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STOCHASTIC SURVIVAL ANALYSIS OF AN EPIDEMIC MODEL WITH INNATE IMMUNITY AND TREATMENT*

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Abstract The innate immunity helps the individuals in the exposed compartment return into the ones in the susceptible compartment when a pathogen or virus invades the local population of having four compartments: the susceptible, the exposed, the infected and the recovered. In this study, we propose a stochastic SEIR model with innate immunity and treatment. Here, Holling type II functional responses are used to describe the saturated effects of the innate immunity and treatment. Then, we obtain the extinction of the exposed and the infected when the basic reproduction number $\mathcal{R}_0 < 1$ and the exponential decline rate $\nu < 0$ are valid. Moreover, we conclude that when innate immunity and treatment increase, the time that the exposed and the infected approach zero reduces. We also find that the deterministic SEIR model reaches extinction a bit faster than the stochastic SEIR model. Further, the persistence in the mean and stationary distribution of stochastic SEIR model are obtained under suitable conditions. Finally, the numerical investigations with two methods and a case study of Fuzhou COVID-19 epidemic of 2022 are discussed.

Keywords SEIR epidemic model, Holling type II functional response, innate immunity, extinction and persistence, COVID-19.

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1. Model formulation

The reliable mathematical epidemic models were proposed to predict and control the spread of infectious diseases since the pioneering work was discussed by Kermack and McKendrick in [12]. Later, some compartment models were extensively studied in the recent contributions [1, 6, 15, 16, 30, 33–35, 38, 39, 41, 50]. The research results showed that the transmission mechanisms of infectious diseases played a very important role when establishing the mathematical models. Especially, the incidence rates between the susceptible and the infected quantitatively were governed to describe the transmission mechanisms of infectious diseases, such as the bilinear incidence rate, the standard incidence rate, the Beddington-DeAngelis incidence rate, and the generalized nonlinear incidence rate etc in [3, 10–13, 17, 18, 20–22, 25, 26, 38, 41, 45–47].

The innate immunity, one of the main defense mechanisms formed by organisms in the process of long-term evolution, is extensively discussed on cellular level in recent studies [2, 7, 14, 28, 29, 31, 32, 36, 43]. Usually, the innate immunity of the individuals plays crucial roles because the responses of innate immunity work immediately or within hours after pathogens or virus invade the hosts. Meanwhile, the innate immunity of different individuals presents distinct timelines after they are invaded by pathogens or viruses. However, from the best knowledge of the authors, the studies of innate immunity on population level are few for epidemic models. For instance, the effects of immunity are considered in an impulsive stochastic model, in which the authors of [3] mainly study the saturated tumor-immune responses. And the nonlinear innate immunity and saturated treatment in the forms of Holling type II are governed for an SEIS epidemic model (1.3) in [10], in which the authors of [10] further compare the dynamics of model (5.1) with linear innate immunity and linear treatment. In this study, we believe that the role of innate immunity is of importance for epidemic models, we further are motivated by recent contributions to establish an SEIR model with nonlinear innate immunity and nonlinear treatment.

The aforementioned innate immunity helps the exposed getting recovery and returning into the susceptible again, despite the impacts of innate immunity are limited to the exposed. In this study, we use Holling type II functional response $g(E) = aE/(1 + kE)$ with $a > 0, k \geq 0$ to describe the increasing tendency of the impacts of innate immunity, where $1/a$ is the average returning period that the exposed remove from the exposed compartment and return into the susceptible compartment due to the existence of innate immunity; k is a non-negative constant measuring the limitation in innate immunity; $1/(1+kE)$ declines the exposed returning into the susceptible compartment due to low immunoglobulin and neutrophil deficiency as studied in [17, 46]. Meanwhile, the treatment of the infected is an effective and important measure for controlling infectious diseases. We assume that the infected return into the susceptible compartment due to the temporary immunity after they receive the treatment in medical agents, by governing Holling type II functional response $h(I) = \gamma I/(1 + bI)$ with $\gamma > 0, b \geq 0$. Here, $1/\gamma$ is the average loss period of temporary immunity that the infected lose their temporary immunity after they receive the treatment and return into the susceptible compartment; b is a non-negative constant measuring the limitation in treatment availability; $1/(1+bI)$ prevents the infected returning into the susceptible compartment due to the limited medical resources. When $b = 0$, $h(I)$ degenerates the linear forms in [34, 35]. Moreover, some infected individuals get the lifelong immunity

after they receive the treatment and return into the recovered compartment with the recovery rate μ. Thus, we derive the following model:

$$\begin{aligned}
\dot{S}(t) &= \Gamma - \delta S - \frac{\beta SI}{\phi(I)} + \frac{aE}{1+kE} + \frac{\gamma I}{1+bI},\\
\dot{E}(t) &= \frac{\beta SI}{\phi(I)} - (\delta+\sigma)E - \frac{aE}{1+kE},\\
\dot{I}(t) &= \sigma E - (\delta+\mu)I - \frac{\gamma I}{1+bI},\\
\dot{R}(t) &= \mu I - \delta R.
\end{aligned} \tag{1.1}$$

In the model (1.1), Γ is the constant recruitment rate, δ denotes the natural death rate of the total population, β is the contact rate between the susceptible and the infected, $\phi(I)$ is an increasing function with the properties that $\phi(0)=1$ and $\phi'(I)\geq 0$, σ represents the positive infection rate from the exposed to the infected. We derive that $\dot{N}(t)=\dot{S}(t)+\dot{E}(t)+\dot{I}(t)+\dot{R}(t)=\Gamma-\delta N(t)$, here $N(0)=S(0)+E(0)+I(0)+R(0)\geq 0$. Then, it follows that $\limsup_{t\to\infty} N(t)<\frac{\Gamma}{\delta}$. Therefore, we derive a positive invariant set $\Omega=\{(S,E,I,R)\in\mathbb{R}^4_+ : N(t)<\frac{\Gamma}{\delta}\}$. The disease-free equilibrium point of model (1.1) is denoted by $P_0(\frac{\Gamma}{\delta},0,0,0)$. By the next generation matrix method in [4,5], the matrices F and V^{-1} are respectively written as

$$F=\begin{pmatrix} 0 & \frac{\beta\Gamma}{\delta} \\ 0 & 0 \end{pmatrix}, \quad V^{-1}=\frac{1}{(\delta+\sigma+a)(\delta+\mu+\gamma)}\begin{pmatrix} \delta+\mu+\gamma & 0 \\ \sigma & \delta+\sigma+a \end{pmatrix}.$$

By Theorem 2 in [27], the basic reproduction number of model (1.1) is

$$\mathcal{R}_0=\rho(FV^{-1})=\frac{\Gamma\beta\sigma}{\delta(\delta+\sigma+a)(\delta+\mu+\gamma)}. \tag{1.2}$$

In fact, the endemic equilibrium point $P^*(S^*,E^*,I^*,R^*)$ of (1.1) is calculated as follows:

$$\begin{aligned}
S^* &= \frac{1}{\beta}(b\delta I^*+\mu bI^*+\mu+\gamma+\delta)\phi(I^*)\\
&\quad\times\Big(\frac{\delta+\sigma}{b\sigma I^*+\sigma}+\frac{a}{kI^*(b\delta I^*+\gamma+\delta)+\mu kI^*(bI^*+1)+\sigma+b\sigma I^*}\Big),\\
E^* &= \frac{b(\delta+\mu)(I^*)^2+(\delta+\mu+\gamma)I^*}{\sigma(1+bI^*)}, \quad R^*=\frac{\mu I^*}{\delta},
\end{aligned}$$

and I^* is the solution of $A_5\phi(I^*)+A_4(I^*)^4+A_3(I^*)^3+A_2(I^*)^2+A_1(I^*)+A_0=0$, where

$$\begin{aligned}
A_5 &= -\delta kb^2(\delta+\sigma)(\delta+\mu)^2-\delta b(\delta+\mu)(\delta+\sigma)(k\gamma+k\delta+k\mu+b\sigma)\\
&\quad-\delta\sigma b(\delta+\sigma)(\delta+\mu)-kb\delta(\delta+\sigma)(\delta+\mu)(\mu+\gamma+\delta)-a\delta\sigma b^2(\delta+\mu)\\
&\quad-\delta(\delta+\sigma)(\mu+\gamma+\delta)(k\gamma+k\delta+k\mu+b\sigma)-ab\delta\sigma(2\mu+\gamma+2\delta)\\
&\quad-\delta\sigma(\delta+\sigma)(\mu+\gamma+\delta)-a\delta\sigma(\mu+\gamma+\delta),\\
A_4 &= \beta b^2k(\delta+\sigma)(\delta+\mu)^2,
\end{aligned}$$

$$
\begin{aligned}
A_3 &= \Gamma\beta\sigma b^2 k(\delta+\mu) - \beta(\delta+\sigma)(\mu+\gamma+\delta)(k\gamma+k\delta+k\mu+b\sigma)\\
&\quad - \beta kb(\delta+\sigma)(\delta+\mu)(\mu+\gamma+\delta) + \beta\sigma\gamma kb(\delta+\mu),\\
A_2 &= \Gamma\beta\sigma b(k\gamma+k\delta+k\mu+b\sigma) + \Gamma\beta\sigma kb(\delta+\mu) - \beta\sigma b(\delta+\sigma)(\delta+\mu)\\
&\quad - \beta(\delta+\sigma)(\mu+\gamma+\delta)(k\gamma+k\delta+k\mu+b\sigma) + \beta\sigma\gamma(k\gamma+k\delta+k\mu+b\sigma),\\
A_1 &= \Gamma\beta\sigma(k\gamma+k\delta+k\mu+b\sigma) + \Gamma\beta\sigma^2 b - \beta\sigma(\delta+\sigma)(\mu+\gamma+\delta) + \beta\sigma^2\gamma,\\
A_0 &= \Gamma\beta\sigma^2.
\end{aligned}
$$

In the real circumstances, the existences of environmental fluctuations and population mobility lead to the variations on persistence and extinction of epidemic models in [1,6,18,20,23,35,38,39,41,47–49]. Therefore, it is necessary to introduce the environmental fluctuations into model (1.1). The independent environmental fluctuations are proportional to S, E, I and R of model (1.1), which are provided by multiplicative white noises. More precisely, we consider a Markov process $X(t) = (S(t), E(t), I(t), R(t))^T$ when $\triangle t \to 0$, and we derive the following descriptions:

$$
\begin{aligned}
&\mathbb{E}[S(t+\Delta t) - S(t)|X_t = x] \approx \Big(\Gamma - \delta S - \frac{\beta SI}{\phi(I)} + \frac{aE}{1+kE} + \frac{\gamma I}{1+bI}\Big)\Delta t,\\
&\mathbb{E}[E(t+\Delta t) - E(t)|X_t = x] \approx \Big(\frac{\beta SI}{\phi(I)} - (\delta+\sigma)E - \frac{aE}{1+kE}\Big)\Delta t,\\
&\mathbb{E}[I(t+\Delta t) - I(t)|X_t = x] \approx \Big(\sigma E - (\delta+\mu)I - \frac{\gamma I}{1+bI}\Big)\Delta t,\\
&\mathbb{E}[R(t+\Delta t) - R(t)|X_t = x] \approx (\mu I - \delta R)\Delta t,
\end{aligned}
$$

and

$$
\begin{aligned}
&\mathrm{Var}[S(t+\Delta t) - S(t)|X_t = x] \approx \sigma_1^2 S^2(t)\Delta t,\\
&\mathrm{Var}[E(t+\Delta t) - E(t)|X_t = x] \approx \sigma_2^2 E^2(t)\Delta t,\\
&\mathrm{Var}[I(t+\Delta t) - I(t)|X_t = x] \approx \sigma_3^2 I^2(t)\Delta t,\\
&\mathrm{Var}[R(t+\Delta t) - R(t)|X_t = x] \approx \sigma_4^2 R^2(t)\Delta t,
\end{aligned}
$$

where $\sigma_i^2 > 0$ are the intensities of white noises for $i = 1,\ 2,\ 3,\ 4$. Thus, we obtain the following stochastic epidemic model as follows:

$$
\begin{aligned}
\mathrm{d}S(t) &= \Big(\Gamma - \delta S - \frac{\beta SI}{\phi(I)} + \frac{aE}{1+kE} + \frac{\gamma I}{1+bI}\Big)\mathrm{d}t + \sigma_1 S\mathrm{d}B_1(t),\\
\mathrm{d}E(t) &= \Big(\frac{\beta SI}{\phi(I)} - (\delta+\sigma)E - \frac{aE}{1+kE}\Big)\mathrm{d}t + \sigma_2 E\mathrm{d}B_2(t),\\
\mathrm{d}I(t) &= \Big(\sigma E - (\delta+\mu)I - \frac{\gamma I}{1+bI}\Big)\mathrm{d}t + \sigma_3 I\mathrm{d}B_3(t),\\
\mathrm{d}R(t) &= (\mu I - \delta R)\mathrm{d}t + \sigma_4 R\mathrm{d}B_4(t),
\end{aligned}
\tag{1.3}
$$

where $B_i(t)$ are mutually independent standard Brownian motions with $i = 1, 2, 3, 4$.

Remark 1.1. We take $a = 0$ and $\gamma = 0$, model (1.3) turns into model (6) in [41]. When $\phi(I) = 1 + \alpha_1 S + \alpha_2 I$, $a = 0$ and $b = 0$, model (1.3) becomes model (4) in [18]. When $\phi(I) = 1 + \alpha I$, $a = 0$ and $b = 0$, model (1.3) turns into model (1.4) in [44].

Remark 1.2. When $a = 0$ and $\gamma = 0$ in (1.2), the expressions of the basic reproduction number $\mathcal{R}_0$ of model (6) in [41], model (4) in [18] and model (1.4) in [44] are same. That is, $\mathcal{R}_0 = \Gamma\beta\sigma/[\delta(\delta+\sigma)(\delta+\mu)]$. Moreover, $\mathcal{R}_0 \leq 1$ and $\mathcal{R}_0 > 1$ determine the extinction and the persistence of these three models therein. While, the values of a and γ of model (1.3) do not vanish, two indices $\mathcal{R}_0$ and $\mathcal{R}_0^s$ are given in the next investigations.

Noticing that the first three equations of model (1.1) are independent of the recovered, we therefore investigate the dynamics of the equivalent model (1.4) as follows:

$$\begin{aligned} \mathrm{d}S(t) &= \Big(\Gamma - \delta S - \frac{\beta SI}{\phi(I)} + \frac{aE}{1+kE} + \frac{\gamma I}{1+bI}\Big)\mathrm{d}t + \sigma_1 S\mathrm{d}B_1(t), \\ \mathrm{d}E(t) &= \Big(\frac{\beta SI}{\phi(I)} - (\delta+\sigma)E - \frac{aE}{1+kE}\Big)\mathrm{d}t + \sigma_2 E\mathrm{d}B_2(t), \\ \mathrm{d}I(t) &= \Big(\sigma E - (\delta+\mu)I - \frac{\gamma I}{1+bI}\Big)\mathrm{d}t + \sigma_3 I\mathrm{d}B_3(t). \end{aligned} \tag{1.4}$$

Let $(\Omega, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ which is increasing and right continuous while $\mathcal{F}_0$ contains all $\mathbb{P}$-null sets. Next, we will show a unique global positive solution to model (1.4). Then, the extinction and the persistence in the mean for model (1.4) will be considered under proper conditions in Section 2 and Section 3, respectively. The ergodic stationary distribution of model (1.4) is also verified in Section 4. Finally, several numerical simulations in Section 5 are carried out by two methods.

Remark 1.3. We take $\phi(I) = 1$ and $\sigma_1 = \sigma_2 = \sigma_3 = 0$, model (1.4) turns into model (1.3) in [10]. Moreover, we take $k = b = 0$, model (1.4) turns into model (5.1) in [10]. The expressions of basic reproduction number of model (1.3) and model (5.1) in [10] are same with that of model (1.4) in this study.

2. Stochastic extinction

Before we discuss the dynamical behaviors of model (1.4), the unique global positive solution to model (1.4) should be checked firstly, the corresponding proof is similar to the proof in [20], so we only write down the following Theorem 2.1 without details.

Theorem 2.1. *For any given initial value $(S(0), E(0), I(0))$ in $\mathbb{R}^3_+$, there is a unique solution $(S(t), E(t), I(t))$ to model (1.4) on $t \geq 0$ and the solution will remain in $\mathbb{R}^3_+$ with probability one.*

Proof. We write down the lines and sentences as we did in the existence-and-uniqueness theorem in [20]. We just omit the details of proof hereby. □

Throughout this paper, we denote $\langle x(t)\rangle = \frac{1}{t}\int_0^t x(s)\mathrm{d}s$.

Theorem 2.2. *Let $(S(t), E(t), I(t))$ be the solution of model (1.4) with any initial value $(S(0), E(0), I(0))$ in $\mathbb{R}^3_+$. If the basic reproduction number satisfies $\mathcal{R}_0 < 1$, $\mu > 0.5(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2)$ and*

$$\nu = (\gamma+\mu+\delta)\sqrt{\mathcal{R}_0} + \min\{a+\delta+\sigma, \gamma+\mu+\delta\}(\sqrt{\mathcal{R}_0} - 1)$$

$$
\begin{aligned}
&+\max\{a,\gamma\}-[2(\sigma_2^{-2}+\sigma_3^{-2})]^{-1}\\
&<0,
\end{aligned}
$$

then, the infectious disease goes to extinction. That is, $\lim_{t\to\infty} I(t)=0$.

Proof. We define a C^2-function $Z:\mathbb{R}_+^2\to\mathbb{R}_+$ as follows:

$$
Z(E,I)=\frac{1}{\gamma+\mu+\delta}\Big(\frac{\sigma}{a+\delta+\sigma}E(t)+\sqrt{\mathcal{R}_0}I(t)\Big)=\theta_1E(t)+\theta_2I(t).
$$

By Itô's formula, we have

$$
\begin{aligned}
\mathrm{d}\ln Z(E(t),I(t)) &= \frac{1}{Z}\big(\theta_1\mathrm{d}E+\theta_2\mathrm{d}I\big)-\frac{1}{2Z^2}\big[\theta_1^2(\mathrm{d}E)^2+\theta_2^2(\mathrm{d}I)^2\big]\\
&= \mathcal{L}(\ln Z(E(t),I(t)))\mathrm{d}t+\frac{1}{Z}\big[\theta_1\sigma_2E\mathrm{d}B_2(t)+\theta_2\sigma_3I\mathrm{d}B_3(t)\big].
\end{aligned}
$$

Based on $(a^2+b^2)(c^2+d^2)\geq(ac+bd)^2$, for $a>0$, $b>0$, $c>0$, $d>0$, then, we get that

$$
(\theta_1E+\theta_2I)^2=\Big(\theta_1\sigma_2E\frac{1}{\sigma_2}+\theta_2\sigma_3I\frac{1}{\sigma_3}\Big)^2\leq(\theta_1^2\sigma_2^2E^2+\theta_2^2\sigma_3^2I^2)\Big(\frac{1}{\sigma_2^2}+\frac{1}{\sigma_3^2}\Big). \quad (2.1)
$$

Therefore, from (1.4) and (2.1), we have

$$
\begin{aligned}
\mathcal{L}\ln Z(E(t),I(t)) &= \frac{1}{Z}\Big(\theta_1\frac{\beta SI}{\phi(I)}-\theta_1\delta E-\theta_1\sigma E-\theta_1\frac{aE}{1+kE}+\theta_2\sigma E\\
&\quad -\theta_2(\delta+\mu)I-\theta_2\frac{\gamma I}{1+bI}\Big)-\frac{\theta_1^2\sigma_2^2E^2+\theta_2^2\sigma_3^2I^2}{2(\theta_1E+\theta_2I)^2}\\
&:= I_1-I_2.
\end{aligned}
$$

Indeed, we estimate each item by the following approaches

$$
\begin{aligned}
I_1 &\leq \frac{1}{Z}\Big\{\theta_1\beta SI-\theta_1\delta E-\theta_1\sigma E-\theta_1aE+\theta_1aE-\theta_1\frac{aE}{1+kE}+\theta_2\sigma E-\theta_2(\delta+\mu)I\\
&\quad -\theta_2\gamma I+\theta_2\gamma I-\theta_2\frac{\gamma I}{1+bI}\Big\}\\
&\leq \frac{1}{Z}\Big\{\theta_1\beta SI+\theta_1\Big[\frac{\Gamma\beta I}{\delta}-(\delta+\sigma+a)E\Big]+\theta_2\big[\sigma E-(\delta+\mu+\gamma)I\big]\\
&\quad +\theta_1aE+\theta_2\gamma I\Big\}\\
&\leq \frac{\theta_1\beta S}{\theta_2}+\frac{1}{Z}\Big(\mathcal{R}_0I-\frac{\sigma}{\gamma+\mu+\delta}E+\frac{\sqrt{\mathcal{R}_0}\sigma E}{\gamma+\mu+\delta}-\sqrt{\mathcal{R}_0}I\Big)+\max\{a,\gamma\}\\
&= \frac{\theta_1\beta S}{\theta_2}+\frac{1}{Z}(\sqrt{\mathcal{R}_0}-1)\big[(a+\delta+\sigma)\theta_1E+(\gamma+\mu+\delta)\theta_2I\big]+\max\{a,\gamma\}\\
&\leq \frac{\theta_1\beta S}{\theta_2}+\min\{a+\delta+\sigma,\gamma+\mu+\delta\}(\sqrt{\mathcal{R}_0}-1)+\max\{a,\gamma\},
\end{aligned}
$$

and $I_2 \geq [2(\sigma_2^{-2} + \sigma_3^{-2})]^{-1}$. Therefore, we derive

$$\begin{aligned} \mathrm{d}\ln Z(E(t), I(t)) \leq \Big\{ & \frac{\theta_1 \beta S}{\theta_2} + \min\{a+\delta+\sigma, \gamma+\mu+\delta\}(\sqrt{\mathcal{R}_0} - 1) + \max\{a, \gamma\} \\ & - [2(\sigma_2^{-2} + \sigma_3^{-2})]^{-1} \Big\} \mathrm{d}t + \frac{\theta_1 \sigma_2}{Z} E \mathrm{d}B_2(t) + \frac{\theta_2 \sigma_3}{Z} I \mathrm{d}B_3(t). \end{aligned} \tag{2.2}$$

Taking integration on both sides of (2.2) and divided by t, one implies that

$$\begin{aligned} \frac{1}{t}\ln Z(E(t), I(t)) \leq & \frac{1}{t}\ln Z(E(0), I(0)) + \frac{1}{t}\int_0^t \frac{\theta_1 \beta}{\theta_2} S(s) \mathrm{d}s \\ & + \min\{a+\delta+\sigma, \gamma+\mu+\delta\}(\sqrt{\mathcal{R}_0} - 1) + \max\{a, \gamma\} \\ & - \left[2(\sigma_2^{-2} + \sigma_3^{-2})\right]^{-1} + \frac{1}{t} M_1(t) + \frac{1}{t} M_2(t), \end{aligned} \tag{2.3}$$

where $M_1(t) = \int_0^t \frac{\theta_1 \sigma_2 E(s)}{Z(s)} \mathrm{d}B_2(s)$, $M_2(t) = \int_0^t \frac{\theta_2 \sigma_3 I(s)}{Z(s)} \mathrm{d}B_3(s)$ are local martingales, and their quadratic variations are $\langle M_1(t), M_1(t)\rangle \leq \sigma_2^2 t$, $\langle M_2(t), M_2(t)\rangle \leq \sigma_3^2 t$. By Lemma 2 in [23], we derive that

$$\limsup_{t\to\infty} \frac{1}{t} M_i(t) = 0, \quad i = 1, 2 \quad \text{a.s..} \tag{2.4}$$

By (2.3) and (2.4), we take the upper limit on both sides, it then follows

$$\begin{aligned} \limsup_{t\to\infty} \frac{1}{t}\ln Z(E(t), I(t)) \leq & \frac{\theta_1 \beta \Gamma}{\theta_2 \delta} + \min\{a+\delta+\sigma, \gamma+\mu+\delta\}(\sqrt{\mathcal{R}_0} - 1) \\ & + \max\{a, \gamma\} - [2(\sigma_2^{-2} + \sigma_3^{-2})]^{-1} \\ := & \ \nu \quad \text{a.s..} \end{aligned}$$

If $\nu < 0$, then we have $\lim_{t\to\infty} I(t) = 0$ and $\lim_{t\to\infty} E(t) = 0$. The proof is complete. □

Remark 2.1. As claimed in Theorem 3.2 of [10] and Theorem 2.2 of this study, the basic reproduction number $\mathcal{R}_0 < 1$ determines the extinction of model (1.3) of [10], also determines the extinction of model (1.4) of this study. We investigate the extinction time of the solution of model (1.4) with $\phi(I) = 1$ in numerical simulation section. Further, we compare the differences of the extinction time for model (1.4) with $\phi(I) = 1$ and model (1.3) of [10].

3. Stochastic persistence

The stochastic persistence of the infectious disease will be demonstrated in this section.

Lemma 3.1. *For any initial value* $(S(0), E(0), I(0)) \in \mathbb{R}^3_+$, *the solution* $(S(t), E(t), I(t))$ *of model* (1.4) *has the following properties:*

$$\lim_{t\to\infty} \frac{1}{t} S(t) = 0, \quad \lim_{t\to\infty} \frac{1}{t} E(t) = 0, \quad \lim_{t\to\infty} \frac{1}{t} I(t) = 0 \quad a.s.,$$

and

$$\limsup_{t\to\infty} \frac{1}{t}\ln S(t) \leq 0, \quad \limsup_{t\to\infty} \frac{1}{t}\ln E(t) \leq 0, \quad \limsup_{t\to\infty} \frac{1}{t}\ln I(t) \leq 0 \quad a.s..$$

Moreover, if $\mu > 0.5(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2)$, *then*

$$\lim_{t\to\infty} \frac{1}{t}\int_0^t S(s)dB_1(s) = 0, \quad \lim_{t\to\infty} \frac{1}{t}\int_0^t E(s)dB_2(s) = 0,$$
$$\lim_{t\to\infty} \frac{1}{t}\int_0^t I(s)dB_3(s) = 0 \ a.s..$$

Proof. The proof is similar to the approaches used in [48,49]. Therefore we make variable replacement that replace $E(t)$ with $I(t)$, replace $I(t)$ with $V(t)$, and $S(t)$ remains unchanged, then we easily obtain the results of Lemma 3.1. We omit the details hereby. □

Theorem 3.1. *Let* $(S(t), E(t), I(t))$ *be a solution of model* (1.4) *with any initial value in* $\mathbb{R}^3_+$. *If*

$$\mathcal{R}_0^s = \frac{\Gamma\beta\sigma}{(\delta + 0.5\sigma_1^2)(\delta + \sigma + a + 0.5\sigma_2^2)(\delta + \mu + \gamma + 0.5\sigma_3^2)} > \phi(\Gamma\delta^{-1}) > 1,$$

then, model (1.4) *satisfies*

$$\liminf_{t\to\infty} \langle I\rangle_t \geq \frac{1}{h}\big(\mathcal{R}_0^s - \phi(\Gamma\delta^{-1})\big) > 0, \quad h = \frac{\beta}{\delta + 0.5\sigma_1^2}\mathcal{R}_0^s.$$

In other words, the infectious disease will prevail in the long run.

Proof. Define $Z_1 = -c_1 \ln S - c_2 \ln E - c_3 \ln I$, where c_1, c_2, c_3 are positive constants determined later. By applying Itô's formula, which gives that

$$\begin{aligned}
\mathcal{L}Z_1(t) &= -\frac{c_1\Gamma}{S} + c_1\delta + \frac{c_1\beta I}{\phi(I)} - \frac{c_1 aE}{(1+kE)S} - \frac{c_1\gamma I}{(1+bI)S} - \frac{c_2\beta SI}{E\phi(I)} + c_2(\delta+\sigma) \\
&\quad + \frac{ac_2}{1+kE} - \frac{c_3\sigma E}{I} + c_3\delta + c_3\mu + \frac{c_3\gamma}{1+bI} + 0.5c_1\sigma_1^2 + 0.5c_2\sigma_2^2 + 0.5c_3\sigma_3^2 \\
&< -\frac{c_1\Gamma}{S} - \frac{c_2\beta SI}{E\phi(I)} - \frac{c_3\sigma E}{I} - \phi(I) + \phi(I) + c_1\delta + c_1\beta I \\
&\quad + c_2\delta + c_2\sigma + ac_2 + c_3\delta + c_3\mu + c_3\gamma + 0.5c_1\sigma_1^2 + 0.5c_2\sigma_2^2 + 0.5c_3\sigma_3^2 \\
&\leq -4(c_1c_2c_3\Gamma\beta\sigma)^{\frac{1}{4}} + \phi(I) + c_1\big(\delta + 0.5\sigma_1^2\big) + c_1\beta I \\
&\quad + c_2\big(\delta + \sigma + a + 0.5\sigma_2^2\big) + c_3\big(\delta + \mu + \gamma + 0.5\sigma_3^2\big).
\end{aligned}$$

Let

$$c_1 = \frac{\mathcal{R}_0^s}{\delta + 0.5\sigma_1^2}, \quad c_2 = \frac{\mathcal{R}_0^s}{\delta + \sigma + a + 0.5\sigma_2^2}, \quad c_3 = \frac{\mathcal{R}_0^s}{\delta + \mu + \gamma + 0.5\sigma_3^2}.$$

Then $\mathcal{L}Z_1(t) \leq -\mathcal{R}_0^s + \phi(I) + hI$, $h = c_1\beta$, we thus derive that

$$\mathrm{d}Z_1(t) \leq (-\mathcal{R}_0^s + \phi(I) + hI)\mathrm{d}t - c_1\sigma_1\mathrm{d}B_1(t) - c_2\sigma_2\mathrm{d}B_2(t) - c_3\sigma_3\mathrm{d}B_3(t). \quad (3.1)$$

By Lemma 2.1, $M_3(t) = \int_0^t c_1\sigma_1\mathrm{d}B_1(s) + \int_0^t c_2\sigma_2\mathrm{d}B_2(s) + \int_0^t c_3\sigma_3\mathrm{d}B_3(s)$ is a local martingale, and satisfies $\lim_{t\to\infty} \frac{1}{t}M_3(t) = 0$. Consequently, by (3.1), the integra-

tion implies that

$$\begin{aligned}
&\liminf_{t\to\infty} h\langle I\rangle_t \\
&\geq \mathcal{R}_0^s - \limsup_{t\to\infty}\langle\phi(I)\rangle_t + \liminf_{t\to\infty}\frac{1}{t}Z_1(t) - \limsup_{t\to\infty}\frac{1}{t}Z_1(0) + \liminf_{t\to\infty}\frac{1}{t}M_3(t) \\
&\geq \mathcal{R}_0^s - \phi(\Gamma\delta^{-1}) \\
&> 0.
\end{aligned}$$

The proof is complete. □

Remark 3.1. Let $\sigma_1 = \sigma_2 = \sigma_3 = 0$. Then $\mathcal{R}_0^s$ degenerates $\mathcal{R}_0$ as demonstrated in (1.2).

4. Existence of stationary distribution

In this section, the existence of an ergodic stationary distribution will be proved and sufficient conditions for the stationary will be established.

Theorem 4.1. *Let $(S(0), E(0), I(0)) \in \mathbb{R}^3_+$ be any positive initial value, If $\mathcal{R}_0^s > \phi(\Gamma\delta^{-1}) > 1$, then, there exists a stationary distribution $\mu(\cdot)$ for model (1.4), which is ergodic.*

Proof. The diffusion matrix of model (1.4) is given by $\widetilde{B} = \mathrm{diag}\{\sigma_1^2S^2, \sigma_2^2E^2, \sigma_3^2I^2\}$. Meanwhile, we select $L = \min_{(S,E,I)\in D_m\subset\mathbb{R}^3_+}\{\sigma_1^2S^2, \sigma_2^2E^2, \sigma_3^2I^2\}$. We get

$$\begin{aligned}
\sum_{i,j=1}^{3} a_{ij}(S,E,I)\xi_i\xi_j &= (\xi_1,\xi_2,\xi_3)\widetilde{B}(\xi_1,\xi_2,\xi_3)^T \\
&= \sigma_1^2S^2\xi_1^2 + \sigma_2^2E^2\xi_2^2 + \sigma_3^2I^2\xi_3^2 \\
&\geq L \parallel \xi \parallel^2,
\end{aligned}$$

for any $(S, E, I) \in D_m$, $\xi = (\xi_1, \xi_2, \xi_3) \in \mathbb{R}^3_+$, where $m > 1$ is a sufficiently large integer and $D_m = [\frac{1}{m}, m] \times [\frac{1}{m}, m] \times [\frac{1}{m}, m]$. Consequently, we take l-dimensional Euclidean space $E_l = \mathbb{R}^3_+$ and $U = D_m$. That is, condition (A1) of [37] holds. For $M > 0$ and a sufficiently small constant $n > 0$, we construct a nonnegative C^2-function $\widetilde{W} : \mathbb{R}^3_+ \to \mathbb{R}$ as follows

$$\widetilde{W}(S,E,I) = M(-c_1\ln S - c_2\ln E - c_3\ln I) - \ln S - \ln E + \frac{1}{n+1}(S+E+I)^{n+1}.$$

It is easy to check that $\liminf_{m\to\infty,(S,E,I)\in\mathbb{R}^3_+\setminus D_m}\widetilde{W}(S,E,I) = +\infty$. Besides, $\widetilde{W}(S,E,I)$ is a continuous function, and there is a minimum point $P^*(S^*,E^*,I^*)$ of the function $\widetilde{W}(S,E,I)$ in the interior of $\mathbb{R}^3_+$. Then a nonnegative C^2-function $\overline{W}$ is constructed as follows:

$$\begin{aligned}
\overline{W}(S,E,I) &= \widetilde{W}(S,E,I) - \widetilde{W}(S^*,E^*,I^*) \\
&= MZ_1 + Z_2 + Z_3 + Z_4 - \widetilde{W}(S^*,E^*,I^*),
\end{aligned}$$

where Z_1 is presented above, and define $Z_2 = -\ln S$, $Z_3 = -\ln E$, $Z_4 = \frac{1}{n+1}(S + E+I)^{n+1}$. Next, we choose a positive constant M such that the following conditions hold

$$\eta = \delta - 0.5n(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) > 0,$$

$$\Xi(S,E,I) := 3M\lambda + 2\delta + \sigma + a + 0.5(\sigma_1^2+\sigma_2^2) + B - 0.5\eta(S^{n+1}+E^{n+1}+I^{n+1}) \le -2,$$

where

$$\lambda = \mathcal{R}_0^s, \quad B = \sup_{(S,E,I)\in\mathbb{R}^3_+} \big\{\Gamma(S+E+I)^n - 0.5\eta(S^{n+1}+E^{n+1}+I^{n+1})\big\}.$$

According to the similar discussions as shown in Theorem 3.1, we re-estimate Z_1 here

$$\begin{aligned}\mathcal{L}Z_1(t) <& -\frac{c_1\Gamma}{S} - \frac{c_2\beta SI}{E\phi(I)} - \frac{c_3\sigma E}{I} + c_1\delta + c_1\beta I + c_2\delta + c_2\sigma + ac_2\\ &+ c_3\delta + c_3\mu + c_3\gamma + 0.5(c_1\sigma_1^2 + c_2\sigma_2^2 + c_3\sigma_3^2)\\ \le& -3\Big(\frac{c_1c_2c_3\Gamma\beta\sigma}{\phi(I)}\Big)^{\frac{1}{3}} + c_1\big(\delta + 0.5\sigma_1^2\big) + c_1\beta I\\ &+ c_2\big(\delta+\sigma+a+0.5\sigma_2^2\big) + c_3\big(\delta+\mu+\gamma+0.5\sigma_3^2\big),\end{aligned}$$

substituting c_1, c_2, c_3 into the above expression, which gives the simplified form

$$\mathcal{L}Z_1(t) < -3\lambda\Big(\frac{\lambda}{\phi(I)}\Big)^{\frac{1}{3}} + 3\lambda + \frac{\beta\lambda}{\delta+0.5\sigma_1^2}I.$$

By the same approaches, we get

$$\begin{aligned}\mathcal{L}Z_2 &= -\frac{\Gamma}{S} + \delta + \frac{\beta I}{\phi(I)} - \frac{aE}{S(1+kE)} - \frac{\gamma I}{S(1+bI)} + \frac{\sigma_1^2}{2} < -\frac{\Gamma}{S} + \delta + \beta I + \frac{\sigma_1^2}{2},\\ \mathcal{L}Z_3 &= -\frac{\beta SI}{E\phi(I)} + \delta + \sigma + \frac{a}{1+kE} + \frac{\sigma_2^2}{2} < -\frac{\beta SI}{E\phi(I)} + \delta + \sigma + a + \frac{\sigma_2^2}{2},\end{aligned}$$

and

$$\begin{aligned}\mathcal{L}Z_4 &= (S+E+I)^n\big[\Gamma - \delta(S+E+I) - \mu I\big]\\ &\quad + \frac{n}{2}(S+E+I)^{n-1}\big(\sigma_1^2S^2 + \sigma_2^2E^2 + \sigma_3^2I^2\big)\\ &< \Gamma(S+E+I)^n - \Big[\delta - \frac{n}{2}(\sigma_1^2\vee\sigma_2^2\vee\sigma_3^2)\Big](S+E+I)^{n+1}\\ &\le \Gamma(S+E+I)^n - \eta(S^{n+1}+E^{n+1}+I^{n+1})\\ &< B - \frac{\eta}{2}(S^{n+1}+E^{n+1}+I^{n+1}).\end{aligned}$$

Therefore

$$\mathcal{L}\widetilde{W} \le -3M\lambda\Big(\frac{\lambda}{\phi(I)}\Big)^{\frac{1}{3}} + \Big(\frac{M\beta\lambda}{\delta+0.5\sigma_1^2} + \beta\Big)I - \frac{\Gamma}{S} - \frac{\beta SI}{E\phi(I)} + \Xi(S,E,I).$$

Next, we construct the following bounded closed set

$$D = \Big\{(S,E,I)\in\mathbb{R}^3_+ : \varepsilon_1 \le S \le \frac{1}{\varepsilon_1}, \varepsilon_2 \le E \le \frac{1}{\varepsilon_2}, \varepsilon_3 \le I \le \frac{1}{\varepsilon_3}\Big\}$$

such that condition (A2) of [37] is valid, where $\varepsilon_i > 0$ $(i = 1, 2, 3)$ are sufficiently small constants satisfying the expressions (4.1)– (4.6) as follows:

$$-\frac{\Gamma}{\varepsilon_1} + Q \leq -1, \tag{4.1}$$

$$-3M\lambda\Big(\frac{\lambda}{\phi(\varepsilon_3)}\Big)^{\frac{1}{3}} + \Big(\frac{M\beta\lambda}{\delta + 0.5\sigma_1^2} + \beta\Big)\varepsilon_3 + T \leq -1, \tag{4.2}$$

$$-\frac{\beta\varepsilon_1\varepsilon_3}{\varepsilon_2\phi(\varepsilon_3)} + Q \leq -1, \tag{4.3}$$

$$-\frac{\eta}{4\varepsilon_1^{n+1}} + U \leq -1, \tag{4.4}$$

$$-\frac{\eta}{4\varepsilon_3^{n+1}} + Y \leq -1, \tag{4.5}$$

$$-\frac{\eta}{4\varepsilon_2^{n+1}} + G \leq -1, \tag{4.6}$$

where

$$f(I) = 3M\lambda + \Big(\frac{M\beta\lambda}{\delta + 0.5\sigma_1^2} + \beta\Big)I + 2\delta + \sigma + a + \frac{\sigma_1^2 + \sigma_2^2}{2} + B,$$

$$Q = \sup_{(S,E,I)\in\mathbb{R}^3_+}\Big\{f(I) - \frac{\eta}{2}(S^{n+1} + E^{n+1} + I^{n+1})\Big\},$$

$$T = \sup_{(S,E,I)\in\mathbb{R}^3_+}\Big\{\Xi(S, E, I)\Big\},$$

$$U = \sup_{(S,E,I)\in\mathbb{R}^3_+}\Big\{f(I) - \frac{\eta}{4}S^{n+1} - \frac{\eta}{2}(E^{n+1} + I^{n+1})\Big\},$$

$$Y = \sup_{(S,E,I)\in\mathbb{R}^3_+}\Big\{f(I) - \frac{\eta}{4}I^{n+1} - \frac{\eta}{2}(S^{n+1} + E^{n+1})\Big\},$$

$$G = \sup_{(S,E,I)\in\mathbb{R}^3_+}\Big\{f(I) - \frac{\eta}{4}E^{n+1} - \frac{\eta}{2}(S^{n+1} + I^{n+1})\Big\}.$$

For convenience, we divide $\mathbb{R}^3_+ \setminus D$ into six domains:

$$D_1 = \{(S, E, I) \in \mathbb{R}^3_+ : 0 < S < \varepsilon_1\},$$
$$D_2 = \{(S, E, I) \in \mathbb{R}^3_+ : 0 < I < \varepsilon_3\},$$
$$D_3 = \{(S, E, I) \in \mathbb{R}^3_+ : S \geq \varepsilon_1, 0 < E < \varepsilon_2, I \geq \varepsilon_3\},$$
$$D_4 = \Big\{(S, E, I) \in \mathbb{R}^3_+ : S > \frac{1}{\varepsilon_1}\Big\},$$
$$D_5 = \Big\{(S, E, I) \in \mathbb{R}^3_+ : I > \frac{1}{\varepsilon_3}\Big\},$$
$$D_6 = \Big\{(S, E, I) \in \mathbb{R}^3_+ : E > \frac{1}{\varepsilon_2}\Big\}.$$

Clearly, $D^c = D_1 \cup D_2 \cup \cdots \cup D_6$. Thus, we only need to prove $\mathcal{L}\widetilde{W}(S, E, I) \leq -1$ on D^c.

Case 1. When $(S, E, I) \in D_1$, by (4.1), we get that

$$\mathcal{L}\widetilde{W} < -\frac{\Gamma}{S} + \Big(\frac{M\beta\lambda}{\delta + 0.5\sigma_1^2} + \beta\Big)I + \Xi(S, E, I) \leq -\frac{\Gamma}{\varepsilon_1} + Q \leq -1.$$

Case 2. When $(S,E,I)\in D_2$, by (4.2), we have that

$$\mathcal{L}\widetilde{W}<-3M\lambda\Big(\frac{\lambda}{\phi(\varepsilon_3)}\Big)^{\frac{1}{3}}+\Big(\frac{M\beta\lambda}{\delta+0.5\sigma_1^2}+\beta\Big)I+\Xi(S,E,I)\leq -1.$$

Case 3. When $(S,E,I)\in D_3$, by (4.3), we have that

$$\mathcal{L}\widetilde{W}<\Big(\frac{M\beta\lambda}{\delta+0.5\sigma_1^2}+\beta\Big)I-\frac{\beta SI}{E\phi(I)}+\Xi(S,E,I)\leq -\frac{\beta\varepsilon_1\varepsilon_3}{\varepsilon_2\phi(\varepsilon_3)}+Q\leq -1.$$

Case 4. When $(S,E,I)\in D_4$, by (4.4), we get that

$$\mathcal{L}\widetilde{W}<-\frac{\eta}{4}S^{n+1}+f(I)-\frac{\eta}{4}S^{n+1}-\frac{\eta}{2}(E^{n+1}+I^{n+1})\leq -\frac{\eta}{4\varepsilon_1^{n+1}}+U\leq -1.$$

Case 5. When $(S,E,I)\in D_5$, by (4.5), we get that

$$\mathcal{L}\widetilde{W}<-\frac{\eta}{4}I^{n+1}+f(I)-\frac{\eta}{4}I^{n+1}-\frac{\eta}{2}(S^{n+1}+E^{n+1})\leq -\frac{\eta}{4\varepsilon_3^{n+1}}+Y\leq -1.$$

Case 6. When $(S,E,I)\in D_6$, by (4.6), we get that

$$\mathcal{L}\widetilde{W}<-\frac{\eta}{4}E^{n+1}+f(I)-\frac{\eta}{4}E^{n+1}-\frac{\eta}{2}(S^{n+1}+I^{n+1})\leq -\frac{\eta}{4\varepsilon_2^{n+1}}+G\leq -1.$$

The proof is complete. □

5. Examples with numerical simulations

The numerical simulations are demonstrated by two examples in this section. The comparison investigations of Milstein's higher order method (MHOM in [8]) and positivity preserving truncated Euler-Maruyama method (PPTEM in [24]) are operated in the first example. A case study is extensively discussed on Fuzhou COVID-19 epidemic of 2022 as the second example. The parameter values of the simulations in Example 5.1, Example 5.2 and Example 5.3 are provided in Table 1.

5.1. Two types of discretization equations

(i) Discretization equation with MHOM. With MHOM in [8], model (1.4) is written as follows:

$$\begin{aligned}
S(t_{k+1})&=S(t_k)+\Big[\Gamma-\delta S(t_k)-\frac{\beta S(t_k)I(t_k)}{\phi(I(t_k))}+\frac{aE(t_k)}{1+kE(t_k)}+\frac{\gamma I(t_k)}{1+bI(t_k)}\Big]\Delta t\\
&\quad+\sigma_1 S(t_k)\xi_1\sqrt{\Delta t}+\frac{\sigma_1^2}{2}S(t_k)(\xi_1^2-1)\Delta t,\\
E(t_{k+1})&=E(t_k)+\Big[\frac{\beta S(t_k)I(t_k)}{\phi(I(t_k))}-\delta E(t_k)-\sigma E(t_k)-\frac{aE(t_k)}{1+kE(t_k)}\Big]\Delta t\\
&\quad+\sigma_2 E(t_k)\xi_2\sqrt{\Delta t}+\frac{\sigma_2^2}{2}E(t_k)(\xi_2^2-1)\Delta t,\\
I(t_{k+1})&=I(t_k)+\Big[\sigma E(t_k)-(\delta+\mu)I(t_k)-\frac{\gamma I(t_k)}{1+bI(t_k)}\Big]\Delta t\\
&\quad+\sigma_3 I(t_k)\xi_3\sqrt{\Delta t}+\frac{\sigma_3^2}{2}I(t_k)(\xi_3^2-1)\Delta t,
\end{aligned}$$

Table 1. Parameter values of the simulations in Example 5.1, Example 5.2 and Example 5.3.

Group	Γ	β	δ	a	γ	σ
(I)	0.20	0.01	0.04	0.15	0.10	0.08
(II)	0.65	0.70	0.15	0.25	0.20	0.25
(III)	488.39	1.99×10^{-6}	1.67×10^{-5}	0.22	0.18	0.25
Group	σ_1	σ_2	σ_3	k	b	$\phi(I)$
(I)	0.0080	0.006	0.03	1	1	$1+0.25I$
(II)	0.0100	0.006	0.02	1	1	$1+0.25I$
(III)	0.0005	0	0	1/70	1/360	$1+0.25I$
Group	μ	Δt	$S(0)$	$E(0)$	$I(0)$	
(I)	0.20	0.001	0.8	0.7	0.6	
(II)	0.25	0.001	0.8	0.7	0.6	
(III)	0.03	0.001	8,291,268	350	20	

[1] For Group (III), the main parameters of model (1.4) are computed as $\Gamma = 0.0215 \times 8291268/365 = 488.39$, $\delta = 0.0061/365 = 1.67 \times 10^{-5}$, where 0.0215 is yearly growth rate and 0.0061 is yearly natural death rate by Fujian Bureau of Statistics in [51]. By the idea of Least Square, $\beta = 1.99 \times 10^{-6}$ is obtained. By surveillance data from Fujian CDC, $\mu = 0.03$ is derived. The initial values $S(0)$, $E(0)$, $I(0)$ are from the reported data released by Fujian Provincial Health Commission in [52].

where $\sigma_i^2 > 0$ $(i = 1, 2, 3)$ are the intensities of the white noises, the time increment Δt is positive, and ξ_i $(i = 1, 2, 3)$ are independent Gaussian random variables which follow the normal distribution $\mathcal{N}(0, 1)$ for $k = 0, 1, 2, \cdots, n$.

(ii) Discretization equation with PPTEM. By PPTEM in [24], model (1.4) is written below:

$$\begin{aligned} S(t_{k+1}) &= S(t_k) + (\Gamma + f_{11} + f_{12})\Delta t + g_1\sqrt{\Delta t}, \\ E(t_{k+1}) &= E(t_k) + (f_{21} + f_{22})\Delta t + g_2\sqrt{\Delta t}, \\ I(t_{k+1}) &= I(t_k) + (f_{31} + f_{32})\Delta t + g_3\sqrt{\Delta t}, \end{aligned}$$

where

$$\begin{aligned} &f_{11} = -\delta\hat{\pi}_0(S(t_k)), \quad f_{12} = -\frac{\beta\hat{\pi}_0(S(t_k)I(t_k))}{\phi(\hat{\pi}_0(I(t_k)))} + \frac{a\hat{\pi}_0(E(t_k))}{1 + k\hat{\pi}_0(E(t_k))} + \frac{\gamma\hat{\pi}_0(I(t_k))}{1 + b\hat{\pi}_0(I(t_k))}, \\ &f_{21} = -(\delta + \sigma)\hat{\pi}_0(E(t_k)), \quad f_{22} = \frac{\beta\hat{\pi}_0(S(t_k)I(t_k))}{\phi(\hat{\pi}_0(I(t_k)))} - \frac{a\hat{\pi}_0(E(t_k))}{1 + k\hat{\pi}_0(E(t_k))}, \\ &f_{31} = \sigma\hat{\pi}_0(E(t_k)) - (\delta + \mu)\hat{\pi}_0(I(t_k)), \quad f_{32} = -\frac{\gamma\hat{\pi}_0(I(t_k))}{1 + b\hat{\pi}_0(I(t_k))}, \\ &g_1 = \sigma_1\hat{\pi}_0(S(t_k))r_{1,k}, \quad g_2 = \sigma_2\hat{\pi}_0(E(t_k))r_{2,k}, \\ &g_3 = \sigma_3\hat{\pi}_0(I(t_k))r_{3,k}, \quad \hat{\pi}_0(u) = \max\{0, u\}, \end{aligned}$$

and $r_{i,k}$ $(i = 1, 2, 3, k = 0, 1, 2, 3, \cdots)$ are independent random variables with the normal distribution $\mathcal{N}(0, 1)$.

5.2. Numerical simulations of stochastic extinction and persistence of model (1.4)

Example 5.1. (Stochastic extinction) We choose the parameter values in Group (I) in Table 1, according to Theorem 2.2, then, model (1.4) admits a disease-free

equilibrium point $P_0(5,0,0)$, and $\mathcal{R}_0 = 0.0435 < 1$, $\mu = 0.2 > 0.5(\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2) = 0.00005$. The corresponding simulations are demonstrated by MHOM and PPTEM in Figure 1 as $n = 30000$. Moreover, the extinction time for model (1.4) with $\sigma_1 = \sigma_2 = \sigma_3 = 0$ are compared, from which we conclude that the extinction time for the exposed and the infected of model (1.4) becomes a bit later as presented in Figure 2. We also notice that when the values of b and k turn out to be smaller, the extinction time that the infected individuals spent gets earlier as shown in Figure 3. When the larger fluctuations $\sigma_1 = 0.1$, $\sigma_2 = 0.1$, $\sigma_3 = 0.2$ and $n = 20000$ are given, the corresponding simulations show that the disease reaches extinction faster in Figure 4.

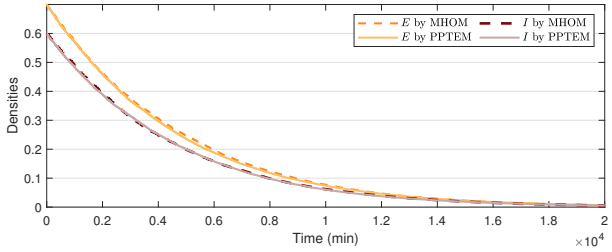

Figure 1. Stochastic extinction of E and I under Group (I) of model (1.4) with MHOM and PPTEM.

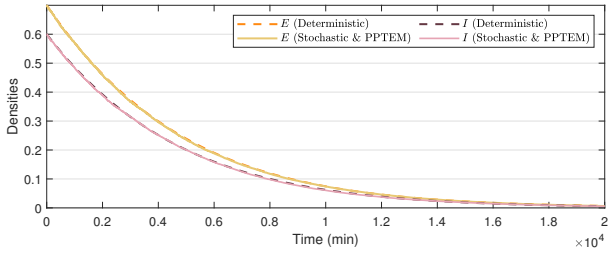

Figure 2. Comparisons of the extinctions of E and I for deterministic model and stochastic model under PPTEM.

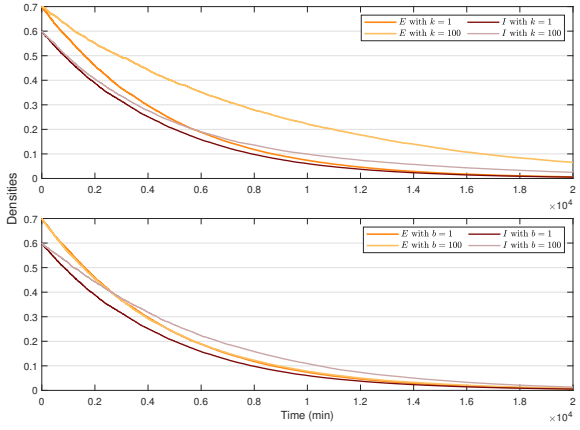

Figure 3. Stochastic extinctions of E and I to model (1.4) with PPTEM. Top for changes of k, bottom for changes of b.

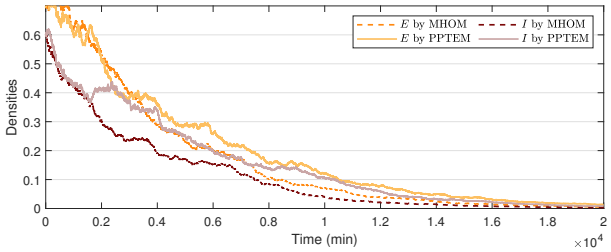

Figure 4. Stochastic extinction of E and I to model (1.4) under MHOM and PPTEM with larger fluctuations $\sigma_1 = 0.1$, $\sigma_2 = 0.1$, $\sigma_3 = 0.2$.

Example 5.2. (Stochastic persistence) We use the parameter values in Group (II) in Table 1, then, model (1.4) admits an endemic equilibrium point $P^*(S^*, E^*, I^*) = (1.6884, 1.1348, 0.5228)$ by PPTEM. Moreover, we have $\mathcal{R}_0^s = 2.1652 > 1$, and $\liminf_{t\to\infty}\langle I\rangle_t \geq 0.3818$. Therefore, we take $n = 100000$, model (1.4) admits a stationary distribution presented by Theorem 3.1 and Theorem 4.1, and the stochastic persistence of model (1.4) is demonstrated in Figure 5.

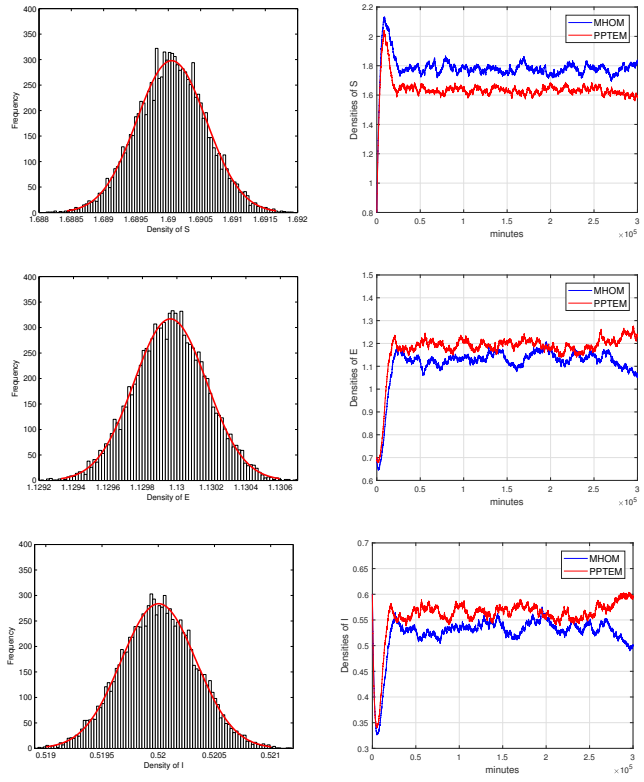

Figure 5. Left for histogram of S, E, and I to model (1.4), right for stochastic persistence of S, E and I to model (1.4) with MHOM and PPTEM.

5.3. Numerical simulations of Fuzhou COVID-19 epidemic in 2022

Example 5.3. (A case study of Fuzhou COVID-19 epidemic). Since Chinese government issued Twenty Measures on November 11 of 2022, and New Ten Measures on December 7 of 2022, the tendency of Fuzhou COVID-19 epidemic fluctuated during the period from November 22 to December 12 of 2022. We take the parameter values of Group (III) in Table 1 and the awareness delay is set as 6 days by [9,40,42]. The numerical simulations are operated by PPTEM based on the surveillance data from Fujian Provincial Center for Disease Control and Prevention (short for, Fujian CDC) in Figure 6.

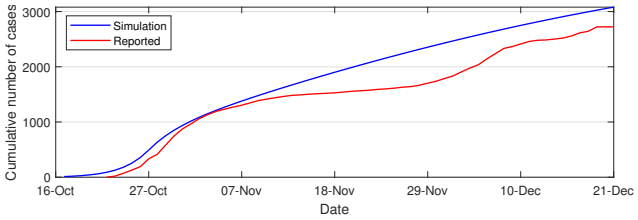

Figure 6. Cumulative number of infection cases for Fuzhou COVID-19 epidemic led by SARS-CoV-2 Omicron BA.5.2 from October 23 to December 22 of 2022.

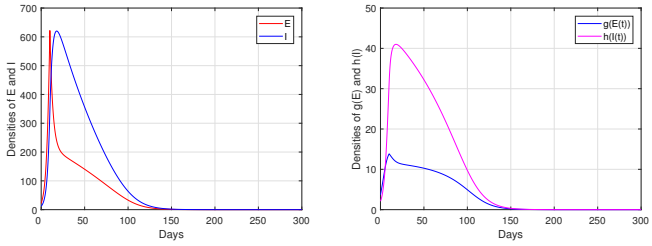

Figure 7. Numerical simulations of E, I, $g(E)$ and $h(I)$ for 300 days to Fuzhou epidemic by using model (1.4) with $\sigma_1 = 0.0005, \sigma_2 = 0, \sigma_3 = 0$.

The results of numerical simulations reveal the peak of E on the 10th day with 622 cases, and the peak of I on the 18th day with 620 cases as shown in Figure 7. Meanwhile, the decreasing of k implies the increasing of the innate immunity, the decreasing of b implies the increasing of the treatment availability, further, the time that both E and I approach zero reduces in Figure 8, which are consistent with the results in Figure 3.

6. Conclusion

The innate immunity of the individuals is usually regarded as the protection abilities during the progress that pathogen or virus invades the hosts. So, the innate immunity plays a vital role in controlling of the infectious diseases. We consider the innate immunity in the form of Holling type II functional response in this paper, instead of linear form, to establish a nonlinear stochastic epidemic model with innate immunity. Then, the stochastic extinction and stochastic persistence under

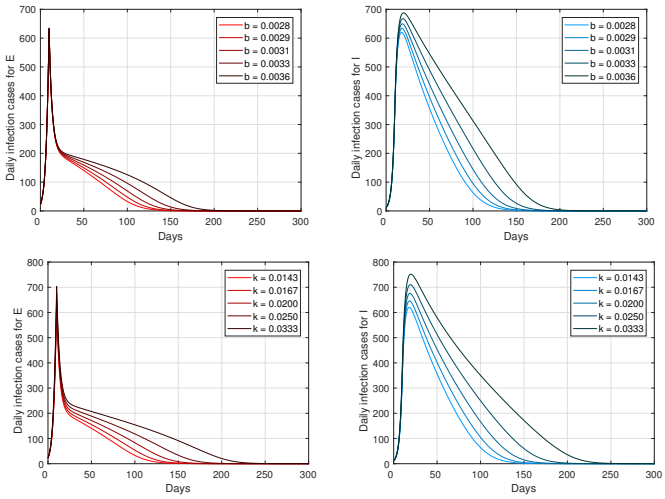

Figure 8. Stochastic extinctions of E and I to Fuzhou epidemic by using model (1.4) with $\sigma_1 = 0.0005$, $\sigma_2 = 0$, $\sigma_3 = 0$. Top for changes of b, bottom for changes of k.

moderate conditions, the existence of stationary distribution are extensively discussed. Therefore, the main numerical simulations by two methods are performed to demonstrate the differences therein.

We show that model (1.4) admits a unique global positive solution with the positive initial value. Further, we derive that the infectious disease approaches the extinction when $\mathcal{R}_0 < 1$, $\nu < 0$ and $2\mu > \sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2$ are valid as shown in Figure 1. Meanwhile, the persistence in the mean and the existence of ergodic stationary distribution to model (1.4) are obtained when $\mathcal{R}_0^s > \phi(\Gamma\delta^{-1})$ held. The results show that the parameters a and k of innate immunity are involved into the sufficient conditions of the extinction, persistence in the mean and ergodic stationary distribution to model (1.4). We explore that the increasing values of k and b make the time that the exposed and the infected approaches zero earlier as demonstrated in Figure 3 and Figure 8. That is to say, the individuals with low innate immunity in the population take high risks against infection, these individuals should be treated promptly by medical agents. The main results of this study improve the ones in model (6) of [41], model (4) of [18] and model (1.4) of [44] for the case of $a = 0$ and $\gamma = 0$, which provides a new biological perspective to the respiratory infectious diseases. Moreover, we are motivated by results of two-group SEIR model in [19], intend to propose a multi-group stochastic SEIR with time delay in the next investigation.

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We declare that all authors conceive of the study and carry out the proof. All authors of this paper read and approve the final manuscript.

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