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The effect of media coverage on threshold dynamics for a stochastic SIS epidemic model

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**HIGHLIGHTS**

- A stochastic SIS epidemic model incorporating media coverage and noise is analyzed.
- The completed threshold dynamic behaviors with respect to \(R_s^0\) are derived.
- Environmental fluctuation may significantly affect threshold dynamical behavior.
- Media coverage plays an important role in affecting the stationary distribution of model.

**ABSTRACT**

Media coverage is one of the important measures for controlling infectious diseases, but the effect of media coverage on diseases spreading in a stochastic environment still needs to be further investigated. Here, we present a stochastic susceptible–infected–susceptible (SIS) epidemic model incorporating media coverage and environmental fluctuations. By using Feller’s test and stochastic comparison principle, we establish the stochastic basic reproduction number \(R_s^0\), which completely determines whether the disease is persistent or not in the population. If \(R_s^0 \leq 1\), the disease will go to extinction; if \(R_s^0 = 1\), the disease will also go to extinction in probability, which has not been reported in the known literatures; and if \(R_s^0 > 1\), the disease will be stochastically persistent. In addition, the existence of the stationary distribution of the model and its ergodicity are obtained. Numerical simulations based on real examples support the theoretical results. The interesting findings are that (i) the environmental fluctuation may significantly affect the threshold dynamical behavior of the disease and the fluctuations in different size scale population, and (ii) the media coverage plays an important role in affecting the stationary distribution of disease under a low intensity noise environment.

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1. Introduction

Infectious diseases are caused by a variety of pathogens, which can transmit in human, animals or between human and animals, and are a major global cause of death resulting in a heavy economic burden all over the world. According to the world health organization (WHO)’s report [1], lower respiratory infections remained to be the most deadly infectious disease,
causing 3.2 million deaths worldwide in 2015. Therefore, understanding the transmission process of infectious diseases can provide evidences for prevention and control, and reduce the economic burden of disease [2].

Once an infectious disease appears and outbreaks in one area, the department for disease control and prevention will take some actions to prevent the epidemic spreading. One strategy is to disseminate the correct preventive knowledge of diseases through the media with the least delay possible [3–5]. The mass media developing rapidly, especially, internet-based is becoming very popular, also take the profound influence to the information dissemination way as well as people’s life style. Kinds of mass media (such as TV, radio, billboards, Wechat, microblog, etc.) can improve the protection awareness and change the behaviors of the susceptible populations [6]. Generally, media education consequently changing the human behaviors plays a tremendous role in limiting the spread of infectious disease [3]. Human behavior change can result in the reduction in number of real susceptible individuals or effective contact rates. People became more aware of the protection against influenza and other types of infectious diseases during this period.

The media coverage is one of the most key factors to establish the prevention and control measure, some other factors like the medical treatment level [7] or climate change [8] can also affect the spread of infectious diseases. For preventing and controlling the infectious diseases, the influences of media coverage on the spread of the disease are mainly from the following two ways:

- People can get disease related information (including the causes, the route of infection, etc.) through different media coverage (for example, television, Internet, newspapers and other media ways) from the beginning of the outbreak of disease. Thus, health education through media coverage may reduce the contact rate of human being as we observed during the spread of severe acute respiratory syndrome (SARS) during 2002 and 2004 [9].
- Authoritative information, including the potential risk of the diseases and prevention measures before the outbreak of disease, can be released to public directly. Such as H1N1 flu virus that broke out in Mexico in 2009, and before the disease outbreak spread widely across the globe, the centers for disease control and prevention (USA) provided an 9–1-1 public safety answering points (PSAPs) for management of patients with confirmed or suspected swine-origin H1N1 infection [10].

To evaluate the potential impact of media coverage on the infectious disease, mathematical modeling can provide a better understanding of the spreading mechanism of infectious diseases. For example, Cui et al. [11] investigated the transmission process of infectious diseases, the disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0)$ is global asymptotic stability, if $R^d_0 < 1$, the disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0)$ is global asymptotic stability, if $R^d_0 > 1$, the endemic equilibrium $E_0 = (S^*, I^*)$, which satisfies the following equations, is globally asymptotic stable [9].
On the other hand, in real life, the transmissions of infectious diseases are inevitable affected by the variations of environmental factors, such as temperature, humidity, etc. Recent experimental research result [13] showed that the virus transmission is closely related to the temperature and humidity. Semenza and Menne’s experimental results [14] presented that the effects of global climate change on infectious diseases are hypothetical until more is known about the degree of change in temperature and humidity that will occur. Thomas and Kevin [8] pointed out that global climate change may cause considerable uncertainty about the rates of change. Therefore, the environmental fluctuation (uncertainty change in temperature or humidity) may translate to affect the transmission rate of infectious diseases [15–23]. If we assume that the uncertainty change of transmission rate of infectious disease\( \beta_1 \) is subjected to a Gaussian white noise, with \( \beta_1 \rightarrow \beta_1 + \sigma dB(t) \), where \( B(t) \) is a standard Brownian motion, and \( \sigma \) is the intensity of white noise, then we can rewrite model (1.1) as the following form:

\[
\begin{align*}
\frac{dS}{dt} &= \left( -\beta_1 - \beta_2 \frac{I}{S + I} \right) S - \mu S dt + \sigma \frac{S}{S + I} dB(t), \\
\frac{dI}{dt} &= \left( \beta_1 - \beta_2 \frac{I}{S + I} \right) S - (\mu + \gamma) I dt + \sigma \frac{I}{S + I} dB(t).
\end{align*}
\]

(1.2)

Notice that \( S + I \rightarrow \frac{1}{\beta} \triangleq N \) as \( t \rightarrow \infty \). Thus model (1.2) has the invariant set \( \mathcal{I}_{\gamma} = \{(S, I) \in \mathbb{R}^2_+ : S(t) + I(t) = N \} \), on which model (1.2) is reduced to

\[
\frac{dI}{dt} = \left( \beta_1 - \beta_2 \frac{I}{N} \right) (N - I) dt + \sigma \frac{(N - I)I}{N} dB(t),
\]

(1.3)

with initial value

\[
I(0) = I_0 \in (0, N).
\]

(1.4)

In this paper, we pay attention on the question that how does the environmental fluctuation affect the threshold dynamics of the SIS epidemic model (1.3) incorporating media coverage. More precisely, the main aim is to obtain the completed threshold results and the existence of stationary distribution of stochastic epidemic model (1.3). As far as we know, there is rare result of model (1.3) with respect to a completely threshold and its statistical characters (i.e., the existence of stationary distribution).

As well known, there are different potential methods to introduce the environmental noise into a deterministic model. In [24], the authors used a white noise type that is directly proportional to \( S(t), I(t) \) and influences on \( \frac{dS(t)}{dt}, \frac{dI(t)}{dt} \), respectively. However, model (1.2) in this paper, we focus on the effect of environmental fluctuation on the key parameter \( \beta_1 \) in epidemic models. The diffusion term of model (1.2) is not only dependent on \( S(t) \) but also on \( I(t) \) in a nonlinear form \( \sigma \frac{S}{S+I} dB(t) \). Moreover, the random noises for the two subpopulations \( \{S(t), I(t)\} \) are assumed to be correlated, which corresponds to the real situation when the system is suffered from the same environmental factor (such as meteorological conditions etc.). Mathematically, the random perturbations of model (2.4) in [24] are described by two independent standard Brownian motions \( \gamma(t), i = 1, 2 \), while model (1.2) uses a degenerate type \( B(t) \). Since Fokker–Planck equation corresponding to model (1.2) is of degenerate type, it is difficult to establish the existence of stationary distribution (unless the Markov semigroup theory is used for a two-dimensional system with degenerate type white noise, please see [16,25]). Fortunately, there exists an invariant set \( \mathcal{I}_{\gamma} \) for stochastic model (1.2), and thus it can be reduced to a one-dimensional system (1.3) with initial value (1.4). Then, using Feller’s test we can establish the complete threshold, and applying the Khasminskii’s method we can further prove the existence of stationary distribution.

The rest of this paper are organized as follows. In the next Section, by using of Feller’s test, we obtain the extinction results of infectious disease if \( R_0^e \leq 1 \). Then, in Sections 3 and 4, by using of stochastic comparison principle and Khasminskii’s method, we prove the stochastic persistence and the existence of the stationary distribution of model (1.3) when \( R_0^e > 1 \). Some numerical simulations based on influenza parameters are carried out. Finally, a brief discussion and some biological implications are given in the last Section.

2. Disease extinction

To establish the extinction results of model (1.3), we first give the existence and unique result of global positive solution of model (1.3), and some useful lemmas.

**Lemma 2.1.** For any given initial value (1.4), there is a unique positive solution \( I(t) \) of model (1.3) on \( t \geq 0 \), and the solution will remain in \( \mathbb{R}_+ \) almost surely.

Consider the following one-dimensional stochastic differential equations:

\[
\frac{dX}{dt} = b(X)dt + a(X)dB(t)
\]

(2.1)

satisfying the following conditions:

1. \( \alpha(x)^2 > 0 \) for any \( X \in K = (l, r) \), where \(-\infty \leq l \leq r \leq \infty \),
2. For any \( X \in K \), there exists a \( \varepsilon > 0 \) such that \( \int_{X_{-\varepsilon}}^{X_{+\varepsilon}} \frac{1 + |a(x)|}{\alpha(x)^2} dx < \infty \).
Lemma 2.2 ([22,26]). Assume that (1) and (2) hold, and let $X(t)$ be a weak solution of system (2.1) in $(l, r)$. For some fixed constant $c \in K$, the scale function is defined as $q(x) = \int_{c}^{x} e^{-\int_{c}^{x} \frac{2N \beta_{2} v}{\sigma^{2}(u)} du} dv$. If $q(t^{+}) > -\infty$ and $q(r^{-}) = \infty$ hold, then

$$
P(\lim_{t \to \infty} X(t) = l) = P(\lim_{t \to 0} X(t) < r) = 1.
$$

We have the following result.

Theorem 2.1. Let $l(t)$ be the solution of model (1.3) with initial value (1.4), if $R_{0}^{e} \triangleq \frac{\beta_{1}}{\mu + \beta_{2}} - \frac{\sigma^{2}}{2(\mu + \gamma)} < 1$ hold, then we have

$$
P(\lim_{t \to \infty} l(t) = 0) = 1.
$$

Proof. For (1.3), we denote

$$
b(v) = v(t)[(\beta_{1} - \beta_{2}) \frac{v}{b + v}] \frac{N - v}{N} - (\mu + \gamma)],
$$

and

$$
\alpha(v) = \sigma \frac{(N - v)v}{N}.
$$

Then, we can calculate that

$$
\frac{\int_{c}^{x} 2b(v) \alpha(v)^{2} dv}{\int_{c}^{x} \frac{2(\beta_{1} - \beta_{2}v \frac{v}{b + v})(N - v)v}{N} - 2(\mu + \gamma)v}{du} = \int_{c}^{x} 2(\beta_{1} - \beta_{2}v \frac{v}{b + v})(N - v)v - 2(\mu + \gamma)v}{du}
$$

$$
= \frac{2N}{\sigma^{2}} \int_{c}^{x} \frac{[\beta_{1}(b + v) - \beta_{2}v](N - v)(N - x) - (\mu + \gamma)(b + v)N}{(b + v)(N - x)^{2}v}\, dv
$$

$$
= \frac{2N}{\sigma^{2}} \int_{c}^{x} \frac{\beta_{1}N}{N + b} \ln x - \frac{\beta_{1}N}{N + b} \ln(N - x) - \frac{\beta_{1}N}{N + b} \ln(b + x) + \frac{\beta_{1}}{N + b} \ln(N - x)
$$

$$
+ \frac{\beta_{1} - \beta_{2}}{N + b} \ln(b + x) + \frac{\beta_{1} - \beta_{2}}{N + b} \ln(N - x)
$$

$$
+ \frac{1}{N} \frac{N}{N - x} - \frac{\mu + \gamma}{N} \ln x] + C(c)
$$

$$
= \frac{2\beta_{1} - 2(\mu + \gamma)}{\sigma^{2}} \ln x + \frac{2\beta_{1}(N - b) - 2N\beta_{2}}{\sigma^{2}(N + b)} \ln(N - x)
$$

$$
- \frac{2N\beta_{2}}{\sigma^{2}(N + b)} \ln(b + x) - \frac{2(1 + N - 2N^{2})(\mu + \gamma)}{\sigma^{2}} \frac{1}{N - x} + C(c),
$$

where $C(c)$ is a constant, and then the scale function is

$$
q(x) = e^{-C(c)} \int_{c}^{x} e^{-\frac{2\beta_{1} - 2(\mu + \gamma)}{\sigma^{2}} \ln y + \frac{2\beta_{1}(N - b) - 2N\beta_{2}}{\sigma^{2}(N + b)} \ln(N - y) - \frac{2N\beta_{2}}{\sigma^{2}(N + b)} \ln(b + y)} e^{\frac{2N\beta_{2}}{\sigma^{2}(N + b)} \frac{1}{N - y}} dy
$$

$$
= e^{-C(c)} \int_{c}^{x} e^{-\frac{2\beta_{1} - 2(\mu + \gamma)}{\sigma^{2}} \ln y - \frac{2\beta_{1}(N - b) - 2N\beta_{2}}{\sigma^{2}(N + b)} \ln(N - y) - \frac{2N\beta_{2}}{\sigma^{2}(N + b)} \ln(b + y)} e^{\frac{2N\beta_{2}}{\sigma^{2}(N + b)} \frac{1}{N - y}} dy.
$$

Letting $x \to N^{-}$ and $s = \frac{1}{N - y}$ yields

$$
q(N^{-}) \geq e^{-C(c)} N \frac{2\beta_{1}}{\sigma^{2}} \frac{c}{\sigma^{2}} (b + c) \frac{2N\beta_{2}}{\sigma^{2}(N + b)} (N - c) \frac{2N\beta_{2} + 2\beta_{1}}{\sigma^{2}(N + b)}
$$

$$
\times \int_{c}^{x} (N - y) \frac{-2\beta_{1}N}{\sigma^{2}(N + b)} e^{\frac{2N\beta_{2}}{\sigma^{2}(N + b)} \frac{1}{N - y}} dy
$$

$$
\geq e^{-C(c)} N \frac{2\beta_{1}}{\sigma^{2}} \frac{c}{\sigma^{2}} (b + c) \frac{2N\beta_{2}}{\sigma^{2}(N + b)} (N - c) \frac{2N\beta_{2} + 2\beta_{1}}{\sigma^{2}(N + b)}
$$

$$
\times \int_{\frac{1}{N - y}}^{+\infty} \frac{2\beta_{1}N}{\sigma^{2}(N + b)} e^{\frac{2N\beta_{2}}{\sigma^{2}(N + b)} \frac{1}{N - y}} ds = \infty.
$$

Notice that $R_{0}^{e} < 1$ implies $\frac{2\beta_{1} - 2(\mu + \gamma)}{\sigma^{2}} < 1$. Letting $x \to 0^{+}$, we can obtain

$$
-q(0^{+}) \leq e^{-C(c)} (N - c) \frac{-2\beta_{1}(N - b) - 2N\beta_{2}}{\sigma^{2}(N + b)} (b + c) \frac{2N\beta_{2}}{\sigma^{2}(N + b)}
$$

$$
\times e^{-\frac{2N\beta_{2}}{\sigma^{2}(N + b)} \frac{1}{N - y}} \int_{0}^{y} y \frac{2\beta_{1} + 2(\mu + \gamma)}{\sigma^{2}} dy
$$

$$
= e^{-C(c)} (N - c) \frac{-2\beta_{1}(N - b) - 2N\beta_{2}}{\sigma^{2}(N + b)} (b + c) \frac{2N\beta_{2}}{\sigma^{2}(N + b)}
$$

$$
\times e^{-\frac{2N\beta_{2}}{\sigma^{2}(N + b)} \frac{1}{N - y}} \int_{0}^{y} y \frac{2\beta_{1} + 2(\mu + \gamma)}{\sigma^{2}} dy.$$
\[\begin{align*}
e^{-C \gamma X} & \leq \frac{e^{-2b_1(N+b)+2N\beta_1\omega}}{\sigma^2(N+b)} \frac{(b + c)^{2N\beta_2}}{\sigma^2(N+b)} \times e^{-\frac{2N^2(\mu + \gamma)}{\sigma^2}} \frac{-2b_1 + 2(\mu + \gamma)}{\sigma^2} \frac{1}{\pi^2} + 1. \\
\end{align*}\]

\[< \infty.\]

That is to say, \(q(0^+) = -\infty\). It follows from Lemma 2.2 that \(P(\lim_{t \to \infty} X(t) = 0) = 1\). The proof is completed. \(\square\)

Now, we are in a position to prove the extinction fate of disease in the case when \(R_0^* = 1\). To do this, we first present the following another useful lemma.

**Lemma 2.3** ([27]). Assume that \(\alpha(X) \equiv 1\) and system (2.1) admits a non-explosive solution which is unique in the sense of a probability law, denoted by \(X(t)\). If the scale function \(\rho(\cdot) = \int_0^{\infty} e^{x \psi} dv\) and \(\theta(\cdot) = \int_0^x e^{x \psi} dv\) satisfy \(\rho(-\infty) = -\infty,\ \rho(\infty) < -\infty, \ \theta(-\infty) = -\infty \text{ and } \theta(\infty) = \infty\), then \(X(t) \to -\infty\) in probability, namely, for any \(y \in \mathbb{R}\),

\[\lim_{t \to \infty} P(X(t) < y) = 1. \tag{2.5}\]

**Theorem 2.2.** Suppose that \(I(t)\) is the solution of model (1.3) with initial value (1.4). If \(R_0^* = 1\), then \(\lim_{t \to \infty} I(t) = 0\) in probability.

**Proof.** Define \(X = \eta(I) = \eta(x) = \frac{1}{\sigma} \ln x - \frac{1}{\sigma} \ln (N - x)\). It is obvious that \(\eta(x)\) is a continuous and increasing function on \((0, N)\). Utilizing the Itô formula to \(X\), we can get

\[dX = b(X)dt + dB(t), \tag{2.6}\]

with \(x = \eta^{-1}(X), b(X) = \frac{1}{\sigma}(\beta_1 - \beta_2 \frac{\eta^{-1}(X)}{\eta(\eta^{-1}(X))} - \frac{N(\mu + \gamma)}{\sigma(1 - \eta^{-1}(X))} + \frac{\eta^{-1}(X)}{N} - \frac{\eta}{\sigma}).\)

Taking \(y = \eta(s)\), we can compute that

\[\int_0^X b(y)dy \approx \int_0^\eta^{-1}(X) \left( (\beta_1 - \beta_2 \frac{\eta^{-1}(y)}{b + \eta^{-1}(y)}) - \frac{N(\mu + \gamma)}{\sigma(1 - \eta^{-1}(y))} \right) dy \]

\[= \int_{\eta^{-1}(0)}^\eta^{-1}(X) \left( \frac{1}{\sigma} \frac{2(\beta_1 - \beta_2)}{b + s} - \frac{N(\mu + \gamma)}{\sigma(1 - s)} \right) \frac{N}{s} ds \]

\[= C_0 + \left( \frac{2\beta_1}{\sigma^2} - \frac{2N(\mu + \gamma)}{\sigma^2} - 1 \right) \ln(N - \eta^{-1}(X)) - \frac{2N\beta_1}{\sigma^2(N + b)} \ln(b + \eta^{-1}(X)).\]

Then, taking \(y = \eta(\omega)\) can result in

\[dy = \frac{1}{\sigma} \left( \frac{1}{\omega + 1} \right) d\omega.\]

So we have

\[\begin{align*}
= & e^\int_0^X \left( \frac{2\beta_1}{\sigma^2} - \frac{2N(\mu + \gamma)}{\sigma^2} - 1 \right) \ln(N - \eta^{-1}(X)) + \left( \frac{2N\beta_1}{\sigma^2} + \frac{2N(\mu + \gamma)}{\sigma^2} - 1 \right) \ln(N - \eta^{-1}(X)) \\
& \times e^{-\frac{2N\beta_1}{\sigma^2(N + b)} \ln(b + \eta^{-1}(X))} dy, \\
= & \int_0^X \frac{2b_1}{\omega^2} \ln(N - \eta^{-1}(X)) \left( \frac{2\beta_1}{\sigma^2} - \frac{2N(\mu + \gamma)}{\sigma^2} - 1 \right) dy, \\
= & \int_0^X \frac{2b_1}{\omega^2} \ln(N - \eta^{-1}(X)) \left( \frac{2\beta_1}{\sigma^2} - \frac{2N(\mu + \gamma)}{\sigma^2} - 1 \right) d\omega. \\
\end{align*}\]

Notice that \(\eta^{-1}(0) \in (0, N)\), \(\lim_{x \to -\infty} \eta^{-1}(0) = 0\), \(\lim_{x \to \infty} \eta^{-1}(0) = N\), and \(\frac{b_1}{\mu + \gamma} - \frac{\sigma^2}{2(\mu + \gamma)} = 1\). Thus we have

\[\rho(-\infty) \leq \frac{\beta_1}{\sigma} \left[ N - \eta^{-1}(0) \right] \left( \frac{2\beta_1}{\sigma^2(N + b)} + \frac{2N(\mu + \gamma)}{\sigma^2} - 1 \right) + \frac{1}{N} \int_0^\eta^{-1}(0) \frac{2b_1 - 2N(\mu + \gamma)}{\sigma^2} d\omega, \]

\[= -\infty.\]
Theorem 3.1. The disease will be stochastically persistent, which is one of the important issues from public health point of view.

3. Stochastic persistence of disease

In this section, we shall investigate the stochastic persistence of model (1.3). It is proved that when $R_0^d > 1$, the disease will be stochastically persistent, which is one of the important issues from public health point of view.

**Theorem 3.1.** Assume $I(t)$ is the solution of model (1.3) with initial value (1.4). If $R_0^d > 1$, then $I(t)$ satisfies

$$\limsup_{t \to \infty} I(t) \geq \xi,$$

and

$$\liminf_{t \to \infty} I(t) \leq \xi,$$

where $\xi$ is the positive root in $(0, N)$ of

$$\beta_1 - \frac{\beta_2x}{b+x} \frac{N-x}{N} - \mu - \gamma - \frac{\sigma^2(N-x)^2}{2N^2} = 0. \quad (3.3)$$

**Proof.** By the Itô formula, we can obtain

$$\ln I(t) = \ln I_0 + \int_0^t f(I(s)) ds + \int_0^t \frac{\sigma(N-I(s))}{N} dB(s), \quad (3.4)$$

where $f : R \to R$:

$$f(x) \equiv (\beta_1 - \frac{\beta_2x}{b+x} \frac{N-x}{N} - \mu - \gamma - \frac{\sigma^2(N-x)^2}{2N^2}).$$

For $x = N$, we have

$$f(N) = -\mu - \gamma < 0.$$  

Note that $R_0^d = \frac{\beta_1}{\mu+\gamma} - \frac{\sigma^2}{2(\mu+\gamma)} > 1$, thus we can get

$$f(0) = \beta_1 - \mu - \gamma - \frac{1}{2} \sigma^2 > 0.$$  

Then there exists a $\xi, \hat{x} \in (0, N)$ such that $f(\xi) = 0$ and $\max_{(0,N)} f(x) = f(\hat{x})$, thus

- $f(x) > 0$ is strictly increasing in $x \in (0, 0 \vee \hat{x})$,
- $f(x) > 0$ is strictly decreasing in $x \in (0 \vee \hat{x}, \xi)$,
- $f(x) < 0$ is strictly decreasing in $x \in (\xi, N)$.

Next, we prove assertion (3.1). If it is not true, then there is a sufficiently small $\varepsilon \in (0, 1)$ such that

$$\Pr(\Omega_1) > \varepsilon,$$

where $\Omega_1 = \{ \limsup_{t \to \infty} I(\omega, t) \leq \xi - 2\varepsilon \}$. For every $\omega \in \Omega_1$, there is a $T = T(\omega) > 0$ such that when $t \geq T(\omega)$,

$$I(t, \omega) \leq \xi - \varepsilon.$$  

Clearly, we can choose $\varepsilon$ small enough such that $f(0) > f(\xi - \varepsilon)$. According to (3.5)–(3.7), when $t \geq T(\omega)$ we have

$$f(I(t, \omega)) \geq f(\xi - \varepsilon).$$  

(3.10)
By the large number theorem for martingales [28], there is an $\Omega_2 \subset \Omega$ and $P(\Omega_2) = 1$ such that for every $\omega \in \Omega_2$:
\[
\lim_{t \to \infty} \frac{1}{t} \int_0^t \sigma(N - I(s, \omega))dB(s, \omega) = 0.
\] (3.11)

Now, for any fixed $\omega \in \Omega_1 \cap \Omega_2$, it then follows from (3.4) and (3.10) that for $t \geq T(\omega)$, we have
\[
\ln(I(t, \omega)) \geq \ln(I_0) + \int_0^{T(\omega)} f(I(s, \omega))ds + f(\xi + \varepsilon)(t - T(\omega)) + \int_0^t \frac{\sigma(N - I(s, \omega))}{N}dB(s, \omega).
\]

This results in
\[
\liminf_{t \to \infty} \frac{1}{t} \ln(I(t, \omega)) \geq f(\xi + \varepsilon) > 0.
\]

Thus,
\[
\lim_{t \to \infty} I(t, \omega) = \infty.
\]

However, this contradicts with (3.10). We therefore must have the desired assertion (3.1).

Similarly, we can prove assertion (3.2). If it is not true, then there is a sufficiently small $\kappa \in (0, 1)$ such that
\[
P(\Omega_3) > \kappa,
\]
where $\Omega_3 = \{\liminf_{t \to \infty} I(\omega, t) \geq \xi + \kappa\}$. Hence, for every $\omega \in \Omega_3$, there is a $T = T(\omega) > 0$ such that when $t \geq T(\omega)$,
\[
I(t, \omega) \geq \xi + \kappa.
\] (3.12)

Now, for any fixed $\omega \in \Omega_3 \cap \Omega_2$. It then follows from (3.4) and (3.11) that for $t \geq T(\omega)$,
\[
\ln(I(t, \omega)) \leq \ln(I_0) + \int_0^{T(\omega)} f(I(s, \omega))ds + f(\xi + \kappa)(t - T(\omega)) + \int_0^t \frac{\sigma(N - I(s, \omega))}{N}dB(s, \omega).
\]

That is,
\[
\limsup_{t \to \infty} \frac{1}{t} \ln(I(t, \omega)) \leq f(\xi + \kappa) < 0.
\]

Whence
\[
\lim_{t \to \infty} I(t, \omega) = 0.
\]

This contradicts with (3.12), we thus have the desired assertion (3.2). □

**Remark 3.1.** From Theorems 2.1, 2.2 and 3.1, we know that evolution behaviors of the disease is completely determined by the stochastic basic reproduction number $R_0^s$; if $R_0^s \leq 1$, the disease will go to extinction in probability 1; if $R_0^s > 1$, the disease will be persistent in some range, which provides for us a formula to estimate the fluctuation range of the infected number.

### 4. Stationary distribution and ergodic property

In this section, we shall further discuss the existence of stationary distribution and ergodic property in the case of $R_0^s > 1$, which can reflect the statistical characteristic of the sample trajectories. We first give the following useful definition and lemma.

**Definition 4.1 (See [29]).** Let $P(t, X_0, \cdot)$ denote the probability measure induced by $X(t) = I(t)$ with initial value $X_0 = I_0$; that is,
\[
P_{X_0}(x \in B) = P(X(t) \in B : X(0) = X_0)
\]
for any Borel set $B \in \mathbb{R}_+^2$.

If there is a probability measure $\pi(\cdot)$ on the measurable space $(\mathbb{R}_+^2, \mathcal{B}(\mathbb{R}_+^2))$ such that
\[
\lim_{t \to \infty} P_{X_0}(x \in B) = \pi(B)
\]
for any $X_0 \in \mathbb{R}_+^2$, then we say that model (1.3) has a stationary distribution.

**Lemma 4.1 (See [30]).** Let $X(t)$ be a time-homogeneous solution of model (2.1) on $E_1$, Assume that
(A1) In the domain $U \subset E_1$ and some neighborhood thereof, the diffusion $\sigma(X)$ is bounded away from zero;
(A2) If for all $X \in E_1 \setminus U$ the mean time $\tau_X$ at which a path emerging from $X$ reaches the set $U$ is finite, and $\sup_{X \in K} E(\tau_X) < \infty$ for every compact subset $K \subset E_1$,

Then the Markov process $X(t)$ has a stationary distribution $\pi(x)$, and for an integrable function $f(\cdot)$,
\[
P\left(\lim_{t \to \infty} \frac{1}{t} \int_0^t f(x(s))ds = \int_{-\infty}^{\infty} f(x)\pi(dx)\right) = 1.
\] (4.2)
Theorem 4.1. If \( R_0^* > 1 \), then model (1.3) admits a unique stationary distribution \( \pi(\cdot) \) and the solution \( I(t) \) is ergodic.

Proof. To verify (A1) of Lemma 4.1, according to Zhu and Yin [31], let \( \kappa \) be a sufficiently large number and \( \Gamma' \) be a bounded open subset with a regular boundary such that
\[
\{ I : \frac{1}{\kappa} \leq i \leq N - \frac{1}{\kappa} \} \in \Gamma' \in (0, N),
\]
where \( \Gamma' \) is the closure of \( \Gamma' \). Define a \( C^2 \)-function \( V : \mathbb{R}_+ \rightarrow \mathbb{R}_+ \):
\[
V(I) = pl^{-p} + \frac{1}{N - l} = V_1(I) + V_2(I),
\]
where \( p \in (0, 1) \). By utilizing the Itô formula, we have
\[
dV(I(t)) = LV(I) - \left[ \frac{\sigma(N - I)}{lpN} + \frac{2\sigma}{N(N - I)^2} \right] dB(t),
\]
where
\[
LV_1(I) = -\frac{p}{l} \left[ (\beta_1 - \beta_2 I) \frac{N - l}{N} - (\mu + \gamma) \right] - \frac{(1 + p)\sigma^2}{2l^p} \left( \frac{N - l}{N} \right)^2
\]
and
\[
LV_2(I) = \frac{1}{(N - I)^2} \left[ (\beta_1 - \beta_2 I) \frac{N - l}{N} - (\mu + \gamma) \right] + \frac{\sigma^2}{(N - I)^3} \frac{I^2(N - I)}{N^2}
\]
It follows from (4.5)–(4.7) that
\[
LV(I) \leq -\frac{p}{l} \left[ (\beta_1 - (\mu + \gamma) - \frac{1}{2}(1 + p)\sigma^2 \right] - \frac{(\mu + \gamma)N}{(N - I)^2} - \frac{\beta_2I^2}{N(b + I)(N - I)} + p\beta_2N^{-p} + \frac{1}{N} \left[ (\beta_1 + (\mu + \gamma) + \sigma^2 \right].
\]
Since \( R_0^* > 1 \), we can choose \( p \) small enough such that \( \beta_1 - (\mu + \gamma) - \frac{1}{2}(1 + p)\sigma^2 > 0 \). Thus for sufficiently \( \kappa \), we have
\[
LV(I) \leq -1 \text{ for all } I \in (0, N) \setminus \Gamma',
\]
which implies that (A2) in Lemma 4.1 is satisfied. In addition, we can check that condition (A1) in Lemma 4.1 is also satisfied. As a consequence, model (1.3) admits a stationary distribution. \( \square \)

Remark 4.1. Theorem 4.1 shows that model (1.3) has a stationary distribution and has ergodic property:
\[
P \left( \lim_{t \to \infty} \frac{1}{t} \int_0^t I(s) ds = \int_0^\infty I(\pi(dt)) \right) = 1,
\]
which provides a formula to estimate the average infected number in probability sense.
Influenza, generally known as ‘the flu’, is an infectious disease caused by an influenza virus, which circulate in all parts of the world. Usually, the virus is spread through the air from coughs or sneezes [33]. Recently, SIS models have been widely used to describe the transmission of influenza, e.g., Mao et al. [28,34], Li et al. [15], Li and Cui [35], Cai et al. [21] etc. Thus, in the following, we shall discuss the effect of media coverage from two aspects: the reported strength \( \beta_2 \) and the audience scope \( N = \frac{\lambda}{\mu} \) based on the following parameters of model (1.3) with respect to influenza.

We can calculate \( R_0 = 1.6397 > 1 \), according to Theorems 3.1 and 4.1, the disease will be stochastically persistent and model (1.3) has a stationary distribution.
Fig. 2. The sample paths of stochastic model (1.3) and its corresponding deterministic model (1.1) when $R_0 = 1$: (a) $S(t)$, (b) $I(t)$. The parameter values are the same as in Example 5.1 and the initial value is $(21, 4)$.

Fig. 3. (a) The sample paths of $S(t)$, and (b) the sample paths of $I(t)$ of stochastic model (1.3) and its corresponding estimated range of the value of $\xi$ when $R_0 > 1$. The parameter values is used in Example 5.1 and initial value is $(21, 4)$.

Fig. 4. The sample paths $I(t)$ of stochastic model (1.3) and its corresponding probability density function (PDF) for different $\beta_2$, the other parameter values is used in Example 5.2 and initial value is $I_0 = 300000$.

(i) Firstly, we explore the effect of media coverage on the stationary distribution. The only difference between the following cases is $\beta_2$ (the maximum reduced contact rate due to media coverage), which can be viewed as a related measurement of media coverage strength. From Fig. 4, we can know that with the increase of $\beta_2$ from 0.35 to 0.45, the infected number $I(t)$ is decreased. The position of stationary distribution (denoted by the central tendency of infected number $I(t)$) moves to left and the dispersion becomes smaller with the increase of $\beta_2$. Thus, the media coverage plays an important role in determining the statistics of stationary distribution.
Fig. 5. The sample paths $I(t)$ of stochastic model (1.3) for different size scale of population: $N = 13827000$ (blue line) and $N = 1382700$ (red line), the other parameter values are the same as in Example 5.2 and initial value is $I_0 = 300000$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

| Table 1 |
| The parameters of model (1.3) based on the flu. |
| Parameters | Value | Unit | Source |
| $\beta_1$ | 0.1298 | day$^{-1}$ | [36] |
| $\mu$ | 0.007 | day$^{-1}$ | [37] |
| $\gamma$ | 0.0714 | day$^{-1}$ | [38] |
| $b$ | 0.1 | day$^{-1}$ | [34] |
| $N$ | 13827000 | person | assumed |
| $\beta_2$ | 0.03 | day$^{-1}$ | assumed |
| $\sigma$ | 0.05 | – | assumed |

| Table 2 |
| Effects of $N$, $\beta_1$, $\beta_2$, $\sigma$ on the infected level $I(t)$. |
| $N$ ↑ | $\beta_1$ ↑ | $\beta_2$ ↑ | $\sigma$ ↑ |
| Infected level $I(t)$ | ↑ | ↑ | ↓ | ↓ |

(ii) Secondly, due to the fact that the effect of media coverage may be affected by the audience scope, we discuss the effect of environmental fluctuation on different audience scope. In China, there are more than 100 large-scale cites and over 1000 middle-scale cites [39], and the total population of China in 2016 is about 1.3827 billion [37]. So we may assume that the average population number in large-scale cites is about $N' = 13827000$, and in middle-scale cites, it is about $N = 1382700$. Therefore, for model (1.3), we choose two different size scale population $N' = 13827000$ and $N = 1382700$, which represent respectively two kinds of audience scopes of media coverage. The others parameters are the same as in Table 1. It can be obviously observed from Fig. 5 that the effect of same intensity environmental fluctuation ($\sigma$) may result in a more stronger fluctuation of $I(t)$ for the larger size scale population ($N' = 13827000$), that is to say, the smaller size scale of the population is less sensitivity to the environmental fluctuation. In summary, the same intensity fluctuation caused by variation of environment (e.g., the climate change, season alternation, etc.) may produce diverse fluctuation effects in different size scale population.

From above simulation analysis, we can summarize the effect of $N$, $\beta_1$, $\beta_2$, $\sigma$ on the average infected level $I(t)$ as in Table 2. It can be seen from Table 2 that the decrease of susceptible number $N$ or the increase of media coverage $\beta_2$ is benefit to the control of infectious disease. Therefore, we can draw the conclusion that the media coverage may significantly affect the stationary distribution of $I(t)$ and fluctuation effects in different size scale population.

6. Discussion

The emerging and reemerging diseases have led to an extensive interest in investigation of infectious disease [40]. The medical coverage as an effective preventive measure plays an important role in controlling the disease spread [4]. Dynamical modeling of infectious disease has become a powerful tool to improve our understanding of the pattern of epidemic spread and develop better controlling strategies [41]. Meanwhile, in real situation, the environmental fluctuation should not be ignored. In this paper, we investigated a stochastic SIS epidemic model with both media coverage and environmental fluctuation. By using the Feller’s test method, we obtained that the dynamical behavior of model (1.3) is completely determined by the stochastic basic reproduction number $R_0^s := R_0^d - \sigma^2/2(\mu + \gamma)$. More precisely, if $R_0^s \leq 1$, the
The media coverage may play an important role in determining the persistence of the diseases and its statistical characteristics, such as the change of the position of central tendency of infected number \( I(t) \) with the strength of media coverage increasing. Although media coverage cannot completely eliminate the disease spread, it can decrease the contact rate of disease effectively during the early stage of infection [3]. Thus, taking media coverage as an effective preventive measure before the outbreak of the infectious diseases is reasonable in a fluctuation environment.

- The environmental fluctuation may significantly affect the threshold dynamics of the disease and fluctuation effects in different size scale population. If we ignore the influence of the environmental fluctuation, the epidemic may be overestimated. In other words, the environmental stochasticity may be benefit to the disease extinction and result in diverse variation of the infected numbers \( I(t) \) for different size scale \( N \).

From the practical viewpoint, identifying the role of media coverage and stochasticity in the transmission of infectious disease may be conducive to the understanding of the potential control measures, and to configure the media coverage’s audience scope and coverage strength more reasonable.

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