mathbb{X}, \Sigma, m)$.

Now, let $\mathbb{Y} = \mathbb{X} \times \mathbb{S}$, F be the σ -algebra of Borel subsets of $\mathbb{Y}$, and let $\hat{m}$ be the product measure on $(\mathbb{Y}, F)$ given by $\hat{m}(B \times \{i\}) = m(B)$ for each $B \in \Sigma$ and $i \in \mathbb{S}$. Obviously, $\mathcal{A} u$

generates a continuous Markov semigroup $\{\mathcal{T}(t)\}_{t \geq 0}$ on the space $L^1(\mathbb{Y}, F, \hat{m})$, given by the formula

$$\mathcal{T}(t)g = (\mathcal{T}_1(t)g(S, I, R, 1), \mathcal{T}_2(t)g(S, I, R, 1), \dots, \mathcal{T}_N(t)g(S, I, R, 1)), \quad g \in L^1(\mathbb{Y}, F, \hat{m}).$$

Now, let λ be a constant such that $\lambda > \max_{1 \leq i \leq N} \{-\gamma_{ii}\}$ and $(q_{ij})_{N \times N} = Q = \lambda^{-1} \Gamma' + \text{Id}$. Then Eq. (4.2) can be written in the form

$$\frac{\partial u}{\partial t} = \lambda Qu - \lambda u + Au, \quad (4.3)$$

and Q is a Markov operator on $L^1(\mathbb{Y}, F, \hat{m})$.

From the Philips perturbation theorem [58], Eq. (4.2) with the initial condition $u(0, S, I, R, k) = g(S, I, R, k)$ generates a continuous semigroup of Markov operators $\{P(t)\}_{t \geq 0}$ on the space $L^1(\mathbb{Y})$ given by

$$P(t)g = e^{-\lambda t} \sum_{n=0}^{\infty} \lambda^n \Phi^{(n)}(t)g, \quad (4.4)$$

where $\Phi^{(0)}(t) = \mathcal{T}(t)$ and

$$\Phi^{(n+1)}(t)g = \int_0^t \Phi^{(0)}(t-s)Q\Phi^{(n)}(s)gds, \quad n \geq 0, \quad (4.5)$$

The semigroup $\{P(t)\}_{t \geq 0}$ satisfies the integral equation

$$P(t)g = e^{-\lambda t} \mathcal{T}(t)g + \lambda \int_0^t e^{-\lambda s} \mathcal{T}(s)QP(t-s)gds. \quad (4.6)$$

Remark 4.4. By virtue of Remark 4.3 and (4.6), it follows that semigroup $\{P(t)\}_{t \geq 0}$ is partially integral, where $P(t)$ and is given in Eq. (4.4).

Next, we will prove that solutions of model (1.4) are converging to the endemic dynamics almost surely by Lemmas 4.3–4.5.

Lemma 4.3. For every $g \in \mathbb{D}$

$$\int_0^\infty P(t)gdt > 0 \quad \text{almost everywhere on } \mathbb{Y},$$

where $\mathbb{D}$ is given in Eq. (A.1) (see Appendix A).

Proof. By Remark 4.4, $\{P(t)\}_{t \geq 0}$ is a partially integral Markov semigroup. In view of Remark 4.2, Eq. (1.1) and $q_{ij} > 0 (i \neq j)$, we know that, for every nonnegative $g \in L^1(\mathbb{Y})$ with $\|g\| = 1$,

$$\int_0^\infty P(t)gdt > 0 \quad \text{almost everywhere on } \mathbb{Y}.$$

This completes the proof. $\square$

Lemma 4.4. The semigroup $\{P(t)\}_{t \geq 0}$ is asymptotically stable or is sweeping with respect to compact sets.

Proof. If the semigroup $\{P(t)\}_{t \geq 0}$ has an invariant density g^* , then from Lemma 4.3 it follows that $g^* > 0$ almost everywhere. If a Markov semigroup has two different invariant densities then it has two invariant densities with disjoint supports, which is impossible in our case.

Thus the semigroup $\{P(t)\}_{t \geq 0}$ has at most one invariant density. Fix $t > 0$ and $(S_0, I_0, R_0) \in \Delta$. Since

$$\iiint_{\Delta} k_i(t, S, I, R; S_0, I_0, R_0) dS dI dR = 1, \quad i \in \mathbb{S},$$

where $k_i(t, S, I, R; S_0, I_0, R_0)$ is a continuous kernel of the Markov semigroup $\{\mathcal{T}_i(t)\}_{t \geq 0}$, there exist an $(S_0, I_0, R_0) \in \Delta$ and a $\lambda > 0$ such that

$$k_i(t, S, I, R; S_0, I_0, R_0) > \lambda.$$

From continuity of the kernel k_i it follows that there exists an ϵ such that $k_i(t, S, I, R; S_0, I_0, R_0) > \lambda$ for $(S, I, R) \in B_\epsilon(S_0, I_0, R_0)$ and $(U, V, W) \in B_\epsilon(S_0, I_0, R_0)$. Let $\eta(S, I, R) = \lambda \mathbf{1}_{B_\epsilon(S_0, I_0, R_0)}(S, I, R)$, where $\mathbf{1}_{B_\epsilon(S_0, I_0, R_0)}$ is the indicator function of $B_\epsilon(S_0, I_0, R_0)$. Then

$$k_i(t, S, I, R; U, V, W) \geq \eta(S, I, R)$$

for $(S, I, R) \in \Delta$ and $(U, V, W) \in B_\epsilon(S_0, I_0, R_0)$. By virtue of Eq. (4.6), condition (b) of Lemma A.2 (see the Appendix A) holds, which completes the proof. $\square$

Lemma 4.5. *If $\mathcal{R}_0^s > 1$, that is $\sum_{i=1}^N \pi_i \mathcal{R}_i > 0$, then the semigroup $\{P(t)\}_{t \geq 0}$ is asymptotically stable, where $\mathcal{R}_i = \frac{\Lambda_i \beta_i}{\mu_i} - (\mu_i + \gamma_i + \delta_i) - \frac{\Lambda_i^2 \sigma_i^2}{2\mu_i^2}$.*

Proof. We will construct a nonnegative C^2 -function V and a closed set $U \in \Sigma$ (which lies entirely in Δ) such that for any $i \in \mathbb{S}$,

$$\sup_{(S, I, R) \in \Delta \setminus U} \mathcal{A}V(S, I, R, i) < 0,$$

where

$$\begin{aligned} \mathcal{A}V(S, I, R, i) = & \frac{\sigma_i^2 S^2 I^2}{2(1 + \alpha I^2)^2} \left[\frac{\partial^2 V}{\partial S^2} - 2 \frac{\partial^2 V}{\partial S \partial I} + \frac{\partial^2 V}{\partial I^2} \right] + f_i^1 \frac{\partial V}{\partial S} + f_i^2 \frac{\partial V}{\partial I} + f_i^3 \frac{\partial V}{\partial R} \\ & + \sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij} (V(S, I, R, j) - V(S, I, R, i)) \end{aligned}$$

and

$$f_i^1(S, I, R) = \Lambda_i - \mu_i S_t - \frac{\beta_i S_t I_t}{1 + \alpha_i I_t^2} + \theta_i R_t,$$

$$f_i^2(S, I, R) = \frac{\beta_i S_t I_t}{1 + \alpha_i I_t^2} - (\mu_i + \gamma_i) I_t,$$

$$f_i^3(S, I, R) = \gamma_i I_t - (\mu_i + \theta_i) R_t.$$

In fact, $\mathcal{A}$ is the adjoint operator of the infinitesimal generator of the semigroup $\{P(t)\}_{t \geq 0}$.

From the irreducibility of the generator matrix Γ , one can get that for $\tilde{\mathcal{R}} = (\mathcal{R}_1, \mathcal{R}_2, \dots, \mathcal{R}_N)'$, there exists $\varpi = (\varpi_1, \varpi_2, \dots, \varpi_N)'$ satisfying the following Poisson system (see [59], Lemma 2.3)

$$\Gamma \varpi - \tilde{\mathcal{R}} = - \sum_{i=1}^N \pi_i \mathcal{R}_i \mathbf{1},$$

where $\mathbf{1} = (1, 1, \dots, 1)'$. Therefore, we have

$$\sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij} (\varpi_j - \varpi_i) - \mathcal{R}_i = - \sum_{i=1}^N \pi_i \mathcal{R}_i < 0.$$

Define a C^2 -function

$$H(S, I, R, i) = M(-\ln I - \ell_1 \ln S + \ell_2 I^2 + \ell_3(\varpi_i + |\varpi|)) \\ - \ln S - \ln(L - \tilde{N}) - \ln(\tilde{N} - I), \quad i \in \mathbb{S},$$

where $L = \max\{\frac{\Lambda_i}{\mu_i}\}$, $l = \min\{\frac{\Lambda_i}{\mu_i + \alpha_i}\}$, $\ell_1 = \frac{c_i}{\mu_i}$, $\ell_2 = \frac{\alpha_i c_i}{2(\mu_i + \gamma_i + \delta_i)}$, $\ell_3 = \frac{3}{\left(\frac{\Lambda_i \beta_i}{\mu_i c_i}\right)^{\frac{2}{3}} + \left(\frac{\Lambda_i \beta_i}{\mu_i c_i}\right)^{\frac{1}{3}} + 1}$, $c_i = \mu_i + \gamma_i + \delta_i + \frac{\sigma_i^2 \Lambda_i^2}{2\mu_i^2}$, $\tilde{N} = S + I + R$ and $M > 0$ satisfies the following condition constant

$$-M\ell_3 \sum_{i=1}^N \pi_i \mathcal{R}_i + \mu_i + \frac{\beta_i \Lambda_i}{\mu_i} + \frac{\sigma_i^2}{4\alpha_i} + \mu_i + \theta_i + \mu_i + \mu_i + \delta_i \leq -2.$$

It is easy to see that $H(S, I, R, i)$ has a unique minimum point in Δ and its minimum value is denoted by M_1 . We define a nonnegative C^2 -function V as follows:

$$V(S, I, R, i) = H(S, I, R, i) - M_1 \\ = M(-\ln I - \ell_1 \ln S + \ell_2 I^2 + \ell_3(\varpi_i + |\varpi|)) - \ln S - \ln(L - \tilde{N}) - \ln(\tilde{N} - I) - M_1 \\ = MV_1 + V_2 + V_3 + V_4 - M_1,$$

where $V_1 = -\ln I - \ell_1 \ln S + \ell_2 I^2 + \ell_3(\varpi_i + |\varpi|)$, $V_2 = -\ln S$, $V_3 = -\ln(L - \tilde{N})$, $V_4 = -\ln(\tilde{N} - I)$.

By the generalized Itô's formula, one can get that

$$\mathcal{A}V_2 = -\frac{\Lambda_i}{S} + \mu_i + \frac{\beta_i I}{1 + \alpha_i I^2} - \frac{\theta_i R}{S} + \frac{\sigma_i^2 I^2}{2(1 + \alpha_i I^2)^2} \\ \leq -\frac{\Lambda_i}{S} + \mu_i + \beta_i I + \frac{\sigma_i^2}{4\alpha_i} \leq -\frac{\Lambda_i}{S} + \mu_i + \frac{\beta_i \Lambda_i}{\mu_i} + \frac{\sigma_i^2}{4\alpha}, \\ \mathcal{A}V_3 = -\frac{\gamma_i I - (\mu_i + \theta_i)R}{R} = \mu_i + \theta_i - \frac{\gamma_i I}{R}, \\ \mathcal{A}V_4 = -\frac{\Lambda_i - \mu_i \tilde{N} - \delta_i I}{\tilde{N} - I} \leq \mu_i + \delta_i - \frac{\delta_i S}{\tilde{N} - I}, \\ \mathcal{A}V_5 = \frac{\Lambda_i - \mu_i \tilde{N} - \delta_i I}{L - \tilde{N}} \leq \mu_i - \frac{\delta_i I}{L - \tilde{N}}.$$

Moreover, we also have

$$\mathcal{A}V_1 = \frac{-\beta_i S}{1 + \alpha_i I^2} + (\mu_i + \gamma_i + \delta_i) + \frac{\sigma_i^2 S^2}{2(1 + \alpha_i I^2)^2} - \frac{\ell_1 \Lambda}{S} + \ell_1 \mu_i + \frac{\ell_1 \beta_i I}{1 + \alpha_i I^2} - \frac{\ell_1 \theta_i R}{S} + \frac{\ell_1 \sigma_i^2 I^2}{2(1 + \alpha_i I^2)^2} \\ + 2\ell_2 I \left(\frac{\beta_i S I}{1 + \alpha_i I^2} - (\mu_i + \gamma_i + \delta_i) I \right) + \frac{\ell_2 \sigma_i^2 S^2 I^2}{(1 + \alpha_i I^2)^2} + \ell_3 \sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij} (\varpi_j - \varpi_i) \\ \leq \frac{-\beta_i S}{1 + \alpha_i I^2} - \frac{\ell_1 \Lambda_i}{S} - \frac{2\ell_2 (\mu_i + \gamma_i + \delta_i) (1 + \alpha_i I^2)}{\alpha_i} + \frac{2\ell_2 (\mu_i + \gamma_i + \delta_i)}{\alpha_i} + (\mu_i + \gamma_i + \delta_i) + \frac{\sigma_i^2 \Lambda_i^2}{2\mu_i^2} \\ + \ell_1 \mu_i + \ell_1 \beta_i I + \frac{\ell_1 \sigma_i^2 I^2}{2} + 2\ell_2 \beta_i S I^2 + \ell_2^2 \sigma_i^2 S^2 I^2 + \ell_3 \sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij} (\varpi_j - \varpi_i)$$

$$\begin{aligned}
&\leq -3\sqrt[3]{\frac{2\ell_1\ell_2\Lambda_i\beta_i(\mu_i+\gamma_i+\delta_i)}{\alpha_i}} + c_i + \ell_1\mu_i + \frac{2\ell_2(\mu_i+\gamma_i+\delta_i)}{\alpha_i} + \ell_3 \sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij}(\varpi_j - \varpi_i) \\
&\quad + \ell_1\beta_i I + \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) I^2 \\
&= -3c_i \left(\sqrt[3]{\frac{\Lambda_i\beta_i}{\mu_i c_i}} - 1 \right) + \ell_3 \sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij}(\varpi_j - \varpi_i) + \ell_1\beta_i I + \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) I^2 \\
&= \frac{-3c_i \left(\frac{\Lambda_i\beta_i}{\mu_i c_i} - 1 \right)}{\left(\frac{\Lambda_i\beta_i}{\mu_i c_i} \right)^{\frac{2}{3}} + \left(\frac{\Lambda_i\beta_i}{\mu_i c_i} \right)^{\frac{1}{3}} + 1} + \ell_3 \sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij}(\varpi_j - \varpi_i) + \ell_1\beta_i I + \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) I^2 \\
&= -\ell_3 \left(\mathcal{R}_i - \sum_{j \neq i, j \in \mathbb{S}} \gamma_{ij}(\varpi_j - \varpi_i) \right) + \ell_1\beta_i I + \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) I^2 \\
&= -\ell_3 \mathcal{R}_0^s + \ell_1\beta_i I + \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) I^2,
\end{aligned}$$

Hence

$$\begin{aligned}
\mathcal{A}V &\leq -M\ell_3\mathcal{R}_0^s + \ell_1 M\beta_i I + M \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) I^2 - \frac{\Lambda_i}{S} + \mu_i + \frac{\beta_i\Lambda_i}{\mu_i} + \frac{\sigma_i^2}{4\alpha_i} \\
&\quad + \mu_i + \theta_i - \frac{\gamma_i I}{R} + \mu_i + \delta_i - \frac{\delta_i S}{\tilde{N} - l} + \mu_i - \frac{\delta_i I}{L - \tilde{N}} \\
&\leq \ell_1 M\beta_i I + M \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) I^2 - \frac{\Lambda_i}{S} - \frac{\gamma_i I}{R} - \frac{\delta_i S}{\tilde{N} - l} - \frac{\delta_i I}{L - \tilde{N}} - 2.
\end{aligned}$$

Define a closed set

$$D_\varepsilon = \{(S, I, R) \in \Delta : \varepsilon \leq S \leq L, \varepsilon \leq I \leq L, \varepsilon^2 \leq R \leq L, l + \varepsilon^2 \leq \tilde{N} \leq L - \varepsilon^2\},$$

where $0 < \varepsilon < 1$ is a sufficiently small constant such that

$$\begin{aligned}
&\ell_1 M\beta_i \varepsilon + M \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) \varepsilon^2 - 2 \leq -1, \\
&\ell_1 M\beta_i \frac{\Lambda_i}{\mu_i} + M \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) \frac{\Lambda_i^2}{\mu_i^2} - \frac{\Lambda_i}{\varepsilon} - 2 \leq -1, \\
&\ell_1 M\beta_i \frac{\Lambda_i}{\mu_i} + M \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) \frac{\Lambda_i^2}{\mu_i^2} - \frac{\gamma_i}{\varepsilon} - 2 \leq -1, \\
&\ell_1 M\beta_i \frac{\Lambda_i}{\mu_i} + M \left(\frac{\ell_1\sigma_i^2}{2} + \frac{2\ell_2\beta_i\Lambda_i}{\mu_i} + \frac{\ell_2^2\sigma_i^2\Lambda_i^2}{\mu_i^2} \right) \frac{\Lambda_i^2}{\mu_i^2} - \frac{\delta_i}{\varepsilon} - 2 \leq -1,
\end{aligned}$$

For convenience, we divide $\Delta \setminus D_\varepsilon$ into five domains

$$\begin{aligned}
D_1 &= \{(S, I, R) \in \Delta : 0 < I < \varepsilon\}, \quad D_2 = \{(S, I, R) \in \Delta : 0 < S < \varepsilon\}, \\
D_3 &= \{(S, I, R) \in \Delta : 0 < R < \varepsilon^2, I \geq \varepsilon\}, \quad D_4 = \{(S, I, R) \in \Delta : l < \tilde{N} < l + \varepsilon^2, \\
&\quad S \geq \varepsilon_i, I \geq \varepsilon, R \geq \varepsilon^2\}, \\
D_5 &= \{(S, I, R) \in \Delta : L - \varepsilon^2 < \tilde{N} < L, S \geq \varepsilon, I \geq \varepsilon, R \geq \varepsilon^2\}.
\end{aligned}$$

Clearly, $\Delta \setminus D_\varepsilon = D_1 \cup D_2 \cup D_3 \cup D_4 \cup D_5$. Next, we will prove $LV(S, I, R) \leq -1$ for any $(S, I, R) \in \Delta \setminus D_\varepsilon$.

Case 1. On domain D_1 , we get

$$\begin{aligned} \mathcal{A}V &\leq \ell_1 M \beta_i I + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) I^2 - \frac{\Lambda_i}{S} - \frac{\gamma_i I}{R} - \frac{\delta_i S}{\tilde{N} - l} - \frac{\delta_i I}{L - \tilde{N}} - 2 \\ &\leq \ell_1 M \beta_i \varepsilon + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) \varepsilon^2 - 2 \\ &\leq -1. \end{aligned}$$

Case 2. On domain D_2 , one can see that

$$\begin{aligned} \mathcal{A}V &\leq \ell_1 M \beta_i I + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) I^2 - \frac{\Lambda_i}{S} - \frac{\gamma_i I}{R} - \frac{\delta_i S}{\tilde{N} - l} - \frac{\delta_i I}{L - \tilde{N}} - 2 \\ &\leq \ell_1 M \beta_i \frac{\Lambda_i}{\mu_i} + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) \frac{\Lambda_i^2}{\mu_i^2} - \frac{\Lambda_i}{\varepsilon} - 2 \\ &\leq -1. \end{aligned}$$

Case 3. On domain D_3 yields

$$\begin{aligned} \mathcal{A}V &\leq \ell_1 M \beta_i I + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) I^2 - \frac{\Lambda_i}{S} - \frac{\gamma_i I}{R} - \frac{\delta_i S}{\tilde{N} - l} - \frac{\delta_i I}{L - \tilde{N}} - 2 \\ &\leq \ell_1 M \beta_i \frac{\Lambda_i}{\mu_i} + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) \frac{\Lambda_i^2}{\mu_i^2} - \frac{\gamma_i \varepsilon}{\varepsilon^2} - 2 \\ &\leq -1. \end{aligned}$$

Case 4. On domain D_4 , one can get that

$$\begin{aligned} \mathcal{A}V &\leq \ell_1 M \beta_i I + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) I^2 - \frac{\Lambda_i}{S} - \frac{\gamma_i I}{R} - \frac{\delta_i S}{\tilde{N} - l} - \frac{\delta_i I}{L - \tilde{N}} - 2 \\ &\leq \ell_1 M \beta_i \frac{\Lambda_i}{\mu_i} + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) \frac{\Lambda_i^2}{\mu_i^2} - \frac{\delta_i \varepsilon}{\varepsilon^2} - 2 \\ &\leq -1, \end{aligned}$$

Case 5. On domain D_5 , one can get that

$$\begin{aligned} \mathcal{A}V &\leq \ell_1 M \beta_i I + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) I^2 - \frac{\Lambda_i}{S} - \frac{\gamma_i I}{R} - \frac{\delta_i S}{\tilde{N} - l} - \frac{\delta_i I}{L - \tilde{N}} - 2 \\ &\leq \ell_1 M \beta_i \frac{\Lambda_i}{\mu_i} + M \left(\frac{\ell_1 \sigma_i^2}{2} + \frac{2\ell_2 \beta_i \Lambda_i}{\mu_i} + \frac{\ell_2^2 \sigma_i^2 \Lambda_i^2}{\mu_i^2} \right) \frac{\Lambda_i^2}{\mu_i^2} - \frac{\delta_i \varepsilon}{\varepsilon^2} - 2 \\ &\leq -1. \end{aligned}$$

Consequently

$$\mathcal{A}V(S, I, R) \leq -1, \quad \text{for } \forall (S, I, R) \in \Delta \setminus D_\varepsilon.$$

Table 5.1

Parameter values in numerical simulations for model (1.4).

Parameters	Values	Units	References
Λ : The constant recruitment of new susceptibles	0.33	days ⁻¹	[61]
μ : Natural mortality rate	0.006	days ⁻¹	[61]
α : Measures the psychological or inhibitory effect	0.001		[15]
θ : Rate of appearance of new antigenic variants	0.021	days ⁻¹	[61]
δ : The disease-induced death rate	0.06	days ⁻¹	[61]
γ : Recovery rate of infected individuals	0.04	days ⁻¹	[61]
β : Transmission rate	0.0013 or 0.0056	days ⁻¹	[61]

By using similar arguments to those in [55], the existence of a Khasminskii function implies that the semigroup is not sweeping from the set D_ε . According to Lemma 4.4, the semigroup $\{P(t)\}_{t \geq 0}$ is asymptotically stable, which completes the proof. $\square$

5. Numerical results

In this section we provide numerical simulation results to substantiate the analytical findings for the stochastic model reported in the previous sections. Using the Milsteins Higher Order Method mentioned in Higham [60], we obtain the following discretization equations

$$\begin{cases} S_{k+1} = S_k + \left(\Lambda(i) - \mu(i)S_k - \frac{\beta(i)S_k I_k}{1 + \alpha(i)I_k^2} - \theta(i)R_k \right) \Delta t - \frac{\sigma(i)S_k I_k}{1 + \alpha(i)I_k^2} \sqrt{\Delta t} \xi_k - \frac{\sigma^2(i)}{2} \frac{S_k I_k}{1 + \alpha(i)I_k^2} (\xi_k^2 - 1) \Delta t, \\ I_{k+1} = I_k + \left(\frac{\beta(i)S_k I_k}{1 + \alpha(i)I_k^2} - (\mu(i) + \gamma(i) + \delta(i))I_k \right) \Delta t + \frac{\sigma(i)S_k I_k}{1 + \alpha(i)I_k^2} \sqrt{\Delta t} \xi_k + \frac{\sigma^2(i)}{2} \frac{S_k I_k}{1 + \alpha(i)I_k^2} (\xi_k^2 - 1) \Delta t, \\ R_{k+1} = R_k + \left(\gamma(i)I_k - (\mu(i) + \theta(i))R_k \right) \Delta t, \end{cases} \quad (5.1)$$

where time increment $\Delta t > 0$ and $\xi_k (k = 1, 2, \dots, n)$ are independent Gaussian random variables which follow $N(0, 1)$. Here we assume $r(t)$ is a right-continuous Markov chain taking values in a finite state space $\mathbb{S} = \{1, 2\}$ with the generator $\Gamma = \begin{pmatrix} -73 & 73 \\ 292 & -292 \end{pmatrix}$. Then the unique stationary distribution of $r(t)$ is given by $\pi = (\pi_1, \pi_2) = (0.8, 0.2)$. In this section, we only focus on the transmission rate β of model (1.4) is disturbed by a random switching because in reality it is more sensitive to environmental fluctuations than other parameters for human populations. Motivated by Cai et al. [15] and Anderson and May [61] the parameter values in model (1.4) are chosen as follows (in this section the unit of time is one day): $\Lambda(1) = \Lambda(2) = 0.33$, $\mu(1) = \mu(2) = 0.006$, $\alpha(1) = \alpha(2) = 0.001$, $\theta(1) = \theta(2) = 0.021$, $\gamma(1) = \gamma(2) = 0.04$, $\delta(1) = \delta(2) = 0.06$ (See, Table 5.1 for details). In addition, we always assume that the initial value of system (1.4) is $(S_0, I_0, R_0, r(0)) = (50, 1, 0, 1) \in \Delta \times \mathbb{S}$. We then divide our simulations into two cases:

Case 1: firstly, fix $\beta(1) = 0.0013$ and $\beta(2) = 0.0056$ which are taken from the work [61]. Then we use different vector fields values of $\sigma = (\sigma(1), \sigma(2))'$ in order to investigate the effect of the white noise on the transmission dynamics of the disease. we consider three different fields values of σ : $(1 \times 10^{-3}, 1 \times 10^{-3})'$, $(2 \times 10^{-3}, 2 \times 10^{-3})'$ and $(4 \times 10^{-3}, 4 \times 10^{-3})'$. The corresponding values of $\mathcal{R}_0^s$ are 1.1065, 1.0637 and 0.8925. By Remark 2.3, we

Fig. 5.1. The evolution of infected individuals of the stochastic model (1.4) is graphed for yield fields σ : $(1 \times 10^{-3}, 1 \times 10^{-3})$, $(2 \times 10^{-3}, 2 \times 10^{-3})$ and $(4 \times 10^{-3}, 4 \times 10^{-3})$.

see that the disease is persistent in the first two cases, while the disease is extinctive in the last case (i.e. $\sigma_i^2 < \frac{\beta_i \mu_i}{\Lambda_i}$ for any $i \in \{1, 2\}$ and $\mathcal{R}_0^s < 1$). The computer simulations shown in Figs. 5.1 and 5.2 clearly support these results.

Fig. 5.1 shows trends of the evolution of $I(t)$, one can see that as the noise intensities $\sigma(1)$, $\sigma(2)$ increases, the variability of the stochastic model increases. (b) of Fig. 5.2 shows the corresponding probability density function (PDF) of the path $I(t)$ at $t = 1000$ for model (1.2) based on 100,000 stochastic simulations. As can be seen in (b) of Fig. 5.2, for first two cases (i.e., $\mathcal{R}_0^s > 1$), both appear skew to the right and the skewness becomes larger as the intensities $\sigma(1)$, $\sigma(2)$ of the white noise increases; in the last case ($\mathcal{R}_0^s < 1$), the PDF of the distribution of $I(t)$ is concentrated on the small neighborhood of zero. Here we can conclude that the small environmental perturbations can generate the irregular cycling phenomena of recurrent diseases, while the large ones will eradicate diseases. This means the small perturbations of the white noise can sustain the irregular recurrence of the disease (such as influenza, SARS) in humans between two pandemics, and larger ones may be beneficial, leading to the extinction of the disease. In order to further illustrate the effect of the random switching on the transmission dynamics of the disease, we carry out another case.

Case 2: for comparison, we also plot the component-wise sample paths and PDF of I -class in Figs. 5.3 and 5.4. Here, we assume the transmission rate $\beta(1) = 0.0023$, $\beta(2) = 0.0056$, $\sigma(1) = \sigma(2) = 4 \times 10^{-3}$. Then the corresponding values of $\mathcal{R}_0^s$ are 0.9651, 2.6774 and 1.3075 when there is only one state $r = 1$, $r = 2$ and switching back and forth from one state $r = 1$ to another state $r = 2$, respectively. The computer simulations in Figs. 5.3 and 5.4 support these results clearly.

In Fig. 5.3 represent the sample paths of I of state 1, state 2 and hybrid system. It is clear that the path of hybrid system is between the paths of state 1 and state 2, that is, the hybrid system is stochastically permanent if the two subsystems are stochastically

Fig. 5.2. (a) The evolution of a single path of $I(t)$ for model (1.4). (b) The corresponding probability density function (PDF) of the path $I(t)$ at $t = 1000$ for model (1.4) based on 100,000 stochastic simulations.

permanent and is extinctive if the two subsystems are extinctive. (b) of Fig. 5.4 shows the probability density function (PDF) of the path $I(t)$ in state 1, state 2 and hybrid system, respectively. In addition, Figs. 5.3 and 5.4 indicate that an interesting fact: if one of the subsystems is extinctive, another is stochastically permanent, then the hybrid system may be stochastically permanent if the Markov chain spends enough time on the stochastically permanent state. From biological point of view, the Markov chain $r(t)$ is conducive to the survival of the disease. That is to say, the Markov chain can suppress the extinction of the disease.

Fig. 5.3. The red line, blue line and black line represent the paths of hybrid system, state 1 and state 2, respectively. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

6. Concluding remarks and future directions

This article formulates a stochastic SIRS epidemic model with regime switching and the infectious forces under intervention strategies and applied our theoretical results to the inter-pandemic evolution dynamic of the disease in the host population based on realistic parameter values obtained from previous literatures. Combining analytical results with numerical simulations, a threshold dynamic determined by the basic reproduction number

$\mathcal{R}_0^s := \frac{\sum_{i=1}^N \pi_i (\frac{\Lambda_i \beta_i}{\mu_i} - \frac{\Lambda_i^2 \sigma_i^2}{2\mu_i^2})}{\sum_{i=1}^N \pi_i (\mu_i + \gamma_i + \delta_i)}$ is established: the disease can be eradicated almost surely if $\mathcal{R}_0^s < 1$ and under mild extra conditions; whereas if $\mathcal{R}_0^s > 1$, the densities of the distributions of the solution can converge to the unique stationary distribution.

Compared with previous researches, the main contributions of the present study are as follows:

- We can see that SDE (1.4) is an extension of Eq. (1.2) and the basic reproduction number $\mathcal{R}_0^s$ is an extension of $\bar{R}$. Particularly, the SDE (1.4) becomes to Eq. (1.2), and $\mathcal{R}_0^s$ becomes to $\bar{R}$ when $N = 1$. Mathematical deduction in present paper faces greater challenges than Cai et al. [15]. Moreover, we found the interesting fact: if one of the subsystems is extinctive, another is stochastically permanent, then the hybrid system may be stochastically permanent if the Markov chain spends enough time on the stochastically permanent state. From biological point of view, the Markov chain $r(t)$ is conducive to the survival of the disease. That is to say, the Markov chain can suppress the extinction of the disease. Hence, the telegraph noise is an important factor, it has a direct effect on the evolution of the epidemic (see Figs. 5.3 and 5.4).
- Compared with results in Liu et al. [42], Han et al. [43] for a stochastic epidemic model with the fluctuation by both white and telegraph noises in the transmission rate of disease, the advantages in this paper lie in: (1) the existence of endemic stationary distribution is

Fig. 5.4. (a) The sample paths of I of state 1, state 2 and hybrid system. (b) The corresponding probability density function (PDF) of the path $I(t)$ at $t = 1000$ for model (1.4) based on 100,000 stochastic simulations.

proved in the case of persistence; (2) the effect of the white and telegraph noises on the epidemiological consequences is studied based on realistic parameter values.

- The effect of the white noise was not studied in Li et al. [53]. The present paper shows that white noise play a very important role in determining persistence or extinction of diseases in the transmission of infectious disease. And it also reveals an interesting result: the small perturbations of the white noise can sustain the irregular recurrence of the disease in the host population during two pandemics, and larger ones may be beneficial, leading to the extinction of the disease (see Figs. 5.1 and 5.2).

Some interesting topics deserve further investigation. In this paper, the threshold for stochastic model (1.2) under consideration is determined merely when the intensities of white

noise is relatively small. In fact, we also attempts to study the problem which if $\mathcal{R}_0^s < 1$ and without any other condition, the disease I_t tends to zero exponentially. Unfortunately, there are some technical obstacles that cannot be overcome at present stage. On the other hand, one may propose some more realistic but complex models, such as considering the impact of the number of hospital beds [62] on system (1.4). We leave these investigations for future research.

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Appendix A

As the proof of our result is based on the theory of integral Markov semigroups, we need some auxiliary definitions and results concerning Markov semigroups [54–57]. For the convenience of the reader, we present these definitions and results in the appendix.

Definition A.1 (Markov operator). Let $\Sigma = \mathcal{B}$ be the σ -algebra of Borel subset of $\mathbb{X}$, and m the Lebesgue measure on $(\mathbb{X}, \Sigma)$. $\mathbb{D} = \mathbb{D}(\mathbb{X}, \Sigma, m)$ denotes the subset of the space $L^1 = L^1(\mathbb{X}, \Sigma, m)$ which contains all densities, that is

$$\mathbb{D} = \{g \in L^1 : g \geq 0, \|g\| = 1\}, \quad (\text{A.1})$$

where $\|\cdot\|$ represents the norm in L^1 and $\mathbb{X}$ is the state space of SDE model (4.1). A linear mapping $P: L^1 \rightarrow L^1$ is called a Markov operator if $P(\mathbb{D}) \subset \mathbb{D}$.

Definition A.2 (Integrable Markov operator). Suppose that $k: \mathbb{X} \times \mathbb{X} \rightarrow [0, +\infty)$ is a measurable function such that

$$\int_{\mathbb{X}} k(\mathbf{x}, \mathbf{y}) m(d\mathbf{y}) = 1, \quad (\text{A.2})$$

for almost all $\mathbf{x} \in \mathbb{X}$, then $Pg(\mathbf{x}) = \int_{\mathbb{X}} k(\mathbf{x}, \mathbf{y}) g(\mathbf{y}) m(d\mathbf{y})$ is an integral Markov operator. The function k is called a kernel of the Markov operator P .

Definition A.3 (Markov semigroup). A family $\{P(t)\}_{t \geq 0}$ of Markov operator which satisfies conditions

- (a) $P(0) = \text{Id}$ (Id denotes identity matrix);
- (b) $P(t+s) = P(t)P(s)$ for $s, t \geq 0$;
- (c) The function $t \rightarrow P(t)g$ is continuous for every $g \in L^1$,

then $\{P(t)\}_{t \geq 0}$ is called a Markov semigroup.

Definition A.4 (Integrable Markov semigroup). A Markov semigroup $\{P(t)\}_{t \geq 0}$ is called integral, if for every $t > 0$, the operator $P(t)$ is an integral Markov operator, that is, there exists a measurable function $k: (0, \infty) \times \mathbb{X} \times \mathbb{X} \rightarrow [0, \infty)$ such that

$$P(t)g(\mathbf{x}) = \int_{\mathbb{X}} k(t, \mathbf{x}, \mathbf{y}) g(\mathbf{y}) m(d\mathbf{y}),$$

for each density g .

Definition A.5 (Asymptotically stable). For a Markov semigroup $\{P(t)\}_{t \geq 0}$, a density g^* is called invariant, if $P(t)g^* = g^*$ for $t > 0$. The Markov semigroup is called asymptotically stable if there is an invariant density g^* such that

$$\lim_{t \rightarrow \infty} \|P(t)g - g^*\| = 0 \quad \text{for } g \in \mathbb{D}.$$

Remark A.1. If the Markov semigroup $\{P(t)\}_{t \geq 0}$ is formed by a differential equation (e.g. model (1.2)), then the asymptotic stability of Markov semigroup implies that all of solutions of the equation starting from a density converge to the invariant density.

Definition A.6 (Partially integral). A Markov semigroup $\{P(t)\}_{t \geq 0}$ is called partially integral if there exist $t_0 > 0$ and a measurable nonnegative function $k(\mathbf{x}, \mathbf{y})$ such that

$$\int_{\mathbb{X}} \int_{\mathbb{X}} k(\mathbf{x}, \mathbf{y}) m(d\mathbf{x}) m(d\mathbf{y}) > 0 \quad (\text{A.3})$$

and

$$P(t_0)g(\mathbf{x}) \geq \int k(\mathbf{x}, \mathbf{y}) g(\mathbf{y}) m(d\mathbf{y}) \quad \text{for every } g \in \mathbb{D}. \quad (\text{A.4})$$

Lemma A.1. Let $\{P(t)\}_{t \geq 0}$ be a partially integral Markov semigroup. Assume that $\{P(t)\}_{t \geq 0}$ has only one invariant density g^* . If $g^* > 0$ then this semigroup $\{P(t)\}_{t \geq 0}$ is asymptotically stable.

Definition A.7 (Sweeping). If for every $g \in \mathbb{D}$ and a set $B \in \Sigma$

$$\lim_{t \rightarrow \infty} \int_B P(t)g(\mathbf{x}) m(d\mathbf{x}) = 0,$$

then the Markov semigroup $\{P(t)\}_{t \geq 0}$ is called sweeping with respect to B .

Lemma A.2. Assume that an integral Markov semigroup $\{P(t)\}_{t \geq 0}$ has the following properties:

- (a) for every $g \in \mathbb{D}$ we have $\int_0^\infty P(t)g dt > 0$ almost everywhere,
- (b) for every $\mathbf{y}_0 \in \mathbb{X}$ there exists $\epsilon > 0$ and a measurable function $\eta \geq 0$ such that $\int \eta dm > 0$ and

$$k(\mathbf{x}, \mathbf{y}) \geq \eta(\mathbf{x}) \mathbf{1}_{B(\mathbf{y}_0, \epsilon)}(\mathbf{y}),$$

where k is a function satisfying Eqs. (A.3) and (A.4), $B(\mathbf{y}_0, \epsilon)$ denotes an open ball of radius ϵ centered at $\mathbf{y}_0$, $\mathbf{1}_{B(\mathbf{y}_0, \epsilon)}$ is the indicator function of $B(\mathbf{y}_0, \epsilon)$. If the semigroup $\{P(t)\}_{t \geq 0}$ has no invariant density then it is sweeping with respect to compact sets.

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