



Research article

Threshold dynamics of a stochastic SIHR epidemic model of COVID-19 with general population-size dependent contact rate

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Abstract: In this paper, we propose a stochastic SIHR epidemic model of COVID-19. A basic reproduction number R_0^s is defined to determine the extinction or persistence of the disease. If $R_0^s < 1$, the disease will be extinct. If $R_0^s > 1$, the disease will be strongly stochastically permanent. Based on realistic parameters of COVID-19, we numerically analyze the effect of key parameters such as transmission rate, confirmation rate and noise intensity on the dynamics of disease transmission and obtain sensitivity indices of some parameters on R_0^s by sensitivity analysis. It is found that: 1) The threshold level of deterministic model is overestimated in case of neglecting the effect of environmental noise; 2) The decrease of transmission rate and the increase of confirmed rate are beneficial to control the spread of COVID-19. Moreover, our sensitivity analysis indicates that the parameters β , σ and δ have significantly effects on R_0^s .

Keywords: stochastic SIHR epidemic model; COVID-19; the basic reproduction number; extinction; stochastically permanent

1. Introduction

The COVID-19 has been significantly impacting our lives since the emerge of the first case in early December 2019 in Wuhan, China. As of 14 January 2022, there have been 318,648,834 confirmed cases, including 5,518,343 deaths in the world [1]. Multiple mutant strains of COVID-19 have emerged, such as Alpha, Beta, Gamma, Delta, Omicron and so on. Among these strains, Omicron, the newly discovered strain, is known as the fast speed of transmission and the strong ability of infection. Therefore it is extremely urgent to study and control the transmission of COVID-19.

To better understand the transmission and develop efficient control strategies, researchers have employed mathematical models to analyze the dynamic behavior and control the outbreak of COVID-19 [2–10]. Recently, Allegretti et al. [3] considered a modified SIR model of COVID-19 and

found that a high fraction of avoided contacts leads to the stability of the disease free equilibrium. Naik et al. [4] proposed a COVID-19 epidemic model and indicated that reducing transmission rate of the coronavirus is the most essential strategy to prevent the virus further spread. Okuonghae et al. [5] formulated a mathematical model and examined the impact of various non-pharmaceutical control measures on the population dynamics of COVID-19 by using the available data from Lagos, Nigeria. Fatma et al. [6] studied the interactions between COVID-19 and diabetes by using real data from Turkey and numerically visualized the population dynamics of COVID-19. Tang et al. [7] proposed a Filippov SIR model to investigate the impacts of three control strategies (media coverage, vaccination and treatment) and choose the switching policy properly to reduce the infected size. Humphrey et al. [8] developed an SEIRL model and found that testing and tracing asymptomatic individuals frequently can help in controlling new cases. Jin et al. [9] proposed a generalised SEIR model to seek optimal strategies for disease control, finding that reducing the transmission rates and increasing contact tracing are possible to hinder the fast spread of COVID-19. It is found that in the early stages of COVID-19, isolating confirmed cases was considered as a more effective control measure due to the inability to quickly produce highly effective vaccines [11]. Hence, Jiao et al. [11] proposed an SIHR model incorporating confirmed cases with general population-size dependent contact rate as follows:

$$\begin{cases} \frac{dS}{dt} = A - \beta f(N)SI - \mu S, \\ \frac{dI}{dt} = \beta f(N)SI - (\gamma + \delta + \mu + \mu_1)I, \\ \frac{dH}{dt} = \delta I - (m + \mu + \mu_2)H, \\ \frac{dR}{dt} = \gamma I + mH - \mu R. \end{cases} \quad (1)$$

Here the infectious cases are divided into two sub-populations: non-confirmed cases (I) and confirmed cases (H). The non-confirmed cases are infected individuals who have not been tested by medical institutions. Once the nucleic acid tests are positive, they would become confirmed cases and be isolated. In model (1), $I(t)$ and $H(t)$ are the non-confirmed infected individuals and the confirmed individuals at time t , respectively; and $S(t)$, $R(t)$ and $N(t)$ denote respectively the susceptible individuals, the recovered individuals and the total population. A is the recruitment rate of the population and it is assumed that all the newcomers are susceptible. $\beta f(N)SI$ is the general population-size dependent incidence, in which the parameter β is the transmission rate from the infectious class to the susceptible class, and $f(N)$ is a function of N and it comes in many forms, see [11–13]. μ and γ denote the natural death rate and the natural recovery rate, respectively. δ is the confirmation rate from the infected population to confirmed cases. μ_1, μ_2 are respectively the extra disease-related death rate constants in compartments I and H . m is the transform rate from the confirmed population to the removed population. All parameters are nonnegative constants. Model (1) always exists the disease-free equilibrium $E_0 = (\frac{A}{\mu}, 0, 0, 0)$, whose stability is determined by the basic reproduction number $R_0 = \frac{A\beta f(\frac{A}{\mu})}{\mu(\delta + \gamma + \mu + \mu_1)}$. If $R_0 < 1$, then E_0 is globally asymptotically stable; and if $R_0 > 1$, E_0 becomes unstable and an endemic equilibrium $E^*(S^*, I^*, H^*, R^*)$ appears and it is locally asymptotically stable, see [11] for details.

However, deterministic model (1) has certain limitation, it cannot describe the effects of random environment. In fact, there are many stochastic factors that can effect the transmission of disease. For example, Jamshidi et al. [14] investigated the impact of mobility, urban density, population, homestay,

and mask-wearing separately on COVID-19 by conducting a multiple regression analysis and found that a higher level of population mobility and traveling can increase the transmission rate. Sabbir Hossain et al. [15] studied the impact of weather on COVID-19 in part of South Asian countries through adopting the Autoregressive Integrated Moving Average with Explanatory Variables (ARIMAX) model and revealed that maximum wind speed had a significant negative effect on the transmission rate in India, while rainfall, relative humidity, temperature and maximum air pollutants matter PM 2.5 had different influence on COVID-19 in different areas. We also refer the readers to Habeebullah [16], Baniasad [17] and Damette [18] for learning more about the effect of weather, pollution and mobility to the transmission efficiency of COVID-19. Therefore, the effects of these random factors can be translated to the fluctuations in the transmission rate β [19]. It has been well established in literatures that introducing parameter perturbations can affect the dynamic behavior of population. Gray et al. [20] studied the effect of stochastic parameter perturbation on SIS model and fill previous gap. Li et al. [21] applied similar parameter perturbation to investigate the effect of stochastic environmental variability on inter-pandemic transmission dynamics of influenza A. Cai et al. [22] showed that appropriate parameter perturbation to the system is useful in controlling the spread of the disease. Motivated by these, in this paper we suppose that the transmission rate β fluctuates around an average value due to the continuous fluctuations in the environment by the white noise $\beta + \sigma \dot{B}(t)$, and then obtain the following stochastic model:

$$\begin{cases} dS = [A - \beta f(N)SI - \mu S]dt - \sigma f(N)S I dB(t), \\ dI = [\beta f(N)SI - (\gamma + \delta + \mu + \mu_1)I]dt + \sigma f(N)S I dB(t), \\ dH = [\delta I - (m + \mu + \mu_2)H]dt, \\ dR = (\gamma I + mH - \mu R)dt, \end{cases} \quad (2)$$

where $B(t)$ is a standard Brownian motion, which is defined on the complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbb{P})$, and σ represents the noise intensity; the function $f(\cdot) : \mathbb{R}_+ \rightarrow \mathbb{R}_+$ satisfies the following assumptions:

$$f'(x) \leq 0, (f(x)x)' \geq 0, \quad (3)$$

where $f'(x)$ and $(f(x)x)'$ respectively denote the derivative of $f(x)$ and $(f(x)x)$.

The main purpose of this paper is to explore the effect of random variability in the environments on the spread of COVID-19 based on realistic parameters from [11] and [23], and obtain the strict threshold condition of the disease. The main contributions of our study can be summarized as follows.

- It has been shown in [24] that uncertainty is certain in the disease transmission rate of COVID-19 and there are large variation in its range. Therefore, it seems necessary and important to consider random factors in the context of COVID-19.
- Under the setting of more general population-size dependent contact rate, we obtain the threshold condition of disease extinction and persistence by constructing suitable Lyapunov functions. In this sense, we extend the previous studies such as [25] and [26], where the standard incidence form is used.
- We have proved that the basic reproduction R_0^s for our stochastic model completely determines the extinction or persistence of the disease. This is contrasted with the existing literatures [27–32],

where except for the conditions required for the basic reproduction number, there still have some additional conditions for noise to ensure the extinction or persistence of disease. Therefore our results can be regard as an significant extension of the previous articles and also can provide effective information to the control of COVID-19.

The organization of this paper is as follows. In Section 2, we prove the existence and uniqueness of the positive solution. In Section 3, using techniques in [33–36] we derive the threshold R_0^s which completely determines the extinction and strongly permanent of the disease. Finally, in Section 4, numerical simulations are carried out to illustrate our theoretical results by analyzing the effect of key parameters on disease and obtain sensitivity indices of some parameters on R_0^s by sensitivity analysis. We also discuss the impact of some measures (media coverage, government intervention, testing and tracking) on COVID-19 and give a summary.

2. Existence and uniqueness of the positive solution

In order to investigate the dynamics of stochastic model (2), we first need to show that the model has a unique global positive solution. Denote by $\Phi(t) = (S(t), I(t), H(t), R(t))$ the solution of model (2) and $\mathbb{R}_+^4 := \{(x_1, x_2, x_3, x_4) \in \mathbb{R}^4 : x_i > 0, i = 1, 2, 3, 4\}$. Moreover, for any function $V \in C^2(\mathbb{R}^4, \infty); \mathbb{R}_+)$, define the differential operator L associated with model (2) as

$$LV = V_\Phi(\Phi)\tilde{f}(\Phi) + \frac{1}{2}\tilde{g}^T(\Phi)V_{\Phi\Phi}(\Phi)\tilde{g}(\Phi),$$

where $V_\Phi(\Phi)$ and $V_{\Phi\Phi}(\Phi)$ are the gradient and Hessian of $V(\cdot)$; \tilde{f} and \tilde{g} are respectively the drift and diffusion coefficients of model (2). By Itô's formula,

$$dV = LVdt + V_\Phi(\Phi)\tilde{g}(\Phi)dB(t).$$

We have the following theorem.

Theorem 2.1. *For any initial value $(S(0), I(0), H(0), R(0)) \in \mathbb{R}_+^4$, there is a unique solution $(S(t), I(t), H(t), R(t))$ of model (2) on $t \geq 0$, and the solution will remain in \mathbb{R}_+^4 with probability 1.*

Proof. Notice that the coefficients of model (2) satisfy the local Lipschitz condition. Then there is a unique local solution $(S(t), I(t), H(t), R(t))$ on $[0, \tau_e)$ for any initial value $(S(0), I(0), H(0), R(0))$ in \mathbb{R}_+^4 , where τ_e is the explosion time [37]. In order to show the global existence of the positive solution, we need to prove that $\tau_e = \infty$ almost surely (a.s.).

By summing all the equations in model (2) we get that

$$dN(t) = (A - \mu N - \mu_1 I - \mu_2 H)dt, \quad (4)$$

where $N(t) = S(t) + I(t) + H(t) + R(t)$. It then follows that for all $t < \tau_0$,

$$N(t) \leq \max \left\{ S(0) + I(0) + H(0) + R(0), \frac{A}{\mu} \right\} := C_1, \quad (5)$$

where

$$\tau_0 := \inf\{t \geq 0 : S(t) \leq 0 \text{ or } I(t) \leq 0 \text{ or } H(t) \leq 0 \text{ or } R(t) \leq 0\}.$$

Obviously, $\tau_0 \leq \tau_\varepsilon$, a.s. To prove $\tau_\varepsilon = \infty$, we only need to prove $\tau_0 = \infty$, a.s. For this, we set $\varepsilon_0 > 0$ such that $S(0) > \varepsilon_0$, $I(0) > \varepsilon_0$, $H(0) > \varepsilon_0$, $R(0) > \varepsilon_0$. For any $\varepsilon > 0$ satisfying $\varepsilon \leq \varepsilon_0$, define the stopping time

$$\tau_\varepsilon = \inf\{t \in [0, \tau_\varepsilon) : S(t) \leq \varepsilon \text{ or } I(t) \leq \varepsilon \text{ or } H(t) \leq \varepsilon \text{ or } R(t) \leq \varepsilon\}, \quad (6)$$

where we let $\inf \emptyset = \infty$ (as usual \emptyset denotes the empty set). It is easy to see that τ_ε increases to τ_0 as ε tends to 0, that is $\tau_0 = \lim_{\varepsilon \rightarrow 0} \tau_\varepsilon$, a.s. Now we prove that $\tau_0 = \infty$, a.s.

Suppose that this statement is false, then there exist a pair of constants $T > 0$ and $\rho \in (0, 1)$ such that

$$\mathbb{P}\{\tau_0 \leq T\} > \rho.$$

Thus there is a positive constant $\varepsilon_1 \leq \varepsilon_0$ such that $\mathbb{P}\{\tau_\varepsilon \leq T\} > \rho$ for any positive $\varepsilon \leq \varepsilon_1$. Define a function $V : \mathbb{R}_+^4 \rightarrow \mathbb{R}_+$ by

$$V(\Phi(t)) = -\ln \frac{S}{C_1} - \ln \frac{I}{C_1} - \ln \frac{H}{C_1} - \ln \frac{R}{C_1}.$$

Clearly, V is positive definite. Applying Itô's formula in Appendix, we obtain

$$dV = LVdt + \sigma f(N)(I - S)dB(t),$$

where

$$\begin{aligned} LV = & -\frac{A}{S} + \beta f(N)I + \mu + \frac{1}{2}\sigma^2 I^2 f^2(N) - \beta f(N)S + (\gamma + \delta + \mu + \mu_1) \\ & + \frac{1}{2}\sigma^2 S^2 f^2(N) - \frac{\delta I}{H} + (m + \mu + \mu_2) - \frac{\gamma I}{R} - \frac{mH}{R} + \mu. \end{aligned}$$

By using models (5) and (3), we can obtain

$$\begin{aligned} LV & \leq \beta f(N)N + \frac{1}{2}\sigma^2(I^2 + S^2)f^2(N) + 4\mu + \gamma + \delta + \mu_1 + m + \mu_2 \\ & \leq \beta f(C_1)C_1 + \frac{1}{2}\sigma^2(C_1 f(C_1))^2 + 4\mu + \gamma + \delta + \mu_1 + m + \mu_2 := C_2. \end{aligned}$$

Therefore

$$dV \leq C_2 dt + \sigma f(N)(I - S)dB(t).$$

Integrating both sides from 0 to $\tau_\varepsilon \wedge T$ and taking expectations, yields

$$\mathbb{E}V(\Phi(\tau_\varepsilon \wedge T)) \leq V(\Phi(0)) + C_2 T.$$

Set $\Omega_\varepsilon = \{\tau_\varepsilon \leq T\}$ for any positive $\varepsilon \leq \varepsilon_1$, then we have $\mathbb{P}(\Omega_\varepsilon) > \rho$. Note that for every $\omega \in \Omega_\varepsilon$, there is at least one of $S(\tau_\varepsilon, \omega)$, $I(\tau_\varepsilon, \omega)$, $H(\tau_\varepsilon, \omega)$ and $R(\tau_\varepsilon, \omega)$ equals ε , then

$$V(\Phi(\tau_\varepsilon)) \geq -\ln \frac{\varepsilon}{C_1}.$$

Consequently,

$$V(\Phi(0)) + C_2 T \geq \mathbb{E}[I_{\Omega_\varepsilon} V(\Phi(\tau_\varepsilon \wedge T))] = \mathbb{P}(\Omega_\varepsilon)V(\Phi(\tau_\varepsilon)) > -\rho \ln \frac{\varepsilon}{C_1},$$

where I_{Ω_ε} is the indicator function of Ω_ε . Letting $\varepsilon \rightarrow 0$, we obtain the contradiction

$$\infty > V(\Phi(0)) + C_2 T = \infty.$$

So $\tau_0 = \infty$, a.s. The proof of Theorem 2.1 is thus completed.

Denote

$$\Delta = \{(S, I, H, R) \in \mathbb{R}_+^4 : \frac{A}{\mu + \mu_1 + \mu_2} \leq S + I + H + R \leq \frac{A}{\mu}\}. \quad (7)$$

It is easy to see from model (4) that Δ is the positive invariant set of stochastic model (2). Thus, in the sequel of this paper, we only need to consider the dynamics of model (2) constrained in Δ .

3. Threshold dynamics of the disease

In this section, we perform the persistence and extinction analysis of stochastic model (2). Define

$$R_0^s = \frac{A\beta f\left(\frac{A}{\mu}\right)}{\mu\left(\delta + \gamma + \mu + \mu_1 + \frac{1}{2}f^2\left(\frac{A}{\mu}\right)\left(\frac{A}{\mu}\right)^2\sigma^2\right)}. \quad (8)$$

We can see from below that R_0^s plays the similar role of basic reproduction number of disease as defined in classical deterministic epidemic models, called stochastic basic reproduction number, which completely determines the dynamics of stochastic model (2).

Denote

$$g(S, I, H, R) = -\frac{1}{2}\sigma^2 f^2(N)S^2 + \beta f(N)S - (\gamma + \delta + \mu + \mu_1) \quad (9)$$

and let

$$\lambda := g\left(\frac{A}{\mu}, 0, 0, 0\right) = -\frac{1}{2}\sigma^2 f^2\left(\frac{A}{\mu}\right)\left(\frac{A}{\mu}\right)^2 + \beta f\left(\frac{A}{\mu}\right)\frac{A}{\mu} - (\gamma + \delta + \mu + \mu_1). \quad (10)$$

It is easy to check that $R_0^s = 1$ implies $\lambda = 0$, and moreover $R_0^s < 1$ if and only if $\lambda < 0$.

3.1. Extinction

Consider the following stochastic differential equation of $x(t) \in \mathbb{R}^n$:

$$dx(t) = a(x(t))dt + b(x(t))d\tilde{B}(t), \quad (11)$$

where $a(\cdot) : \mathbb{R}^n \rightarrow \mathbb{R}^n$ and $b(\cdot) : \mathbb{R}^n \rightarrow \mathbb{R}^{n \times d}$; $\tilde{B}(t)$ is a \mathcal{F}_t -adapted \mathbb{R}^d -valued standard Brownian motion. Assume that $x(t) = 0$ is the trivial solution of model (11). The following lemma presents a proper adaptation of Theorem 3.1 in Dang and Yin [38], which will be used later to establish the condition for the extinction of disease.

Lemma 3.1. *Let D be a neighborhood of $0 \in \mathbb{R}^n$ and $V : D \rightarrow \mathbb{R}_+$ which satisfies that $V(x) = 0$ if and only if $x = 0$ and that $V(x)$ is continuous on D , twice continuously differentiable in $D \setminus \{0\}$. Then the trivial solution is asymptotically stable in probability provided there exists a negative constant number c such that for any $x \in D \setminus \{0\}$,*

$$LV(x) \leq cV(x).$$

The following result is about the extinction of disease.

Theorem 3.2. *If $R_0^s < 1$, then for any initial value $(S(0), I(0), H(0), R(0)) \in \Delta$, $\Phi(t) \rightarrow (\frac{A}{\mu}, 0, 0, 0)$ a.s. as $t \rightarrow \infty$, i.e., the disease will go to extinction. Moreover,*

$$\mathbb{P}\left\{\lim_{t \rightarrow \infty} \frac{\ln I(t)}{t} = \lambda < 0\right\} = 1. \quad (12)$$

Proof. Notice that $R_0^s < 1$ implies that $\lambda < 0$. Thus we can choose a sufficiently small number $\xi > 0$ such that $\lambda + \xi < 0$. Consider the Lyapunov function

$$V(S, I, H, R) = \left(\frac{A}{\mu} - S\right)^2 + I^p + H^2 + qR^2, \quad (13)$$

where $q = \frac{\mu^2}{2m^2}$ and $p \in (0, 1)$ will be determined later. Obviously, $V(S, I, H, R) = 0$ if and only if $(S, I, H, R) = (\frac{A}{\mu}, 0, 0, 0)$. Applying Itô's formula, it then follows from model (13) that

$$\begin{aligned} LV(S, I, H, R) &= -2\left(\frac{A}{\mu} - S\right)(A - \beta f(N)SI - \mu S) + pI^{p-1}[\beta f(N)SI - (\gamma + \delta + \mu + \mu_1)I] \\ &\quad + \sigma^2 f^2(N)S^2I^2 + \frac{1}{2}p(p-1)I^p\sigma^2 f^2(N)S^2 + 2H[\delta I - (m + \mu + \mu_2)H] \\ &\quad + 2qR(\gamma I + mH - \mu R) \\ &= -2\mu\left(\frac{A}{\mu} - S\right)^2 - 2(m + \mu + \mu_2)H^2 - 2q\mu R^2 + I\left[2\left(\frac{A}{\mu} - S\right)\beta f(N)S \right. \\ &\quad \left. + \sigma^2 f^2(N)S^2I + 2\delta H + 2q\gamma R\right] + pI^p g(S, I, H, R) + 2qmHR \\ &\quad + \frac{1}{2}p^2\sigma^2 f^2(N)S^2I^p. \end{aligned} \quad (14)$$

We first perform some estimates on the items appeared in model (14). Notice that

$$-2\mu\left(\frac{A}{\mu} - S\right)^2 - (2m + 2\mu_2 + \mu)H^2 - q\mu R^2 \leq p(\lambda + \xi)\left[\left(\frac{A}{\mu} - S\right)^2 + H^2 + qR^2\right], \quad (15)$$

provided p is sufficiently small such that

$$\min\{2\mu, 2m + 2\mu_2 + \mu, q\mu\} \geq p|\lambda + \xi|. \quad (16)$$

Now denote $U_{\delta_1} := (\frac{A}{\mu} - \delta_1, \frac{A}{\mu}] \times [0, \delta_1]^3$ for $\delta_1 \in (0, \frac{A}{\mu})$. Notice the continuity of functions $g(\cdot)$ and $f(\cdot)$. We can take δ_1 and p sufficiently small such that model (16) holds and for any $(S, I, H, R) \in U_{\delta_1}$, the following two inequalities hold:

$$pI^p g(S, I, H, R) \leq p\left[g\left(\frac{A}{\mu}, 0, 0, 0\right) + \xi_1\right]I^p = p(\lambda + \xi_1)I^p$$

and

$$I\left[2\left(\frac{A}{\mu} - S\right)\beta f(N)S + \sigma^2 f^2(N)S^2I + 2\delta H + 2q\gamma R\right] + \frac{1}{2}p^2\sigma^2 f^2(N)S^2I^p \leq p\xi_2 I^p,$$

where $0 < \xi_1, \xi_2 < \xi$ and $\xi_1 + \xi_2 = \xi$. Consequently, we have

$$pI^p g(S, I, H, R) + I \left[2 \left(\frac{A}{\mu} - S \right) \beta f(N) S + \sigma^2 f^2(N) S^2 I + 2\delta H + 2q\gamma R \right] + \frac{1}{2} p^2 \sigma^2 f^2(N) S^2 I^p \leq p(\lambda + \xi) I^p. \quad (17)$$

Moreover, we can easily check that

$$-q\mu R^2 + 2qmHR - \mu H^2 \leq 0. \quad (18)$$

Combining models (15), (17) and (18), we know that if we take p and δ_1 both sufficiently small, it then follows from model (14) that for any $(S, I, H, R) \in U_{\delta_1}$, we have

$$LV(S, I, H, R) \leq p(\lambda + \xi)V(S, I, H, R).$$

According to Lemma 3.1, we know that the disease free equilibrium $(\frac{A}{\mu}, 0, 0, 0)$ is asymptotically stable in probability. That is, for any $\varepsilon > 0$, there exists a δ_2 , $0 < \delta_2 < \delta_1$ such that

$$\mathbb{P} \left\{ \lim_{t \rightarrow \infty} \Phi(t) = \left(\frac{A}{\mu}, 0, 0, 0 \right) \right\} \geq 1 - \varepsilon \quad (19)$$

for any $(S(0), I(0), H(0), R(0)) \in U_{\delta_2}$, where $U_{\delta_2} = (\frac{A}{\mu} - \delta_2, \frac{A}{\mu}] \times [0, \delta_2]^3$. Now we are in a position to prove that any solution starting in Δ will eventually enter U_{δ_2} .

Define $\tau_{\delta_2} = \inf\{t \geq 0 : S(t) \geq \frac{A}{\mu} - \delta_2\}$. Consider the Lyapunov function $V_1(\Phi(t)) = c_1 - (S + 1)^{c_2}$, where c_1 and c_2 are two positive constants to be specified. By calculating we obtain

$$LV_1(\Phi(t)) = -c_2(S + 1)^{c_2-2} \left[(S + 1) \left(\mu \left(\frac{A}{\mu} - S \right) - \beta f(N) S I \right) + \frac{c_2 - 1}{2} \sigma^2 f^2(N) S^2 I^2 \right].$$

For any $S \in (0, \frac{A}{\mu} - \delta_2]$, we have $(S + 1)\mu(\frac{A}{\mu} - S) \geq \mu\delta_2$ and $\inf\{\sigma^2 f^2(N)\} > 0$, then we can choose a sufficiently large c_2 such that

$$(S + 1) \left[\mu \left(\frac{A}{\mu} - S \right) - \beta f(N) S I \right] + \frac{c_2 - 1}{2} \sigma^2 f^2(N) S^2 I^2 \geq \frac{1}{2} \mu \delta_2.$$

Hence

$$LV_1(\Phi(t)) \leq -\frac{1}{2} \mu \delta_2.$$

By Dynkin's formula [39], we obtain

$$\mathbb{E}[V_1(\Phi(\tau_{\delta_2} \wedge t))] = V_1(\Phi(0)) + \mathbb{E} \int_0^{\tau_{\delta_2} \wedge t} LV_1(\Phi) ds \leq V_1(\Phi(0)) - \frac{1}{2} \mu \delta_2 \mathbb{E}(\tau_{\delta_2} \wedge t).$$

Letting $t \rightarrow \infty$ and using Fatou's lemma yields that

$$\mathbb{E}[V_1(\Phi(\tau_{\delta_2}))] \leq V_1(\Phi(0)) - \frac{1}{2} \mu \delta_2 \mathbb{E}(\tau_{\delta_2}).$$

Due to V_1 is bounded on \mathbb{R}_+^4 , then $\mathbb{E}(\tau_{\delta_2}) < \infty$. By the strong Markov property, from model (19) and $\mathbb{E}(\tau_{\delta_2}) < \infty$ we have that

$$\mathbb{P} \left\{ \lim_{t \rightarrow \infty} \Phi(t) = \left(\frac{A}{\mu}, 0, 0, 0 \right) \right\} \geq 1 - \varepsilon$$

for any $\varepsilon > 0$ and $(S(0), I(0), H(0), R(0)) \in \Delta$. Therefore

$$\mathbb{P} \left\{ \lim_{t \rightarrow \infty} \Phi(t) = \left(\frac{A}{\mu}, 0, 0, 0 \right) \right\} = 1 \quad (20)$$

for any $(S(0), I(0), H(0), R(0)) \in \Delta$. Applying the Itô's formula, we have

$$d \ln I = \left(\beta f(N)S - (\gamma + \delta + \mu + \mu_1) - \frac{1}{2} \sigma^2 f^2(N)S^2 \right) dt + \sigma f(N)S dB(t). \quad (21)$$

Integrating both sides of model (21) from 0 to t leads to

$$\ln I(t) - \ln I(0) = \int_0^t g(\Phi(u)) du + \int_0^t \sigma f(N)S dB(u). \quad (22)$$

By the strong law of large numbers for martingales, we obtain from models (10) and (20) that

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t g(\Phi(u)) du = \lambda$$

and

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \sigma f(N)S(u) dB(u) = 0, \quad a.s.$$

It then follows from model (22) that $\lim_{t \rightarrow \infty} \frac{\ln I(t)}{t} = \lambda$, which implies model (12). The proof is thus completed.

3.2. Persistence

In this section, we prove that the disease will be persistent provided $R_0^s > 1$. We first present the following useful lemma.

Lemma 3.3. *Let $\partial\Delta := \{(S, I, H, R) \in \Delta : I = 0\}$. Then for any $(S(0), I(0), H(0), R(0)) \in \partial\Delta$, there exists $T > 0$ such that*

$$\mathbb{E} \int_0^T g(S(t), I(t), H(t), R(t)) dt \geq \frac{3\lambda}{4} T. \quad (23)$$

Proof. If $I(0) = 0$, then $I(t) = 0$ for all $t \geq 0$ and model (2) becomes

$$\begin{cases} dS = (A - \mu S) dt, \\ dI = 0, \\ dH = -(m + \mu + \mu_2) H dt, \\ dR = (mH - \mu R) dt. \end{cases} \quad (24)$$

Obviously, for any $(S(0), I(0), H(0), R(0)) \in \partial\Delta$, $(S(t), I(t), H(t), R(t))$ tends asymptotically to the disease free equilibrium $(\frac{A}{\mu}, 0, 0, 0)$. Therefore,

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t g(S(s), I(s), H(s), R(s)) ds = g\left(\frac{A}{\mu}, 0, 0, 0\right) = \lambda.$$

Then there exists a $T > 0$ such that model (23) holds.

The following theorem is about the persistence of the disease.

Theorem 3.4. *If $R_0^s > 1$, then for any initial value $(S(0), I(0), H(0), R(0)) \in \Delta$, the disease is strongly stochastically permanent, namely, for any $\varepsilon > 0$, there exists a $\alpha > 0$ such that*

$$\liminf_{t \rightarrow \infty} \mathbb{P}\{I(t) \geq \alpha\} > 1 - \varepsilon. \quad (25)$$

Proof. Notice that $R_0^s > 1$ implies that $\lambda > 0$. Consider the Lyapunov function $V_\theta = I^\theta$, where $\theta \in \mathbb{R}$ is a constant. Applying the Itô's formula, we have

$$LV_\theta = \theta[\beta f(N)S - (\gamma + \delta + \mu + \mu_1) + \frac{\theta - 1}{2} \sigma^2 f^2(N)S^2] I^\theta. \quad (26)$$

Denoting $Q_\theta = \sup_{(S,I,H,R) \in \Delta} \{\theta[\beta f(N)S - (\gamma + \delta + \mu + \mu_1) + \frac{\theta - 1}{2} \sigma^2 f^2(N)S^2]\}$, then we have from model (26) that $LV_\theta \leq Q_\theta I^\theta$ for any initial value $(S(0), I(0), H(0), R(0)) \in \Delta$. By integrating both sides of model (26) from 0 to t and taking expectation yields

$$\mathbb{E}(I^\theta(t)) = \mathbb{E}(I^\theta(0)) + \mathbb{E} \int_0^t LV_\theta ds \leq \mathbb{E}(I^\theta(0)) + \int_0^t Q_\theta \mathbb{E}(I^\theta(s)) ds.$$

Using Gronwall inequality, for any $t \geq 0$ and $(S(0), I(0), H(0), R(0)) \in \Delta$, we have

$$\mathbb{E}(I^\theta(t)) \leq I^\theta(0) \exp(Q_\theta t). \quad (27)$$

Similarly, we have for $t \geq nT$,

$$\mathbb{E}(I^\theta(t)) \leq \mathbb{E}(I^\theta(nT)) \exp[Q_\theta(t - nT)]. \quad (28)$$

Denote $\ln I(0) - \ln I(t) = W(t)$. It then follows from model (22) that

$$W(t) = - \int_0^t g(\Phi(u)) du - \int_0^t \sigma f(N)S(u) dB(u). \quad (29)$$

By Feller property and Lemma 3.3, it then follows from model (29) that there exists a sufficiently small $\delta_3 > 0$ such that for any $(S(0), I(0), H(0), R(0)) \in \Delta$ with $I(0) < \delta_3$, we have

$$\mathbb{E}(W(T)) = -\mathbb{E} \int_0^T g(\Phi(t)) dt \leq -\frac{\lambda}{2} T. \quad (30)$$

Notice also from model (27) that for any fixed $t \geq 0$,

$$\mathbb{E}(e^{W(t)} + e^{-W(t)}) = \mathbb{E}\left(\frac{I(0)}{I(t)} + \frac{I(t)}{I(0)}\right) \leq \mathbb{E}(e^{Q_\theta t} + e^{-Q_\theta t}) < \infty. \quad (31)$$

Using Lemma A.1 in Appendix, we obtain

$$\ln \mathbb{E}(e^{\theta W(T)}) \leq \mathbb{E}(\theta W(T)) + \hat{Q}_1 \theta^2, \quad \theta \in [0, 0.5],$$

where \hat{Q}_1 is a constant which depends on T , Q_{-1} and Q_1 . For a sufficiently small θ satisfying $\hat{Q}_1 \theta^2 \leq \frac{\lambda \theta}{4} T$, by model (30), we have

$$\mathbb{E}\left(\frac{I^\theta(0)}{I^\theta(T)}\right) = \mathbb{E}(e^{\theta W(T)}) \leq \exp\left(-\frac{\lambda \theta}{2} T + \hat{Q}_1 \theta^2\right) \leq \exp\left(-\frac{\lambda \theta}{4} T\right).$$

Then

$$\mathbb{E}(I^{-\theta}(T)) \leq I^{-\theta}(0) \exp\left(-\frac{\lambda \theta}{4} T\right) = q I^{-\theta}(0) \quad (32)$$

for $I(0) < \delta_3$, where $q = \exp(-\frac{\lambda \theta}{4} T)$.

Next, by model (27), we obtain that

$$\mathbb{E}(I^{-\theta}(T)) \leq \delta_3^{-\theta} \exp(Q_{-\theta} T) := C \quad (33)$$

for any $I(0) > \delta_3$. Then model (33) together with model (32) implies that

$$\mathbb{E}(I^{-\theta}(T)) \leq q I^{-\theta}(0) + C$$

for any $(S(0), I(0), H(0), R(0)) \in \Delta$. By the Markov property, we have

$$\mathbb{E}[I^{-\theta}((k+1)T)] \leq q \mathbb{E}(I^{-\theta}(kT)) + C.$$

Using this recursively, we obtain

$$\mathbb{E}[I^{-\theta}((k+2)T)] \leq q^2 \mathbb{E}(I^{-\theta}(kT)) + qC + C$$

and

$$\mathbb{E}(I^{-\theta}(nT)) \leq q^n I^{-\theta}(0) + \frac{C(1-q^n)}{1-q}.$$

This together with model (28) leads to

$$\mathbb{E}(I^{-\theta}(t)) \leq \left(q^n I^{-\theta}(0) + \frac{C(1-q^n)}{1-q}\right) \exp(Q_{-\theta} T), \quad t \in [nT, (n+1)T].$$

Letting $n \rightarrow \infty$, we obtain

$$\limsup_{t \rightarrow \infty} \mathbb{E}(I^{-\theta}(t)) \leq \frac{C}{1-q} \exp(Q_{-\theta} T) := Z.$$

For any $\varepsilon > 0$, let $\alpha = \frac{\varepsilon^{\frac{1}{\theta}}}{Z^{\frac{1}{\theta}}}$. By Chebyshev's inequality we obtain

$$\mathbb{P}\{|I(t)| < \alpha\} = \mathbb{P}\left\{\frac{1}{|I(t)|^\theta} > \frac{1}{\alpha^\theta}\right\} \leq \alpha^\theta \mathbb{E}(|I^{-\theta}(t)|).$$

That is,

$$\limsup_{t \rightarrow \infty} \mathbb{P}\{|I(t)| < \alpha\} \leq \alpha^\theta Z = \varepsilon.$$

Therefore

$$\liminf_{t \rightarrow \infty} \mathbb{P}\{I(t) \geq \alpha\} > 1 - \varepsilon.$$

The proof is thus completed.

Table 1. Parameter values used in the simulation.

Parameters	Description	Values	Sources
A	The recruitment rate	1319.2940	[23]
μ	The natural death rate	0.000042578	[23]
γ	The natural recovery rate	0.0185	[11]
m	The recovery rate of confirmed individuals	0.0667	[11]
δ	The confirmed rate	0.1836	[11]
μ_1	The disease-induced death rate of infected individuals	0.0044	[23]
μ_2	The disease-induced death rate of confirmed cases	0.0044	[23]
R_0^s	The basic reproduction number of COVID-19	1.9900	[23]

Remark 1. In this section, we obtain the threshold condition R_0^s which completely determines the extinction and persistence of disease. By contrast, in [40], besides the conditions required on basic reproduction number R_0 , there is one additional condition $\mu > \frac{\sigma_1^2 \vee \sigma_2^2 \vee \sigma_3^2 \vee \sigma_4^2}{2}$ to make the disease extinct or persistent. Thus, our results can be regard as an significant extension of COVID-19 and could provide government effective information to the control of of disease transmission.

4. Numerical results

In this section, model (2) with the incidence function $f(N) = \frac{1}{1+bN+\sqrt{1+2bN}}$ [12] is applied to verify/extend our analytical results based on the realistic parameter values of COVID-19 from Asamoah [23] and Jiao [11]. We fix the initial values $(S(0), I(0), H(0), R(0)) = (50000, 5, 2, 1)$ except for other specification. We divide our simulations into the following two subsections.

4.1. The effect of β , σ and δ on the dynamics of model (2)

In the subsection, we numerically simulate the solution of model (2) using MATLAB R2016b to illustrate the theoretical results obtained in Section 3, mainly revealing the effect of σ , β and δ on the dynamics of model (2). The numerical scheme is obtained through Milstein's higher order method [41].

Case 1. The effect of noise intensity

We first suppose $\beta = 0.1860$ and $b = 0.3900$ and other parameter values are shown in Table 1. In this case one can get that $R_0 = 2.3081$. It then follows from [11] that deterministic model (1) has a unique endemic equilibrium $E^*(12824304.8094, 3743.8322, 9661.8313, 16762296.1178)$, which is stable. It follows from $R_0^s = 1.9900$ that we have $\sigma = 0.1000$. By Theorem 3.4, we can get that the disease is strongly stochastically permanent. The computer simulations shown in Figure 1(a) clearly support the result. To show how the noise affects the dynamics of disease, now we take $\sigma = 0.0100$, and other parameters remain unchanged. In this case, we obtain $R_0^s = 2.3045 > 1$. The similar simulation result is shown in Figure 1(b). We observed that the path of $I(t)$ for model (2) is oscillating around the steady state value $I^* = 3743.8322$. Compared with Figure 1(a), one can get that when $R_0^s > 1$, the small noise does not change the stability of the equilibrium state of model (2), but with the intensity of white noise increasing, the volatility of $I(t)$ is getting larger. Finally, we consider $\sigma = 0.3000$. It is easy to compute

that $R_0^s = 0.9494 < 1$, according to Theorem 3.2, the disease will go extinct almost surely as shown in Figure 1(c). However, deterministic model (1) claims the persistence of the disease. This discrepancy highlights the impact of stochastic environmental to the disease dynamics.

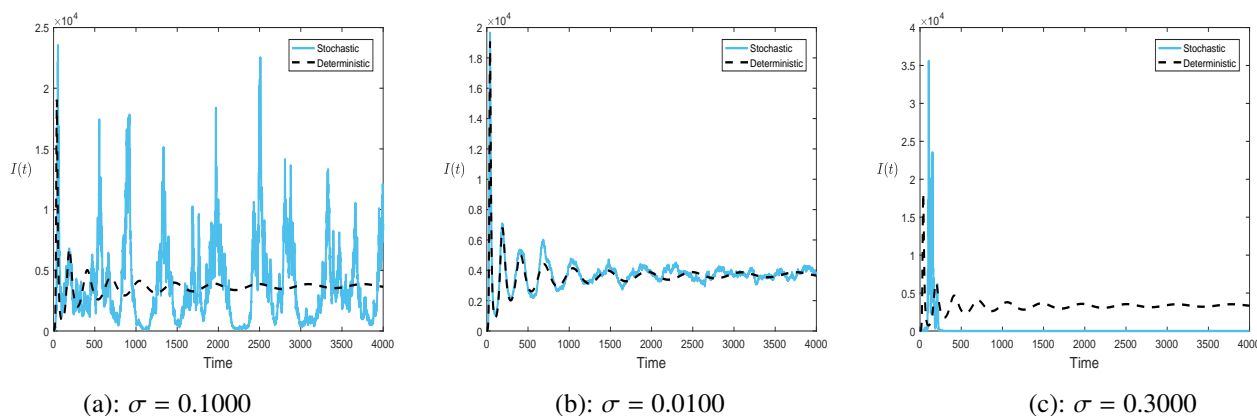


Figure 1. The evolution of a single path of $I(t)$ of models (1) and (2) is graphed for different values of σ (0.1000, 0.0100, 0.3000). Here we take $\beta = 0.1860$, $b = 0.3900$ and other parameter values are tabulated in Table 1 (Color figure online).

Case 2. The effect of transmission rate

Here we assume $b = 0.3900$, $\sigma = 0.1000$, and other parameters take values as in Table 1. In this case we consider three different values of $\beta = 0.0500, 0.1860, 0.3500$ to see the effect of transmission rate on the spread of infectious disease. The corresponding values of R_0^s are respectively 0.5353, 1.9900 and 3.7473. By Theorems 3.2 and 3.4, we can get that the disease is strongly stochastically permanent when $\beta = 0.1860$ and $\beta = 0.3500$, while the disease is extinctive when $\beta = 0.0500$. The computer simulations are shown in Figure 2(a) which clearly support these results. Figure 2(b) shows the corresponding persistence level of $I(t)$ for various values of β . It is observed that when $R_0^s > 1$, the persistence level of $I(t)$ is reduced gradually with the decrease of transmission rate. This indicates that decreasing transmission rate is beneficial to the control of the spread of COVID-19. So we can take some measures to reduce the scale of outbreaks by decreasing transmission rate. For example, transmission rate can be reduced through improving the media response rate to reports on the severity of COVID-19 and encouraging citizens to actively prevent disease. Moreover, government can adopt a series of policies including wearing masks, avoiding farm and wild animals, travel restrictions, stay at home, lockdowns, and so on to decline transmission rate. These measures could effectively reduce the number of infected cases and suppress the outbreak of disease.

Case 3. The effect of confirmed rate

Here we assume $\beta = 0.1860$, $b = 0.3900$, $\sigma = 0.1000$, and other parameters taking values as in Table 1. In this case we choose three different values of $\delta = 0.1000, 0.1836, 0.6000$ to see the effect of confirmed rate on the spread of infectious diseases. The corresponding values of R_0^s are respectively 3.0601, 1.9900, 0.7270. According to Theorems 3.2 and 3.4, the disease persists when $\delta = 0.1000$ and $\delta = 0.1836$, while the disease will be extinct when $\delta = 0.6000$. Figure 3(a) clearly support these results. Figure 3(b) shows that the corresponding persistence level of $I(t)$ for various values of δ . We can see that when $R_0^s > 1$, the persistence level of $I(t)$ is reduced gradually with the increase of confirmed rate. This indicates that the increase of confirmed rate is beneficial to control the spread of COVID-19, while

blind testing is not desirable, it will cause a huge burden on society. Therefore, in order to enhance confirmation rate, a positive tracking and testing strategy should be carried out to control the spread of disease [8]. Frequently testing in smaller scale populations, such as schools, factories, community, etc., where virus is more easier to spread, and test less frequently parts of the population who are not as exposed. Detecting continually close contacts also leads to the increase of confirmed cases.

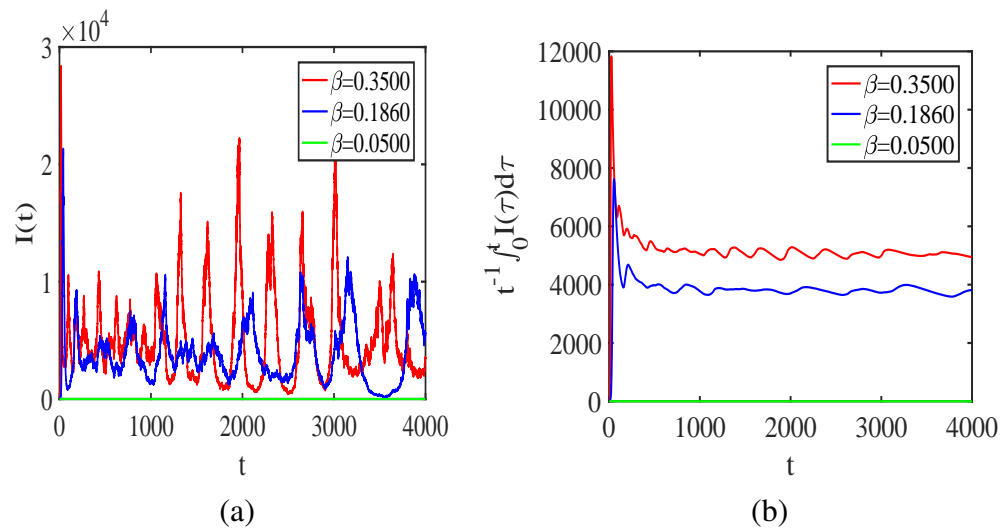


Figure 2. (a) The evolution of a single path of $I(t)$ of stochastic model (2) is graphed for different values of β (0.3500, 0.1860, 0.0500). (b) The corresponding persistence level of infected individuals of model (2) is graphed for various values of β . Here we take $\sigma = 0.1000$, $b = 0.3900$ and other parameter values are shown in Table 1 (Color figure online).

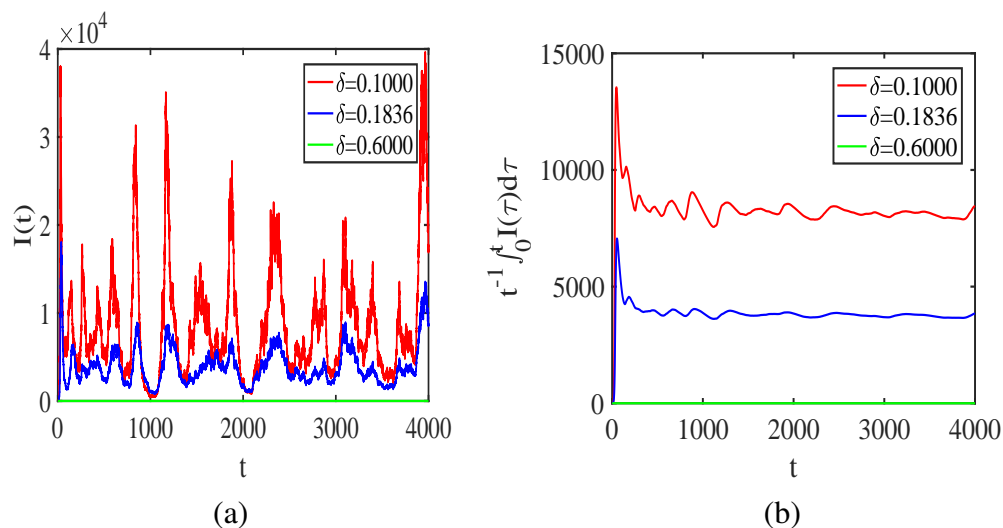


Figure 3. (a) The evolution of a single path of $I(t)$ of stochastic model (2) is graphed for different values of δ (0.1000, 0.1836, 0.6000). (b) The corresponding persistence level of infected individuals of model (2) is graphed for various values of δ . Here we take $\beta = 0.1860$, $\sigma = 0.1000$, $b = 0.3900$ and other parameter values are given in Table 1 (Color figure online).

Table 2. Normalized sensitivity index for some parameters for R_0^s .

Parameters	Description	Sensitivity Index
A	The recruitment rate	+0.0001477
μ	The natural death rate	-0.0003257
γ	The natural recovery rate	-0.0773
β	The transmission rate	+1.0000
δ	The confirmed rate	-0.7675
σ	The noise intensity	-0.2746
μ_1	The disease-induced death rate of infected individuals	-0.0184

4.2. Sensitivity analysis

Varying parameter values will have different effects on the output of model (2). In order to qualitatively analyze the influence of some parameters on the output of model (2), the sensitivity analysis method is adopted. The normalized forward sensitivity index of a variable, \mathcal{R} , that depends on a parameter, h , is defined as:

$$\Upsilon_h^{\mathcal{R}} = \frac{\partial \mathcal{R}}{\partial h} \times \left| \frac{h}{\mathcal{R}} \right|.$$

Using the above formula, we analyze the sensitivity of state variable R_0^s to the following parameters of model (2):

$$\begin{aligned} A &= 1319.2940, \beta = 0.1860, \delta = 0.1836, \sigma = 0.1000 \\ \mu &= 0.000042578, \mu_1 = 0.0044, \gamma = 0.0185. \end{aligned} \quad (34)$$

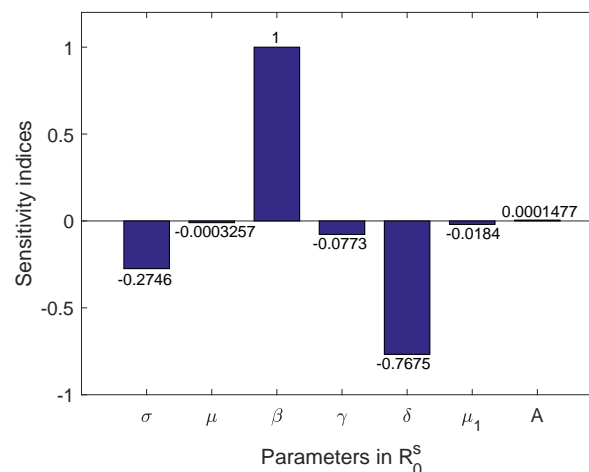


Figure 4. The sensitivity indices of state variable R_0^s with respect to some parameters for model (2).

From Table 2, parameters with positive sensitivity index, A and β , indicate that the transmission of COVID-19 increases with the increase of these two parameters. Similarly, parameters with negative

sensitivity index, μ , γ , δ , σ and μ_1 , mean that the transmission of COVID-19 decreases with the increase of these parameters. As shown in Figure 4, we observe that β , σ and δ have significant effects on R_0^s . This verifies that our analysis of parameters is meaningful in Subsection 4.1.

5. Conclusions

In reality, there exist many random environmental factors, like weather, relative humidity, temperature and population mobility, which may have significant effects on the transmission of COVID-19. Therefore, considering stochastic influences into the epidemic model seems necessary and important. In this paper, we propose and investigate a stochastic SIHR epidemic model with the environmental variability in the transmission rate to describe the transmission of COVID-19, and based on which we numerically illustrate the evolution dynamic of COVID-19 using the realistic parameter values from literatures.

The main contribution of our paper can be summarized as the following two aspects. Mathematically, we prove that the stochastic dynamics of stochastic model (2) is completely determined by the reproduction number R_0^s : If $R_0^s < 1$, the disease will go to extinction ultimately, and if $R_0^s > 1$, the disease stochastically permanent. Epidemiologically, we can conclude that: (i) The presence of environmental noises can sustain the irregular recurrence of disease and the volatility of infected population increases with the increasing noise intensity if $R_0^s > 1$. When the noise increases to a certain level such that $R_0^s < 1$, the disease will go to extinction (See Figure 1). And also it can be seen from Figure 1(c) that white noise may reshape the solution behavior of corresponding deterministic model (1). In other words, noise may change the evolution tendency of disease. (ii) The decrease of transmission rate and the increase of confirmed rate are beneficial to the control of COVID-19 spread (See Figures 2 and 3). (iii) Our sensitivity analysis indicates that the transmission rate β , noise intensity σ and confirmed rate δ are the most sensitive parameters to R_0^s (See Figure 4).

More than two years have lasted since the emergence of COVID-19 in the world. It is well known the transmission of disease will necessarily be affected by other factors such as media coverage, seasonal changes and so on [42–47]. Considering the seasonal effect or the switching of environments in model (2) will be an interesting research topic. We leave this for our future investigation.

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Conflict of interest

The authors declare there is no conflict of interest.

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Appendix

The following lemma is from [33], which is used in the establishment of conditions for the persistence of disease.

Lemma A.1. *Let Y be a random variable, suppose $\mathbb{E} \exp(Y) + \mathbb{E} \exp(-Y) \leq K_1$. Then the log-Laplace transform $\phi(\theta) = \ln \mathbb{E} \exp(\theta Y)$ is twice differentiable on $[0, 0.5]$ and $\frac{d\phi}{d\theta}(0) = \mathbb{E}Y$, $0 \leq \frac{d^2\phi}{d\theta^2}(\theta) \leq 2K_2$, $\theta \in [0, 0.5]$ for some $K_2 > 0$ depending only on K_1 . Thus, it follows from Taylor's expansion that*

$$\phi(\theta) \leq \theta \mathbb{E}Y + K_2 \theta^2, \quad \theta \in [0, 0.5].$$



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