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A STOCHASTIC EPIDEMIC MODEL INCORPORATING MEDIA COVERAGE*

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Abstract. In this paper, we investigate the effects of environment fluctuations on disease dynamics through studying the stochastic dynamics of an SIS model incorporating media coverage. The value of this study lies in two aspects: Mathematically, we show that the disease dynamics the SDE model can be governed by its related basic reproduction number R_0^S: if $R_0^S \leq 1$, the disease will die out stochastically, but if $R_0^S > 1$, the disease will break out with probability one. Epidemiologically, we partially provide the effects of the environment fluctuations affecting spread of the disease incorporating media coverage. First, noise can suppress the disease outbreak. Notice that $R_0^S < R_0$, and it is possible that $R_0^S < 1 < R_0$. This is the case when the deterministic model has an endemic while the SDE model has disease extinction with probability one. Second, two stationary distribution governed by R_0^S: If $R_0^S < 1$, it has disease-free distribution which means that the disease will die out with probability one; while $R_0^S > 1$, it has endemic stationary distribution, which leads to the stochastically persistence of the disease. In order to understand the role of media coverage on disease dynamics, we present some numerical simulations to validate the analytical findings. It is interesting to note that although some parameters have no role in determining R_0^s, however the strength of noise to the susceptible population and the parameters characterizing media affect play crucial role in determining the long term dynamics of the system.

Key words. Epidemic model, Lyapunov function, stochastic asymptotic stability, ergodic property.

AMS subject classifications. 92D30, 60H10, 93E15.

1. Introduction

Infectious diseases are the second leading cause of death worldwide, after heart disease, and are responsible for more deaths annually than cancer [31]. Understanding the mechanism that underlies the spread of an infectious disease can give important insights to help in the fight against the disease itself [4].

When an infectious disease appears and spreads in a region, the departments for disease control and prevention will do everything possible to prevent the disease from spreading. One of the measures is to tell people the correct preventive knowledge of the disease as soon as possible through media and education [15, 16, 33, 36, 45, 54]. Mass media (television, radio, newspapers, billboards, and booklets) have been used as a way of delivering preventive health messages, as they have the potential to influence people's behavior, and deter them from risky behavior or from taking precautionary measures in relation to a disease outbreak, as concurrent presentation of objective information about the diseases can mitigate its severity [8, 11, 57].

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Media coverage of an infectious outbreak can be seen as following two major routes. The first route is when the media report directly to the public on facts that they (the media) observe; the second has public health authorities using mass media or the Internet to communicate about the outbreak [3, 49]. The responsibilities of the media are to disseminate health information and to frequently cover health-related topics, and as such, the media are the leading source of information about important health issues for many individuals [8], and the mass media have been used as a way of delivering preventive health messages due to their potential influence on people's behavior [11, 20, 55]. People's response to the threat of disease is dependent on their perception of risk, which is influenced by public and private information disseminated widely by the media [41, 50]. For instance, mass media campaigns based on communication for behavioral impact and social change have been shown to be an effective intervention for smoking cessation in adults [8]. In the case of a vaccine-preventable disease, people may opt to vaccinate more (and promptly), when the perceived risk from the disease is high, and little (and later) otherwise [17]. Human behavior change consequently leads to a reduction in the number of real susceptible individuals or effective contact rates. The study showed that media coverage and education may reduce the contact rate of human beings as we have observed during the spreading of severe acute respiratory syndrome (SARS) in 2002 and 2004 [16, 35]. Another study showed that during the outbreak of influenza A (H1N1) in 2009, media coverage played an important role in helping both the government authority make interventions to contain the disease and people respond to the disease [11, 55].

Media coverage is obviously not the most important factor responsible for the transmission of the infectious disease, but it is a very important issue that has to be taken care of seriously. In the case of a large number of infected cases, the media coverage may cause the panic of the society, but it can certainly reduce the opportunity for and probability of contact transmission among the alerted susceptible populations, which in turn helps to control and prevent the disease from spreading further [15]. To examine the role of media coverage on disease outbreaks and curb the spread of infectious diseases, mathematical modeling can therefore play an important role in helping understand the potential effects of media coverage on infectious disease transmission. In [54], Xiao and Ruan formulated an SIR (susceptible-infectious-recovered) model, proposed a non-linear incidence rate to describe the effect of mass media coverage, and showed that media coverage did not have any obvious effect on disease dynamics. Cui *et al.* [15, 16, 33, 45] presented a series study on the epidemic models incorporating media coverage and concluded that media coverage was critical in disease eradication. Sun *et al.* [49] formulated an SIS epidemic model on two patches and found that media coverage can reduce the burden of the epidemic and shorten the duration of the disease outbreak. In [41], the authors formulated an SIS model to study the impact of awareness programs conducted by a media campaign on the spread of an infectious disease and showed that the spread of an infectious disease can be controlled by using awareness programs but the disease remains endemic due to immigration.

On the other hand, in many instances, environmental variations have a critical influence on the development of an epidemic [42, 51]. For human disease, the nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts [47] and because the population is subject to a continuous spectrum of disturbances [2, 9]. Hence the variability and randomness of the environment is fed through to the state of the epidemic [52]. And in epidemic dynamics, stochastic differential equation (SDE) models could be a more appropriate way of modeling epidemics

in many circumstances, and many realistic stochastic epidemic models can be derived based on their deterministic formulations [1, 2, 5, 6, 7, 10, 12, 13, 14, 18, 21, 22, 26, 27, 28, 30, 32, 34, 37, 38, 39, 40, 42, 43, 44, 51, 52, 53, 56].

And there comes a question: How do environmental fluctuations affect the spreading of disease, when incorporating media coverage?

In this paper, we will focus on the effects of environment fluctuations on the disease's dynamics through studying the stochastic dynamics of an SIS model incorporating media coverage. The rest of this article is organized as follows: In Section 2, based on the results of Cui *et al.* [16], we derive a stochastic differential SIS model incorporating media coverage. In Section 3, we give the conditions of stochastic extinction of the model. In Section 4, following the method given by Khasminskii [29], we give the conditions of the existence of a unique stationary distribution of the SDE model. In Section 5, we provide one example to support our research results. In the last section, we provide a brief discussion and summary of our main results.

2. Model derivation

Let $S(t)$ and $I(t)$ be the number of susceptible and infectious individuals at time t, respectively. In the absence of media effect, we assume a classic standard (or proportional) incidence, with the rate at which new infections arise given by $\frac{\beta SI}{S+I}$, β being the infection coefficient. When media coverage is present, social distancing mechanisms come into effect. Reporting by the media is assumed to be an increasing function of the number of infectious cases present, and as a consequence, the contact rate between susceptible and infectious individuals is a decreasing function of the number of infectious cases present. We take similar non-linear functions as in [16] and denote the effective contact rate as

$$\beta(I) = \beta_1 - \beta_2 f(I),$$

where β_1 is the usual contact rate without considering the infectious individuals and β_2 is the maximum reduced contact rate due to the presence of the infected individuals. But we know that everyone cannot avoid contact with others in any case, so we assume that $\beta_1 > \beta_2$. The function $f(I)$ satisfies

(A1) $f(0) = 0$, $\lim_{I\to\infty} f(I) = 1$, $0 < f'(I) \le 1$ and $f''(I) < 0$.

It follows from the work of Cui *et al.* [16] that an SIS epidemic model incorporating media coverage takes the following form

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \mu S - (\beta_1 - \beta_2 f(I))\dfrac{SI}{S+I} + \gamma I, \\ \dfrac{dI}{dt} = (\beta_1 - \beta_2 f(I))\dfrac{SI}{S+I} - (\mu+\gamma)I, \end{cases} \tag{2.1}$$

where Λ is the recruitment rate of the population, μ the natural death rate of the population, and γ the recovery rate of infectious individuals.

For model (2.1), the basic reproduction number is defined as

$$R_0 = \frac{\beta_1}{\mu+\gamma}. \tag{2.2}$$

It is easy to see that

$$\lim_{t\to\infty} N(t) = S(t) + I(t) = \frac{\Lambda}{\mu}.$$

Hence, the plane $S+I=\dfrac{\Lambda}{\mu}$ is an invariant manifold of model (2.1). Throughout this paper, we always assume that N is fixed.

A simple calculation shows that model (2.1) has two equilibrium points: one is disease-free equilibrium $E_0=(\Lambda/\mu,0)$ which exists for all parameter values and is globally stable when $R_0<1$, and the other is the endemic equilibrium $E^*=(S^*,I^*,)$ satisfying

$$S^*=\frac{\Lambda}{\mu}-I^*,\quad \frac{\mu I^*}{\Lambda}-\left(1-\frac{\mu+\gamma}{\beta_1-\beta_2 f(I^*)}\right)=0 \tag{2.3}$$

if $R_0>1$. The endemic equilibrium $E^*=(S^*,I^*,)$ is global stable when it exists. For more details, see [16].

There are different possible approaches to including random effects in the model, from both a biological and a mathematical perspective [25]. In this article, we adopt the approach by Beddington and May [9], which has been pursued in [2, 13, 26, 39, 52, 56]. Mathematically speaking, this approach is based on the assumption that the noise is uniform over the state space and over time [19]. Thus, stochastic perturbation in our model is a white noise type that is directly proportional to $S(t)$, $I(t)$, and $R(t)$ and is influenced on the $\frac{\mathrm{d}S(t)}{dt}$, $\frac{\mathrm{d}I(t)}{dt}$ and $\frac{\mathrm{d}R(t)}{dt}$, respectively. Following this approach, we obtain the following SDE epidemic model (2.4) that is analog to its deterministic version (2.1) by introducing stochastic perturbation terms to the growth equations of susceptible, infectious, and recovered individuals to incorporate the effect of randomly fluctuating environments:

$$\begin{cases} dS(t)=\left(\Lambda-\mu S-(\beta_1-\beta_2 f(I))\dfrac{SI}{S+I}+\gamma I\right)dt+\sigma_1 S dB_1(t),\\ dI(t)=\left((\beta_1-\beta_2 f(I))\dfrac{SI}{S+I}-(\mu+\gamma)I\right)dt+\sigma_2 I dB_2(t), \end{cases} \tag{2.4}$$

where $\sigma_i\,(i=1,2)$ is a real constant, $\sigma_i^2\,(i=1,2)$ is known as the intensity of environmental fluctuations, and $B_i(t)\,(i=1,2)$ independent standard Brownian motions.

Throughout this paper, let $(\mathbb{R}^2_+,\mathcal{F},\mathcal{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\in\mathbb{R}_+}$ satisfying the usual conditions (i.e., it is right continuous and increasing while $\mathcal{F}_0$ contains all $\mathcal{P}$-null sets). We also denote $\mathbb{R}^2_+=\{(S,I)|S,I>0\}$.

For the existence of the positive global solution of model (2.4), one can obtain following results.

THEOREM 2.1. *For any given initial value $(S(0),I(0))\in\mathbb{R}^2_+$, there is a unique solution $(S(t),I(t))$ of model (2.4) on $t\geq 0$ that will remain in $\mathbb{R}^2_+$ with probability one.*

The proof of this theorem is rather standard and hence is omitted.

3. Stochastic disease-free dynamics

In this section, we focus on the stochastic disease-free dynamics as well as the feature of the stochastic dynamics of $S(t)$ of model (2.4) when the disease extinct. First, we give the following theorem regarding the extinction of the disease.

THEOREM 3.1. *If either of the following conditions hold*

$$\sigma_2^2<2\beta_1\quad\text{and}\quad R_0^s:=\frac{\beta_1}{\mu+\gamma}-\frac{\sigma_2^2}{2(\mu+\gamma)}=R_0\left(1-\frac{\sigma_2^2}{2\beta_1}\right)<1, \tag{3.1}$$

$$\sigma_2^2\geq 2\beta_1, \tag{3.2}$$

then, for any given initial value $(S(0),I(0))=(S_0,I_0)\in\mathbb{R}^2_+$, $I(t)$ *obeys*

$$\limsup_{t\to\infty}\frac{1}{t}\log I(t)\le(\mu+\gamma)(R_0^S-1)<0\quad a.s.$$

Namely, $I(t)$ *tends to zero exponentially almost surely. In other words, the disease dies out with probability one.*

Proof. By the Itô formula, we have

$$d\log I(t)=\left((\beta_1-\beta_2 f(I))\frac{S}{S+I}-(\mu+\gamma)-\frac{\sigma_2^2}{2}\right)dt+\sigma_2 dB_2(t). \tag{3.3}$$

Hence,

$$\begin{aligned}\log I(t)&=\log I_0+\int_0^t\left((\beta_1-\beta_2 f(I))\frac{S}{S+I}-(\mu+\gamma)-\frac{\sigma_2^2}{2}\right)ds+\int_0^t\sigma_2 dB_2(s)\\&\le\log I_0+\int_0^t\left(\beta_1-\beta_2 f(0)-(\mu+\gamma)-\frac{\sigma_2^2}{2}\right)ds+\int_0^t\sigma_2 dB_2(s)\\&=\log I_0+\left(\beta_1-(\mu+\gamma)-\frac{\sigma_2^2}{2}\right)t+G(t),\end{aligned} \tag{3.4}$$

where $G(t)$ is a martingale defined by

$$G(t)=\int_0^t\sigma_2 dB_2(s).$$

This implies

$$\langle G,G\rangle_t=\int_0^t\sigma_2^2 ds=\sigma_2^2 t.$$

By the strong law of large numbers for martingales [39], we have

$$\limsup_{t\to\infty}\frac{G(t)}{t}=0\ a.s.$$

It finally follows from (3.4) by dividing t on the both sides and then letting $t\to\infty$ that

$$\begin{aligned}\limsup_{t\to\infty}\frac{1}{t}\log I(t)&\le\beta_1-(\mu+\gamma)-\frac{\sigma_2^2}{2}\\&=(\mu+\gamma)\left(R_0^S-1\right)<0\quad a.s.\end{aligned} \tag{3.5}$$

which is the required assertion. ∎

Remark 3.1. Notice that $R_0^s=\dfrac{\beta_1}{\mu+\gamma}-\dfrac{\sigma_2^2}{2(\mu+\gamma)}$. Thus, according to Theorem 3.1, the condition of the extinction of a disease is independent of the maximum reduced contact rate β_2.

Next, we will focus on the stochastic dynamics of $S(t)$ of model (2.4) when $R_0^S<1$.

THEOREM 3.2. *If $R_0^s<1$, for model (2.4), then distribution of the process $\psi(t)=\ln S(t)$ converges weakly to the measure which has the density*

$$g_*(\psi)=K_1\exp\left\{-\frac{1}{\sigma_1^2}(2\Lambda\exp\{-\psi\}+2\mu\psi+\sigma_1^2\psi)\right\}, \tag{3.6}$$

where $K_1=\left(\int_{-\infty}^{\infty}\exp\left\{-\frac{1}{\sigma_1^2}(2\Lambda\exp\{-\psi\}+2\mu\psi+\sigma_1^2\psi)\right\}d\psi\right)^{-1}$.

Proof. According to Theorem 3.1, if $R_0^s<1$, we get $\lim_{t\to\infty} I(t)=0$, a.s.. That is to say, for $\forall 0<\varepsilon\ll 1$, there exists a constant $T_1=T_1(\omega)$ and a set Ω_ε such that $\mathcal{P}(\Omega_\varepsilon)>1-\varepsilon$, $0\le I(t)\le\varepsilon$ for $t>T_1$ and $\omega\in\Omega_\varepsilon$. Then

$$\Lambda-\mu S-\beta_1\varepsilon+\sigma_1 S dB_1(t)\le dS(t)\le\Lambda-\mu S+\gamma\varepsilon+\sigma_1 S dB_1(t).$$

For arbitrary ε, we obtain

$$dS(t)=\Lambda-\mu S+\sigma_1 S dB_1(t). \tag{3.7}$$

By putting $S(t)=\exp\{\psi(t)\}$, we have

$$d\psi(t)=\left(\Lambda\exp\{-\psi(t)\}-\mu-\frac{\sigma_1^2}{2}\right)dt+\sigma_1 dB_1(t).$$

Then the above equation has a unique stationary distribution which has a density $g_*(\psi)$ satisfying the Fokker–Planck equation

$$\frac{1}{2}\sigma_1^2\frac{d^2g_*(\psi)}{d\psi^2}-\frac{d}{d\psi}\left(\left(\Lambda\exp\{-\psi\}-\mu-\frac{\sigma_1^2}{2}\right)g_*(\psi)\right)=0. \tag{3.8}$$

The general solution to equation (3.8) is

$$\begin{aligned}g_*(\psi)=&\exp\left\{-\frac{1}{\sigma_1^2}(2\Lambda\exp\{\psi\}+2\mu\psi+\sigma_1^2\psi)\right\}\\&\cdot\left(K_1-K_2\int_0^r\exp\left\{\frac{1}{\sigma_1^2}(2\Lambda\exp\{\psi\}+2\mu\psi+\sigma_1^2\psi)\right\}dr\right),\end{aligned}$$

where K_1,K_2 are two constants. It follows easily from the conditions

$$g_*(\psi)\ge 0,\ \int_{-\infty}^{\infty}g_*(\psi)d\psi=1,$$

that $K_2=0$ and

$$K_1=\left(\int_{-\infty}^{\infty}\exp\left\{-\frac{1}{\sigma_1^2}(2\mu\exp\{-\psi\}+2\mu\psi+\sigma_1^2\psi)\right\}d\psi\right)^{-1}.$$

Therefore,

$$g_*(\psi)=K_1\exp\left\{-\frac{1}{\sigma_1^2}(2\Lambda\exp\{-\psi\}+2\mu\psi+\sigma_1^2\psi)\right\}.$$

By the existence of a stationary distribution [46], $\psi(t)=\ln S(t)$ converges to the measure with the density $g_*(\psi)$ as $t\to\infty$. ∎

4. Stochastic endemic dynamics

The deterministic SIS model (2.1) is globally stable at its endemic equilibrium E^* whenever $R_0 = \dfrac{\beta_1}{\mu+\gamma} > 1$ [16]. Since model (2.4) is the perturbed system of model (2.1), it seems reasonable to consider that the disease will prevail if the solution of model (2.4) has the ergodic property. In what follows, unless otherwise specified, we assume that S^* and I^* satisfy condition (2.3).

4.1. Persistence of the disease. In the following, we will consider the persistence of the stochastic model (2.4).

THEOREM 4.1. *If* $R_0^s := \dfrac{\beta_1}{\mu+\gamma} - \dfrac{\sigma_2^2}{2(\mu+\gamma)} > 1$, *then for any given initial values* $I(0) \in (0, \Lambda/\mu)$, *the solution of the stochastic differential equation (2.4) obeys*

$$\limsup_{t\to\infty} I(t) \geq \xi, a.s. \tag{4.1}$$

and

$$\liminf_{t\to\infty} I(t) \leq \xi, a.s., \tag{4.2}$$

where ξ *is the positive root of*

$$g(I) := (\beta_1 - \beta_2 f(I))\left(1 - \frac{I}{N}\right) - (\mu+\gamma) - \frac{\sigma_2^2}{2} = 0.$$

Proof. In view of $R_0^s > 1$, we have $g(0) = (\beta_1 - (\mu+\gamma) - \dfrac{\sigma_2^2}{2} = (\mu+\gamma)(R_0^s - 1) > 0$ and $g(N) = -(\mu+\gamma) - \dfrac{\sigma_2^2}{2} < 0$. Then $g(I)$ admits a root $\xi \in (0, N)$. Moreover $g(I)$ is decreasing around ξ, so we can easily show that, for any sufficiently small $\varepsilon > 0$, we have

$$g(\xi+\varepsilon) < 0 < g(\xi-\varepsilon). \tag{4.3}$$

We now begin to prove assertion (4.1). If it is not true, then there is a sufficiently small $\epsilon > 0$ such that $\mathbb{P}(\Omega_1) > 0$, where $\Omega_1 = \left\{\limsup_{t\to\infty} I(t) \leq \xi - 2\epsilon\right\}$. Hence, for every $\omega \in \Omega_1$, there is a $T(\omega) > 0$ such that

$$0 \leq I(t,\omega) \leq \xi - \epsilon, \forall t \geq T(\omega). \tag{4.4}$$

It therefore follows from (4.3) and (4.4) that

$$g(I(t,\omega) \geq g(\xi-\epsilon), \forall t \geq T(\omega). \tag{4.5}$$

Moveover, by the strong law of large numbers for martingales [39], there is a $\Omega_2 \subset \Omega$ with $\mathbb{P}(\Omega_2) = 1$ such that for every $\omega \in \Omega_2$,

$$\limsup_{t\to\infty} \frac{G(t)}{t} = 0 \ a.s.$$

Now, fix any $\omega \in \Omega_1 \cap \Omega_2$. It then follows from (4.5), for $t \geq T(\omega)$

$$\log I(t,\omega) = \log I_0 + \int_0^{T(\omega)} (g(I(s)))\,ds + \int_{T(\omega)}^{t} (g(I(s)))\,ds + G(t)$$

$$\geq \log I_0 + \int_0^{T(\omega)} (g(I(s)))\,ds + g(\xi-\epsilon)(t-T(\omega)) + G(t). \tag{4.6}$$

This yields

$$\liminf_{t\to\infty} \frac{1}{t}\log I(t,\omega) \geq g(\xi-\epsilon) > 0,$$

whence, $\lim_{t\to\infty} I(t,\omega) = \infty$. This contradicts (4.4). The required assertion, (4.1), must therefore hold.

Similarly, if (4.2) were not true, we could then find an $\tilde{\epsilon} > 0$ sufficiently small such that $\mathbb{P}(\Omega_3) > 0$, where $\Omega_3 = \left\{\liminf_{t\to\infty} I(t) \geq \xi + 2\tilde{\epsilon}\right\}$. Hence, for every $\omega \in \Omega_3$, there is a $\tau(\omega) > 0$ such that

$$I(t,\omega) \geq \xi + \tilde{\epsilon}, \forall t \geq \tau(\omega). \tag{4.7}$$

Now, fix any $\omega \in \Omega_3 \cap \Omega_2$. It then follows from (4.5), for $t \geq T(\omega)$

$$\log I(t,\omega) \leq \log I_0 + \int_0^{\tau(\omega)} (g(I(s)))\,ds + g(\xi+\tilde{\epsilon})(t-T(\omega)) + G(t). \tag{4.8}$$

This yields

$$\limsup_{t\to\infty} \frac{1}{t}\log I(t,\omega) \leq g(\xi+\epsilon) < 0,$$

whence, $\lim_{t\to\infty} I(t,\omega) = 0$. This contradicts (4.7). This completes the proof of assertion (4.2). ∎

4.2. Stochastic asymptotic stability.

THEOREM 4.2. *If the following two conditions are satisfied,*

(i) $R_0^S > 1$*;*

(ii) $\sigma_1^2 < 2\mu$, $\sigma_2^2 < 2\mu$,

then for any initial value $(S(0),I(0)) \in \mathbb{R}_+^2$*, the solution* $(S(t),I(t))$ *of model (2.4) has the property*

$$\limsup_{t\to\infty} \frac{1}{t}\mathbb{E}\int_0^t \left(\left(S - \frac{2\mu}{2\mu-\sigma_1^2}S^*\right)^2 - \left(I - \frac{2\mu}{2\mu-\sigma_2^2}I^*\right)^2\right) d\tau \leq \frac{\Psi}{\Theta}.$$

where

$$\Psi = \frac{\mu\sigma_1^2}{2\mu-\sigma_1^2}S^{*2} + \frac{\mu\sigma_2^2}{2\mu-\sigma_2^2}I^{*2} + \frac{2\sigma_2^2\mu(S^*+I^*)}{2(\beta_1-\beta_2 f(I^*))}I^*,$$
$$\Theta = \min\left\{\mu - \frac{1}{2}\sigma_1^2, \mu - \frac{1}{2}\sigma_2^2\right\}.$$

Proof. Since $R_0^S > 1$, hence $R_0 = R_0^S + \dfrac{\sigma_2^2}{2(\mu+\gamma)} > 1$, there is an endemic equilibrium $E^* = (S^*,I^*)$ of model (2.1). Then we have

$$\Lambda = \mu S^* + (\beta_1 - \beta_2 f(I^*))\frac{S^*I^*}{S^*+I^*} - \gamma I^*,\ (\beta_1-\beta_2 f(I^*))\frac{S^*I^*}{S^*+I^*} = (\mu+\gamma)I^*. \tag{4.9}$$

Set

$$V(S,I)=\frac{1}{2}(S-S^*+I-I^*)^2+\lambda\left(I-I^*-I^*\ln\frac{I}{I^*}\right):=V_1(S,I)+\lambda V_2(I), \tag{4.10}$$

where λ is positive constants to be determined later. V is a nonnegative C^2–function. From (4.9), by the Itô formula, we have

$$\begin{aligned}
dV_1=&\left((S-S^*+I-I^*)(\Lambda-\mu S-(\mu+\ )I)+\frac{1}{2}(\sigma_1^2S^2+\sigma_2^2I^2)\right)dt\\
&+(S-S^*+I-I^*)(\sigma_1SdB_1(t)+\sigma_2IdB_2(t))\\
=&((S-S^*+I-I^*)(-\mu(S-S^*)-(\mu+\ )(I-I^*))+\frac{1}{2}(\sigma_1^2S^2+\sigma_2^2I^2))dt\\
&+(S-S^*+I-I^*)(\sigma_1SdB_1(t)+\sigma_2IdB_2(t))\\
=&(-\mu(S-S^*)^2-\mu(I-I^*)^2-2\mu(S-S^*)(I-I^*)+\frac{1}{2}(\sigma_1^2S^2+\sigma_2^2I^2))dt\\
&+(S-S^*+I-I^*)(\sigma_1SdB_1(t)+\sigma_2IdB_2(t))
\end{aligned}$$

and

$$\begin{aligned}
dV_2=&\left((I-I^*)\left((\beta_1-\beta_2f(I))\frac{S}{S+I}-(\mu+\gamma)\right)+\frac{1}{2}\sigma_2^2I^*\right)dt+(I-I^*)\sigma_2dB_2(t)\\
=&(I-I^*)\left((\beta_1-\beta_2f(I))\frac{S}{S+I}-(\beta_1-\beta_2f(I^*))\frac{S^*}{S^*+I^*}\right)+\frac{1}{2}\sigma_2^2I^*\\
&+(I-I^*)\sigma_2dB_2(t)\\
=&-\frac{\beta_2S}{S+I}(I-I^*)(f(I)-f(I^*))+(I-I^*)\frac{(\beta_1-\beta_2f(I^*))S(S^*-S+I^*-I)}{(S^*+I^*)(S+I)}\\
&+\frac{(\beta_1-\beta_2f(I^*))(S-S^*)(I-I^*)}{S^*+I^*}+\frac{1}{2}\sigma_2^2I^*+(I-I^*)\sigma_2dB_2(t)\\
\le&-\frac{(\beta_1-\beta_2f(I^*))S}{(S^*+I^*)(S+I)}(S^*-S)(I-I^*)+\frac{(\beta_1-\beta_2f(I^*))}{S^*+I^*}(S-S^*)(I-I^*)+\frac{1}{2}\sigma_2^2I^*\\
&+(I-I^*)\sigma_2dB_2(t)\\
\le&\frac{(\beta_1-\beta_2f(I^*))}{S^*+I^*}(S-S^*)(I-I^*)\text{sign}(S-S^*)(I-I^*)+\frac{1}{2}I^*\sigma_2^2+(I-I^*)\sigma_2dB_2(t).
\end{aligned}$$

Here, we use the facet that $f(I)$ is non-decreasing. Choose

$$\lambda=\frac{2\mu(S^*+I^*)}{\beta_1-\beta_2f(I^*)}$$

then

$$\begin{aligned}
dV\le&\left(-\mu(S-S^*)^2-\mu(I-I^*)^2+\frac{1}{2}(\sigma_1^2S^2+\sigma_2^2I^2)+\frac{\lambda}{2}\sigma_2^2I^*\right)dt\\
&+\sigma_1S(S-S^*+I-I^*)dB_1(t)+\sigma_2(I(S-S^*+I-I^*)+\lambda(I-I^*))dB_2(t)\\
:=&LV\,dt+\sigma_1S(S-S^*+I-I^*)dB_1(t)+\sigma_2(I(S-S^*+I-I^*)+\lambda(I-I^*))dB_2(t),
\end{aligned}$$

where

$$LV=-\mu(S-S^*)^2-\mu)(I-I^*)^2+\frac{1}{2}(\sigma_1^2S^2+\sigma_2^2I^2)+\frac{\lambda}{2}\sigma_2^2I^*$$

$$= -\left(\mu - \frac{1}{2}\sigma_1^2\right)\left(S - \frac{2\mu}{2\mu-\sigma_1^2}S^*\right)^2 - \left(\mu - \frac{1}{2}\sigma_2^2\right)\left(I - \frac{2\mu}{2\mu-\sigma_2^2}I^*\right)^2 + \frac{\mu\sigma_1^2}{2\mu-\sigma_1^2}S^{*2} + \frac{\mu\sigma_2^2}{2\mu-\sigma_2^2}I^{*2} + \frac{\lambda}{2}\sigma_2^2 I^*.$$

Thus integrating both sides of the equality from 0 to t and taking expectations, yields

$$\begin{aligned}&\mathbb{E}\tilde{V}(S(t),I(t)) - \mathbb{E}\tilde{V}(S(0),I(0))\\ &\le -\mathbb{E}\int_0^t \left(\left(\mu - \frac{1}{2}\sigma_1^2\right)\left(S - \frac{2\mu}{2\mu-\sigma_1^2}S^*\right)^2 - \left(\mu - \frac{1}{2}\sigma_2^2\right)\left(I - \frac{\mu}{2\mu-\sigma_2^2}I^*\right)^2\right)d\tau\\ &\qquad + \left(\frac{\mu\sigma_1^2}{2\mu-\sigma_1^2}S^{*2} + \frac{\mu\sigma_2^2}{2\mu-\sigma_2^2}I^{*2} + \frac{\lambda}{2}\sigma_2^2 I^*\right)t, \quad (4.11)\end{aligned}$$

Now take

$$\Theta = \min\left\{\mu - \frac{1}{2}\sigma_1^2, \mu - \frac{1}{2}\sigma_2^2\right\}.$$

Through dividing both sides of (4.11) by t and letting $t \to \infty$, we obtain

$$\limsup_{t\to\infty}\frac{1}{t}\mathbb{E}\int_0^t \left(\left(S - \frac{2\mu}{2\mu-\sigma_1^2}S^*\right)^2 - \left(I - \frac{2\mu}{2\mu-\sigma_2^2}I^*\right)^2\right)d\tau \le \frac{\Psi}{\Theta}.$$

The proof is complete. ∎

4.3. Stationary distribution and Ergodic property. Before giving the main theorem about the endemic stationary distribution, we first give a definition about stationary distribution [23] and some lemmas.

DEFINITION 4.3 (Stationary distribution [23]). *Let $P(t,X_0,\cdot)$ denote the probability measure induced by $X(t) = (S(t),I(t))$ with initial value $X_0 = (S(0),I(0))$; that is,*

$$P_{X_0}(X \in \boldsymbol{B}) = \mathcal{P}\{X(t)) \in \boldsymbol{B} : X(0) = X_0\} \text{ for any Borel set } \boldsymbol{B} \subset \mathbb{R}_+^2.$$

If there is a probability measure $\pi(\cdot)$ on the measurable space $(\mathbb{R}_+^2, \mathcal{B}(\mathbb{R}_+^2))$ such that

$$\lim_{t\to\infty} P_{X_0}(X \in \boldsymbol{B}) = \pi(\boldsymbol{B}) \text{ for any } X_0 \in \mathbb{R}_+^2,$$

we then say that model (2.4) has a stationary distribution $\pi(\cdot)$.

Let $X(t)$ be a regular temporally homogeneous Markov process in $\mathbb{R}_+^2$ described by the stochastic differential equation

$$dX(t) = F(X,t)dt + \sum_{r=1}^{2}\sigma_r(X)dB_r(t)$$

and the diffusion matrix is defined as follows

$$A(x) = ((a_{ij}(x))),\ a_{ij}(x) = \sum_{r=1}^{2}\sigma_r^i(x)\sigma_r^j(x).$$

For model (2.4), the diffusion matrix is

$$A(x) = \operatorname{diag}\left(\sigma_1^2 S^2, \sigma_2^2 I^2\right).$$

LEMMA 4.4 ([29]). *We assume that there exists a bounded domain $U \subset \mathbb{R}^2_+$ with regular boundary, which has the following properties:*

(i) In the domain U and some neighborhood thereof, the smallest eigenvalue of the diffusion matrix $A(x)$ is bounded away from zero.

(ii) If $x \in \mathbb{R}^2_+ \setminus U$, the mean time τ at which a path issuing from x reaches the set U is finite, and $\sup_{x\in K} \mathbb{E}_x \tau < \infty$ for every compact subset $K \subset \mathbb{R}^2_+$.

LEMMA 4.5 ([29]). *Suppose that Lemma 4.4 holds. Then the Markov process $X(t)$ has a unique stationary distribution $\pi(\cdot)$. Moreover, if $F(X,t)$ is a function integrable with respect to the measure π, then*

$$\mathcal{P}_x\left\{\lim_{T\to\infty} \frac{1}{T}\int_0^T F(X(t))dt = \int_{\mathbb{R}^2_+} F(t)\pi(dx)\right\} = 1$$

for all $x \in \mathbb{R}^2_+$.

We here omit the proof of the above lemmas, as the proofs can be found in [29].

Applying Theorem 4.2 and Lemmas 4.4 and 4.4, we can get the following result.

THEOREM 4.6. *Consider the stochastic model (2.4) with initial condition $(S(0), I(0)) \in \mathbb{R}^2_+$. Suppose that the assumptions in Theorem 4.2 and*

$$0 < \Psi < \min\left\{\frac{2\mu^2}{2\mu - \sigma_1^2} S^{*2}, \frac{2\mu+^2}{2\mu - \sigma_2^2} I^{*2}\right\}$$

hold, where Ψ has the same definitions as it does in Theorem 4.2. Then there exists a unique stationary distribution $\pi(\cdot)$, and the solution $((S(t), I(t))$ of model (2.4) is ergodic.

Proof. To verify (i) of Lemma 4.4, with reference to Zhu and Yin [58], it is sufficient to show that there exists some neighborhood U and a nonnegative C^2–function $V(x)$ such that, for some constants $C > 0$,

$$LV(x) < -C \text{ for any, } x \in \mathbb{R}^2_+ \setminus U.$$

To this end, we use the nonnegative C^2–function $V(S,I)$ as Theorem 4.2. Hence, it follows from Theorem 4.2 that

$$LV \le -\left(\mu - \frac{1}{2}\sigma_1^2\right)\left(S - \frac{2\mu}{2\mu - \sigma_1^2} S^*\right)^2 - \left(\mu - \frac{1}{2}\sigma_2^2\right)\left(I - \frac{2\mu}{2\mu - \sigma_2^2} I^*\right)^2 + \Psi.$$

Now since Ψ satisfies the following conditions,

$$0 < \Psi < \min\left\{\frac{2\mu^2}{2\mu - \sigma_1^2} S^{*2}, \frac{2\mu^2}{2\mu - \sigma_2^2} I^{*2}\right\},$$

the ellipsoid

$$\left(\mu - \frac{1}{2}\sigma_1^2\right)\left(S - \frac{2\mu}{2\mu - \sigma_1^2} S^*\right)^2 + \left(\mu - \frac{1}{2}\sigma_2^2\right)\left(I - \frac{2\mu}{2\mu - \sigma_2^2} I^*\right)^2 = \Psi$$

lies entirely in $\mathbb{R}^2_+$. One can then take U as any neighborhood of the ellipsoid such that $\bar{U}\subset\mathbb{R}^2_+$, where $\bar{U}$ is the closure of U. Thus, we have $LV(S,I)<0$ for $(S,I)\in\mathbb{R}^2_+\setminus U$, which implies that condition (ii) in Lemma 4.4 is satisfied.

On the other hand, there is $M=\min\{\sigma_1^2S^2,\sigma_2^2I^2\}>0$ such that

$$\operatorname{diag}\left(\sigma_1^2S^2,\sigma_2^2I^2\right)\xi_i\xi_j=\sigma_1^2S^2\xi_1^2+\sigma_2^2I^2\xi_2^2\geq M|\xi|^2,$$

for all $(S,I)\in\bar{U}$, $\xi\in\mathbb{R}^2_+$. Thus, by Rayleigh's principle (see [48], p.342), condition (i) in Lemma 4.4 is verified for model (2.4). As a consequence, the stochastic model (2.4) has a stationary distribution $\pi(\cdot)$ and is ergodic. □

5. An application

In this section, we apply our results, obtained in Section 4, to a stochastic SIS epidemic model, and we discuss the influence of noise intensity on disease transmission. We fix the function $f(I)$ as

$$f(I)=\frac{I}{b+I},\tag{5.1}$$

motivated by Cui *et al.* [16]. With this assumption the model (2.4) becomes

$$\begin{cases} dS(t)=\left(\Lambda-\mu S-\left(\beta_1-\dfrac{\beta_2 I}{b+I}\right)\dfrac{SI}{S+I}+\gamma I\right)dt+\sigma_1 S dB_1(t),\\ dI(t)=\left(\left(\beta_1-\dfrac{\beta_2 I}{b+I}\right)\dfrac{SI}{S+I}-(\mu+\gamma)I\right)dt+\sigma_2 I dB_2(t). \end{cases}\tag{5.2}$$

It is easy to verify that the condition (A1) is satisfied for the chosen form of $f(I)$. Model (5.2) without noise intensity has the disease-free equilibrium $E_0=(\Lambda/\mu,0)$ and the endemic equilibrium $E^*=(S^*,I^*)$ satisfying, if $R_0>1$,

$$S^*=\frac{\Lambda}{\mu}-I^*,\quad \frac{\mu I^*}{\Lambda}-\left(1-\frac{(\mu+\gamma)(b+I^*)}{(\beta_1-\beta_2)I^*+b\beta_1}\right)=0.$$

Now, we present some numerical simulation results to show the effect of noise on the dynamics of SIS models by using the Milstein method mentioned in Higham [24]. For this purpose, the SDE model (5.2) can be rewritten as the following discretized equations:

$$\begin{cases} S_{k+1}=S_k+\left(\Lambda-\mu S-\left(\beta_1-\dfrac{\beta_2 I}{b+I}\right)\dfrac{S_kI_k}{S_k+I_k}+\gamma I\right)\Delta t+\sigma_1 S_k\sqrt{\Delta t}\xi_k\\ \qquad\qquad +\dfrac{\sigma_1^2}{2}S_k(\xi_k^2-1)\Delta t,\\ I_{k+1}=I_k+\left(\left(\beta_1-\dfrac{\beta_2 I}{b+I}\right)\dfrac{S_kI_k}{S_k+I_k}-(\mu+\gamma)I\right)\Delta t+\sigma_2 I_k\sqrt{\Delta t}\eta_k+\dfrac{\sigma_2^2}{2}I_k(\eta_k^2-1)\Delta t, \end{cases}$$

where ξ_k and η_k, $k=1,2,\cdots,n$, are the kth realization of two independent Gaussian random normal variate $N(0,1)$.

Throughout the rest of this manuscript, the choice for the following parameters remain unaltered:

$$\Lambda=1,\quad \mu=0.05,\quad \beta_1=0.15,\quad \beta_2=0.1,\quad \gamma=0.02,b=10.\tag{5.3}$$

For the stochastic model (5.2), we choose $\sigma_1 = 0.3, \quad \sigma_2 = 0.5$, then we have

$$R_0^s = \frac{\beta_1}{\mu+\gamma} - \frac{\sigma_2^2}{2(\mu+\gamma)} = 0.357 < 1.$$

Thus according to Theorem 3.1, we can conclude that for any initial value $(S(0), I(0)) \in \mathbb{R}^2_+$, $I(t)$ obeys

$$\limsup_{t\to\infty} \frac{1}{t} \log I(t) \le -0.045 \ a.s.$$

That is, $I(t)$ will tend to zero exponentially with probability one (see Figure 5.1).

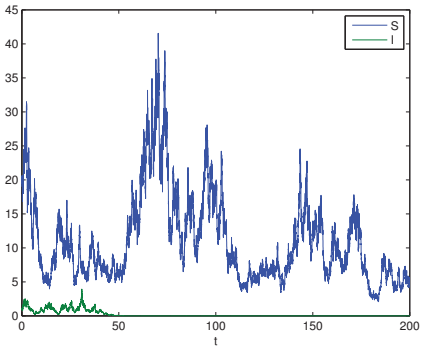

FIG. 5.1. *The path $S(t)$ and $I(t)$for the stochastic model (5.2) with initial values $(S(0), I(0)) = (19, 1)$. The parameters are taken as (5.3) and $\sigma_1 = 0.3, \sigma_2 = 0.5$.*

For the stochastic model (5.2), the basic reproduction number is given by

$$R_0^s = \frac{\beta_1}{\mu+\gamma} - \frac{\sigma_2^2}{2(\mu+\gamma)},$$

hence the important parameters (namely β_2, b, and σ_1) have no role in determining R_0^s. However, the strength of noise to the susceptible population and the parameters characterizing media effect play a crucial role in determining the long-term dynamics of the system. In order to understand the role, we can proceed step by step.

First we calculate the average time for extinction of I for three different values of σ_1 keeping other parameters fixed as mentioned earlier for which the condition $R_0^s < 1$ is satisfied. We have noted the time for which $I(t)$ is becoming zero for the first time and then calculated the average over 1000 simulations. The average extinction times are 89.91, 83.79, 78.73 for $\sigma_1 = 0.1, 0.2, 0.3$ respectively. It clearly shows that the extinction of infected species accelerated with the increasing noise strength on the susceptible population. Next we have collected the values of S at $t = 200$ from 1000 simulations for three different values of σ_1 and presented their distribution in Figure 5.2. Significant change in distribution of $S(200)$ with the variation of σ_1 explains the role of noise strength on the system dynamics.

To see the disease dynamics of (5.2) when $R_0^s > 1$, we decrease the noise intensity σ_2 of infectious I to be 0.25, i.e., $\sigma_2 = 0.25$, and keeping the other parameters unchanged. Then we have

$$R_0^s = \frac{\beta_1}{\mu+\gamma} - \frac{\sigma_2^2}{2(\mu+\gamma)} = 1.696 > 1.$$

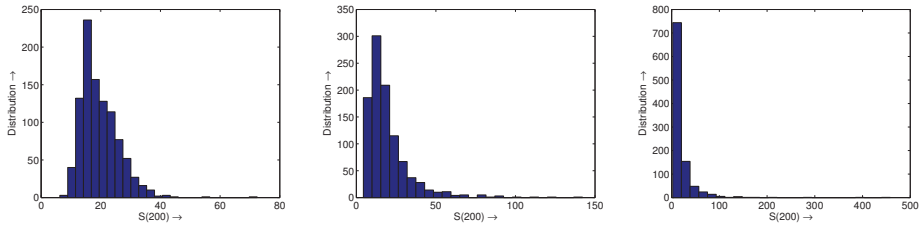

FIG. 5.2. *Distribution of $S(t)$ at $t = 200$ obtained from 1000 simulations and for three different values of σ_1, (a) $\sigma_1 = 0.1$; (b) $\sigma_1 = 0.2$; (c) $\sigma_1 = 0.3$.*

Therefore, the condition of Theorem 3.1 is not satisfied. In this case, our simulations suggest that $I(t)$ is stochastically persistent (see Figure 5.3).

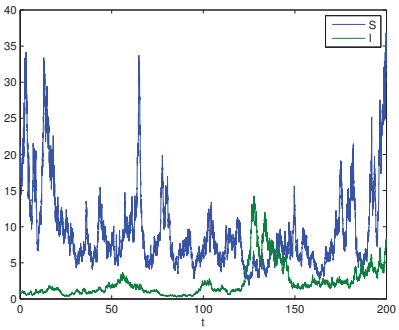

FIG. 5.3. *The path $S(t)$ and $I(t)$for the stochastic model (5.2) with initial values $(S(0), I(0)) = (19,1)$. The parameters are taken as (5.3) and $\sigma_1 = 0.3, \sigma_2 = 0.25$.*

Now we can check that how the magnitude of maximum reduced contact rate (β_2) due to coverage on media, change the system dynamics and in particular accelerate the extinction of infectious individuals. The average time required for the extinction of infectious individuals reduces with the increasing magnitude of β_2 when the parameter values and noise strengths satisfy the condition $R_0^s < 1$. We have collected the values of $I(t)$ at $t = 200$ from 1000 simulation runs for different magnitudes of β_2, with equal noise strengths $\sigma_1 = 0.01 = \sigma_2$ and other parameter values kept unaltered. This choice of σ_2 satisfies the condition $R_0^s > 1$ and hence both the susceptible and infected species coexist at all future time. It is interesting to observe that the mean value of $I(t = 200)$ decreases gradually with the increase in magnitude of β_2, the distribution of $I(t)$ obtained from 1000 simulation runs, for each chosen values of β_2, also changes significantly (see Figure 5.4). The level of infectious individuals sometimes attains the magnitude 20–30 when $\beta_2 = 0$ and 0.1, but they never cross the level $I = 10$ for $\beta_2 > 2.5$. The simulation results in favor of our claim can be verified from the range of horizontal axis at Figure 5.4(c) and (d).

In order to verify the analytical conditions mentioned in Theorem 4.6, we choose the parameter values as follows:

$$\Lambda = 1, \quad \mu = 0.05, \quad \beta_1 = 0.15, \quad \beta_2 = 0.5, b = 10, \quad \gamma = 0.02, \quad \sigma_1 = 0.01, \quad \sigma_2 = 0.04. \tag{5.4}$$

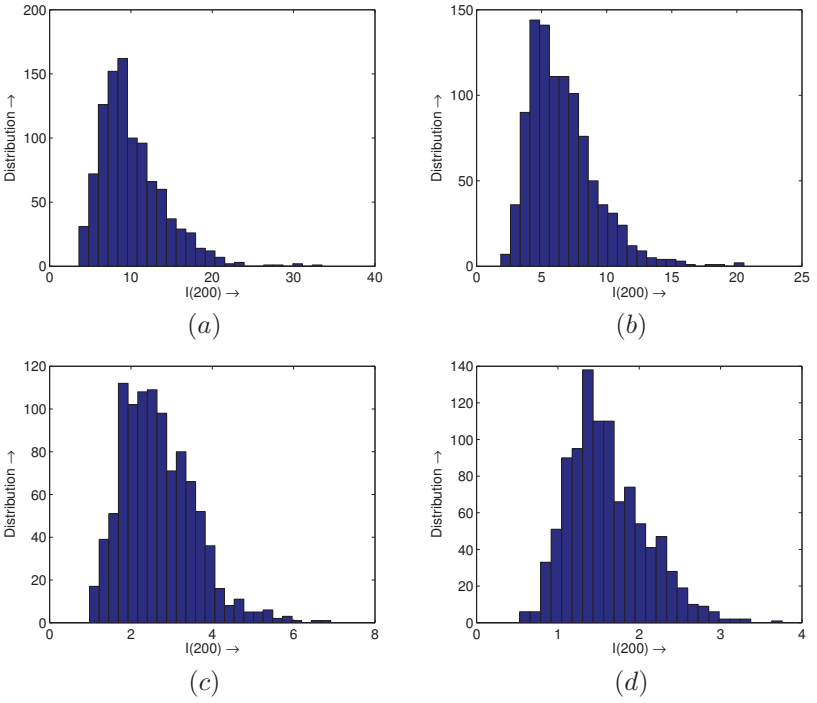

FIG. 5.4. *Distribution of $I(t)$ at $t = 200$ obtained from 1000 simulations and for four different values of β_2, (a) $\beta_2 = 0.0$; (b) $\beta_2 = 0.1$; (c) $\beta_2 = 0.3$; (d) $\beta_2 = 0.5$.*

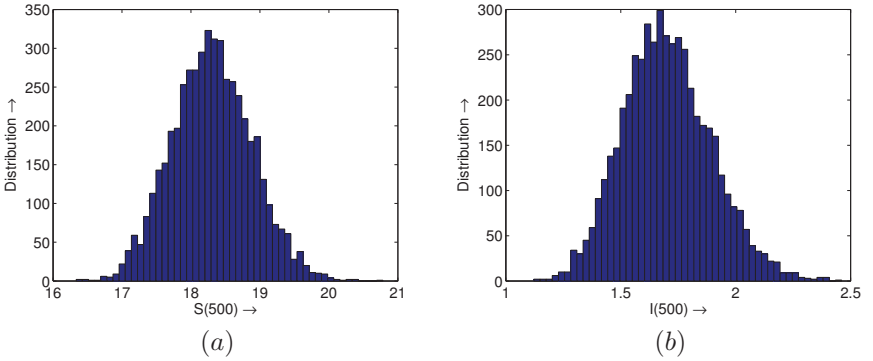

FIG. 5.5. *Distribution of $S(t)$ and $I(t)$ at $t = 500$ obtained from 5000 simulations of model (5.2) and for parameter values mentioned in (5.4).*

With this choice of the parameters, we find $\Psi = 0.05508$, $\frac{2\mu^2}{2\mu-\sigma_1^2}S^{*2} = 16.72308$, $\frac{2\mu^2}{2\mu-\sigma_2^2}I^{*2} = 16.72308$ and hence the desired condition for the existence of stationary distribution is satisfied. We have run the numerical simulation 5000 times and collected the values of $S(t)$ and $I(t)$ at $t = 500$, and their distributions are presented in Figure 5.5. The distributions presented at Figure 5.5 do not change with time and, hence they are

stationary in nature. It is important to mention here that the distribution should change a little bit with the variation of parameter values.

6. Discussion

Epidemic models of SIS type have received enormous attention in much research during a long period of time, but recently the main focus of research in this direction is to investigate the possible control mechanisms. As part of such an attempt here we have reconsidered a stochastic SIS model with the degradation of rate of infection due to media coverage. Recently Cui *et al.* [16] considered a model where the rate of infection lowered due to the coverage in media about the disease. It is assumed that the media coverage cannot eradicate the disease but possesses the capability to lower the rate of infection. Keeping this idea in mind, the deterministic model is extended to a stochastic differential equation model by incorporating multiplicative noise terms. Here we have derived the conditions for stochastic extinction and the existence of stationary distribution. In both the cases, the obtained conditions are expressions involving the system parameters and intensities of noise terms.

In order to understand the role of media coverage towards the disease dynamics we have presented some numerical simulation results in validating the analytical findings. It is interesting to note that the expression for R_0^S does contain any parameter involved with the functional form modeling the effect media coverage. However, the role of media coverage for the extinction of infected species is explained here with the help of numerical simulations with varying values of β_2. In the case of an endemic situation, the average value of infected individuals also decreases with the increasing magnitude of β_2.

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