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The threshold of a deterministic and a stochastic SIQS epidemic model with varying total population size

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ABSTRACT

In this paper, a stochastic and a deterministic SIS epidemic model with isolation and varying total population size are proposed. For the deterministic model, we establish the threshold $R_0$. When $R_0$ is less than 1, the disease-free equilibrium is globally stable, which means the disease will die out. While $R_0$ is greater than 1, the endemic equilibrium is globally stable, which implies that the disease will spread. Moreover, there is a critical isolation rate $\delta^*$, when the isolation rate is greater than it, the disease will be eliminated. For the stochastic model, we also present its threshold $R_{0i}$. When $R_{0i}$ is less than 1, the disease will disappear with probability one. While $R_{0i}$ is greater than 1, the disease will persist. We find that stochastic perturbation of the transmission rate (or the valid contact coefficient) can help to reduce the spread of the disease. That is, compared with stochastic model, the deterministic epidemic model overestimates the spread capacity of disease. We further find that there exists a critical the stochastic perturbation intensity of the transmission rate $\sigma^*$, when the stochastic perturbation intensity of the transmission rate is bigger than it, the disease will disappear. At last, we apply our theories to a realistic disease, pneumococcus amongst homosexuals, carry out numerical simulations and obtain the empirical probability density under different parameter values. The critical isolation rate $\delta^*$ is presented. When the isolation rate $\delta$ is greater than $\delta^*$, the pneumococcus amongst will be eliminated.

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1. Introduction

Isolation (quarantine) is one of the important intervention measures to control the spread of infectious diseases. Over the decades, isolation has been used to reduce the transmission of numerous emerging and re-emerging human diseases such as pandemic influenza, smallpox, leprosy, tuberculosis, plague, cholera, measles, typhus, yellow fever, ebola. More recently, by virtue of effective isolation, the Chinese government had eliminated successfully SARS (Severe Acute Respiratory Syndrome) which was reported firstly in late 2002 in Guangdong Province [1,2]. Since December 2019, an outbreak of pneumonia caused by a novel coronavirus (COVID-19) has occurred in Wuhan, Hubei Province, China. Cases have been exported to other places in China, as well as almost all countries around the world. By isolating the infected, close contacts, susceptibles at home and other measures, the Chinese government has basically controlled the spread of COVID-19. As of March 23, 2020, the National Health Commission (NHC) of China had confirmed a total of 81,093 cases of COVID-19 in mainland China, including 3,270 deaths, and 72,703 recoveries [3].

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Numerous mathematical models had been designed to study the effects of isolation (quarantine) on controlling the spread of infectious disease in human and animal populations (see [4–11] and the references therein). Hsieh et al. [12] analyzed laboratory-verified SARS case data and the detailed quarantine data in Taiwan during the outbreak. Then, by dividing the intensity of quarantine implemented in Taiwan into Level A (of potentially exposed contacts of suspected SARS patients) and Level B (of travelers arriving at borders from SARS affected areas), they established the corresponding model. Their research results showed that Level A quarantine prevented approximately 461 additional SARS cases and 62 additional deaths, while the effect of Level B quarantine was comparatively minor. Hethcote et al. [11] proposed three SIS and SIR epidemic models with isolation measures and three different incidence functions (standard incidence, mass action, and quarantine-adjusted incidence), respectively. They found that for most of these models, the endemic equilibrium is asymptotically stable, but for the SIQR model with the quarantine-adjusted incidence, the endemic equilibrium is an unstable spiral for some parameter values and periodic solutions arise by Hopf bifurcation.

Many studies showed that environmental fluctuations have a huge impact on the development of an epidemic. Due to environmental fluctuations, the parameters involved in the system show stochastic perturbation [13–16]. Stochastic differential equation (SDE) models could be a more appropriate way of modeling epidemics in many circumstances [17–19]. There are different possible approaches to include stochastic effects in the model, both from a biological and from a mathematical perspective [21]. Mao et al. [22] assumed that the parameters involved in the model fluctuate around a mean value due to continuous fluctuations of environment. This kind of modeling idea was adopted by many scholars [23–33]. In this paper, we follow this idea and introduce environmental fluctuations to an SIQS epidemic model.

For epidemic models, the key parameter is the threshold which determine extinction or persistence of the disease. Naturally, there comes an important question: How do environmental fluctuations affect the dynamics of the disease, especially the threshold? Recently, there are already some literatures focusing on this question [34–36]. For example, Cai et al. [37] extended a classical SIRS epidemic model from a deterministic model to a stochastic model through introducing stochastic perturbations and established the sufficient conditions for the extinction and persistence of the disease. Moreover, they found that the stochastic perturbation can suppress disease outbreak. Gray et al. [38] analyzed an SDE version of the classical SIS epidemic model, with noise introduced in the disease transmission term. They found that the threshold of the SDE version is less than the corresponding deterministic version. Dieu et al. [39] solved the threshold of a stochastic SIR model. However, there existed few works on the thresholds of stochastic epidemic models, except [39,40]. That is, most studies just established the sufficient conditions of the extinction and persistence of the disease, and did not give the sufficient and necessary conditions.

The main goal of this article is to establish the thresholds of a stochastic and deterministic SIQS epidemic model and investigate how the stochastic perturbation of the transmission rate of the disease affect disease’s dynamics, and further analyze the global dynamics of the stochastic and deterministic SIQS epidemic model.

The organization is arranged as follows. In Section 2, the model is derived. In Section 3, we analyze the existence and uniqueness of disease-free equilibrium and endemic equilibrium of deterministic model (2.4). Then, using Liapunov functions, we investigate their global stability. In Section 4, first, we show the existence and uniqueness of a global positive solution of the model (2.6). Next, we study the extinction of the model (2.6). Moreover, we discuss the persistence of the system (2.6). In Section 5, we provide the application of the results to realistic pneumococcus amongst homosexuals to support our findings, by numerical simulations. In Section 6, we give the summary of the main results briefly. In Section 7, we provide a brief discussion.

2. Background

Hethcote et al. [11] introduced the following SIQS model

\[
\begin{align*}
\frac{dS(t)}{dt} &= \lambda - \frac{\beta IS}{N} - \mu S + \gamma l + \varepsilon Q, \\
\frac{dl(t)}{dt} &= \frac{\beta IS}{N} - (\mu + \alpha + \delta + \gamma)l, \\
\frac{dQ(t)}{dt} &= \delta l - (\mu + \alpha + \varepsilon)Q.
\end{align*}
\]

(2.1)

The meanings of all variables and parameters in the above model are as follows:

- \(S(t)\): the number of susceptible individuals in the population at time \(t\),
- \(I(t)\): the number of infectious individuals in the population at time \(t\),
- \(Q(t)\): the number of isolated (quarantined) individuals in the population at time \(t\),
- \(N(t)\): the number of total population at time \(t\), namely \(N(t) = S(t) + I(t) + Q(t)\),
- \(\beta\): the transmission rate of the disease,
- \(b\): the natural birth rate,
- \(\mu\): the natural death rate,
- \(\delta\): the rate of individuals leaving the infective compartment \(I\) for the quarantined compartment \(Q\),
- \(\alpha\): the disease-related death rate,
\( \gamma \) and \( \varepsilon \): the rate of individuals recovering and returning to susceptible compartment \( S \) from compartments \( I \) and \( Q \), respectively.

In the model (2.1), Herbert et al. [11] assumed that the recruitment rate of susceptibles is constant. This assumption is reasonable for diseases of short duration with limited effects on mortality. However, it clearly fails to hold for diseases which result in a decrease in population or raise the mortality rate substantially (see [41-46]). Hence, in this paper, we supposed that the recruitment rate of susceptibles of the model (2.1) is proportional to the total population as in [47]. Consequently, we get the following model

\[
\begin{align*}
\frac{dS(t)}{dt} &= bN - \frac{\beta IS}{N} - \mu S + \gamma I + \varepsilon Q, \\
\frac{dI(t)}{dt} &= \frac{\beta IS}{N} - (\mu + \alpha + \delta + \gamma)I, \\
\frac{dQ(t)}{dt} &= \delta I - (\mu + \alpha + \varepsilon)Q. 
\end{align*}
\] (2.2)

It is easy to see that \( \frac{dN(t)}{dt} = (b - \mu)N - \alpha I - \alpha Q \). Let \( x = \frac{S}{N} \), \( y = \frac{I}{N} \), \( z = \frac{Q}{N} \) denote the fractions of the classes \( S \), \( I \) and \( Q \) in the population, respectively. It is easy to identify that \( x \), \( y \) and \( z \) satisfy the following equations system:

\[
\begin{align*}
\frac{dx(t)}{dt} &= b - \beta yx + \gamma x + \varepsilon z - bx + \alpha xy + \alpha xz, \\
\frac{dy(t)}{dt} &= \beta yx - (b + \alpha + \delta + \gamma) y + \alpha xy^2 + \alpha yz, \\
\frac{dz(t)}{dt} &= \delta y - (b + \alpha + \varepsilon)z + \alpha yz + \alpha z^2. 
\end{align*}
\] (2.3)

Since \( x + y + z = 1 \), the first two equations of system (2.3) can be written as

\[
\begin{align*}
\frac{dx(t)}{dt} &= (b + \varepsilon + \alpha x)(1 - x) - (\beta x - \gamma - \varepsilon) y, \\
\frac{dy(t)}{dt} &= (\beta - \alpha)xy - (b + \delta + \gamma)y. 
\end{align*}
\] (2.4)

Following the approach in Mao et al. [22], we suppose that the transmission rate of the disease will fluctuate around some average value due to continuous fluctuation in the environment as in [37,38]. In detail, \( \beta dt \) in system (2.2) which represents the number of potential infectious contacts that a single infected individual makes with another individual in the small time range \( dt \), is replaced by \( \beta dt + \sigma dB(t) \). Here \( dB(t) = B(t + dt) - B(t) \) is the increment of a standard Brownian motion. This means that the number of potential infectious contacts that a single infected individual makes with another individual in the small time range \( dt \) obeys normal distribution with mean \( \mathbb{E}(\beta dt + \sigma dB(t)) = \beta dt \) and variance \( \mathbb{V}(\beta dt + \sigma dB(t)) = \sigma^2 dt \). \( \mathbb{V}(\beta dt + \sigma dB(t)) \rightarrow 0 \) as \( dt \rightarrow 0 \), this is a biologically reasonable description (see [37] and the references therein).

Hence, we obtain the following stochastic SIQS epidemic model

\[
\begin{align*}
\frac{dS(t)}{dt} &= \left(bN - \frac{\beta IS}{N} - \mu S + \gamma I + \varepsilon Q\right)dt - \sigma \frac{SI}{N}dB(t), \\
\frac{dI(t)}{dt} &= \left(\frac{\beta IS}{N} - (\mu + \alpha + \delta + \gamma)I\right)dt + \sigma \frac{SI}{N}dB(t), \\
\frac{dQ(t)}{dt} &= \left(\delta I - (\mu + \alpha + \varepsilon)Q\right)dt, 
\end{align*}
\] (2.5)

where \( \sigma > 0 \) represents the intensity of the white noise.

Recently, some scholars have paid attention to stochastic SIQS epidemic models [48-52]. Cao and Zhou [50] discussed a stochastic SIQR epidemic model with quarantine-adjusted incidence. It should be pointed out that our research is different from Cao and Zhou [50]. First, we consider quarantine measures in the SIS epidemic model, while Cao and Zhou consider quarantine measures in the SIR epidemic model. That is to say, our model is used for the nonpermanent immune diseases, while their model is used for the permanent immune diseases. Second, some of our research methods is different from Cao and Zhou. Our methods establish the threshold of disease without additional conditions. However, they needed some additional conditions besides a threshold to obtain the extinction and the permanence of the disease. Pang et al. [52] studied a model similar to (2.5), but they used the bilinear incidence \( \beta SI \) and we use the standard incidence \( \frac{\beta IS}{N} \). The bilinear incidence \( \beta SI \) supposes that the adequate contacts is proportional to the total population, while the standard incidence \( \frac{\beta IS}{N} \) supposes that the adequate contacts is constant. Since the patterns of daily encounters are largely independent of community size within a given country, Data [53] suggested that the standard incidence is more realistic for human diseases than the bilinear incidence. For more formation about the differences of using two forms of the incidence, reader see [11] and the references therein. In addition, Pang et al. [52] presented sufficient conditions for extinction exponentially and persistence in the mean while our condition is almost necessary and sufficient.
Letting
\[ x = \frac{S}{N}, y = \frac{I}{N}, z = \frac{Q}{N}, \]
then the model (2.5) becomes
\[
\begin{align*}
  dx(t) &= (b - \beta xy + \gamma y + \varepsilon z - bx + \alpha xy + \alpha xz)dt - \sigma xydB(t), \\
  dy(t) &= (\beta y-x - (b+\alpha + \delta + \gamma)y + \alpha y^2 + \alpha yz)dt + \sigma xydB(t), \\
  dz(t) &= (\delta y - (b+\alpha + \varepsilon)z + \alpha yz + \alpha z^2)dt.
\end{align*}
\] (2.6)

Since \( x(t) + y(t) + z(t) = 1 \), system (2.6) is equivalent to
\[
\begin{align*}
  dx(t) &= ([b + \varepsilon + \alpha x](1-x) - (\beta x - \gamma + \varepsilon)y)dt - \sigma xydB(t), \\
  dy(t) &= ((\beta - \alpha)xy - (b + \delta + \gamma)y)dt + \sigma xydB(t),
\end{align*}
\] (2.7)
and
\[
x(t) + y(t) + z(t) = 1. \] (2.8)

3. The asymptotic behaviors of the deterministic system (2.4)

In this section we will study the asymptotic behaviors of system (2.4).
It is easy to confirm that the domain \( D = \{(x, y) \mid x > 0, y \geq 0, x + y \leq 1\} \) is the positively invariant set of system (2.4).
Obviously, there is always the disease-free equilibrium \( E_0(1, 0) \) for system (2.4).
To get the endemic equilibrium for system (2.4), let the right side of system (2.4) be 0, we have
\[
\begin{align*}
  (b + \varepsilon + \alpha x)(1-x) - (\beta x - \gamma + \varepsilon)y &= 0, \\
  (\beta - \alpha)x - (b + \delta + \gamma) &= 0.
\end{align*}
\] (3.1)

Solving the first equation of (3.1), we get \( x^* = \frac{b + \delta + \gamma}{\beta - \alpha}. \)
Let
\[ R_0 = \frac{\beta}{b + \delta + \alpha + \gamma}. \]
It is easy to see that if \( R_0 > 1, \)
\[ \beta - \alpha > 0 \text{ and } 0 < x^* = \frac{b + \delta + \gamma}{\beta - \alpha} < 1. \] (3.2)

From the first equation of (3.1), we obtain
\[ y^* = \frac{(b + \varepsilon + \alpha x^*)(1 - x^*)}{\beta x^* - \gamma + \varepsilon}. \]
Clearly, \((b + \varepsilon + \alpha x^*)(1 - x^*) > 0.\) In addition, by (3.2) we have
\[ \beta x^* - \gamma + \varepsilon = \beta \frac{b + \delta + \gamma}{\beta - \alpha} - \gamma + \varepsilon > \beta \frac{b + \delta + \gamma}{\beta} - \gamma + \varepsilon > 0. \] (3.3)

Hence,
\[ y^* > 0. \] (3.4)

It follows from (3.2) and (3.4) that \( E_1(x^*, y^*) \) is unique endemic equilibrium in domain \( D. \)
Now, let us discuss the local stability of the disease-free equilibrium \( E_0 \) and endemic equilibrium \( E_1. \)

3.1. The local stability of system (2.4)

At any point \( E(x, y), \) Jacobian matrix of system (2.4) is
\[ J(E) = \begin{pmatrix}
  \alpha - (b + \varepsilon + 2\alpha x) & \beta y & -(\beta x - \gamma + \varepsilon) \\
  (\beta - \alpha)y & (\beta - \alpha)x - (b + \delta + \gamma) & \beta - \alpha \\
  -b & -\beta - \gamma + \varepsilon & \beta - \alpha - (R_0 - 1)(b + \delta + \alpha + \gamma)
\end{pmatrix}. \] (3.5)

Then, Jacobian matrix of system (2.4) at the disease-free equilibrium \( E_0 \) is
\[ J(E_0) = \begin{pmatrix}
  -(b + \varepsilon + \alpha) & -(\beta - \gamma + \varepsilon) & 0 \\
  (b + \varepsilon + \alpha) & (\beta - \gamma + \varepsilon) & (R_0 - 1)(b + \delta + \alpha + \gamma)
\end{pmatrix}. \]
The two eigenvalues of \( J(E_0) \) are \(-b + \varepsilon + \alpha\) and \((R_0 - 1)(b + \delta + \alpha + \gamma).\)
Obviously, for system (2.4), the disease-free equilibrium $E_0$ is stable if $R_0 < 1$ and it is unstable if $R_0 > 1$.

At the endemic equilibrium $E_1(x^*, y^*)$, Jacobian matrix of system (2.4) is

$$
J(E_1) = \begin{pmatrix}
\alpha - (b + \varepsilon + 2\alpha x^*) - \beta y^* & -(\beta x^* - \gamma + \varepsilon) \\
(\beta - \alpha)\beta y^* & 0
\end{pmatrix}.
$$

By calculating straightforwardly, we have $\det J(E_1) = (\beta - \alpha)(b + \varepsilon + \alpha x^*) \times (1 - x^*)$. It is clear that when $R_0 > 1$, from (3.2), $\det J(E_1) > 0$ holds. In addition,

$$
\text{tr}(J(E_1)) = \alpha - (b + \varepsilon + 2\alpha x^*) - \beta y^* - \beta x^* - \gamma + \varepsilon
$$

$$
= -\frac{\varepsilon(\beta - \alpha - \gamma + b + 2\alpha x^*) + \varepsilon^2}{\beta x^* - \gamma + \varepsilon} - \frac{\alpha \gamma - b \gamma + \alpha \beta x^2 - 2\alpha x^* \gamma + \beta b}{\beta x^* - \gamma + \varepsilon}
$$

$$
= -\frac{\varepsilon \alpha y + b(\beta - \gamma)(\beta - \gamma)^2 + (\alpha \beta b^2 + \alpha \beta \delta^2 + 2b \delta \alpha \beta) + 2\alpha \gamma(\alpha b + \alpha \delta + \alpha \gamma)}{(\beta x^* - \gamma + \varepsilon)(\beta - \alpha)^2}.
$$

It follows from $R_0 > 1$ that $\beta - \alpha - \gamma > 0$, $\beta - \gamma > 0$ and $\beta x^* - \gamma + \varepsilon > 0$ (from (3.3)) hold which lead to $\text{tr}(J(E_1)) > 0$.

Hence, when $R_0 > 1$, $\det J(E_1) > 0$ and $\text{tr}(J(E_1)) > 0$. This means that the two eigenvalues of the matrix $J(E_1)$ have negative real parts. Hence, the endemic equilibrium $E_1$ is asymptotically stable if $R_0 > 1$.

Summarizing the results above, we have the following.

**Theorem 3.1.** If $R_0 = \frac{\beta}{b + \varepsilon + \alpha \gamma} < 1$, the disease-free equilibrium $E_0(1, 0)$ is asymptotically stable. If $R_0 > 1$, $E_0$ is a saddle point and unstable; the endemic equilibrium $E_1(x^*, y^*)$, is asymptotically stable.

3.2. The Global Stability of Equilibriums

Next, let us discuss the global stability of the disease-free equilibrium $E_0$ and endemic equilibrium $E_1$ of system (2.4).

**Theorem 3.2.** The disease-free equilibrium $E_0$ of system (2.4), is globally stable if $R_0 < 1$.

**Proof.** Let $V = y$, then

$$
\frac{dV(t)}{dt} = [(\beta - \alpha)x - (b + \delta + \gamma)]y
$$

$$
\leq [\beta - \alpha - (b + \delta + \gamma)]y
$$

$$
= [\beta - (b + \delta + \alpha + \gamma)]y
$$

$$
= (b + \delta + \alpha + \gamma)(R_0 - 1)y
$$

$$
\leq 0.
$$

Obviously, $\{(x, y) \mid \frac{dV(t)}{dt} = 0, (x, y) \in D\} = \{(x, y) \mid y = 0, (x, y) \in D\}$ is the largest positively invariant subset of system (2.4) in $D$. By the Lasalle invariant theorem, we have $\lim_{t \to \infty} y(t) = 0$. Then, the limit system of system (2.4) is given by $\frac{dx(t)}{dt} = (b + \varepsilon + \alpha x)(1 - x)$, and is globally asymptotically stable at $x = 1$. By the limiting systems theorem in [54], the disease-free equilibrium $E_0(1, 0)$ of system (2.4) is globally attractive. This combining with the local asymptotic stability of $E_0$ leads to $E_0$ is globally asymptotic stable. \)

**Theorem 3.3.** The endemic equilibrium $E_1(x^*, y^*)$ of system (2.4) is globally stable if $R_0 = \frac{\beta}{b + \varepsilon + \alpha \gamma} > 1$.

**Proof.** It is easy to see that system (2.4) has an endemic equilibrium $E_1(x^*, y^*)$, if and only if system (2.3) has an endemic equilibrium $E_1(x^*, y^*, 1 - x^* - y^*)$. The stability of $E_1(x^*, y^*)$ for system (2.4) is equivalent to that of $E_1(x^*, y^*, 1 - x^* - y^*)$ for system (2.3). Hence, we need prove the global stability of $E_1(x^*, y^*, 1 - x^* - y^*)$ for system (2.3) on the invariant set $D_1 = \{(x, y, z) \mid x \geq 0, y \geq 0, z \geq 0, x + y + z = 1\}$. Since the solution of system (2.3) are bounded, and the equilibrium $E_1(x^*, y^*)$ of system (2.4) is locally asymptotically stable, it is only necessary to prove that system (2.3) has no periodic solution in the invariant domain $D_1$.

It is easy to see that the boundary curve of the domain $D_1$ cannot form the periodic solution of system (2.3). Hence, we only consider in the interior of $D_1$.

Assuming that system (2.3) has a periodic solution $\varphi(t) = \{x(t), y(t), z(t)\}$, the trajectory $\Gamma$ of $\varphi(t)$ is the boundary of a plane domain $\Lambda$ which is in the interior of domain $D_1$.

Let $f_1 = b - \beta xy + \gamma y + \varepsilon z - bx + \alpha xy + \alpha xz$, $f_2 = (\beta - \alpha)xy - (b + \delta + \gamma)z$, $f_3 = \delta y - (b + \alpha + \varepsilon)z + \alpha yz + \alpha z^2$ which are the formulas of the right-hand side in system (2.3), respectively. Let $f = (f_1, f_2, f_3)^T$ (T denotes transpose), $g(x, y, z) = \frac{1}{xyz} \cdot r \times f$, where $r = (x, y, z)^T$.

Obviously,

$$
g \cdot f = 0.
$$
Calculating leads to
\[
\text{rot } g \cdot (1, 1, 1)^T = -\frac{\delta (x^2y + xy^2 + xyz) + y (xyz + y^2z + yz^2) + \varepsilon (xz^2 + yz^2 + z^3) + b(yz + z^2)}{y^2x^2z^2} < 0
\]
(3.7)
in the interior of domain \(D_1\).

Let vector \((1, 1, 1)\) be the normal vector of plane domain direction of plane domain \(\Lambda\). Let the direction of the image \(\Gamma^*\) accord with the right-hand rule with the direction of plane domain \(\Lambda\). By Stokers’ theorem, we get
\[
\frac{1}{\sqrt{3}} \int \int_\Lambda \text{rot} g \cdot (1, 1, 1)^T dS = \int_\Gamma \frac{g \cdot F}{|F|} ds,
\]
which leads to a contradiction between (3.6) and (3.7).

This completes the proof of Theorem 3.3. \(\square\)

4. The asymptotic behaviors of the stochastic system (2.5)

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

In general, the \(d\)-dimensional stochastic system:
\[
dX(t) = f(X(t), t)dt + g(X(t), t)dB_t,
\]
(4.1)
where \(f(X, t)\) is a function in \(\mathbb{R}^d\) defined in \(\mathbb{R}^d \times [t_0, \infty)\), and \(g(X, t)\) is a \(d \times m\) matrix, \(f, g\) are locally Lipschitz functions in \(X, B_t\) is an \(m\)-dimensional standard Wiener process defined on the above probability space. Denote by \(C^{2,1}(\mathbb{R}^d \times [t_0, \infty); \mathbb{R}_+^d)\) the family of all nonnegative functions \(V(X, t)\) defined on \(\mathbb{R}^d \times [t_0, \infty)\) such that they are continuously twice differentiable in \(X\) and once in \(t\). The differential operator \(L\) of Eq. (4.1) is defined [55] by
\[
L = \frac{\partial}{\partial t} + \sum_{i=1}^{d} f_i(X, t) \frac{\partial}{\partial X_i} + \frac{1}{2} \sum_{i,j=1}^{d} \left[ g_i^T(X, t)g_j(X, t) \right]_{ij} \frac{\partial^2}{\partial X_i \partial X_j}.
\]
(4.2)
If \(L\) acts on a function \(V \in C^{2,1}(\mathbb{R}^d \times [t_0, \infty); \mathbb{R}_+^d)\), then
\[
LV(X, t) = V_t(X, t) + V_X(X, t)f(X, t) + \frac{1}{2} \text{trace}[g^T(X, t)V_{XX}(X, t)],
\]
where \(V_t(X, t) = \frac{\partial V}{\partial t}\), \(V_X(X, t) = \left( \frac{\partial V}{\partial X_1}, \ldots, \frac{\partial V}{\partial X_d} \right)\), \(V_{XX} = \left( \frac{\partial^2 V}{\partial X_i \partial X_j} \right)_{d \times d}\). By Itô’s formula, if \(X(t) \in \mathbb{R}^d\), then \(dV(X, t) = LV(X, t)dt + V_X(X, t)g(X, t)dB_t\).

4.1. Existence and uniqueness of the positive solution

In this section, we show there is a unique global and positive solution of model (2.6). That is, the region
\[
\mathcal{A} = \{(x, y, z) : x > 0, y > 0, z > 0, x + y + z = 1\}
\]
is the positively invariant set of the system (2.6) with probability 1. Some researchers have given the similar result [34,37].

**Theorem 4.1.** For any given initial value \((x(0), y(0), z(0)) \in \mathcal{A}\), there is a unique positive solution \((x(t), y(t), z(t))\) of model (2.6) on \(t \geq 0\) and the solution will remain in \(\mathcal{A}\) with probability 1, namely \((x(t), y(t), z(t)) \in \mathcal{A}\) for \(t \geq 0\) almost surely.

**Proof.** Since the coefficients of model are locally Lipschitz continuous, for any given initial value \((x(0), y(0), z(0)) \in \mathbb{R}_+^3\), there is a unique local solution on \(t \in [0, \tau_e]\) where \(\tau_e\) is the explosion time. To show that this solution is global, we need to have \(\tau_e = \infty\) almost surely (briefly a.s.). Let \(k_0 > 1\) be sufficiently large so that \((x(t), y(t), z(t))\) all lie in the interval \(\left[\frac{1}{k_0}, 1\right]\). For each integer \(k \geq k_0\), define the stopping time \(\tau_k\) by
\[
\tau_k = \inf \left\{ t \in [0, \tau_e) : \min(x(t), y(t), z(t)) \leq \frac{1}{k} \right\}
\]
where throughout this paper we set \(\inf \phi = \infty\) (as usual \(\phi\) denotes the empty set). According to the definition, \(\tau_k\) is increasing as \(k \to \infty\). Denote \(\tau_e = \lim_{k \to \infty} \tau_k\), hence \(\tau_e \leq \tau_P\) a.s. If we can show that \(\tau_e = \infty\) a.s., then \(\tau_e = \infty\) and \((x(t), y(t), z(t)) \in \mathbb{R}_+^3\) a.s. for all \(t \geq 0\). In other words, to complete the proof all we need to show is \(\tau_e = \infty\) a.s. For if this statement is false, then there exists a pair of constants \(T > 0\) and \(\epsilon \in (0, 1)\) such that
\[
\mathbb{P}(\tau_e \leq T) > \epsilon.
\]
Hence there is an integer $k_1 \geq k_0$ such that
\[ \mathbb{P}(\tau_k \leq T) \geq \epsilon \text{ for all } k \geq k_1. \]

Define a $C^2$-function $V : \mathbb{R}^3_+ \to \mathbb{R}$ by $V(x, y, z) = \ln xyz$. Applying Itô’s formula to system (2.6), for $\omega \in \{ \tau^\omega \leq T \}$ and $t \in [0, \tau^\omega)$, we obtain
\[ dV(x, y, z) = LVdt + \sigma ydB(t) - \sigma xdB(t), \]
where
\[
LV = \frac{b}{x} - \beta y + \frac{\gamma y}{x} + \frac{\varepsilon z}{x} - b + \alpha y + \alpha z - \frac{1}{2}\sigma^2 y^2
\]
\[ + \beta x - (b + \alpha + \delta + \gamma) + \alpha y + \alpha z - \frac{1}{2}\sigma^2 x^2 + \frac{\delta y}{x} - (b + \alpha + \epsilon) + \alpha y + \alpha z \]
\[ \geq -\beta y - b + \alpha y + \alpha z - \frac{1}{2}\sigma^2 y^2 + \beta x - (b + \alpha + \delta + \gamma) + \alpha y + \alpha z - \frac{1}{2}\sigma^2 x^2 + \frac{\delta y}{x} - (b + \alpha + \epsilon) + \alpha y + \alpha z \]
\[ \triangleq F(x, y, z). \]

By virtue of $x + y + z = 1$ and continuity of $F(x, y, z)$, there exists a constant $M < 0$ such that $F(x, y, z) \geq M$ for $(x, y, z) \in A$. Hence, for any $k \geq k_1$, we have
\[
\mathbb{E}[V(x(T \wedge \tau_k), y(T \wedge \tau_k), z(T \wedge \tau_k))] - V(x(0), y(0), z(0))
\geq \mathbb{E} \int_0^{T \wedge \tau_k} LV(x(u), y(u), z(u))du \geq MT > -\infty.
\]

Set $\Omega_k = \{ \tau_k \leq T \}$ for $k \geq k_1$. Let $I_{\Omega_k}$ be the indicator of $\Omega_k$, we have $\mathbb{P}(\Omega_k) \geq \epsilon$.

On the other hand, noting that $x + y + z = 1$, we have
\[
\mathbb{E}[V(x(T \wedge \tau_k), y(T \wedge \tau_k), z(T \wedge \tau_k))] \leq \mathbb{E} [\ln x(T \wedge \tau_k)]
\leq \mathbb{E} [I_{\Omega_k} \ln x(\tau_k, \omega)]
\leq \epsilon \ln \frac{1}{R_0}.
\]

Letting $k \to \infty$, (4.3) and (4.4) yield the contradiction $-\infty > MT > -\infty$. Therefore we obtain $\tau_* = \infty$ a.s. This completes the proof of Theorem 3.1. $\square$

4.2 Extinction

For an infectious disease, we are concerned about the threshold of the spread and disappearance for the infectious disease. In this section, we will establish the threshold of stochastic system (2.7) denoted as $R_{0s}$ and prove that when $R_{0s} < 1$, the disease will be extinct. In the next section, we will investigate that when $R_{0s} > 1$ the disease will be persistent.

Before proof, we give a remark.

Remark 1. Theorem 4.1 shows that $A = \{ (x, y, z) : x > 0, y > 0, z > 0, x + y + z = 1 \}$ is the positively invariant set of system (2.6). Moreover, from $x(t) + y(t) + z(t) = 1$, we have that $D = \{ (x, y) : x > 0, y > 0, x + y \leq 1 \}$ is the positively invariant set of system (2.7).

Theorem 4.2. Let $(x(t), y(t))$ be any solution of model (2.7) with initial value $(x(0), y(0)) \in D$. If $R_{0s} = \frac{b}{b + \alpha + \gamma} - \frac{\sigma^2}{2(b + \alpha + \gamma)} = R_0 - \frac{\sigma^2}{2(b + \alpha + \gamma)} < 1$ where $R_0$ is the threshold of the deterministic system (2.4), then $(x(t), y(t)) \to (1, 0)$ a.s. (almost surely) as $t \to \infty$. Namely, the disease will be extinct. Moreover,
\[
\mathbb{P} \left\{ \lim_{t \to \infty} \frac{\ln x(t)}{t} = (b + \alpha + \delta + \gamma)(R_{0s} - 1) \right\} = 1, \text{ for } X_0 \in D.
\]

That is, $l(t)$ tends to zero exponentially almost surely.

Proof. For simplicity, let
\[
\Phi(x) = (\beta - \alpha)x - (b + \delta + \gamma) - \frac{\sigma^2 x^2}{2}.
\]

Since $R_{0s} < 1$, we choose sufficiently small $\zeta > 0$ such that $R_{0s} - 1 + \frac{\zeta}{b + \delta + \alpha + \gamma} < 0$. Hence,
\[
\Phi(1) + \zeta = (b + \delta + \alpha + \gamma)(R_{0s} - 1 + \frac{\zeta}{b + \delta + \alpha + \gamma}) < 0.
\]

Consider the Lyapunov function $\Psi(x, y) = \frac{1}{\zeta} (1 - x)^2 + y^\theta$, where $\theta \in (0, 1)$ is a constant to be determined.

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Applying Itô’s formula, we have
\[
L\Psi(x, y) = -(1 - x)((b + \varepsilon + \alpha x)(1 - x) - (\beta x - \gamma + \varepsilon y) + \frac{1}{2}\sigma^2 x^2 y^2 + \theta y^{\lambda - 1}((\beta - \alpha)x y - (b + \delta + \gamma)y)
+ \frac{1}{2}\theta(\theta - 1)y^{\lambda - 2}\sigma^2 x^2 y^2
= -(1 - x)((b + \varepsilon + \alpha x)(1 - x) - (\beta x - \gamma + \varepsilon y) + \frac{1}{2}\sigma^2 x^2 y^2
+ \theta y^{\lambda}((\beta - \alpha)x - (b + \delta + \gamma)) + \frac{1}{2}\theta^2 y^{\lambda}\sigma^2 x^2
= -(b + \varepsilon + \alpha x)(1 - x)^2 + (1 - x)(\beta x - \gamma + \varepsilon y) + \frac{1}{2}\sigma^2 x^2 y^2 + \theta y^{\lambda} \Phi(x) + \frac{1}{2}\theta^2 y^{\lambda}\sigma^2 x^2
\leq -(b + \varepsilon + \alpha x)(1 - x)^2 + (1 - x)(\beta x - \gamma + \varepsilon y) + \frac{1}{2}\sigma^2 x^2 y^2 + \theta y^{\lambda} \Phi(x) + \frac{1}{2}\theta^2 y^{\lambda}\sigma^2 x^2. \tag{4.8}
\]

For a constant \( \omega \in (0, 1) \), denote \( D_\omega = \{(x, y) \mid 1 - \omega < x \leq 1, 0 < y < \omega\} \).

By the continuity of \( \Phi(x) \), we can choose \( \omega \in (0, 1) \) sufficiently small such that for any \( x \in (1 - \omega, 1] \)
\[
\Phi(x) \leq \Phi(1) + \frac{\varepsilon}{2}.
\]

In addition, we let \( \theta \in (0, 1) \) sufficiently small such that for any \( x \in (1 - \omega, 1] \)
\[
\frac{1}{2}\theta\sigma^2 x^2 \leq \frac{\varepsilon}{2}.
\]

Thus, when \( \theta \) and \( \omega \) are small enough, for \( (x, y) \in D_\omega \), we have
\[
\theta y^{\lambda} \Phi(x) + \frac{1}{2}\theta^2 y^{\lambda}\sigma^2 x^2 \leq \theta y^{\lambda} (\Phi(1) + \frac{\varepsilon}{2}).
\]

In addition, when \( \theta \) is sufficiently small, we can get
\[
-(b + \varepsilon + \alpha x)(1 - x)^2 + (1 - x)(\beta x - \gamma y) + \frac{1}{2}\sigma^2 x^2 y^2
\leq \theta (1 - x)^2 (\Phi(1) + \frac{\varepsilon}{2}).
\]

Consequently,
\[
L\Psi(x, y) \leq \theta (\Phi(1) + \frac{\varepsilon}{2})\Psi(x, y) \text{ for } (x, y) \in D_\omega.
\]

By \cite[Theorem 3.3 (Chapter 4) and (4.7)]{55} for any \( \xi > 0 \), there is \( 0 < \omega_1 < \omega \) such that
\[
\mathbb{P}\left[ \lim_{t \to \infty} X(t) = (1, 0) \right] \geq 1 - \xi \quad \text{for } X_0 \in D_{\omega_1}, \tag{4.9}
\]

where \( D_{\omega_1} = \{(x, y) \mid 1 - \omega_1 < x \leq 1, 0 < y < \omega_1\} \).

Next, we further prove that any solution starting in \( D \) will eventually enter \( D_{\omega_1} \). Define \( \tau_{\omega_1} = \inf\{t \geq 0 : X(t) \geq 1 - \omega_1\} \).

Consider the function \( \Theta(x) = -(x + 2)^s \), where \( s \) is a sufficiently large number such that \( \frac{1}{2}(s - 1)\sigma^2 x^2 y^2 + (x + 2) \times \{(b + \varepsilon + \alpha x)(1 - x) - (\beta x - \gamma + \varepsilon y)\} \geq \frac{1}{3}(b + \varepsilon)\omega_1 \) for \( (x, y) \in D \) and \( x \in (0, 1 - \omega_1) \). In fact, since \((b + \varepsilon + \alpha x)(1 - x)(x + 2) > (b + \varepsilon)\omega_1 \), for any \( x \in (0, 1 - \omega_1) \), we can find such a \( s \).

Moreover, we have
\[
L\Theta(x) = -s(x + 2)^{s - 1}((b + \varepsilon + \alpha x)(1 - x) - (\beta x - \gamma + \varepsilon y)) - \frac{1}{2}s(s - 1)(x + 2)^{s - 2}\sigma^2 x^2 y^2
= -s(x + 2)^{s - 2}\left[(b + \varepsilon + \alpha x)(1 - x)(x + 2) - (\beta x - \gamma + \varepsilon y)(x + 2)\right]
+ \frac{1}{2}s(s - 1)\sigma^2 x^2 y^2
\leq -\frac{1}{3}(b + \varepsilon)\omega_1 s \text{ for } (x, y) \in D \text{ and } x \in (0, 1 - \omega_1).
\]

By Dynkin’s formula, we have
\[
\mathbb{E}\Theta(X(\tau_{\omega_1})) = \Theta(X_0) + \mathbb{E}\int_0^{\tau_{\omega_1}} L\Theta(X(t))dt \leq \Theta(X_0) - \frac{1}{3}(b + \varepsilon)\omega_1 \mathbb{E}(\tau_{\omega_1}).
\]

Since \( \Theta(x) \) is bounded on \( D \), we have \( \mathbb{E}(\tau_{\omega_1}) < \infty \). Thanks to the strong markov property, \( \mathbb{E}(\tau_{\omega_1}) < \infty \) and (4.9) leads to for any \( \xi > 0 \),
\[
\mathbb{P}\left[ \lim_{t \to \infty} X(t) = (1, 0) \right] \geq 1 - \xi, \text{ for } X_0 \in D.
\]
Therefore,
\[ P \left\{ \lim_{t \to \infty} (X(t)) = (1, 0) \right\} = 1, \text{ for } X_0 \in D. \]

Applying Itō’s formula, we have
\[ \frac{\ln y(t)}{t} = \frac{\ln y(0)}{t} + \frac{1}{t} \int_{0}^{t} \Phi(x(s))ds + \frac{1}{t} \int_{0}^{t} \sigma xdB(t) \]
(4.10)

By virtue of the large number theorem for martingales and ergodic Markov processes, we have \( \lim_{t \to \infty} \frac{1}{t} \int_{0}^{t} \Phi(X(u))du = \Phi(1) \) and \( \lim_{t \to \infty} \frac{1}{t} \int_{0}^{t} \sigma xdB(t) = 0 \) a.s. This and (4.10) means (4.5).
This completes the proof of Theorem 4.2. \( \Box \)

4.3. Persistence

In this section, we study the persistence of the model (2.6).

**Theorem 4.3.** Let \((x(t), y(t), z(t))\) be any solution of the model (2.6) with initial value \((x(0), y(0), z(0)) \in A\). If \(R_{01} > 1\) then
\[ \lim_{t \to \infty} \inf x(t) \leq x_* \leq \lim_{t \to \infty} \sup x(t) \text{ a.s.}, \]
where \(x_* = \frac{1}{\sigma^2} (\beta - \alpha) - \sqrt{(\beta - \alpha)^2 - 2\sigma^2(b + \delta + \gamma)}\) which is unique root in \((0, 1)\) of \((\beta - \alpha)x - (b + \delta + \gamma) - \frac{\gamma^2}{2} = 0\). That is, \(x(t)\) will rise to or above the level \(x_*\) infinitely often with probability one.

**Remark 2.** Since \(x(t) + y(t) + z(t) = 1\), it follows from \(\lim_{t \to \infty} \inf x(t) \leq x_* \leq \lim_{t \to \infty} \sup x(t) \) a.s. that \(y(t) + z(t)\) is always oscillating between 0 and 1 a.s. This means that the disease is persistent.

**Proof.** Recall the definition of function \(\Phi(x) = (\beta - \alpha)x - (b + \delta + \gamma) - \frac{\gamma^2}{2}\), it is easy to see \(\Phi(0) = -(b + \delta + \gamma) < 0\) and \(\Phi(1) = (b + \alpha + \delta + \gamma)(R_{01} - 1) > 0\). Hence, the equation \(\Phi(x) = 0\) has unique positive root in \((0, 1)\) which is \(x_* = \frac{1}{\sigma^2} (\beta - \alpha) - \sqrt{(\beta - \alpha)^2 - 2\sigma^2(b + \delta + \gamma)}\).

Since \(\frac{d\Phi(x)}{dx} = (\beta - \alpha) - \sigma^2\), \(\Phi(x)\) is monotonically increasing at \(x_*\).

First, we prove that \(\lim_{t \to \infty} \sup x(t) > x_*\). If it is not true, then there exists a sufficiently small \(\epsilon_1 \in (0, 1)\) such that
\[ P(\Omega_1) > \epsilon_1, \]
where \(\Omega_1 = \{\lim_{t \to \infty} \sup x(t) \leq x_* - 2\epsilon_1\}\). Hence, for every \(\omega \in \Omega_1\), there is a \(T_1 = T_1(\omega)\) such that
\[ x(t, \omega) \leq x_* - \epsilon_1, \quad t \geq T_1(\omega). \]
(4.11)

Since \(\Phi(x)\) is monotonically increasing at \(x_*\), we have
\[ \Phi(x(t, \omega)) \leq \Phi(x_* - \epsilon_1) < 0, \quad t \geq T_1(\omega) \]
(4.12)

On the other hand, by the large number theorem for martingales, there is an \(\Omega_2 \subset \Omega\) with \(P(\Omega_2) = 1\) such that for every \(\omega \in \Omega_2\),
\[ \lim_{t \to \infty} \frac{1}{t} \int_{0}^{t} \sigma x(s, \omega)dB(s, \omega) = 0. \]
(4.13)

Now, fix any \(\omega \in \Omega_1 \cap \Omega_2\). From (4.12), for \(t \geq T_1(\omega)\), we get
\[ \ln y(t, \omega) = \ln y(0) + \int_{0}^{T_1(\omega)} \Phi(x(s, \omega))ds + \int_{T_1(\omega)}^{t} \Phi(x(s, \omega))ds + \int_{0}^{t} \sigma x(s, \omega)dB(s, \omega) \]
\[ \leq \ln y(0) + \int_{0}^{T_1(\omega)} \Phi(x(s, \omega))ds + \int_{T_1(\omega)}^{t} \Phi(x_* - \epsilon_1)ds + \int_{0}^{t} \sigma x(s, \omega)dB(s, \omega) \]
\[ = \ln y(0) + \int_{0}^{T_1(\omega)} \Phi(x(s, \omega))ds + (t - T_1(\omega))\Phi(x_* - \epsilon_1) + \int_{0}^{t} \sigma x(s, \omega)dB(s, \omega). \]

This leads to
\[ \lim_{t \to \infty} \sup_{t} \frac{(\ln y(t, \omega))}{t} \leq \Phi(x_* - \epsilon_1) < 0. \]
Therefore,
\[ \lim_{t \to \infty} y(t, \omega) = 0. \]
(4.14)

According to (4.14), the last equation of the system (2.6) is differential system with limit system
\[ dz(t, \omega) = - (b + \alpha + \epsilon)z + \alpha z^2)dt. \]
So we get
\[
z(t, \omega) = \frac{1}{\mu + \alpha + \varepsilon t + \beta} + \left( \frac{1}{\mu + \alpha + \varepsilon t} - \frac{1}{\mu + \alpha + \varepsilon t + \beta} \right) \exp\left(- (\mu + \alpha + \varepsilon)t\right) \rightarrow 0, \text{ as } t \to \infty
\]
This together with the relation \(x(t) + y(t) + z(t) = 1\) leads to \(x(t, \omega) = 1\). However, this contradicts (4.11).

Therefore we must have the desired assertion \(\lim_{t \to \infty} \sup x(t) > x_\ast\).

Next, let us prove assertion \(\lim_{t \to \infty} \inf x(t) \leq x_\ast\). If it is not true, then there is a sufficiently small \(\varepsilon_2 \in (0, 1)\) such that
\[
\mathbb{P}(\Omega_3) > \varepsilon_2. 
\]
where \(\Omega_3 = \{\lim_{t \to \infty} \inf \alpha(t) > x_\ast + 2\varepsilon_2\}\). Hence, for every \(\omega \in \Omega_3\), there is a \(T_2 = T_2(\omega) > 0\) such that
\[
x(t) \geq x_\ast + \varepsilon_2, \quad t > T_2(\omega). \tag{4.15}
\]
Since \(\Phi(x)\) is monotonically increasing at \(x\), we obtain
\[
\Phi(x(t, \omega)) \geq \Phi(x_\ast + \varepsilon_2) > 0, \quad t > T_2(\omega). \tag{4.16}
\]
Now, fix \(\omega \in \Omega_2 \cap \Omega_3\), from (4.16), we get
\[
\ln y(t, \omega) = \ln y(0) + \int_{t_0}^{T_2(\omega)} \Phi(x(s, \omega)) ds + \int_{T_2(\omega)}^{t} \Phi(x(s, \omega)) ds + \int_{t_0}^{t} \sigma x(s, \omega) dB(s, \omega)
\geq \ln y(0) + \int_{t_0}^{T_2(\omega)} \Phi(x(s, \omega)) ds + \int_{t_0}^{t} \sigma x(s, \omega) dB(s, \omega)
= \ln y(0) + \int_{t_0}^{T_2(\omega)} \Phi(x(s, \omega)) ds + (t - T_2(\omega)) \Phi(x_\ast + \varepsilon_2) + \int_{t_0}^{t} \sigma x(s, \omega) dB(s, \omega).
\]
This yields
\[
\lim_{t \to \infty} \inf \frac{\ln y(t(\omega))}{t} \geq \Phi(x_\ast + \varepsilon_2) > 0.
\]
Hence, \(\lim_{t \to \infty} y(t, \omega) = \infty\), that contradicts \(y(t, \omega) \leq 1\). This completes the proof of assertion \(\lim_{t \to \infty} \inf x(t) \leq x_\ast\).

This completes the proof of Theorem 4.3. \(\square\)

Next, when \(R_0 > 1\), we estimate the lower bound of the solution for model (2.6) in mean time.

**Theorem 4.4.** Let \((x(t), y(t), z(t))\) be any solution of model (2.6) with initial value \((x(0), y(0), z(0)) \in A\). If \(R_0 > 1\), then \(\lim_{t \to \infty} \inf (y(t)) \geq \frac{\delta(b + x + b + \gamma)}{\beta(s + b + \varepsilon)} (R_0 - 1), \lim_{t \to \infty} \inf (z(t)) \geq \frac{\delta(b + x + b + \gamma)}{\beta(\delta + b + \varepsilon)} (R_0 - 1), \lim_{t \to \infty} \inf (x(t)) \geq \frac{b}{\beta + \varepsilon} \text{ a.s.}\)

**Proof.** From (2.6)
\[
\frac{\ln y(t) - \ln y(0)}{t} = \frac{1}{t} \int_{0}^{t} \left( \beta x - (b + \alpha + \delta + \gamma) + \alpha y + \alpha z - \frac{\sigma^2 x^2}{2} \right) ds + \frac{1}{t} \int_{0}^{t} \sigma x dB(s)
\]
\[
= \frac{1}{t} \int_{0}^{t} \left( \beta (1 - y - z) - (b + \alpha + \delta + \gamma) + \alpha y + \alpha z - \frac{\sigma^2 x^2}{2} \right) ds + \frac{1}{t} \int_{0}^{t} \sigma x dB(s)
\]
\[
= \beta (b + \alpha + \delta + \gamma) - \frac{\sigma^2 x^2}{2} - \beta (y) - \beta (z) + \alpha (y) + \alpha (z) + \frac{1}{t} \int_{0}^{t} \sigma x dB(s)
\]
\[
\geq \beta (b + \alpha + \delta + \gamma) - \frac{\sigma^2 x^2}{2} - \beta (y) - \beta (z) + \frac{1}{t} \int_{0}^{t} \sigma x dB(s)
\]
\[
= (b + \alpha + \delta + \gamma)(R_0 - 1) - \beta (y) - \beta (z) + \frac{1}{t} \int_{0}^{t} \sigma x dB(s).
\]
Noting that \(x + y + z = 1\). From the third equation of the system (2.6), we have
\[
\frac{z(t) - z(0)}{t} = \frac{1}{t} \int_{0}^{t} \left( \delta y - (b + \alpha + \varepsilon) z + \alpha y z + \alpha z^2 \right) ds
\]
\[
= \frac{1}{t} \int_{0}^{t} \left( \delta y - (b + \alpha + \varepsilon) z + \alpha z(y + z) \right) ds
\]
\[
\leq \frac{1}{t} \int_{0}^{t} \left( \delta y - (b + \alpha + \varepsilon) z + \alpha z \right) ds
\]
\[
= \frac{1}{t} \int_{0}^{t} \left( \delta y - (b + \varepsilon) z \right) ds
\]
\[
= \delta (y) - (b + \varepsilon) (z).
\]

Rewriting (4.18), we get
\[
\langle z \rangle \leq \frac{\delta}{(b + \varepsilon)} (y) - \frac{z(t) - z(0)}{(b + \varepsilon)t}. \tag{4.19}
\]

Similarly, we obtain
\[
\frac{z(t) - z(0)}{t} = \frac{1}{t} \int_0^t (\delta y - (b + \alpha + \varepsilon)z + \alpha y z + \alpha z^2)ds
\]
\[
\geq \frac{1}{t} \int_0^t (\delta y - (b + \alpha + \varepsilon)z)ds
\]
\[
= \frac{1}{t} \int_0^t (\delta y - (b + \alpha + \varepsilon)z)ds
\]
\[
\geq \frac{1}{t} \int_0^t (\delta y - (b + \alpha + \varepsilon)z)ds
\]
\[
= \delta (y) - (b + \alpha + \varepsilon)(z).
\]

That is,
\[
\langle z \rangle \geq \frac{\delta (y)}{(b + \alpha + \varepsilon)} - \frac{z(t) - z(0)}{t(b + \alpha + \varepsilon)}. \tag{4.20}
\]

Substituting (4.19) into (4.17) leads to
\[
\ln y(t) - \ln y(0)
\]
\[
\geq (b + \alpha + \delta + \gamma) (R_0 - 1) - \beta (y) + \frac{z(t) - z(0)}{(b + \varepsilon)t} - \frac{\beta \delta}{(b + \varepsilon)} (y) + \frac{1}{t} \int_0^t \sigma x dB(s)
\]
\[
= (b + \alpha + \delta + \gamma) (R_0 - 1) + \frac{z(t) - z(0)}{(b + \varepsilon)t} - \frac{\beta (\delta + b + \varepsilon)}{(b + \varepsilon)} (y) + \frac{1}{t} \int_0^t \sigma x dB(s)
\]
which deduces
\[
\langle y \rangle \geq \frac{(b + \varepsilon)(b + \alpha + \delta + \gamma)}{\beta (\delta + b + \varepsilon)} (R_0 - 1) + f(t),
\]
where
\[
f(t) = \frac{(b + \varepsilon)}{\beta (b + \varepsilon)} \left[ \frac{z(t) - z(0)}{(b + \varepsilon)t} + \frac{1}{t} \int_0^t \sigma x dB(t) - \frac{\ln y(t)}{t} + \frac{\ln y(0)}{t} \right].
\]
By (4.13) and \(y \leq 1\), we have \(\lim_{t \to \infty} f(t) = 0\) a.s. Therefore,
\[
\liminf_{t \to \infty} (y) \geq \frac{(b + \varepsilon)(b + \alpha + \delta + \gamma)}{\beta (\delta + b + \varepsilon)} (R_0 - 1). \tag{4.21}
\]

Combining (4.21) with (4.20), we have
\[
\liminf_{t \to \infty} (z) \geq \frac{\delta}{b + \alpha + \varepsilon} \liminf_{t \to \infty} (y) = \frac{\delta (b + \varepsilon)(b + \alpha + \delta + \gamma)}{\beta (\delta + b + \varepsilon)(b + \alpha + \varepsilon)} (R_0 - 1). \tag{4.22}
\]

From the first equation of the system (2.6), we obtain
\[
\frac{x(t) - x(0)}{t} = \frac{1}{t} \int_0^t (b - \beta xy - bx + \gamma y + \varepsilon z + \alpha xy + \alpha xz)ds - \frac{1}{t} \int_0^t \sigma xy dB(s)
\]
\[
\geq \frac{1}{t} \int_0^t (b - \beta xy - bx)ds - \frac{1}{t} \int_0^t \sigma xy dB(s)
\]
\[
\geq \frac{1}{t} \int_0^t (b - \beta x - bx)ds - \frac{1}{t} \int_0^t \sigma xy dB(s)
\]
\[
= \frac{1}{t} \int_0^t (b - (\beta + b)x)ds - \frac{1}{t} \int_0^t \sigma xy dB(s)
\]
\[
= b - (\beta + b)(x) - \frac{1}{t} \int_0^t \sigma xy dB(s),
\]
which leads to
\[
\langle x \rangle \geq \frac{b}{\beta + b} - \frac{1}{(\beta + b)t} \int_0^t \sigma xy dB(s) - \frac{1}{\beta + b} \frac{x(t) - x(0)}{t}.
\]

Hence,
\[
\liminf_{t \to \infty} \langle x \rangle \geq \frac{b}{\beta + b}.
\]

This completes the proof of Theorem 4.4. □
Fig. 1. Time series of the solution $x(t)$ and $y(t)$ of the deterministic system (2.4) with $\delta = 0$, $\sigma = 0.1$, $R_0 = 2.1917$.

Fig. 2. Sample paths of the solution $x(t)$ and $y(t)$ of the stochastic system (2.7) with $\delta = 0$, $\sigma = 0.1$ and $R_0 = 1.9178$.

5. Numerical simulations

In this section, using the EM method mentioned [56], we will carry out numerical simulations to apply our results.

This example is motivated by realistic pneumococcus amongst homosexuals [57], with time unit of one day. The exit rate $\mu = \frac{1}{40+365} / \text{day} = 6.849315 \times 10^{-5} / \text{day}$ (average sexually active lifetime), $\gamma = 0.018182 / \text{day}$ [58], $\beta = 0.04$ (based on Gray et. al. [38]). Assumption that $b = \mu = \frac{1}{40+365} / \text{day} = 6.849315 \times 10^{-5} / \text{day}$ and the disease-related death rate $\alpha = 0$. To see the effect of isolation (quarantine) measure $\delta$ and stochastic disturbance intensity $\sigma$, in the next numerical simulations, we only change their values, and do not change other values.

**Example 1.** Let the disturbance intensity $\sigma = 0.1$ and the isolation rate $\delta = 0$.

In this case, $R_0 = 2.1917 > 1$, so the endemic equilibrium $E_1(x^*, y^*) = (0.4563, 0.5437)$ of the deterministic system (2.4) is globally asymptotically stable (Theorem 3.3). Fig. 1 confirm these. Meanwhile, $R_0 = 1.9178 > 1$, the solutions of the stochastic system (2.7) satisfy $\lim_{t \to \infty} \inf x(t) \leq x_* = 0.4858 \leq \lim_{t \to \infty} \sup x(t)$ a.s. (Theorem 4.3) and $\lim_{t \to \infty} \inf \langle y(t) \rangle \geq \frac{\delta (\beta + \gamma)(b \alpha + \alpha + \gamma)}{\beta (\beta + b + \gamma)} (R_0 - 1) = 0.4187$, $\lim_{t \to \infty} \inf \langle z(t) \rangle \geq \frac{\delta (\beta + \gamma)(b \alpha + \alpha + \gamma)}{\beta (\beta + b + \gamma)} (R_0 - 1) = 0.0018$, $\lim_{t \to \infty} \inf \langle x(t) \rangle \geq \frac{b}{\beta + b + \gamma} = 0.0017$ a.s. (Theorem 4.4). This implied that the stochastic system (2.7) is persistent. Fig. 2 confirm these.

In addition, by 100000 time numerical simulation, we collect the values of $x(t)$ and $y(t)$ of the stochastic system (2.7) at $t = 3000$, and give their empirical probability density which are exhibited in Figs. 3 and 4.
Example 2. Now, we choose the same parameters as in Example 1 except $\delta = 0.01$ which means one percent of the infective are isolated. In this situation, $R_0 = 1.4159 > 1$, then the endemic equilibrium $E_1(x^*, y^*) = (0.7063, 0.1898)$ of the deterministic system (2.4) is globally asymptotically stable (Theorem 3.3). Fig. 5 confirm these. Meanwhile, $R_{0b} = 1.2389 > 1$, the solutions of the stochastic system (2.7) satisfy $\lim_{t \to \infty} \inf x(t) \leq x_* = 0.7829 < \lim_{t \to \infty} \sup x(t)$ a.s. (Theorem 4.3) and $\lim_{t \to \infty} \inf y(t) \geq \frac{(b+\epsilon)(b+\alpha+\delta+\gamma)}{\beta(\delta+\beta+\epsilon)} (R_{0b} - 1) = 0.1090$. $\lim_{t \to \infty} \inf Z(t) = \frac{\delta(b+\epsilon)(b+\alpha+\delta+\gamma)}{\beta(\delta+\beta+\epsilon)(b+\alpha+\epsilon)} (R_{0b} - 1) = 0.0017$. $\lim_{t \to \infty} \inf \langle x(t) \rangle \geq \frac{b}{\beta + \epsilon} = 0.0016$ a.s. (Theorem 4.4). This implied that the stochastic system (2.7) is persistent. Fig. 6 confirm these.

Comparing Fig. 1 (b) with Fig. 5 (b), we find that isolation measure can reduce the number of the infective.

In addition, in the same way as Example 1, we give the empirical probability density of $x(t)$ and $y(t)$ of the stochastic system (2.7) which are exhibited in Figs. 7 and 8. Comparing Figs. 3(b) and 7(b), we see that isolation measure increases the probability of few people getting sick and change the shape of empirical probability density.

Example 3. Next, we illustrate that the sufficiently large isolation rate $\delta$ can eliminate the disease. Let the isolation rate $\delta = 0.03$, namely, three percent of the infective are isolated while the other parameter are the same as Example 1.

In this case, $R_0 = 0.8290 < 1$, so the disease-free equilibrium $E_0(1, 0)$ of the deterministic system (2.4) is globally stable (Theorem 3.2). Fig. 9 confirm these.
Fig. 5. Time series of $x(t)$ and $y(t)$ of the deterministic system (2.4) with $\delta = 0.01$, $\sigma = 0.1$, $R_0 = 1.4159$.

Fig. 6. Sample paths of $x(t)$ and $y(t)$ of the stochastic system (2.7) with $\delta = 0.01$, $\sigma = 0.1$ and $R_0 = 1.2389$.

Fig. 7. Empirical density of $(x, y)$ of the stochastic system (2.7) with $\delta = 0.01$, $\sigma = 0.1$, $R_0 = 1.4159$ and $R_0 = 1.2389$. 
Fig. 8. Empirical density of \((x, y)\) of the stochastic system (2.7) with \(\delta = 0.01\), \(\sigma = 0.1\), \(R_0 = 1.4159\) and \(R_{0s} = 1.2389\).

\[ P\left\{ \lim_{t \to \infty} \ln x(t) = (b + \alpha + \delta + \gamma)(R_{0s} - 1) = -0.0133 \right\} = 1 \text{ and } (x(t), y(t)) \to (1, 0) \text{ a.s. as } t \to \infty \text{ (Theorem 4.2).} \]

In addtion, \(R_{0s} = 0.7254\), the solutions of the stochastic system (2.7) satisfy \( P\left\{ \lim_{t \to \infty} \ln x(t) = (b + \alpha + \delta + \gamma)(R_{0s} - 1) = -0.0133 \right\} = 1 \) and \((x(t), y(t)) \to (1, 0) \) a.s. as \(t \to \infty\) (Theorem 4.2). Fig. 10 confirm these.

Example 4. Now, we illustrate that the large stochastic disturbance intensity \(\sigma\) can eliminate the disease. To this end, Let \(\sigma = 0.22\) and the other parameters as in Example 1, then \(R_0 = 2.1917\). Hence, the endemic equilibrium \(E_{1}(x^*, y^*) = (0.4563, 0.5437)\) of the deterministic system (2.4) is globally asymptotically stable (Theorem 3.3), which has been confirmed by Fig. 1.

Meanwhile, \(R_{0s} = 0.8657 < 1\), then the solutions of the stochastic system (2.7) satisfy \( P\left\{ \lim_{t \to \infty} \ln x(t) = (b + \alpha + \delta + \gamma)(R_{0s} - 1) = -0.0233 \right\} = 1 \) and \((x(t), y(t)) \to (1, 0) \) a.s. as \(t \to \infty\) and \( P\left\{ \lim_{t \to \infty} \ln x(t) = (b + \alpha + \delta + \gamma)(R_{0s} - 1) = -0.0233 \right\} = 1 \) (Theorem 4.2). Fig. 11 confirm these.

Example 5. In this example, we illustrate how the isolation rate \(\delta\) and the stochastic disturbance intensity \(\sigma\) affect the threshold \(R_0\) and \(R_{0s}\).

Fig. 12 shows that as the isolation rate \(\delta\) increases, \(R_0\) decreases. We can find that there exists a critical isolation rate \(\delta^* = 0.0217\). If \(\delta > \delta^*\), then \(R_0 < 1\). This means that if more than 2.17% of the infective are isolated, the pneumococcus amongst will be eliminated.
Fig. 10. Sample paths of $x(t)$ and $y(t)$ of the stochastic system (2.7) with $\delta = 0.02$, $\sigma = 0.1$ and $R_0s = 0.7254$.

Fig. 11. Sample paths of $x(t)$ and $y(t)$ of the stochastic system (2.7) with $\delta = 0$, $\sigma = 0.22$ and $R_0s = 0.8657$.

Fig. 12. The relationship between $\delta$ and $R_0$ for the deterministic system (2.4).
Fig. 13. The relationship of $R_{0s}$, $\delta$ and $\sigma$ for the stochastic system (2.7).

Fig. 13 describes that as the isolation rate $\delta$ or the stochastic disturbance intensity $\sigma$ increase, $R_{0s}$ decreases. We can see there is a critical stochastic disturbance intensity $\sigma^*$, when $\sigma > \sigma^*$, $R_{0s} < 1$. This tells us that sufficiently large stochastic disturbance of the transmission rate can eliminate the disease.

6. Conclusion

In this paper, we propose a deterministic and stochastic SIS epidemic model with varying population and isolation. For the deterministic model (2.4), we prove that its threshold is $R_0 = \frac{b}{p + a + \gamma}$. If $R_0 \leq 1$, the disease-free equilibrium $E_0(1, 0)$ of the deterministic model (2.4) is globally stable which means the disease will die out (Theorem 3.2). If $R_0 > 1$, the endemic equilibrium $E_1(x^*, y^*)$ of the deterministic model (2.4) is globally stable which implies that the disease will spread (Theorem 3.3). For the stochastic model (2.7), we also present its threshold $R_{0s} = R_0 - \frac{\sigma^2}{\sigma^2 + \sigma^* + \gamma}$. When $R_{0s} < 1$, the solution of model (2.7) satisfies $\mathbb{P} (\lim_{t \to \infty} \frac{\ln(x(t))}{t} = (b + \alpha + \delta + \gamma)(R_{0s} - 1)) = 1$ which shows that the disease will be extinct almost surely (see Theorem 4.2). When $R_{0s} > 1$, the solution of model (2.7) obeys $\lim_{t \to \infty} x(t) \leq x_*$, $\lim_{t \to \infty} x(t)$ and $\lim_{t \to \infty} \inf [x(t) + z(t)] \leq 1 - x_*$, $\lim_{t \to \infty} \sup [x(t) + z(t)]$ almost surely with $x \in (0, 1)$ which means that $x(t), y(t)$ and $z(t)$ of (2.7) are always oscillating between 0 and 1 with probability 1 (see Theorem 4.3). Namely, the disease is persistent in probability. In addition, we prove that when $R_{0s} > 1$, for the solution $(x(t), y(t), z(t))$ of model (2.7), $\lim_{t \to \infty} \int_0^t x(u)du \geq \frac{b}{p + a + \gamma} \left( R_{0s} - 1 \right)$ and $\lim_{t \to \infty} \int_0^t y(u)du \geq \frac{b + \delta + \gamma}{p + a + \gamma} \left( R_{0s} - 1 \right)$ hold (see Theorem 3.3.2). These results illustrate that the solution of model (2.7) is persistent in mean. It follows from $R_{0s} < R_0$ that the stochastic perturbation of the transmission rate (or the valid contact coefficient) can help to reduce the spread of the disease. In other words, the deterministic epidemic model overestimates the spread capacity of disease. In addition, there is a critical stochastic disturbance intensity $\sigma^*$, when $\sigma > \sigma^*$, $R_{0s} < 1$. This tells us that sufficiently large stochastic disturbance of the transmission rate can eliminate the disease (see Fig. 13). At last, we apply our theories to a realistic disease, pneumococcus amongst homosexuals, and carry out numerical simulations and obtain the empirical probability density under different parameter values. We find that there exists a critical isolation rate $\delta^* = 0.0217$, when $\delta > \delta^*$ the pneumococcus amongst homosexuals will be eliminated. This has the important instructional significance to control pneumococcus amongst.

7. Discussion

Environmental noise has an important impact on the development of epidemics. In this paper, we analyze the dynamics of a stochastic SIQS model. We suppose that the stochastic perturbation is a white noise type which disturbs the transmission coefficient of the disease. This is an accepted method of introducing stochastic environmental noise into the dynamic model of biological population that have been used in [21,38].

In this paper, our contributions is that epidemiologically, we partially answer this question proposed in the introduction: How does environment fluctuations affect the threshold of a SIQS epidemic model? We find that environment fluctuations reduce threshold of a SIQS epidemic model. In other words, the threshold of the stochastic SIQS epidemic model is less than the threshold of the corresponding deterministic version. This means the stochastic perturbation of the transmission rate can suppress the spread of the disease. This is consistent with some existing studies [37]. In addition, in this paper, we establish the almost sufficient and necessary condition of the extinction and the permanence of the disease. Only the critical case when $R_{0s} = 1$ is not studied yet. In contrast, most existing results just are necessary conditions of the extinction and the permanence of the disease. Furthermore, our approach can improve the results of some literatures (e.g. [37,50,52]).
The outbreak of infectious diseases will hinder the normal operation of human society and cause serious economic losses. As a result, quarantine is one of the most popular classification tools to control and eliminate epidemic, especially for new and unknown infections diseases. From the view of application, by the threshold of the SIQS epidemic model, we can get the critical isolation rate $\delta^*$. When the isolation rate $\delta$ is greater than $\delta^*$, the disease will be controlled and eliminated.

It should be noted that in the present paper we consider continuous stochastic perturbations described by white noises. In fact, there are some discontinuous stochastic perturbations which can not be modeled by white noises, but can be done by the telephone and Lévy jumps noise. Recently, stochastic models with these noises have been studied by many authors, and many interesting results have been obtained, for example, see [59–63] and the references therein. These studies motivate us to further analyze system (2.7) including the telephone and Lévy noises. In addition, for stochastic model we establish the threshold condition of persistence and extinction, but do not prove the existence of stationary probability distribution which is similar to the equilibrium state for the deterministic model. We leave these investigations for future work.

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