



Research article

Dynamics of stochastic SIQS model based on nonlinear incidence: disease extinction and stationary distribution under degenerate diffusion

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Abstract: This paper investigated the dynamical behaviors of the SIQS (susceptible, infected, isolated, and again susceptible) infectious disease model with nonlinear incidence rate and degenerate diffusion in a stochastic environment. By introducing nonlinear contagion rate, the model was able to more realistically reflect the complexity of real-world disease transmission, including the effects of social behavior, medical resource constraints, and public health interventions. It was proved that the infectious disease will be extinct when $R_0^s < 1$. Furthermore, by utilizing Markov semigroup theory, we obtained that there existed stationary distribution for the system when $R_0^s > 1$. Numerical simulations were conducted by introducing three different forms of nonlinear incidence rates (standard incidence, non-monotonic incidence, Beddington-DeAngelis incidence) to verify our results.

Keywords: stochastic SIQS model; nonlinear incidence rate; degenerate diffusion; extinction; stationary distribution

1. Introduction

Infectious diseases, as a major challenge in global public health, have always been an important object of scientific research. An in-depth understanding of the dynamics of infectious disease is particularly critical in today's globalized world, where the speed of transmission and the scope of impact of infectious disease have increased dramatically [1]. At the beginning of the 20th century, Kermak and McKendrick developed models of infectious disease [2], then biomathematical modeling has become a key tool for understanding and controlling infectious disease. In recent years, many scholars have studied different types of biomathematical models, such as the SIS (susceptible, infected, and again susceptible) model [3, 4], the SIR (susceptible, infected, and recovered) model [5, 6], the SEIR (susceptible, exposed, infected, and recovered) model [7–9], etc., in order to explore effective prevention and control strategies for infectious disease. As the first line of defense to reduce the spread of infection, isolation has been used as an important means of controlling infectious

disease for centuries. Therefore, the study of SIQS (susceptible, infected, isolated, and again susceptible) infectious disease modeling that includes isolation measures is of particular importance [10–13]. In the SIQS model, infection does not confer immunity on the individual. Susceptible people become infected once they are infected, and a proportion of infected persons return to susceptible groups after remaining infected throughout the period of infection, and the other portion of infected persons are moved to the isolation category until they are no longer infectious and subsequently revert to the susceptible category. In this context, Herbert et al. [13] explored an SIQS model of infectious disease that emphasizes the importance of isolation for controlling the spread of disease and its dynamic mechanisms

$$\begin{cases} \frac{dS(t)}{dt} = A - \beta S(t)I(t) - \mu S(t) + \gamma I(t) + \rho Q(t), \\ \frac{dI(t)}{dt} = \beta S(t)I(t) - (\mu + \gamma + \delta + \alpha)I(t), \\ \frac{dQ(t)}{dt} = \delta I(t) - (\mu + \rho + \alpha)Q(t). \end{cases} \quad (1.1)$$

In mathematical modeling, the form of the incidence function significantly affects the system dynamics characteristics [14, 15]. Wei et al. [16] showed that the equilibrium stability and the time-lag-induced Hopf bifurcation behavior of a predator-prey model using a Beddington-DeAngelis-type functional response function are highly dependent on the parameter structure of this function. Similarly, in the field of human infectious diseases, the introduction of nonlinear incidence rates makes the models more relevant to reality: Liu et al. [17] used generalized incidence rates under random perturbations in a variable population size SEIS (susceptible, exposed, infected, and again susceptible) model to analyze extinction and persistence thresholds and to reveal the modulation mechanism of noise on disease transmission; Wei et al. [18] further constructed two stochastic differential equations (SDEs) perturbed by white noise and one ordinary differential equation (ODE) described by a hybrid model to derive sufficient conditions for extinction and persistence of disease transmission based on an incidence function parameterized by constant exposure rates; Chen et al. [19] investigated the dynamical behavior of a stochastic SIR model with standard incidence rate and demonstrated the existence of stationary distribution of endemic equilibrium points in conjunction with the Lyapunov function. For some infectious diseases, such as avian influenza and dengue fever [20, 21], a bilinear form of incidence based on the mass action law is widely used and has been shown to model the spread of these diseases well. However, in practice, when faced with a disease outbreak, susceptible people tend to take self-protective measures to minimize contact with infected people, mainly due to media reports and individual psychological reactions to the disease [22]. In addition, for situations like sexually transmitted diseases, it may not be accurate to assume that contact between individuals in a population is uniformly distributed [23]. Therefore, in order to more accurately characterize the actual spreading process of a disease, it is common to use incidence functions that have a general nonlinear form [22, 24, 25]. To make the model (1.1) realistic, we use the nonlinear incidence rate $f(S(t), I(t))$, and assume that the mortality rate associated with infectious disease in the infected population is different from the mortality rate associated with infectious disease in the isolated population, respectively α_1, α_2 , i.e.,

$$\begin{cases} \frac{dS(t)}{dt} = A - \beta f(S(t), I(t)) - \mu S(t) + \gamma I(t) + \rho Q(t), \\ \frac{dI(t)}{dt} = \beta f(S(t), I(t)) - (\mu + \gamma + \delta + \alpha_1) I(t), \\ \frac{dQ(t)}{dt} = \delta I(t) - (\mu + \rho + \alpha_2) Q(t). \end{cases} \quad (1.2)$$

Significant progress has been made in characterizing the dynamics of infectious diseases with quarantine mechanisms using stochastic SIQS models. Existing studies, such as Wei et al. [11] developing a stochastic model incorporating a saturated incidence rate, provide valuable insights into complex transmission behaviors. However, such models typically introduce environmental noise solely in an additive form within the state equations, and their analysis is predominantly confined to the stability near the disease-free equilibrium. This approach fails to adequately capture the intrinsic interference of noise on the core parameters governing the transmission process. To construct a more realistic stochastic system, incorporating Brownian motion into deterministic models is a common methodology [22, 26–30]. This study presents a key advancement within the stochastic SIQS framework: we employ a nonlinear incidence rate $f(S(t), I(t))$, which better reflects practical transmission dynamics, while simultaneously accounting for stochastic perturbations affecting the transmission coefficient β , i.e., $\beta \rightarrow \beta + \sigma \dot{B}(t)$. Consequently, the deterministic model (1.2) is transformed into the following stochastic SIQS system, characterized by a nonlinear incidence rate and parametric stochastic perturbation of the transmission coefficient

$$\begin{cases} dS(t) = [A - \beta f(S(t), I(t)) - \mu S(t) + \gamma I(t) + \rho Q(t)]dt - \sigma f(S(t), I(t))dB(t), \\ dI(t) = [\beta f(S(t), I(t)) - (\mu + \gamma + \delta + \alpha_1) I(t)]dt + \sigma f(S(t), I(t))dB(t), \\ dQ(t) = [\delta I(t) - (\mu + \rho + \alpha_2) Q(t)]dt, \end{cases} \quad (1.3)$$

where $S(t)$, $I(t)$, $Q(t)$ indicate susceptible, infected, and isolated population, respectively. $B(t)$ indicates independent standard Brownian motion, and σ indicates the strength of $B(t)$. The definitions of the remaining parameters in the model are outlined in the Table 1.

Table 1. Meaning of parameters in the model (1.3).

Parameters	Meaning
A	Recruitment rate of the population
μ	Natural mortality of the population
α_1	Infectious disease-related mortality in the infected population
α_2	Infectious disease-related mortality in the isolated population
β	Transmission coefficient
δ	Metastasis rates in infected population
γ	Rate of loss of immunity in the infected population
ρ	Rate of loss of immunity in the isolated population

Against this background, the study of the dynamical behavior (extinction and stationary distribution) in such models has become a hot topic in epidemiology and applied mathematics. Extinction is when the infectious disease no longer occurs. Stationary distribution is when the

probability distribution of the system state reaches a steady state on long time scales [21, 31]. It not only reveals the persistence of the disease in a stochastic environment, but also helps us to assess the effectiveness of long-term intervention strategies. However, analyzing the existence and uniqueness of stationary distribution is a complex and difficult task that requires advanced mathematical tools [23, 25, 32, 33].

What follows is organized according to the following structure: the necessary theoretical background information is provided in Section 2. Then, Section 3 reveals the specific conditions under which the disease extinction is under model (1.3). Section 4 further explores the conditions that need to be met to form a stationary distribution for the model (1.3). In order to consolidate the theoretical assumptions made in the previous sections, Section 5 conducts relevant numerical simulation experiments. Finally, Section 6 summarizes and concludes the whole study.

2. Preparations

Let $(\Omega, \{F_t\}_{t \geq 0}, P)$ be a complete probability space whose filters $\{F_t\}_{t \geq 0}$ meet the standard requirements for this work. We consider the formulation of model (1.3) on a probability space $(\Omega, \{F_t\}_{t \geq 0}, P)$.

To facilitate the subsequent analysis, we make the following assumptions:

(Ψ_1) The function $f(S, I)$ is second-order continuously differentiable on the entire positive real two-dimensional space R_+^2 , which suggests that the rate of disease transmission does not change suddenly, but follows a gradual process that is consistent with the nature of disease transmission in the real world. $f(S, I)$ is monotonically increasing for $S \geq 0$, which means that as the number of susceptible or infected population increase, so does the number of transmission events, which is consistent with our basic understanding of the spread of infectious diseases. $f(S, 0) = f(0, I) \equiv 0$ for all $(S, I) \in R_+^2$, the so-called “zero boundary condition”, emphasizes the intuitive fact that in the absence of susceptible or infected population, the spread of disease is necessarily zero. This principle forms the basis of all models of infectious disease and highlights the critical role of susceptible and infected population in the transmission of disease.

(Ψ_2) $\frac{f(S, I)}{I} \leq \frac{\partial f(S^*, 0)}{\partial I}$ for any $0 \leq S \leq S^*$, $I > 0$, and $\frac{\partial f(S^*, 0)}{\partial I} > 0$, where $S^* = \frac{A}{\mu}$. This assumption effectively requires that when an infectious disease outbreak occurs, the actual transmission efficiency of each infected individual does not exceed the peak transmission efficiency at the beginning of the outbreak. This suggests that the transmission of a single infected person decays as the susceptible population decreases or as prevention and control are strengthened.

(Ψ_3) Assuming there exist constants $\eta > 0, \beta_1 \geq 0, \beta_2 \geq 0$ such that they satisfy the condition

$$\inf_{(S, I) \in \Gamma} \left\{ \frac{(\beta_1 + \beta_2 I) f(S, I)}{SI} \right\} \geq \eta. \quad (2.1)$$

This assumption ensures that there is a strictly positive lower bound η on transmission efficiency (within the plausible region Γ). It prevents biologically impractical “complete stagnation of transmission” in the model and ensures that the disease always retains a minimum potential for transmission while there are still susceptible and infected individuals.

With (Ψ_1) and (Ψ_2), we have

(1) For any $(S, I) \in R_+^2$, $\frac{\partial f(S, I)}{\partial I}$ is irreducible to S , and

$$\frac{\partial f(0, I)}{\partial I} = \frac{\partial f(S, 0)}{\partial S} \equiv 0.$$

(2) For any constant $r > 0, s > 0$, let $J = \{(S, I) : S \geq 0, I \geq 0, r \leq S + I \leq s\}$, then

$$\max_{(S, I) \in J} \left\{ \frac{f(S, I)}{S}, \frac{f(S, I)}{I} \right\} < \infty. \quad (2.2)$$

Define

$$\Gamma = \left\{ (S, I, Q) \in R_+^3 : \frac{A}{\mu + \alpha_1 + \alpha_2} \leq S + I + Q \leq \frac{A}{\mu} \right\}.$$

Lemma 2.1. Every trajectory of model (1.3) stays on a tight set Γ .

Lemma 2.2. For all initial values $(S(0), I(0), Q(0)) \in R_+^3$, model (1.3) has a unique solution $(S(t), I(t), Q(t))$ that stays within R_+^3 with probability one for all $t \geq 0$.

A detailed proof of this lemma can be based on [34].

Let m be the Leberger measure on R_+^3 , and let $\mathcal{B}(R_+^3)$ be the σ -algebra of a Borel subset of R_+^3 . The subset of the space $L^1 = L^1(R_+^3, \mathcal{B}(R_+^3), m)$ containing all densities is denoted by D

$$D = \{g \in L^1, g \geq 0, \|g\|_{L^1} = 1\}.$$

where $\|\cdot\|_{L^1}$ denotes the quantity of paradigms within L^1 . Given a linear mapping $P : L^1 \rightarrow L^1$, an operator P is said to be a Markov operator when $P(D) \subset D$.

If $k : R_+^3 \times R_+^3 \times R_+^3 \rightarrow [0, \infty)$ is a measurable kernel function such that

$$\int_{R_+^3} k(S, I, Q; S_1, I_1, Q_1) m(dS, dI, dQ) = 1, \quad (2.3)$$

for all $(S_1, I_1, Q_1) \in R_+^3$ and

$$Pg(S, I, Q) = \int_{R_+^3} k(S, I, Q; S_1, I_1, Q_1) g(S_1, I_1, Q_1) m(dS_1, dI_1, dQ_1),$$

then P is an integral operator and k is the kernel of the integral operator P .

If the following criteria are met by the family of Markov operators $\{P(t)\}_{t \geq 0}$,

(a) $P(0) = Id$, where Id is a constant operator on L^1 .

(b) $P(t + s) = P(t)P(s)$ for every $t, s \geq 0$.

(c) $P(t)g$ is continuous, for $t \geq 0$ and $g \in L^1$, then $\{P(t)\}_{t \geq 0}$ is a Markov semigroup.

Lemma 2.3. Let $\{P(t)\}_{t \geq 0}$ be a continuous kernel $k(t; S, I, Q; S_1, I_1, Q_1)$ and a semigroup of integral Markov chains on L^1 , and let $t > 0$ satisfy (2.3). Suppose that for any $g \in D$,

$$\int_0^\infty P(t)g(S, I, Q) dt > 0. \quad (2.4)$$

Then, with regard to the tight set, the Markov semigroup $\{P(t)\}_{t \geq 0}$ is either sweeping or asymptotically stable [35, 36].

3. Extinction

Define

$$R_0^s = \frac{\partial f(S^*, 0)}{\partial I} \frac{\beta}{\mu + \gamma + \delta + \alpha_1} - \frac{1}{2} \left(\frac{\partial f(S^*, 0)}{\partial I} \right)^2 \frac{\sigma^2}{\mu + \gamma + \delta + \alpha_1}.$$

Theorem 3.1. For all initial value $(S(0), I(0), Q(0)) \in R_+^3$, if (Ψ_1) , (Ψ_2) hold and $R_0^s < 1$, then

$$\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} \leq (\mu + \gamma + \delta + \alpha_1)(R_0^s - 1) < 0,$$

and the infection will be extinct.

Proof. By Ito's formula for $\ln I(t)$, then

$$d \ln I(t) = \left(\frac{\beta f(S(t), I(t))}{I(t)} - (\mu + \gamma + \delta + \alpha_1) - \frac{\sigma^2 f^2(S(t), I(t))}{2I^2(t)} \right) dt + \frac{\sigma f(S(t), I(t))}{I(t)} dB(t).$$

The above equation integrates from 0 to t and divides by t , and we have

$$\begin{aligned} \frac{\ln I(t) - \ln I(0)}{t} &= \frac{1}{t} \int_0^t \left(\frac{\beta f(S(s), I(s))}{I(s)} - (\mu + \gamma + \delta + \alpha_1) - \frac{\sigma^2 f^2(S(s), I(s))}{2I^2(s)} \right) ds \\ &\quad + \frac{1}{t} \int_0^t \frac{\sigma f(S(s), I(s))}{I(s)} dB(s) \\ &\leq \frac{\beta \partial f(S^*, 0)}{\partial I} - (\mu + \gamma + \delta + \alpha_1) - \frac{\sigma^2}{2} \left(\frac{\partial f(S^*, 0)}{\partial I} \right)^2 + \frac{1}{t} \int_0^t \frac{\sigma f(S(s), I(s))}{I(s)} dB(s). \end{aligned}$$

By the powerful number law of the harness [34], we obtain

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

Hence,

$$\begin{aligned} \limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} &\leq \frac{\beta \partial f(S^*, 0)}{\partial I} - (\mu + \gamma + \delta + \alpha_1) - \frac{\sigma^2}{2} \left(\frac{\partial f(S^*, 0)}{\partial I} \right)^2 \\ &\leq (\mu + \gamma + \delta + \alpha_1)(R_0^s - 1). \end{aligned}$$

If $R_0^s < 1$, then $\limsup_{t \rightarrow \infty} \frac{\ln I(t)}{t} < 0$, which means that the infection will be extinct. \square

4. Stationary distribution

Theorem 4.1. Assuming that (Ψ_1) and (Ψ_2) hold, the transfer probability $P(t; S_1, I_1, Q_1; B)$ of a solution $(S(t), I(t), Q(t))$ of model (1.3) has a continuous density $k(t; S, I, Q; S_1, I_1, Q_1) \in C^\infty(R_+ \times R_+^3 \times R_+^3)$.

Proof. Considering two differentiable vector fields $h(S)$ and $l(S)$ on R^n , the Lie bracket defined by $[h, l](S) = ([h, l]_1(S), [h, l]_2(S), \dots, [h, l]_n(S))$ is also a vector field

$$[h, l]_i(S) = \sum_{j=1}^n \left(h_j \frac{\partial l_i}{\partial S_j}(S) - l_j \frac{\partial h_i}{\partial S_j}(S) \right), i = 1, 2, \dots, n,$$

where

$$h(S, I, Q) = \begin{pmatrix} A - \beta f(S, I) - \mu S + \gamma I + \rho Q \\ \beta f(S, I) - (\mu + \gamma + \delta + \alpha_1)I \\ \delta I - (\mu + \rho + \alpha_2)Q \end{pmatrix}, l(S, I, Q) = \begin{pmatrix} -\sigma f(S, I) \\ \sigma f(S, I) \\ 0 \end{pmatrix}.$$

Based on computation, we can obtain $[h, l](S, I, Q) = (E_1, E_2, E_3)^T$, where

$$\begin{aligned} E_1 &= -[A - \mu S + \gamma I + \rho Q]\sigma \frac{\partial f(S, I)}{\partial S} + [(\mu + \gamma + \delta + \alpha_1)I]\sigma \frac{\partial f(S, I)}{\partial I} \\ &\quad - \mu \sigma f(S, I) - \gamma \sigma f(S, I), \\ E_2 &= [A - \mu S + \gamma I + \rho Q]\sigma \frac{\partial f(S, I)}{\partial S} - [(\mu + \gamma + \delta + \alpha_1)I]\sigma \frac{\partial f(S, I)}{\partial I} \\ &\quad + (\mu + \gamma + \delta + \alpha_1)\sigma f(S, I), \\ E_3 &= -\delta \sigma f(S, I). \end{aligned}$$

Continuing with the computation, we have $[h, [h, l]](S, I, Q) = (F_1, F_2, F_3)^T$, where the values of F_1 , F_2 , and F_3 ; see Appendix A. For ease of computation, we denote $\nu_1 = \frac{\partial f(S, I)}{\partial S}$, $\nu_2 = \frac{\partial f(S, I)}{\partial I}$, $\nu_3 = \frac{\partial^2 f(S, I)}{\partial S \partial I}$, $\nu_4 = \frac{\partial^2 f(S, I)}{\partial I \partial S}$, $\nu_5 = \frac{\partial^2 f(S, I)}{\partial S^2}$, $\nu_6 = \frac{\partial^2 f(S, I)}{\partial I^2}$.

By direct computation, we have the determinant

$$| \begin{matrix} l & [h, l] & [h, [h, l]] \end{matrix} | = \begin{vmatrix} -\sigma f(S, I) & E_1 & F_1 \\ \sigma f(S, I) & E_2 & F_2 \\ 0 & E_3 & F_3 \end{vmatrix}.$$

From $E_1 F_3 + E_2 F_3 - E_3 F_1 - E_3 F_2 < 0$, the above determinant is greater than 0. This implies that $l, [h, l], [h, [h, l]]$ is linearly independent on R_+^3 almost everywhere. Therefore, for any R_+^3 , the vector $l, [h, l], [h, [h, l]]$ spans the space R_+^3 . We find that the transfer probability function $P(t; S_1, I_1, Q_1; B)$ has continuous density $k(t; S, I, Q; S_1, I_1, Q_1) \in C^\infty(R_+ \times R_+^3 \times R_+^3)$ by applying the Hormander theorem [37].

In what follows, we examine the positivity of the kernel function $k(t; S, I, Q; S_1, I_1, Q_1)$ according to the support theorem [33]. Considering a fixed constant $T > 0$, for arbitrary point $(S_1, I_1, Q_1) \in R^3$ and function $\phi \in L^3([0, T], R)$,

$$\begin{cases} S_\phi(t) = S_1 + \int_0^t [f_1(S_\phi(s), I_\phi(s), Q_\phi(s)) - \sigma \phi f(S_\phi(s), I_\phi(s))] ds, \\ I_\phi(t) = I_1 + \int_0^t [f_2(S_\phi(s), I_\phi(s), Q_\phi(s)) + \sigma \phi f(S_\phi(s), I_\phi(s))] ds, \\ Q_\phi(t) = Q_1 + \int_0^t f_3(S_\phi(s), I_\phi(s), Q_\phi(s)) ds, \end{cases} \quad (4.1)$$

where

$$\begin{aligned} f_1(S_\phi(s), I_\phi(s), Q_\phi(s)) &= A - \beta f(S_\phi(s), I_\phi(s)) - \mu S_\phi(s) + \gamma I_\phi(s) + \rho Q_\phi(s), \\ f_2(S_\phi(s), I_\phi(s), Q_\phi(s)) &= \beta f(S_\phi(s), I_\phi(s)) - (\mu + \gamma + \delta + \alpha_1)I_\phi(s), \\ f_3(S_\phi(s), I_\phi(s), Q_\phi(s)) &= \delta I_\phi(s) - (\mu + \rho + \alpha_2)Q_\phi(s). \end{aligned}$$

We denote $X = (S, I, Q)^T$, $X_0 = (S_1, I_1, Q_1)^T$, and let $D_{X_0; \phi}$ be the function's Frechet derivative from $c \mapsto X_{\phi+c}(T)$ to X . If for $D_{X_0; \phi}$, the derivative $D_{X_0; \phi}$ has rank 3, then for $X = X_\phi(T)$, there is $k(T; S, I, Q; S_1, I_1, Q_1) > 0$. \square

Let $\psi(t) = f'_i(X_\phi(t)) + \phi g'(X_\phi(t))$, where f'_i is the Jacobi determinant of $f_i = \begin{pmatrix} f_1(S, I, Q) \\ f_2(S, I, Q) \\ f_3(S, I, Q) \end{pmatrix}$ and

$$g = \begin{pmatrix} -\sigma f(S, I) \\ \sigma f(S, I) \\ 0 \end{pmatrix}.$$

Theorem 4.2. Suppose that (Ψ_1) and (Ψ_2) hold, and that for any $(S_1, I_1, Q_1) \in \Gamma$ and $(S, I, Q) \in \Gamma$, there is $T > 0$ such that $k(T; S, I, Q; S_1, I_1, Q_1) > 0$.

Proof. We first prove that the rank of $D_{X_0, \phi}$ is 3. Since we only consider the continuous control function ϕ , then (4.1) can be rewritten as follows:

$$\begin{cases} \dot{S}_{\phi+c}(t) = f_1(S_{\phi+c}(t), I_{\phi+c}(t), Q_{\phi+c}(t)) - \sigma(\phi+c)(t)f(S_{\phi+c}(t), I_{\phi+c}(t)), \\ \dot{I}_{\phi+c}(t) = f_2(S_{\phi+c}(t), I_{\phi+c}(t), Q_{\phi+c}(t)) + \sigma(\phi+c)(t)f(S_{\phi+c}(t), I_{\phi+c}(t)), \\ \dot{Q}_{\phi+c}(t) = f_3(S_{\phi+c}(t), I_{\phi+c}(t), Q_{\phi+c}(t)). \end{cases} \quad (4.2)$$

Calculating the derivatives of the system of equations with respect to c , and setting $c = 0$, we obtain

$$\begin{pmatrix} (\dot{S}_\phi(t))'_c \\ (\dot{I}_\phi(t))'_c \\ (\dot{Q}_\phi(t))'_c \end{pmatrix} = \begin{pmatrix} \frac{\partial f_1}{\partial S} - \sigma(\phi) \frac{\partial f}{\partial S} & \frac{\partial f_1}{\partial I} - \sigma(\phi) \frac{\partial f}{\partial I} & \frac{\partial f_1}{\partial Q} \\ \frac{\partial f_2}{\partial S} + \sigma(\phi) \frac{\partial f}{\partial S} & \frac{\partial f_2}{\partial I} + \sigma(\phi) \frac{\partial f}{\partial I} & \frac{\partial f_2}{\partial Q} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial Q} \end{pmatrix} \begin{pmatrix} (S_\phi(t))'_c \\ (I_\phi(t))'_c \\ (Q_\phi(t))'_c \end{pmatrix} + \begin{pmatrix} -\sigma f(S_\phi(t), I_\phi(t)) \\ \sigma f(S_\phi(t), I_\phi(t)) \\ 0 \end{pmatrix}, \quad (4.3)$$

where

$$\begin{aligned} \left(\frac{\partial f_i}{\partial S}, \frac{\partial f_i}{\partial I}, \frac{\partial f_i}{\partial Q} \right)^T &= \left(\frac{\partial f_i(S, I, Q)}{\partial S}, \frac{\partial f_i(S, I, Q)}{\partial I}, \frac{\partial f_i(S, I, Q)}{\partial Q} \right)^T \Big|_{(S, I, Q) = (S_\phi(t), I_\phi(t), Q_\phi(t))}, \\ \left(\frac{\partial f}{\partial S}, \frac{\partial f}{\partial I} \right)^T &= \left(\frac{\partial f(S, I)}{\partial S}, \frac{\partial f(S, I)}{\partial I} \right)^T \Big|_{(S, I) = (S_\phi(t), I_\phi(t))}. \end{aligned}$$

For $i = 1, 2, 3$, set

$$\psi(t) = \begin{pmatrix} \frac{\partial f_1}{\partial S} - \sigma(\phi) \frac{\partial f}{\partial S} & \frac{\partial f_1}{\partial I} - \sigma(\phi) \frac{\partial f}{\partial I} & \frac{\partial f_1}{\partial Q} \\ \frac{\partial f_2}{\partial S} + \sigma(\phi) \frac{\partial f}{\partial S} & \frac{\partial f_2}{\partial I} + \sigma(\phi) \frac{\partial f}{\partial I} & \frac{\partial f_2}{\partial Q} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial Q} \end{pmatrix}, \quad U(t) = \begin{pmatrix} (S_\phi(t))'_c \\ (I_\phi(t))'_c \\ (Q_\phi(t))'_c \end{pmatrix}.$$

From (4.3), we can get

$$\frac{dU}{dt} = \psi(t)U + uf(S_\phi(t), I_\phi(t)), \quad (4.4)$$

where $u = (-\sigma, \sigma, 0)^T$. Since for any c , $(S_{\phi+c}(0), I_{\phi+c}(0), Q_{\phi+c}(0)) = (S_1, I_1, Q_1)$, the initial condition is established as $U(0) = (0, 0, 0)$. Define $R(t)$ as the principal fundamental matrix solution to the differential system $\frac{dU}{dt} = \psi(t)U$, and let $R(t, s) = R(t)R^{-1}(s)$ for parameters $0 \leq s \leq t \leq T$. Applying the constant variational method, we have

$$U(t) = \int_0^T R(t, s)uf(S_\phi(s), I_\phi(s))ds.$$

Then, we get

$$D_{X_0;\phi}c = \int_0^T R(T, s)uf(S_\phi(s), I_\phi(s))c(s)ds.$$

Let $\xi \in [0, T]$ and $c(t) = 1_{[T-\xi, T]}(t)$ denote the indicator function on the interval $[T - \xi, T]$. Based on the Taylor expansion,

$$R(T, s) = I + \psi(T)(s - T) + \frac{1}{2}\psi^2(T)(s - T)^2 + o((s - T)^2).$$

Hence,

$$\begin{aligned} D_{X_0;\phi}c &= \int_0^T R(T, s)uf(S_\phi(s), I_\phi(s))c(s)ds \\ &= \xi u - \frac{1}{2}\xi^2\psi(T)u + \frac{1}{6}\xi^3\psi^2(T)u + o(\xi^3), \end{aligned}$$

where

$$\begin{aligned} \psi(T)u &= \begin{pmatrix} B_{11} & B_{12} & \rho \\ B_{21} & B_{22} & 0 \\ 0 & \delta & B_{33} \end{pmatrix} \begin{pmatrix} -\sigma \\ \sigma \\ 0 \end{pmatrix} = \begin{pmatrix} B_1 \\ B_2 \\ B_3 \end{pmatrix}, \\ \psi^2(T)u &= \begin{pmatrix} D_{11} & D_{12} & D_{13} \\ D_{21} & D_{22} & D_{23} \\ D_{31} & D_{32} & D_{33} \end{pmatrix} \begin{pmatrix} -\sigma \\ \sigma \\ 0 \end{pmatrix} = \begin{pmatrix} D_1 \\ D_2 \\ D_3 \end{pmatrix}, \end{aligned}$$

with the values of each of the above parameters; see Appendix B.

By calculation, we obtain the determinant

$$| \begin{matrix} u & \psi(T)u & \psi^2(T)u \end{matrix} | = \begin{vmatrix} -\sigma & B_1 & D_1 \\ \sigma & B_2 & D_2 \\ 0 & B_3 & D_3 \end{vmatrix} = -\sigma(B_2D_3 - D_2B_3) - \sigma(B_1D_3 - D_1B_3).$$

It follows from $B_2D_3 - D_2B_3 > 0$ and $B_1D_3 - D_1B_3 > 0$ that the above determinant is less than 0. This means that $u, \psi(T)u, \psi^2(T)u$ is linearly independent everywhere on R^3 . Therefore, the rank of $D_{X_0;\phi}$ is 3.

Then, we prove that given any two points $(S_1, I_1, Q_1) \in \Gamma$ and $(S_2, I_2, Q_2) \in \Gamma$, there is a control function ϕ such that the solution of model (1.3) satisfies $(S_\phi(0), I_\phi(0), Q_\phi(0)) = (S_1, I_1, Q_1)$ and $(S_\phi(T), I_\phi(T), Q_\phi(T)) = (S_2, I_2, Q_2)$.

Let $\Upsilon_\phi = S_\phi + I_\phi + Q_\phi$, then the model (4.2) becomes

$$\begin{cases} \dot{S}_\phi(t) = g_1(S_\phi(t), \Upsilon_\phi(t), Q_\phi(t)) - \sigma\phi f(S_\phi(t), \Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)), \\ \dot{\Upsilon}_\phi(t) = g_2(S_\phi(t), \Upsilon_\phi(t), Q_\phi(t)), \\ \dot{Q}_\phi(t) = g_3(S_\phi(t), \Upsilon_\phi(t), Q_\phi(t)), \end{cases} \quad (4.5)$$

where

$$\begin{aligned} g_1(S, \Upsilon, Q) &= A - \beta f(S, \Upsilon - S - Q) - \mu S + \gamma(\Upsilon - S - Q) + \rho Q, \\ g_2(S, \Upsilon, Q) &= A - \mu\Upsilon - \alpha_1(\Upsilon - S - Q) - \alpha_2 Q, \\ g_3(S, \Upsilon, Q) &= \delta(\Upsilon - S - Q) - (\mu + \rho + \alpha_2)Q. \end{aligned} \quad (4.6)$$

Let

$$\Gamma_0 = \left\{ (S, \Upsilon, Q) \in R_+^3 : 0 < S, Q < \frac{A}{\mu}, \frac{A}{\mu + \alpha_1 + \alpha_2} < \Upsilon < \frac{A}{\mu}, S < \Upsilon, Q < \Upsilon \right\}.$$

Assume that there is $T > 0$ for any $(S_1, \Upsilon_1, Q_1) \in \Gamma_0$ and $(S_2, \Upsilon_2, Q_2) \in \Gamma_0$, such that $(S_\phi(0), \Upsilon_\phi(0), Q_\phi(0)) = (S_1, \Upsilon_1, Q_1)$ and $(S_\phi(T), \Upsilon_\phi(T), Q_\phi(T)) = (S_2, \Upsilon_2, Q_2)$.

To construct the function ϕ , we employ the subsequent methodology. We start by determining a differentiable function

$$\Upsilon_\phi : [0, T] \rightarrow \left(\frac{A}{\mu + \alpha_1 + \alpha_2}, \frac{A}{\mu} \right),$$

which satisfies $\Upsilon_\phi(0) = \Upsilon_1, \Upsilon_\phi(T) = \Upsilon_2, \dot{\Upsilon}_\phi(0) = g_2(S_1, \Upsilon_1, Q_1) = \Upsilon_3, \dot{\Upsilon}_\phi(T) = g_2(S_2, \Upsilon_2, Q_2) = \Upsilon_4$, and

$$A - (\mu + \alpha_1 + \alpha_2)\Upsilon_\phi(t) < \dot{\Upsilon}_\phi(t) < A - \mu\Upsilon_\phi(t), t \in [0, T]. \quad (4.7)$$

In order to satisfy the above condition, we divide the formulation of the function Υ_ϕ into intervals $[0, w]$, $[w, T - w]$, and $[T - w, T]$, where $0 < w < \frac{T}{2}$. Let

$$\vartheta = \frac{1}{2} \min \left\{ \Upsilon_1 - \frac{A}{\mu + \alpha_1 + \alpha_2}, \Upsilon_2 - \frac{A}{\mu + \alpha_1 + \alpha_2}, \frac{A}{\mu} - \Upsilon_1, \frac{A}{\mu} - \Upsilon_2 \right\}.$$

If $\Upsilon_\phi \in \left(\frac{A}{\mu + \alpha_1 + \alpha_2} + \vartheta, \frac{A}{\mu} - \vartheta \right)$, we have

$$A - (\mu + \alpha_1 + \alpha_2)\Upsilon_\phi(t) < -(\mu + \alpha_1 + \alpha_2)\vartheta < 0, A - \mu\Upsilon_\phi(t) > \mu\vartheta > 0. \quad (4.8)$$

Therefore, according to (4.8), we can construct a C^2 function Υ_ϕ :

$$[0, w] \rightarrow \left(\frac{A}{\mu + \alpha_1 + \alpha_2} + \vartheta, \frac{A}{\mu} - \vartheta \right),$$

which satisfies $\Upsilon_\phi(0) = \Upsilon_1, \dot{\Upsilon}_\phi(0) = \Upsilon_3, \dot{\Upsilon}_\phi(w) = 0$, and (4.7) for $t \in [0, w]$.

Meanwhile, we also can construct a C^2 function Υ_ϕ :

$$[T - w, T] \rightarrow \left(\frac{A}{\mu + \alpha_1 + \alpha_2} + \vartheta, \frac{A}{\mu} - \vartheta \right),$$

which satisfies $\Upsilon_\phi(T) = \Upsilon_2, \dot{\Upsilon}_\phi(T) = \Upsilon_4, \dot{\Upsilon}_\phi(T - w) = 0$, and (4.7) for $t \in [T - w, T]$.

$$\Upsilon_\phi : [0, w] \cup [T - w, T] \rightarrow \left(\frac{A}{\mu + \alpha_1 + \alpha_2} + \vartheta, \frac{A}{\mu} - \vartheta \right).$$

Hence, we can expand the function to a C^2 function Υ_ϕ defined on $[0, T]$, provided that T is sufficiently large, such that

$$A - (\mu + \alpha_1 + \alpha_2)\Upsilon_\phi(t) < -(\mu + \alpha_1 + \alpha_2)\vartheta < \dot{\Upsilon}_\phi(t) < \mu\vartheta < A - \mu\Upsilon_\phi(t),$$

for $t \in [w, T - w]$. Then, the function Υ_ϕ satisfies (4.7) on the interval $[0, T]$. Thus, we are able to determine two C^1 -functions S_ϕ and Q_ϕ that meet the requirements of the first equation and third equation of (4.5). Furthermore, a continuous function ϕ can be found from (4.5).

The control mapping $\phi(t) : [0, T] \rightarrow R$ is ultimately defined through the following construction:

$$\phi(t) = \frac{A - \beta f(S_\phi(t), \Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)) - \mu S_\phi(t) + \gamma(\Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)) + \rho Q_\phi(t) - \dot{S}_\phi(t)}{\sigma f(S_\phi(t), \Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t))}.$$

Therefore, on the interval $[0, T]$, we can finally get

$$\begin{aligned}\dot{S}_\phi(t) &= A - \beta f(S_\phi(t), \Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)) - \mu S_\phi(t) + \gamma(\Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)) \\ &\quad + \rho Q_\phi(t) - \sigma \phi f(S_\phi(t), \Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)), \\ \dot{\Upsilon}_\phi(t) &= A - \mu \Upsilon_\phi(t) - \alpha_1(\Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)) - \alpha_2 Q_\phi(t), \\ \dot{Q}_\phi(t) &= \delta(\Upsilon_\phi(t) - S_\phi(t) - Q_\phi(t)) - (\mu + \rho + \alpha_2) Q_\phi(t).\end{aligned}$$

Based on the above discussion, we determine ϕ such that the solutions of (4.1) satisfy $(S_\phi(0), \Upsilon_\phi(0), Q_\phi(0)) = (S_1, \Upsilon_1, Q_1)$, $(S_\phi(T), \Upsilon_\phi(T), Q_\phi(T)) = (S_2, \Upsilon_2, Q_2)$. Therefore, by supporting the theorem [28], we can conclude that for any $(S_1, I_1, Q_1) \in \Gamma$ and $(S, I, Q) \in \Gamma$, there is $T > 0$ such that $k(T; S, I, Q; S_1, I_1, Q_1) > 0$. \square

Theorem 4.3. Suppose that (Ψ_1) and (Ψ_2) hold. If $R_0^s > 1$, then the semigroup $\{P(t)\}_{t \geq 0}$ is sweeping or asymptotically stable with respect to the tight set.

Proof. Theorem 4.1 gives us that $\{P(t)\}_{t \geq 0}$ is a complete Markov semigroup with a continuous kernel for $t > 0$. It follows from Lemma 2.1 that it is sufficient to investigate the semigroup's restriction to the space $L^1(\Gamma)$. Theorem 4.2 gives us the result that for every $g \in D$,

$$\int_0^\infty P(t)g dt > 0.$$

Therefore, we deduce that the Markov semigroup $\{P(t)\}_{t \geq 0}$ is sweeping or asymptotically stable with regard to the tight set from Lemma 2.3. \square

Theorem 4.4. Suppose that (Ψ_1) , (Ψ_2) , and (Ψ_3) hold. If $R_0^s > 1$, then the semigroup $\{P(t)\}_{t \geq 0}$ is not sweeping for any tight set, and there exists a unique stationary distribution.

Proof. To eliminate sweeping, a nonnegative twice continuously differentiable function V and a closed set M must be constructed to satisfy

$$\sup_{(S, I, Q) \in \Gamma \setminus M} \varphi^* V \leq -1.$$

Denote

$$\bar{R}_0 = \frac{\beta \frac{\partial f(S^*, 0)}{\partial I}}{\mu + \gamma + \delta + \alpha_1 + \frac{1}{2} \sigma^2 \left(\frac{\partial f(S^*, 0)}{\partial I} \right)^2}.$$

Then, $\bar{R}_0 > 1$ is equivalent to $R_0^s > 1$. We define the following:

$$\bar{V}(S, I, Q) = NV_1 + V_2 + V_3, \quad (4.9)$$

where $V_1 = S + (1 + \frac{\beta_2}{\mu + \gamma + \delta + \alpha_1})I + 2Q - a_1 \ln S - a_2 \ln I$, $V_2 = -\ln S$, $V_3 = -\ln Q$, and N, a_1, a_2 are positive constants satisfying

$$a_1 = \frac{\partial f(S^*, 0)}{\partial I} \frac{1}{A\eta}, a_2 = \frac{1}{\mu + \gamma + \delta + \alpha_1 + \frac{1}{2} \sigma^2 \left(\frac{\partial f(S^*, 0)}{\partial I} \right)^2},$$

and

$$-N\xi + C_2 \leq -2, \quad (4.10)$$

where

$$\begin{aligned} \xi &= 3[(\bar{R}_0)^{\frac{1}{3}} - 1] > 0, \\ C &= NA + N \frac{\partial f(S^*, 0)}{\partial I} \frac{(\mu + \beta K + \frac{1}{2}\sigma^2 K_1^2)}{A\eta} + N\beta_1 + 2\mu + \rho + \alpha_2 + \beta K_1 + \frac{1}{2}\sigma^2 K_1^2, \\ C_1 &= \sup_{(S, I, Q) \in \Gamma} \left\{ -N(2\mu + \rho + 2\alpha_2)Q + N(2\delta + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I + C \right\}, \\ C_2 &= \sup_{(S, I, Q) \in \Gamma} \{C - N\mu S - N(2\mu + \rho + 2\alpha_2)Q\}. \end{aligned}$$

It is clear that when (S, I, Q) approaches the boundary of R_+^3 , $\bar{V}(S, I, Q)$ converges to $+\infty$. Therefore, $\bar{V}(S, I, Q)$ has a minimum value at point $(S_1, I_1, Q_1) \in \Gamma$. We can define a nonnegative C^2 -function $V(S, I, Q)$

$$V(S, I, Q) = NV_1 + V_2 + V_3 - \bar{V}(S_1, I_1, Q_1).$$

By (2.2), we set $\frac{f(S, I)}{S} \leq K_1$, where K_1 is a positive constant, then

$$\begin{aligned} \varphi^* V_1 &= A - \beta f(S, I) - \mu S + \gamma I + \rho Q + (1 + \frac{\beta_2}{\mu + \gamma + \delta + \alpha_1})(\beta f(S, I) \\ &\quad - (\mu + \gamma + \delta + \alpha_1)I) + 2(\delta I - (\mu + \rho + \alpha_2)Q) \\ &\quad - \frac{a_1}{S}(A - \beta f(S, I) - \mu S + \gamma I + \rho Q) - \frac{a_2}{I}(\beta f(S, I) - (\mu + \gamma + \delta + \alpha_1)I) \\ &\quad + \frac{a_1\sigma^2 (f(S, I))^2}{2} \frac{S^2}{S^2} + \frac{a_2\sigma^2 (f(S, I))^2}{2} \frac{I^2}{I^2} \\ &= A - \mu S - (2\mu + \rho + 2\alpha_2)Q - (\mu + \delta + \alpha_1)I \\ &\quad + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1}f(S, I) - \beta_1 - \beta_2 I + 2\delta I - \frac{a_1 A}{S} + \frac{a_1\beta f(S, I)}{S} \\ &\quad + a_1\mu - \frac{a_1\gamma I}{S} - \frac{a_1\rho Q}{S} - \frac{a_2\beta f(S, I)}{I} + a_2(\mu + \gamma + \delta + \alpha_1) \\ &\quad + \frac{a_1\sigma^2 (f(S, I))^2}{2} \frac{S^2}{S^2} + \frac{a_2\sigma^2 (f(S, I))^2}{2} \frac{I^2}{I^2} + \beta_1 \\ &\leq A - \mu S - (2\mu + \rho + 2\alpha_2)Q + (2\delta + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\ &\quad + a_1(\mu + \beta K_1 + \frac{1}{2}\sigma^2 K_1^2) + a_2(\mu + \gamma + \delta + \alpha_1 + \frac{1}{2}\sigma^2 (\frac{\partial f(S^*, 0)}{\partial I})^2) \\ &\quad - ((\beta_1 + \beta_2 I) + \frac{a_1 A}{S} + \frac{a_2\beta f(S, I)}{I}) + \beta_1. \end{aligned}$$

Using $a + b + c \geq 3(abc)^{\frac{1}{3}}$ and combining (2.1), we obtain

$$\varphi^* V_1 \leq A - \mu S - (2\mu + \rho + 2\alpha_2)Q + (2\delta + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I$$

$$\begin{aligned}
& +a_1(\mu + \beta K_1 + \frac{1}{2}\sigma^2 K_1^2) + a_2(\mu + \gamma + \delta + \alpha_1 + \frac{1}{2}\sigma^2(\frac{\partial f(S^*, 0)}{\partial I})^2) \\
& -3(a_1 a_2 \beta A \frac{(\beta_1 + \beta_2 I)f(S, I)}{SI})^{\frac{1}{3}} + \beta_1 \\
& \leq A - \mu S - (2\mu + \rho + 2\alpha_2)Q + (2\delta + \frac{\beta_2 \beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\
& + \frac{\partial f(S^*, 0)}{\partial I} \frac{(\mu + \beta K_1 + \frac{1}{2}\sigma^2 K_1^2)}{A\eta} - 2 - 3[(\bar{R}_0)^{\frac{1}{3}} - 1] + \beta_1 \\
& \leq A - \mu S - (2\mu + \rho + 2\alpha_2)Q + (2\delta + \frac{\beta_2 \beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\
& + \frac{\partial f(S^*, 0)}{\partial I} \frac{(\mu + \beta K_1 + \frac{1}{2}\sigma^2 K_1^2)}{A\eta} - \xi + \beta_1, \\
\varphi^* V_2 & = -\frac{1}{S}(A - \beta f(S, I) - \mu S + \gamma I + \rho Q) + \frac{\sigma^2 (f(S, I))^2}{2S^2} \\
& \leq -\frac{A}{S} + \beta K_1 + \mu - \frac{\gamma I}{S} - \frac{\rho Q}{S} + \frac{1}{2}\sigma^2 K_1^2, \\
\varphi^* V_3 & = -\frac{1}{Q}(\delta I - (\mu + \rho + \alpha_2)Q) = -\frac{\delta I}{Q} + \mu + \rho + \alpha_2.
\end{aligned}$$

Therefore,

$$\begin{aligned}
\varphi^* V & \leq NA - N\mu S - N(2\mu + \rho + 2\alpha_2)Q + N(2\delta + \frac{\beta_2 \beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\
& + N \frac{\partial f(S^*, 0)}{\partial I} \frac{(\mu + \beta K_1 + \frac{1}{2}\sigma^2 K_1^2)}{A\eta} - N\xi + N\beta_1 - \frac{A}{S} + \beta K_1 + 2\mu \\
& - \frac{\gamma I}{S} - \frac{\rho Q}{S} + \frac{1}{2}\sigma^2 K_1^2 - \frac{\delta I}{Q} + \rho + \alpha_2 \\
& \leq -N\xi - N\mu S - N(2\mu + \rho + 2\alpha_2)Q + N(2\delta + \frac{\beta_2 \beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\
& + C - \frac{A}{S} - \frac{\gamma I}{S} - \frac{\rho Q}{S} - \frac{\delta I}{Q}.
\end{aligned}$$

Define a closed set M in Γ as follows:

$$M = \{(S, I, Q) \in \Gamma : b \leq S \leq S^*, b \leq I \leq S^*, b^2 \leq Q \leq S^*\},$$

where $b > 0$ is a sufficiently small constant and the assumption is

$$\begin{aligned}
& -N\xi - \frac{A}{b} + C_1 \leq -1, \\
& b \leq \frac{1}{N(2\delta + \frac{\beta_2 \beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})}, \\
& -N\xi - \frac{\delta}{b} + C_1 \leq -1.
\end{aligned}$$

Subsequently, $\Gamma \setminus M$ will be decomposed into three different subsets

$$\begin{aligned}
M_1 & = \{(S, I, Q) \in \Gamma : 0 < S < b\}, \\
M_2 & = \{(S, I, Q) \in \Gamma : S \geq b, 0 < I < b\}, \\
M_3 & = \{(S, I, Q) \in \Gamma : S \geq b, I \geq b, 0 < Q < b^2\}.
\end{aligned}$$

Case 1. If $(S, I, Q) \in M_1$, we have

$$\begin{aligned}\varphi^*V &\leq -N\xi - N\mu S - N(2\mu + \rho + 2\alpha_2)Q + N(2\delta + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\ &\quad + C - \frac{A}{S} - \frac{\gamma I}{S} - \frac{\rho Q}{S} \\ &\leq -N\xi - N\mu S + C_1 - \frac{A}{S} \leq -N\xi + C_1 - \frac{A}{b} \leq -1.\end{aligned}\quad (4.11)$$

Case 2. If $(S, I, Q) \in M_2$, we have

$$\begin{aligned}\varphi^*V &\leq -N\xi - N\mu S - N(2\mu + \rho + 2\alpha_2)Q + C + N(2\delta + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\ &\quad - \frac{\gamma I}{S} - \frac{\delta I}{Q} \\ &\leq -N\xi + C_2 + N(2\delta + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})b \leq -1.\end{aligned}\quad (4.12)$$

Case 3. If $(S, I, Q) \in M_3$, we have

$$\begin{aligned}\varphi^*V &\leq -N\xi - N\mu S - N(2\mu + \rho + 2\alpha_2)Q + N(2\delta + \frac{\beta_2\beta}{\mu + \gamma + \delta + \alpha_1} \frac{\partial f(S^*, 0)}{\partial I})I \\ &\quad + C - \frac{\rho Q}{S} - \frac{\delta I}{Q} \\ &\leq -N\xi + C_1 - \frac{\delta}{b} \leq -1.\end{aligned}\quad (4.13)$$

Hence, from (4.11), (4.12), (4.13), we can obtain $b > 0$ sufficiently small such that $\varphi^*V \leq -1$ is not sweeping for all $(S, I, Q) \in \Gamma \setminus M$. Then, due to the argument of [26], we may determine that the semigroup $\{P(t)\}_{t \geq 0}$ is not sweeping for any tight set because of the presence of the Khasminskii function. \square

5. Numerical simulation

This part will involve some examples to test the previous theory. These examples are based on the model (1.3) that we constructed and discussed in Sections 3 and 4. For specific approaches to numerical simulation, we refer to Carletti et al [38] as well as Higman [39]. Furthermore, the discretized system corresponding to model (1.3) is shown below

$$\begin{cases} S_{k+1} = S_k + [A - \beta f(S_k, I_k) - \mu S_k + \gamma I_k + \rho Q_k] \Delta t \\ \quad - \sigma f(S_k, I_k) [\sqrt{\Delta t} \xi_k + \frac{1}{2} \sigma (\xi_k^2 - 1) \Delta t], \\ I_{k+1} = I_k + [\beta f(S_k, I_k) - (\mu + \gamma + \delta + \alpha_1) I_k] \Delta t \\ \quad + \sigma f(S_k, I_k) [\sqrt{\Delta t} \xi_k + \frac{1}{2} \sigma (\xi_k^2 - 1) \Delta t], \\ Q_{k+1} = Q_k + [\delta I_k - (\mu + \rho + \alpha_2) Q_k] \Delta t, \end{cases}$$

where the time increment $\Delta t > 0$, ξ_k is an independent Gaussian random variable, which adheres to the distribution $N(0, 1)$, $k = 1, 2, \dots, n$.

Before carrying out numerical simulation, we perform a sensitivity analysis of the relevant parameters of R_0^s by means of the PRCC (partial rank correlation coefficient) values [40]. From

Table 2. Parameters and values.

Parameters	Example 1	Example 2	Example 3
$f(S, I)$	$\frac{SI}{S+I}$	$\frac{SI}{1+\lambda_2 I^2}$	$\frac{SI}{1+\lambda_1 S+\lambda_2 I}$
A	0.25	0.32	0.18
β	2.1	1.6	1.3
μ	0.05	0.12	0.1
δ	0.35	0.32	0.25
γ	0.3	0.28	0.2
ρ	0.01	0.2	0.15
α_1	0.17	0.1	0.23
α_2	0.15	0.12	0.25
λ_1	0	0	0.1
λ_2	0	0.2	0.2

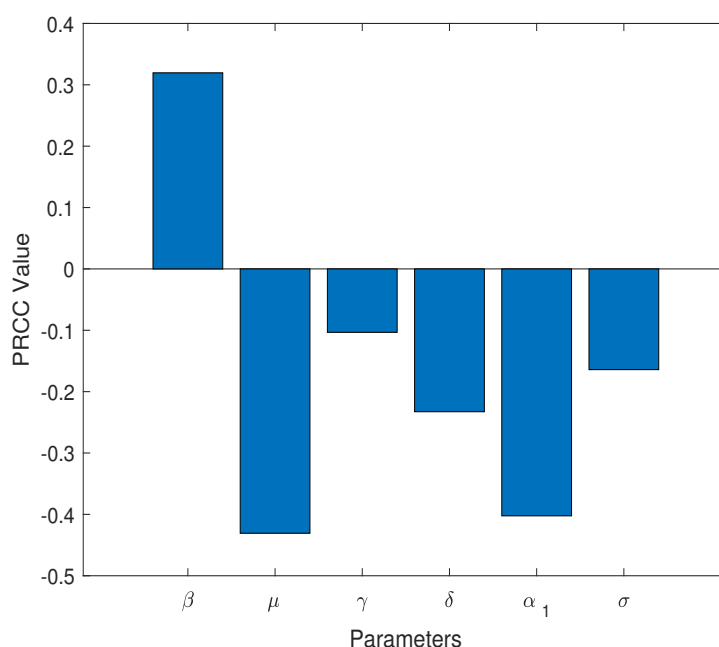
**Figure 1.** The PRCC values for parameters related to R_0^s .

Figure 1, it can be seen that parameters μ , γ , δ , α_1 , σ have negative correlation with R_0^s and parameter β has positive correlation with R_0^s . This means that increasing β may lead to disease outbreaks; on the contrary, increasing μ , γ , δ , α_1 , σ favor blocking the spread of the disease.

Initially, in order to test our theory, we investigate the dynamical behavior of the three nonlinear incidence rates in model (1.3) (satisfying assumptions (Ψ_1) , (Ψ_2) , and (Ψ_3)), i.e., standard incidence (Example 1), non-monotonic incidence (Example 2), and Beddington-DeAngelis incidence (Example 3). The next values taken are shown in Table 2.

Example 1 It is clear that (Ψ_1) and (Ψ_2) hold. When $\beta_1 = 0.5$, $\beta_2 = 0$, $\eta = 0.1$, (Ψ_3) holds. Further, we obtain $R_0^s = 2.4138 - \sigma^2 0.5747$.

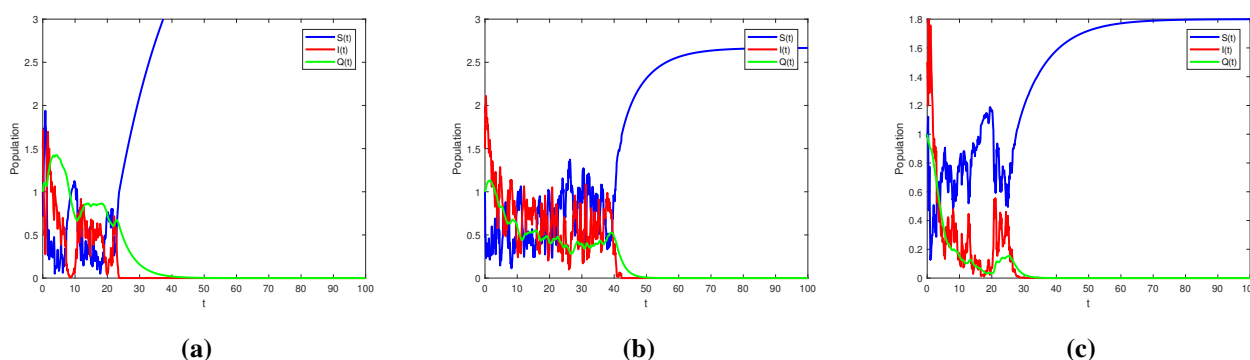


Figure 2. Paths of $I(t)$ of model (1.3) for different σ with initial values of $(S_0, I_0, Q_0) = (1, 1.5, 1)$. (a) Standard incidence ($\sigma = 1.6$); (b) Non-monotonic incidence ($\sigma = 1.0$); (c) Beddington-DeAngelis incidence ($\sigma = 1.1$).

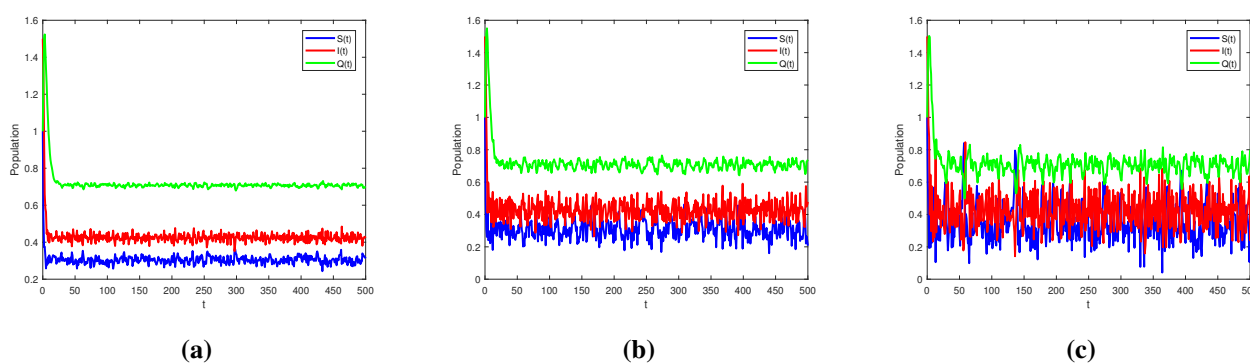


Figure 3. Standard incidence: paths of $S(t)$, $I(t)$, and $Q(t)$ of model (1.3) for different noise intensities (a) $\sigma = 0.1$, (b) $\sigma = 0.3$, (c) $\sigma = 0.5$ with initial values of $(S_0, I_0, Q_0) = (1, 1.5, 1)$.

When σ takes the value of 1.6, the corresponding R_0^s is 0.9427 and the numerical simulation is shown in Figure 2(a). The corresponding R_0^s is 2.4081, 2.3621, and 2.2701 when σ takes the values 0.1, 0.3, and 0.5, respectively, and the numerical simulations are shown in Figures 3 and 4.

Example 2 It is obvious that (Ψ_1) and (Ψ_2) hold. When $\beta_1 = 0.5, \beta_2 = 0, \eta = 0.1$, (Ψ_3) holds. Further, we obtain $R_0^s = 5.2033 - \sigma^2 4.3362$.

When σ takes the value of 1.0, the corresponding R_0^s is 0.8671 and the numerical simulation is shown in Figure 2(b). The corresponding R_0^s is 5.1599, 4.8130, and 4.1193 when σ takes the values 0.1, 0.3, and 0.5, respectively, and the numerical simulations are shown in Figures 5 and 6.

Example 3 It is evident that (Ψ_1) and (Ψ_2) hold. When $\beta_1 = 0.5, \beta_2 = 0, \eta = 0.1$, (Ψ_3) holds. Further, we obtain $R_0^s = 2.5424 - \sigma^2 1.4916$.

When σ takes the value of 1.1, the corresponding R_0^s is 0.7376 and the numerical simulation is shown in Figure 2(c). The corresponding R_0^s is 2.5275, 2.4082, and 2.1695 when σ takes the values 0.1, 0.3, and 0.5, respectively, and the numerical simulations are shown in Figures 7 and 8.

Next, we simulate the infected population $I(t)$ in model (1.3) using different isolation rates δ . The other parameters are detailed in Table 2, and the numerical simulations are shown in Figure 9.

We observe that the behavior of susceptible, infected, and isolates may differ significantly when the noise intensity is different, and higher noise may lead to greater volatility, but even so, these paths

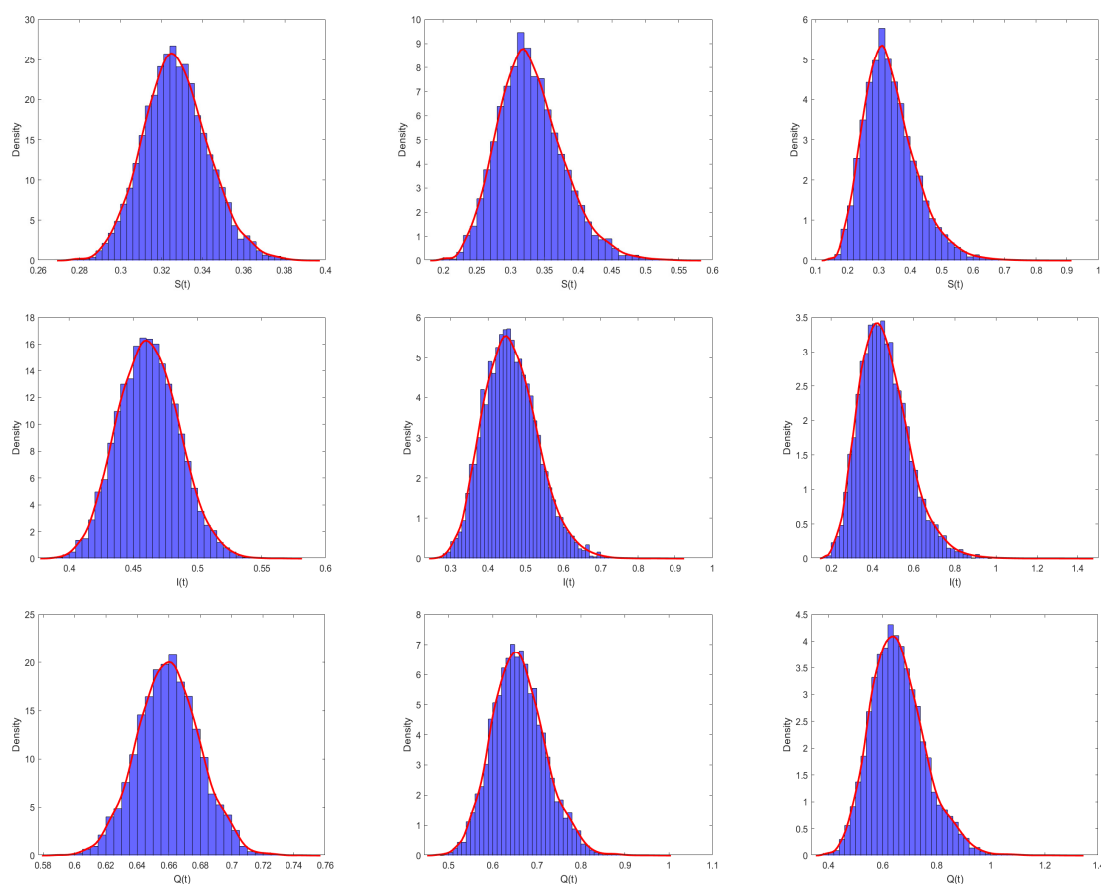


Figure 4. Standard incidence: histograms and stationaried curves of the probability density functions of $S(t)$, $I(t)$, and $Q(t)$ for the stochastic model (1.3) with noise intensities $\sigma = 0.1$ (left panel), $\sigma = 0.3$ (middle panel), and $\sigma = 0.5$ (right panel), respectively.

still show some stability. Figures 4, 6, and 8 show histograms and stationaried curves of the respective probability density functions of susceptible, infected, and isolated at different nonlinear incidence rates and different noise intensities. The form of the probability density function can be seen to change under different noise intensities (the left, middle, and right panels correspond to different levels of noise, respectively): the stronger the noise, the distribution may become broader, with a lower peak reflecting a greater degree of uncertainty, suggesting that stochastic factors increase the variability of the system; however, it forms a stable form of the distribution, suggesting that, even under the influence of stochastic factors, the system is able to maintain its characteristic long-term behavior pattern.

In the case of standard incidence, non-monotonic incidence, and Beddington-DeAngelis incidence, it is shown that when $R_0^S < 1$, the disease will become extinct (see Figure 2). In addition to this, the susceptible population will increase when the infected population becomes extinct. This is due to the fact that the susceptible population will not be infected. From the model (1.3), the source of the isolated population is only the infected population, so the isolated population also becomes zero after a period of time when the infected population becomes extinct. When $R_0^S > 1$, it will be model (1.3) that there exists stationary distribution, and the disease will persist for a long time (see Figures 3, 5, and 7).

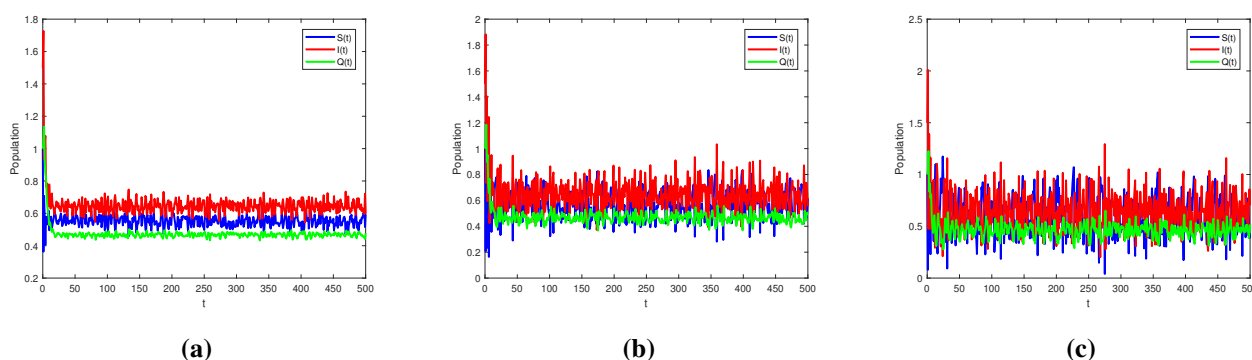


Figure 5. Non-monotonic incidence: paths of $S(t)$, $I(t)$, and $Q(t)$ of model (1.3) for different noise intensities (a) $\sigma = 0.1$, (b) $\sigma = 0.3$, (c) $\sigma = 0.5$ with initial values of $(S_0, I_0, Q_0) = (1, 1.5, 1)$.

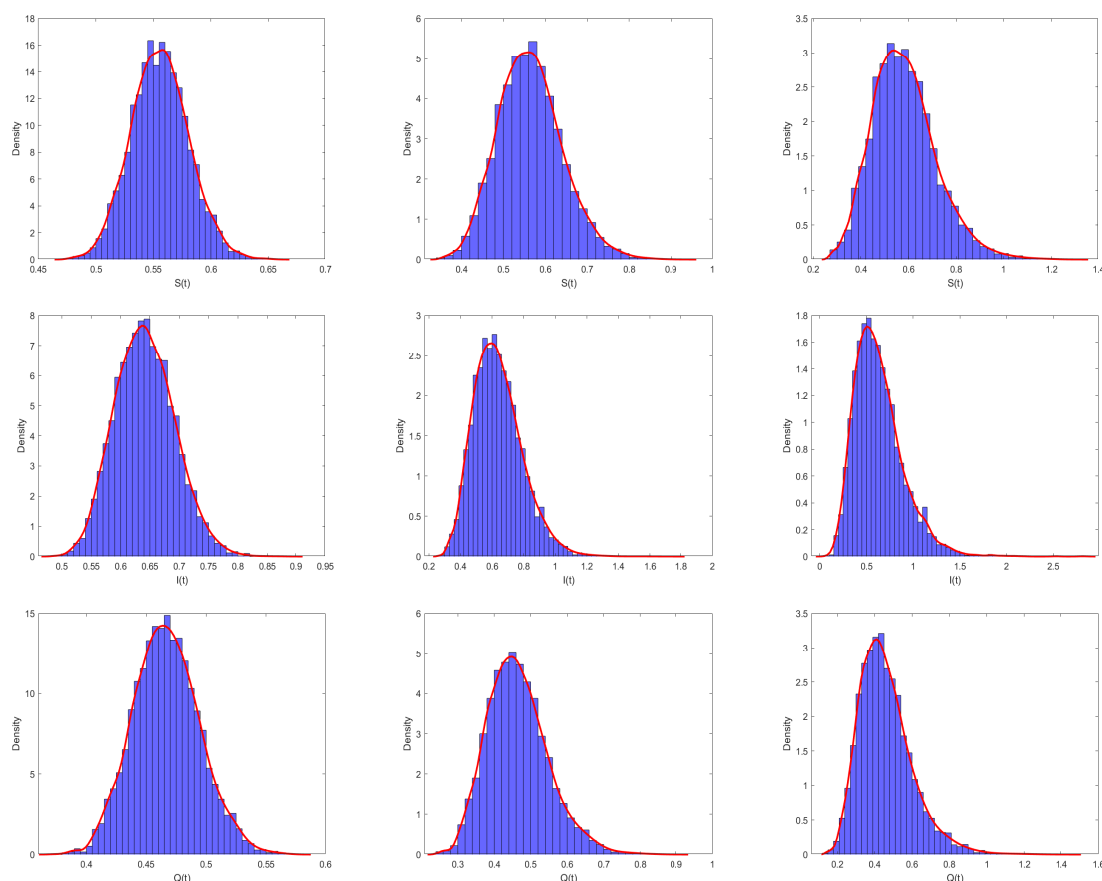


Figure 6. Non-monotonic incidence: histograms and stationary curves of the probability density functions of $S(t)$, $I(t)$, and $Q(t)$ for the stochastic model (1.3) with noise intensities $\sigma = 0.1$ (left panel), $\sigma = 0.3$ (middle panel), and $\sigma = 0.5$ (right panel), respectively.

When the noise intensity σ is set to 0.1, the stochastic fluctuations within the model (1.3) are minimal, resulting in trajectories that exhibit relatively stationary behavior. In contrast, as σ increases to 0.3

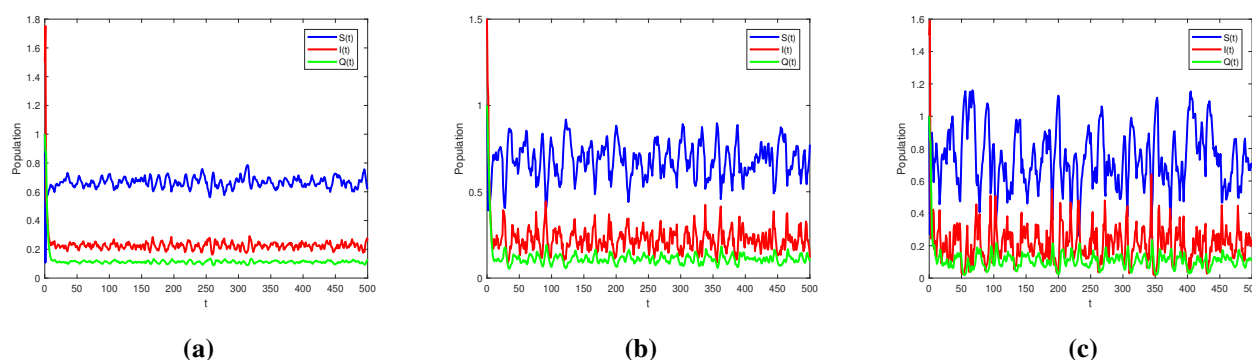


Figure 7. Beddington-DeAngelis incidence: paths of $S(t)$, $I(t)$, and $Q(t)$ of model (1.3) for different noise intensities (a) $\sigma = 0.1$, (b) $\sigma = 0.3$, (c) $\sigma = 0.5$ with initial values of $(S_0, I_0, Q_0) = (1, 1.5, 1)$.

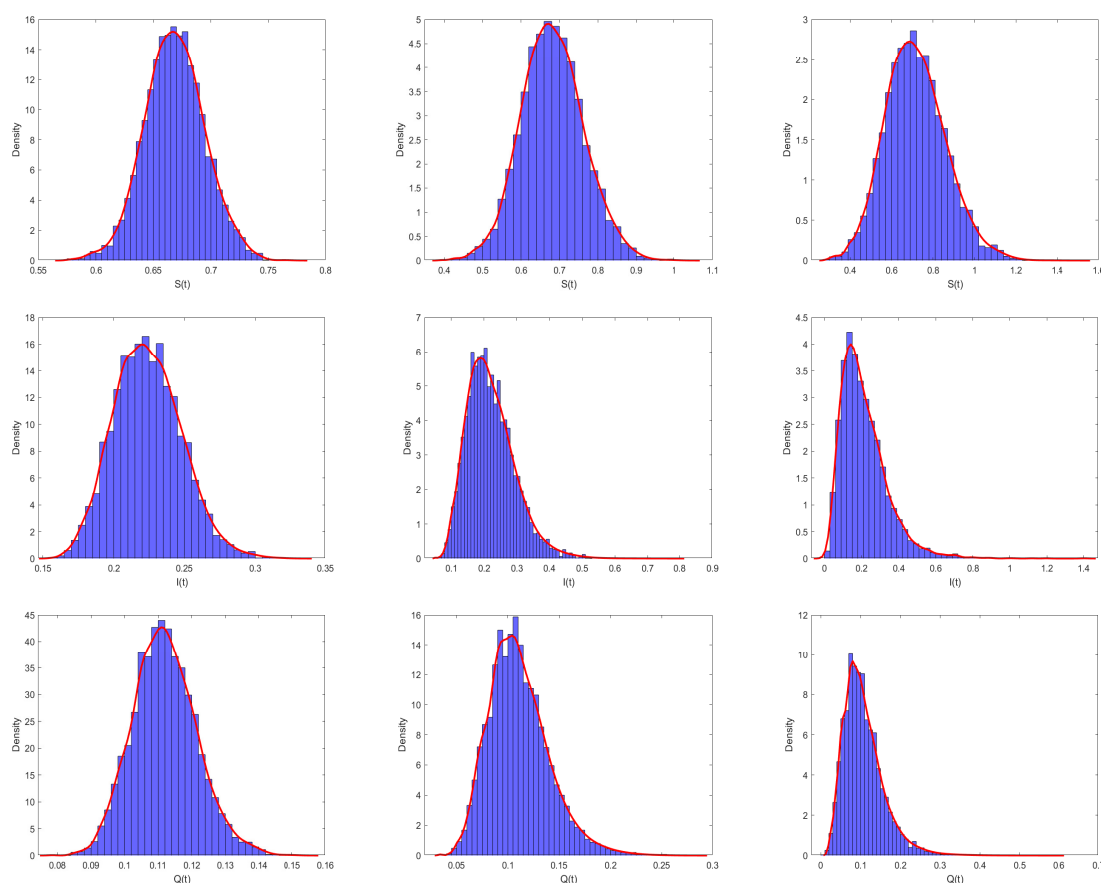


Figure 8. Beddington-DeAngelis incidence: histograms and stationaried curves of the probability density functions of $S(t)$, $I(t)$, and $Q(t)$ for the stochastic model (1.3) with noise intensities $\sigma = 0.1$ (left panel), $\sigma = 0.3$ (middle panel), and $\sigma = 0.5$ (right panel), respectively.

and further to 0.5, the model (1.3) experiences heightened stochastic perturbations, leading to more pronounced irregularity in the trajectories. This demonstrates that the influence of stochastic factors

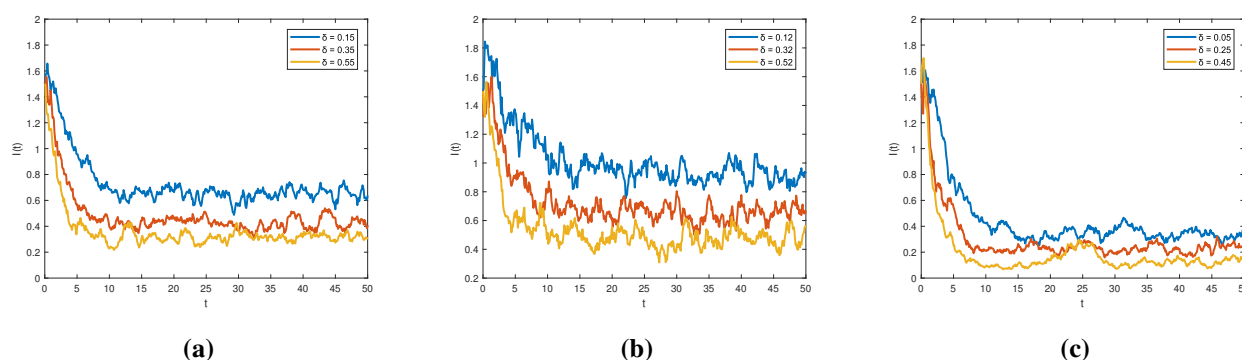


Figure 9. Paths of $I(t)$ of model (1.3) for different δ with initial values of $(S_0, I_0, Q_0) = (1, 1.5, 1)$. (a) Standard incidence; (b) Non-monotonic incidence; (c) Beddington-DeAngelis incidence.

on the model (1.3) becomes increasingly significant with higher noise intensities. In addition, it can be seen that as δ increases, the number of infected population $I(t)$ will decrease (see Figure 9). Combining with the perspective of the threshold \bar{R}_0 , where an increase in δ implies a decrease in \bar{R}_0 , the disease will become extinct, which means that isolation measures can reduce the spread of the disease.

6. Conclusions

Building upon the deterministic SIQS model framework established by Herbert et al. [13], this study incorporates nonlinear incidence rates and stochastic perturbations acting on the transmission coefficient to construct and analyze a stochastic SIQS infectious disease dynamics model. Compared to the stochastic SIQS model proposed by Wei et al. [11], which employs a saturated incidence rate and confines stochastic perturbations to fluctuations around the disease-free equilibrium, our work incorporates a broad class of nonlinear incidence functions. This approach overcomes the limitation of the specific saturated functional form used in [11], significantly enhancing the model's capacity to describe complex transmission mechanisms. Furthermore, distinct from the equilibrium-centric perturbations in [11], our model directly applies environmental noise to the core transmission parameter (the transmission coefficient). This formulation more realistically captures the fundamental impact of stochastic factors on the infection process. The specific investigations conducted are detailed as follows:

First, we obtain the condition for the extinction of infectious disease, i.e., $R_0^s < 1$ (see Theorem 3.1). Subsequently, through Markov semigroup theory, we derive the related Theorems 4.1–4.3. Meanwhile, a suitable Liapunov function is constructed to verify the sufficient condition for the model (1.3) to reach a stationary distribution, i.e., $R_0^s > 1$ (see Theorem 4.4). In this way, we hope to provide new perspectives and tools for understanding and predicting the development of such infectious diseases.

Furthermore, since $f(S(t)I(t))$ is not specified as a concrete function, we support our theoretical findings by introducing three specific forms. Figures 3 and 4 illustrate the dynamics of disease under standard incidence, Figures 5 and 6 depict the dynamics of disease under non-monotonic incidence, and Figures 7 and 8 demonstrate the dynamics of disease under Beddington-DeAngelis incidence. Finally, Figure 9 reveals that as the isolation rate δ increases, the disease transmission rate decreases.

The paper not only improves the understanding of the transmission mechanism of complex infectious diseases, but also provides a scientific basis for the development of effective intervention strategies. By adjusting key parameters in the model (e.g., transmission coefficient, isolation rate, etc.), the effectiveness of different prevention and control measures can be evaluated, and, thus, the allocation of resources can be optimized to control the spread of the epidemic. In conclusion, this work highlights the importance of incorporating nonlinear incidence rate into infectious disease modeling, and validates the proposed model through rigorous mathematical derivation and exhaustive numerical experiments. Future work will further explore the potential of applying the model in more complex real-world scenarios involving multiple stochastic factors and dynamics, e.g., the introduction of Markov chains or the incorporation of jump noise. All of this is the work we need to do in the future.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there is no conflicts of interest.

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Appendices

Appendix A. The values of F_1 , F_2 , and F_3

$$\begin{aligned}
 F_1 = & [A - \beta f(S, I) - \mu S + \gamma I + \rho Q][(A - \mu S + \gamma I + \rho Q)(-\sigma v_5) \\
 & + ((\mu + \gamma + \delta + \alpha_1)I)\sigma v_4 - \gamma \sigma v_1] \\
 & - [(A - \mu S + \gamma I + \rho Q)(-\sigma v_1) + (\mu + \gamma + \delta + \alpha_1)I\sigma v_2] \\
 & - \mu \sigma f(S, I) - \gamma \sigma f(S, I)](-\beta v_1 - \mu) + (\beta f(S, I) \\
 & - (\mu + \gamma + \delta + \alpha_1)I)[- \gamma \sigma v_1 + (A - \mu S + \gamma I + \rho Q)(-\sigma v_3 \\
 & + (\delta + \alpha_1)\sigma v_2 + (\mu + \gamma + \delta + \alpha_1)I\sigma v_6] \\
 & - [(A - \mu S + \gamma I + \rho Q)\sigma v_1 - ((\mu + \gamma + \delta + \alpha_1)I)\sigma v_2 \\
 & + (\mu + \gamma + \delta + \alpha_1)\sigma f(S, I)](-\beta v_2 + \gamma) \\
 & + (\delta I - (\mu + \rho + \alpha_2)Q)(-\rho \sigma v_1) + \delta \sigma f(S, I)\rho,
 \end{aligned}$$

$$\begin{aligned}
 F_2 = & [A - \beta f(S, I) - \mu S + \gamma I + \rho Q][(A - \mu S + \gamma I + \rho Q)\sigma v_5 \\
 & - ((\mu + \gamma + \delta + \alpha_1)I)\sigma v_4 + (\gamma + \delta + \alpha_1)\sigma v_1] \\
 & + [(A - \mu S + \gamma I + \rho Q)\sigma v_1 + (\mu + \gamma + \delta + \alpha_1)I\sigma v_2 - \mu \sigma f(S, I) \\
 & - \gamma \sigma f(S, I)]\beta v_1 + [\beta f(S, I) - (\mu + \gamma + \delta + \alpha_1)I][\gamma \sigma v_1 \\
 & + (A - \mu S + \gamma I + \rho Q)\sigma v_3 - (\mu + \gamma + \delta + \alpha_1)I\sigma v_6] \\
 & - [(A - \mu S + \gamma I + \rho Q)\sigma v_1 - (\mu + \gamma + \delta + \alpha_1)I\sigma v_2 \\
 & + (\mu + \gamma + \delta + \alpha_1)\sigma f(S, I)][\beta v_2 - (\mu + \gamma + \delta + \alpha_1)] \\
 & + (\delta I - (\mu + \rho + \alpha_2)Q)\rho \sigma v_1,
 \end{aligned}$$

$$\begin{aligned}
 F_3 = & [A - \beta f(S, I) - \mu S + \gamma I + \rho Q](-\delta \sigma v_1) \\
 & + [\beta f(S, I) - (\mu + \gamma + \delta + \alpha_1)I](-\delta \sigma v_2) \\
 & - [(A - \mu S + \gamma I + \rho Q)\sigma v_1 - (\mu + \gamma + \delta + \alpha_1)I\sigma v_2 \\
 & + (\mu + \gamma + \delta + \alpha_1)\sigma f(S, I)]\delta - \delta \sigma (\mu + \rho + \alpha_2)f(S, I).
 \end{aligned}$$

Appendix B. The values of each of the parameters

$$\begin{aligned}
 B_{11} &= -(\beta + \sigma\phi)v_1 - \mu, B_{12} = -(\beta + \sigma\phi)v_2 + \gamma, \\
 B_{21} &= (\beta + \sigma\phi)v_1, B_{22} = (\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1), \\
 B_{33} &= -(\mu + \rho + \alpha_2), B_1 = \sigma[(\beta + \sigma\phi)v_1 + \mu] - \sigma[(\beta + \sigma\phi)v_2 - \gamma], \\
 B_2 &= -\sigma(\beta + \sigma\phi)v_1 + \sigma[(\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1)], B_3 = \sigma\delta. \\
 D_{11} &= [(\beta + \sigma\phi)v_1 + \mu]^2 - [(\beta + \sigma\phi)v_2 - \gamma](\beta + \sigma\phi)v_1, \\
 D_{12} &= [(\beta + \sigma\phi)v_1 + \mu][(\beta + \sigma\phi)v_2 - \gamma] - [(\beta + \sigma\phi)v_2 - \gamma] \\
 &\quad [(\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1)] + \rho\delta, \\
 D_{13} &= -[(\beta + \sigma\phi)v_1 + \mu]\rho - (\mu + \rho + \alpha_2)\rho, \\
 D_{21} &= -[(\beta + \sigma\phi)v_1 + \mu][(\beta + \sigma\phi)v_1] \\
 &\quad + [(\beta + \sigma\phi)v_1][(\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1)], \\
 D_{22} &= -[(\beta + \sigma\phi)v_2 - \gamma][(\beta + \sigma\phi)v_1] \\
 &\quad + [(\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1)]^2, \\
 D_{23} &= (\beta + \sigma\phi)v_1\rho, D_{31} = (\beta + \sigma\phi)v_1\delta, \\
 D_{32} &= [(\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1)]\delta - (\mu + \rho + \alpha_2)\delta, D_{33} = (\mu + \rho + \alpha_2)^2, \\
 D_1 &= -\sigma[(\beta + \sigma\phi)v_1 + \mu]^2 - ((\beta + \sigma\phi)v_2 - \gamma)(\beta + \sigma\phi)v_1 \\
 &\quad + \sigma[(\beta + \sigma\phi)v_1 + \mu)((\beta + \sigma\phi)v_2 - \gamma) \\
 &\quad - ((\beta + \sigma\phi)v_2 - \gamma)((\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1)) + \rho\delta], \\
 D_2 &= \sigma[(\beta + \sigma\phi)v_1 + \mu)((\beta + \sigma\phi)v_1) \\
 &\quad + ((\beta + \sigma\phi)v_1)((\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1))] \\
 &\quad - \sigma[(\beta + \sigma\phi)v_2 - \gamma)((\beta + \sigma\phi)v_1) \\
 &\quad + ((\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1))^2], \\
 D_3 &= -\sigma(\beta + \sigma\phi)v_1\delta + \sigma[(\beta + \sigma\phi)v_2 - (\mu + \gamma + \delta + \alpha_1)]\delta - (\mu + \rho + \alpha_2)\delta].
 \end{aligned}$$



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