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Research article

An investigation on the dynamics of a stochastic SEIR epidemic model with psychological effects and Ornstein-Uhlenbeck perturbations

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Abstract: This work proposed a stochastic SEIR epidemic model incorporating psychological effects coupled with an Ornstein-Uhlenbeck (OU) process. The model focuses on the fact that the incidence rate of diseases is disturbed by environmental noise, and it is characterized by a mean-reverting OU process. Unlike traditional white noise, OU noise is consistent with the continuity and stability characteristics of real-world environmental disturbances. First, the existence and stability of equilibria for the corresponding deterministic model were discussed, laying the foundation for the subsequent dynamical analysis. Second, utilizing Itô's formula, the existence and uniqueness of a globally nonnegative solution for the stochastic model were proven, and the suffcient condition for disease extinction was discussed. Subsequently, by constructing appropriate Lyapunov functions and employing ergodic theory, the sufficient condition for the existence of a stationary distribution was established, namely, $R_0^s > 1$, which characterizes the stochastic persistence of the disease. Furthermore, by linearizing the stochastic system around the endemic equilibrium and solving the resulting covariance matrix equation, we derived an explicit expression for the probability density function characterizing the stationary distribution in the vicinity of the endemic equilibrium. Finally, we employed numerical simulations to validate the theoretical findings and to investigate the influence of fluctuation intensity on the system's dynamics.

Keywords: SEIR model; Ornstein-Uhlenbeck process; extinction; stationary distribution; probability density function

1. Introduction

The core of epidemic dynamic modeling lies in the quantitative description of the disease transmission process within a population. The incidence rate function, which characterizes the contact dynamics between susceptible (S) and infected (I) individuals leading to new infections, serves as a

key mathematical determinant of the model's properties and predictive capability. Different disease transmission mechanisms, contact patterns, and intervention measures require distinct forms of incidence rate functions for accurate representation. Under the assumption that the number of susceptible individuals that a patient can infect within a unit time is proportional to the total number of susceptible individuals in this environment, and the population size is relatively stable, with uniform mixing of individual contact behaviors and no significant influence from population density, then the bilinear incidence rate βSI is often adopted, where $\beta > 0$ is the infection rate coefficient. The bilinear incidence rate was first introduced by Kermack and McKendrick to study the Great Plague outbreak in London in 1665 and the plague epidemic in Bombay in 1906, forming the foundation of the renowned Susceptible-Infected-Recovered (SIR) compartmental model [1]. If the population size is large and the number of contacts an infected individual can make per unit time is inherently limited, assuming that the contact rate is constant and independent of the total population N, in this case, then the incidence rate function is typically formulated as $\beta\frac{SI}{N}$. This functional form is referred to as the standard incidence rate. Anderson and May, among others, have noted that for human populations and certain social animal species, the standard incidence rate provides a more realistic representation of transmission dynamics than the bilinear incidence rate [2, 3]. For emerging and sudden infectious diseases such as SARS, COVID-19, and Ebola hemorrhagic fever, which trigger widespread public vigilance and prompt strong intervention measures, the effective contact rate exhibits saturation-type behavior with respect to the infected population I, increasing more slowly and eventually approaching an upper bound. In such cases, the saturated incidence rate $\beta\frac{SI}{1+\alpha I}$ is appropriate for modeling, where the saturation term αI reflects the intensity of inhibitory effects. Capasso and Serio [4] first systematically introduced the saturated incidence rate into the Kermack–McKendrick framework when studying the cholera outbreak in Bari, Italy in 1973, to explain the saturation phenomenon of the contact rate in large-scale epidemics. In the era of highly developed media, for infectious diseases such as H1N1 influenza and COVID-19 that receive extensive coverage, transmission dynamics are significantly influenced by population-wide psychological effects and changes in individual preventive behaviors due to information dissemination. To characterize the increased risk awareness and enhanced self-protective behavior in response to rising infection numbers, Xiao and Ruan proposed a nonmonotone incidence rate $\frac{\beta SI}{1+\alpha I^2}$, where α is a parameter measuring psychological effects or inhibitory impacts. This formulation was employed to develop an SIR epidemic model and conduct a global stability analysis of the system [5].

The transmission of infectious diseases is inevitably influenced by numerous stochastically changing external factors, such as climatic variations, viral mutations, and adjustments in control policies. These factors induce fluctuations in key parameters including infection rates and recovery rates. The fixed parameter settings in deterministic models are unable to capture the impact of such fluctuations on epidemic dynamics, whereas stochastic differential systems can incorporate such uncertainty through the introduction of stochastic terms [6]. Stochastic epidemic models commonly incorporate environmental variability via two primary forms of perturbation: white noise and OU noise. If a system is subject to instantaneous uncorrelated disturbances–such as minor random variations in daily contact numbers, transient measurement errors in case reporting, or unpredictable environmental changes over short time scales–white noise perturbation is an appropriate modeling choice [7, 8]. If a system exhibits persistent environmental fluctuations, such as seasonal or climatic variations, gradual changes in the intensity of public health interventions over time, or stochastic

variations in population mobility or behavioral tendencies, OU noise is more appropriate for modeling [9, 10]. Continuous-time white noise $W(t)$ is typically defined as the formal derivative of Brownian motion $B(t)$:

$$W(t) = \frac{dB(t)}{dt},$$

where $B(t)$ is also known as the Wiener process. The mean of $W(t)$ is $E(W(t)) = 0$, and its autocovariance function is

$$E(W(t)W(s)) = \sigma^2\delta(t-s),$$

where δ is the Dirac delta function and $\sigma^2 > 0$ is referred to as the noise intensity. Interpreting the variance of $W(t)$ as $E[W^2(t)]$, it follows that

$$E[W^2(t)] = \sigma^2\delta(0),$$

which leads to the common statement that "white noise has infinite variance".

In 1930, the physicists Ornstein and Uhlenbeck proposed the OU process while studying the motion of Brownian particles in a frictional medium [11]. The OU process is a stationary, mean-reverting continuous-time stochastic process. A standard OU process $\{X(t), t \geq 0\}$ satisfies the following linear stochastic differential equation:

$$\mathrm{d}X(t) = \theta(\mu - X(t))\mathrm{d}t + \sigma\mathrm{d}B(t). \tag{1.1}$$

Here, $\theta > 0$ is the mean-reversion coefficient, which determines the speed at which the process reverts to its long-term equilibrium level (a larger θ corresponds to a faster reversion). The term μ denotes the stationary mean of the process. Meanwhile, $\sigma > 0$ functions as the diffusion coefficient, governing the intensity of the stochastic fluctuations, and $\mathrm{d}B(t)$ denotes the white-noise driving term. Given the initial condition $X(0) = x_0$, the explicit solution of (1.1) is

$$X(t) = x_0e^{-\theta t} + \mu(1-e^{-\theta t}) + \sigma\int_0^t e^{-\theta(t-s)}\,\mathrm{d}B(s).$$

The mathematical expectation and variance of $X(t)$ are respectively,

$$E[X(t)] = \mu + (x_0-\mu)e^{-\theta t},$$

$$D[X(t)] = \frac{\sigma^2}{2\theta}(1-e^{-2\theta t}) + (x_0-\mu)^2e^{-2\theta t}.$$

It follows directly that as $t \to +\infty$, the OU process described by (1.1) admits a stationary normal distribution $N\left(\mu, \frac{\sigma^2}{2\theta}\right)$, with the probability density function

$$\pi(x) = \frac{\sqrt{\theta}}{\sqrt{\pi}\sigma}e^{-\frac{\theta(x-\mu)^2}{\sigma^2}}.$$

We consider that the infection rates of respiratory infectious diseases such as influenza, COVID-19, and SARS are affected by environmental noise. How can we mathematically describe this noise disturbance? If white noise is directly introduced, then $\beta(t) = \beta + \sigma\dot{B}(t)$, where $\beta(t)$ exhibits independent instantaneous fluctuations at every moment and possesses unbounded variance, a

scenario widely regarded as unrealistic [12]. In comparison, the OU process, as a mean-reverting stochastic process, more effectively captures the fluctuation characteristics of the infection rate around its long-term mean. Although the OU process is Gaussian and retains an unbounded support, its variance is finite at each time instant and remains bounded in the stationary state [13]. More importantly, it captures temporally correlated fluctuations through an exponentially decaying autocorrelation, a feature that aligns more closely with real-world environmental variability than white noise does [14–16]. Consequently, the OU process fluctuates closer to its mean, reducing the frequency of unrealistic parameter values [17]. In practice, whether due to variations in temperature and humidity or phased adjustments of control policies, the infection rate typically evolves as a slowly varying stochastic process over time, and is more appropriately described by OU noise. Considering the above, the infection rate is assumed to be perturbed by OU noise, while accounting for psychological effects on the population induced by media dissemination and a substantial increase in infections. Based on [5], the following Susceptible–Exposed–Infected–Recovered(SEIR) epidemic model is formulated:

$$\begin{cases} dS(t) = \left[\Lambda - \frac{\xi(t)S(t)I(t)}{1+\alpha I^2(t)} - \eta S(t)\right]dt, \\ dE(t) = \left[\frac{\xi(t)S(t)I(t)}{1+\alpha I^2(t)} - (\eta + k)E(t)\right]dt, \\ dI(t) = \left[kE(t) - (\eta + \delta)I(t)\right]dt, \\ dR(t) = \left[\delta I(t) - \eta R(t)\right]dt, \\ d\xi(t) = \theta(\bar{\xi} - \xi(t))dt + \sigma dB(t). \end{cases} \tag{1.2}$$

Here, $S(t)$, $E(t)$, $I(t)$, and $R(t)$ denote the numbers of susceptible, exposed, infectious, and recovered individuals at time t, respectively. The parameter $\xi(t)$ represents the infection rate coefficient at time t, modeled as an OU stochastic process, with $\bar{\xi}$ being its stationary mean. The term $\frac{1}{1+\alpha I^2}$ captures the psychological or inhibitory effect that is triggered by behavioral adaptations within the susceptible population as infection numbers rise. The parameters Λ and η denote the population input rate and natural mortality rate, respectively. The rate at which exposed individuals in the latent period progress to the infectious class is given by k, and δ represents the recovery rate of infectious individuals.

This paper investigates the long-term dynamical behavior of model (1.2). To this end, Section 2 is devoted to discussing the existence and stability of equilibrium points within the corresponding deterministic framework. Section 3 derives the sufficient condition for disease extinction. In Section 4, sufficient conditions for the existence of a stationary distribution are obtained through the construction of Lyapunov functions and the application of Itô's formula. Subsequently, in Section 5, a linearized four-dimensional stochastic system is considered, and the explicit expression for the stationary distribution's probability density function around the endemic equilibrium is obtained by solving the associated covariance algebraic equations. Section 6 interprets the theoretical results through specific examples and numerical simulations, and analyzes the impact of stochastic perturbations. Finally, corresponding conclusions and discussions are presented.

2. The equilibrium points of the deterministic model

If the influence of environmental noise is disregarded, that is, the infection rate coefficient $\xi(t)$ is a constant $\bar{\xi}$, then model (1.2) corresponds to the following deterministic system:

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - \frac{\bar{\xi} S(t) I(t)}{1+\alpha I^2(t)} - \eta S(t), \\ \frac{dE(t)}{dt} = \frac{\bar{\xi} S(t) I(t)}{1+\alpha I^2(t)} - (\eta + k) E(t), \\ \frac{dI(t)}{dt} = kE(t) - (\eta + \delta) I(t), \\ \frac{dR(t)}{dt} = \delta I(t) - \eta R(t). \end{cases} \tag{2.1}$$

Let $N(t) = S(t) + E(t) + I(t) + R(t)$, and then

$$\frac{dN(t)}{dt} = \Lambda - \eta [S(t) + E(t) + I(t) + R(t)] = \Lambda - \eta N(t). \tag{2.2}$$

Clearly, $N(t) = \frac{\Lambda}{\eta}$ is a particular solution of (2.2). For any initial condition $N(t_0) = N_0$, the general solution of (2.2) is

$$N(t) = \frac{\Lambda}{\eta} + \left(N_0 - \frac{\Lambda}{\eta} \right) e^{-\eta(t-t_0)}.$$

Obviously,

$$\lim_{t\to\infty} N(t) = \frac{\Lambda}{\eta}. \tag{2.3}$$

Hence, the hyperplane $S + E + I + R = \frac{\Lambda}{\eta}$ is an invariant manifold of system (2.1) and exhibits attraction within $\mathbb{R}^4_+$.

The equilibrium points of system (2.1) are examined next. A disease-free equilibrium is always present, located at $\left(\frac{\Lambda}{\eta}, 0, 0, 0\right)$. By applying the next-generation matrix approach outlined in [18], we derive the basic reproduction number for the model

$$R_0 = \frac{\bar{\xi} \Lambda k}{\eta(\eta + \delta)(\eta + k)}.$$

Solving the system of equations:

$$\begin{cases} \Lambda - \dfrac{\bar{\xi} S I}{1 + \alpha I^2} - \eta S = 0, \\ \dfrac{\bar{\xi} S I}{1 + \alpha I^2} - (\eta + k) E = 0, \\ kE - (\eta + \delta) I = 0, \\ \delta I - \eta R = 0. \end{cases}$$

After algebraic manipulation, we obtain the equilibrium expressions

$$S^* = \frac{(\eta + \delta)(\eta + k)}{k\bar{\xi}}(1 + \alpha I^{*2}), \quad E^* = \frac{(\eta + \delta) I^*}{k}, \quad R^* = \frac{\delta I^*}{\eta},$$

where I^* satisfies

$$\frac{\alpha\eta(\eta + k)(\eta + \delta)}{k\bar{\xi}} I^{*2} + \frac{(\eta + k)(\eta + \delta)}{k} I^* + \frac{\eta(\eta + k)(\eta + \delta)}{k\bar{\xi}} - \Lambda = 0.$$

The above equation possesses a unique positive root if and only if $R_0 > 1$, given by

$$I^* = \frac{-\bar{\xi} + \sqrt{\bar{\xi}^2 + 4\alpha\eta^2(R_0 - 1)}}{2\alpha\eta}.$$

In conclusion, it can be seen that:

(i) For system (2.1), a disease-free equilibrium always exists at $E_0 = \left(\frac{\Lambda}{\eta}, 0, 0, 0\right)$.

(ii) When $R_0 > 1$, system (2.1) possesses a unique positive equilibrium, referred to as the endemic equilibrium $E^* = (S^*, E^*, I^*, R^*)$.

Regarding the stability of the equilibrium point [19], we have the following:

Theorem 1. *(i) When $R_0 < 1$, the disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\eta}, 0, 0, 0\right)$ of model (2.1) is globally asymptotically stable.*

(ii) When $R_0 > 1$, the endemic equilibrium $E^* = (S^*, E^*, I^*, R^*)$ is locally asymptotically stable.

Proof. (i) The Jacobian matrix of system (2.1) at the equilibrium E_0 is

$$J(E_0) = \begin{pmatrix} -\eta & 0 & -\frac{\bar{\xi}\Lambda}{\eta} & 0 \\ 0 & -(\eta + k) & \frac{\bar{\xi}\Lambda}{\eta} & 0 \\ 0 & k & -(\eta + \delta) & 0 \\ 0 & 0 & \delta & -\eta \end{pmatrix}.$$

Its characteristic polynomial

$$\begin{aligned} \det(J(E_0) - \lambda E) &= \begin{vmatrix} -\eta - \lambda & 0 & -\frac{\bar{\xi}\Lambda}{\eta} & 0 \\ 0 & -(\eta + k) - \lambda & \frac{\bar{\xi}\Lambda}{\eta} & 0 \\ 0 & k & -(\eta + \delta) - \lambda & 0 \\ 0 & 0 & \delta & -\eta - \lambda \end{vmatrix} \\ &= (-\eta - \lambda) \begin{vmatrix} -\eta - \lambda & 0 & -\frac{\bar{\xi}\Lambda}{\eta} \\ 0 & -(\eta + k) - \lambda & \frac{\bar{\xi}\Lambda}{\eta} \\ 0 & k & -(\eta + \delta) - \lambda \end{vmatrix} \\ &= (-\eta - \lambda)^2 \left[\lambda^2 + (2\eta + k + \delta)\lambda + (\eta + k)(\eta + \delta) - \frac{k\bar{\xi}\Lambda}{\eta}\right] \\ &= (-\eta - \lambda)^2 \left[\lambda^2 + (2\eta + k + \delta)\lambda + (\eta + \delta)(\eta + k)(1 - R_0)\right]. \end{aligned}$$

Clearly, when $R_0 < 1$, all eigenvalues of the Jacobian matrix $J(E_0)$ at E_0 possess negative real parts, hence E_0 is locally asymptotically stable.

Construct a Lyapunov function

$$V = kE + (\eta + k)I,$$

and then

$$\begin{aligned} \frac{dV}{dt} &= k\frac{dE}{dt} + (\eta + k)\frac{dI}{dt} = \frac{k\bar{\xi}SI}{1 + \alpha I^2} - (\eta + k)(\eta + \delta)I \\ &\le k\bar{\xi}\frac{\Lambda}{\eta}I - (\eta + k)(\eta + \delta)I \\ &= (\eta + \delta)(\eta + k)(R_0 - 1)I. \end{aligned}$$

Therefore, when $R_0 < 1$, $\frac{dV}{dt} < 0$ holds, which implies that E_0 is globally asymptotically stable.

(ii) The Jacobian matrix of system (2.1) at the equilibrium E^* is

$$J(E^*) = \begin{pmatrix} -\frac{\bar{\xi}I^*}{1+\alpha I^{*2}} - \eta & 0 & -\frac{\bar{\xi}S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2} & 0 \\ \frac{\bar{\xi}I^*}{1+\alpha I^{*2}} & -(\eta+k) & \frac{\bar{\xi}S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2} & 0 \\ 0 & k & -(\eta+\delta) & 0 \\ 0 & 0 & \delta & -\eta \end{pmatrix}.$$

Its characteristic polynomial

$$\det(J(E^*) - \lambda E) = (-\eta-\lambda)\begin{vmatrix} -\frac{\bar{\xi}I^*}{1+\alpha I^{*2}} - \eta - \lambda & 0 & -\frac{\bar{\xi}S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2} \\ \frac{\bar{\xi}I^*}{1+\alpha I^{*2}} & -(\eta+k)-\lambda & \frac{\bar{\xi}S^*(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2} \\ 0 & k & -(\eta+\delta)-\lambda \end{vmatrix}$$

$$\begin{aligned} &= (\lambda+\eta)\left[\left(\lambda+\eta+\frac{\bar{\xi}I^*}{1+\alpha I^{*2}}\right)(\lambda+\eta+k)(\lambda+\eta+\delta) - \frac{(\eta+k)(\eta+\delta)(\lambda+\eta)(1-\alpha I^{*2})}{(1+\alpha I^{*2})^2}\right] \\ &= (\lambda+\eta)(\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3), \end{aligned}$$

where

$$p_1 = 3\eta + k + \delta + \frac{\bar{\xi}I^*}{1+\alpha I^{*2}} > 0,$$

$$p_2 = (2\eta+k+\delta)\left(\frac{\bar{\xi}I^*}{1+\alpha I^{*2}} + \eta\right) + \frac{2\alpha I^{*2}(\eta+k)(\eta+\delta)}{1+\alpha I^{*2}} > 0,$$

$$p_3 = (\eta+k)(\eta+\delta)\left(\frac{\bar{\xi}I^*}{1+\alpha I^{*2}} + \frac{2\eta\alpha I^{*2}}{1+\alpha I^{*2}}\right) > 0,$$

$$\begin{aligned} p_1p_2 - p_3 = &\frac{2\alpha I^{*2}(\eta+k)(\eta+\delta)}{1+\alpha I^{*2}}\left(2\eta+k+\delta+\frac{\bar{\xi}I^*}{1+\alpha I^{*2}}\right) \\ &+ \frac{\bar{\xi}I^*}{1+\alpha I^{*2}}\left[(3\eta+k+\delta)(2\eta+k+\delta) + \eta(2\eta+k+\delta) - (\eta+k)(\eta+\delta)\right] \\ &+ (2\eta+k+\delta)\left(\frac{\bar{\xi}I^*}{1+\alpha I^{*2}}\right)^2 + (3\eta+k+\delta)\eta(2\eta+k+\delta) > 0. \end{aligned}$$

By the Routh–Hurwitz criterion, when $R_0 > 1$, the eigenvalues of the Jacobian matrix $J(E^*)$ at the equilibrium point E^* are all characterized by negative real components. Consequently, local asymptotic stability is confirmed for the equilibrium E^*under the specified conditions.

3. Existence and uniqueness of a global positive solution and extinction conditions

This section examines the well-posedness and extinction behavior of the stochastic system (1.2). Reflecting the practical requirement of non-negative infection rates, the quantity $\xi^+(t) = \max\{\xi(t), 0\}$ is

defined [20, 21]. Furthermore, since $R(t)$ does not appear in the remaining equations, system (1.2) can be simplified as

$$\begin{cases} dS(t) = \left[\Lambda - \frac{\xi^+(t)S(t)I(t)}{1+\alpha I^2(t)} - \eta S(t)\right] dt, \\ dE(t) = \left[\frac{\xi^+(t)S(t)I(t)}{1+\alpha I^2(t)} - (\eta + k)E(t)\right] dt, \\ dI(t) = \left[kE(t) - (\eta + \delta)I(t)\right] dt, \\ d\xi(t) = \theta(\bar{\xi} - \xi(t))dt + \sigma dB(t). \end{cases} \tag{3.1}$$

From Eq (2.3), $N(t) = S(t) + E(t) + I(t) + R(t)$ is bounded. Define

$$\Omega^* = \left\{(S, E, I, \xi) \in \mathbb{R}^3_+ \times \mathbb{R},\ S + E + I \le \frac{\Lambda}{\eta}\right\}.$$

Moreover, for notational convenience, introduce the indicator function of a set A as

$$\mathbf{1}_A = \begin{cases} 1, & \text{if } x \in A, \\ 0, & \text{if } x \notin A. \end{cases}$$

Theorem 2. *For any initial value $(S(0), E(0), I(0), \xi(0)) \in \mathbb{R}^3_+ \times \mathbb{R}$, system (3.1) possesses a unique global positive solution denoted by $(S(t), E(t), I(t), \xi(t))$. Furthermore, this solution stays within $\mathbb{R}^3_+ \times \mathbb{R}$ almost surely.*

Proof. Since the system's parameters fulfill standard local Lipschitz criteria, for any initial state $(S(0), E(0), I(0), \xi(0)) \in \mathbb{R}^3_+ \times \mathbb{R}$, the system (3.1) has a unique local solution $(S(t), E(t), I(t), \xi(t))$, $t \in [0, \tau_e)$, where τ_e denotes the explosion time. Demonstrating rigorous global positivity for this solution, it simply demands to prove $\tau_e = \infty$ with a probability of one. Choose a sufficiently large integer $n_0 > 0$ such that all initial components lie within $[\frac{1}{n_0}, n_0]$. For each integer $n \ge n_0$, define the stopping time

$$\tau_n = \inf\left\{t \in [0, \tau_e) : \min\{S(t), E(t), I(t), \xi(t)\} \le \frac{1}{n} \text{ or } \max\{S(t), E(t), I(t), \xi(t)\} \ge n\right\}.$$

Clearly, τ_n is monotonically increasing as $n \to \infty$. Define $\tau_\infty = \lim_{n\to\infty} \tau_n$, which satisfies $\tau_\infty \le \tau_e$ almost surely. The proof is completed by showing $\tau_\infty = \infty$ almost surely.

Assume the contrary. Then there exist constants $T > 0$ and $\varepsilon \in (0, 1)$ such that $P\{\tau_\infty \le T\} > \varepsilon$. Consequently, there exists an integer $n_1 \ge n_0$ such that

$$P\{\tau_n \le T\} := P(G_n) \ge \varepsilon, \forall n \ge n_1.$$

Moreover, for $t \le \tau_n$, it follows that

$$\begin{aligned} d(S + E + I) &= (\Lambda - \eta(S + E + I) - \delta I)\, dt \\ &\le [\Lambda - \eta(S + E + I)]\, dt. \end{aligned}$$

This implies

$$S(t) + E(t) + I(t) \le \begin{cases} S(0) + E(0) + I(0), & \text{if } S(0) + E(0) + I(0) \ge \frac{\Lambda}{\eta}, \\ \frac{\Lambda}{\eta}, & \text{if } S(0) + E(0) + I(0) < \frac{\Lambda}{\eta}. \end{cases}$$

Let $C = \max\left\{S(0) + E(0) + I(0), \dfrac{\Lambda}{\eta}\right\}$. Then

$$S(t) + E(t) + I(t) \le C.$$

Define the function

$$W = S - 1 - \ln S + E - 1 - \ln E + I - 1 - \ln I + \frac{1}{2}\xi^2.$$

Applying Itô's formula [22] yields

$$\begin{aligned}
\mathcal{L}W &= \left(1 - \frac{1}{S}\right)\left(\Lambda - \frac{\xi^+ SI}{1+\alpha I^2} - \eta S\right) + \left(1 - \frac{1}{E}\right)\left(\frac{\xi^+ SI}{1+\alpha I^2} - (\eta + k)E\right) \\
&\quad + \left(1 - \frac{1}{I}\right)(kE - (\eta + \delta)I) + \theta\xi(\bar{\xi} - \xi) + \frac{1}{2}\sigma^2 \\
&= \Lambda - \frac{\xi^+ SI}{1+\alpha I^2} - \eta S - \frac{\Lambda}{S} + \frac{\xi^+ I}{1+\alpha I^2} + \eta + \frac{\xi^+ SI}{1+\alpha I^2} - (\eta + k)E - \frac{\xi^+ SI}{E(1+\alpha I^2)} \\
&\quad + (\eta + k) + kE - (\eta + \delta)I - \frac{kE}{I} + (\eta + \delta) + \theta\xi(\bar{\xi} - \xi) + \frac{1}{2}\sigma^2 \\
&\le \Lambda + \frac{\xi I}{1+\alpha I^2} + 3\eta + k + \delta + \theta\xi\bar{\xi} - \theta\xi^2 + \frac{1}{2}\sigma^2 \\
&\le \Lambda + \xi C + 3\eta + k + \delta + \theta\xi\bar{\xi} - \theta\xi^2 + \frac{1}{2}\sigma^2 \\
&\le \Lambda + 3\eta + k + \delta + \frac{1}{2}\sigma^2 + \sup_{\xi\in\mathbb{R}}\left\{-\theta|\xi|^2 + (\theta\bar{\xi} + C)|\xi|\right\} \\
&:= M,
\end{aligned}$$

where M is a positive constant. Integrating both sides of the above inequality and taking the mathematical expectation gives

$$\begin{aligned}
&\mathbb{E}\left[W(S(\tau_n \wedge T), E(\tau_n \wedge T), I(\tau_n \wedge T), \xi(\tau_n \wedge T))\right] \\
&= \mathbb{E}\left[W(S(0), E(0), I(0), \xi(0))\right] \\
&\quad + \mathbb{E}\left[\int_0^{\tau_n \wedge T} \mathcal{L}W(S(\tau), E(\tau), I(\tau), \xi(\tau))\, d\tau\right] \\
&\le \mathbb{E}\left[W(S(0), E(0), I(0), \xi(0))\right] + MT.
\end{aligned}$$

For any $\lambda \in G_n$, we have

$$\begin{aligned}
&W(S(\tau_n, \lambda), E(\tau_n, \lambda), I(\tau_n, \lambda), \xi(\tau_n, \lambda)) \\
&\ge (n - 1 - \ln n) \wedge \left(\frac{1}{n} - 1 - \ln\frac{1}{n}\right) \wedge \frac{n^2}{2}.
\end{aligned}$$

Hence,

$$\begin{aligned}
&\mathbb{E}\left[W(S(0), E(0), I(0), \xi(0))\right] + MT \\
&\ge \mathbb{E}\left[W(S(\tau_n \wedge T), E(\tau_n \wedge T), I(\tau_n \wedge T), \xi(\tau_n \wedge T))\right] \\
&\ge P(G_n(\lambda))W(S(\tau_n \wedge T), E(\tau_n \wedge T), I(\tau_n \wedge T), \xi(\tau_n \wedge T)) \\
&\ge \varepsilon\left[(n - 1 - \ln n) \wedge \left(\frac{1}{n} - 1 - \ln\frac{1}{n}\right) \wedge \frac{n^2}{2}\right].
\end{aligned}$$

Letting $n \to +\infty$ yields

$$+\infty \leq \mathbb{E}\left[W(S(0), E(0), I(0), \xi(0))\right] + MT < +\infty,$$

which is a contradiction. Therefore, $\tau_\infty = \infty$ is proved.

Next, we will discuss the extinction of the disease in system (3.1). According to [10, 23, 24], $\xi(t)$ possesses ergodicity, and thus

$$\lim_{t\to+\infty} \frac{1}{t} \int_0^t \left|\xi(s) - \bar{\xi}\right| ds = \int_{-\infty}^{+\infty} \left|x - \bar{\xi}\right| \pi(x)dx = \frac{\sigma}{\sqrt{\pi\theta}}, \tag{3.2}$$

$$\lim_{t\to+\infty} \frac{1}{t} \int_0^t \sqrt[4]{\xi^+(s)}ds = \int_0^{+\infty} \sqrt[4]{x}\pi(x)dx = \sqrt[4]{\tilde{\xi}}, \tag{3.3}$$

where $\tilde{\xi} = \left(\int_0^{+\infty} x^{\frac{1}{4}}\pi(x)dx\right)^4$.

Define the parameter

$$R_0^E = \sqrt{R_0} + \frac{\sigma\Lambda k}{\eta(\eta + k)\sqrt{R_0\pi\theta}\min\{\eta + k, \eta + \delta\}},$$

and it follows that:

Theorem 3. *If $R_0^E < 1$, then the following inequality holds:*

$$\limsup_{t\to+\infty} \frac{\ln\left(\frac{\omega_1}{\eta+k}E + \frac{\omega_2}{\eta+\delta}I\right)}{t} \leq \min\{\eta + k, \eta + \delta\}(R_0^E - 1) < 0,$$

where $\omega_1 = \dfrac{k}{(\eta + \delta)\sqrt{R_0}}$ and $\omega_2 = 1$. This implies that the disease in system (3.1) *will undergo exponential extinction with probability one.*

Proof. Define

$$Q(E, I) = \frac{\omega_1}{\eta + k}E + \frac{\omega_2}{\eta + \delta}I.$$

According to the expression of $Q(E, I)$ given above, we obtain

$$\frac{\omega_1}{\eta + k}\frac{E}{Q} + \frac{\omega_2}{\eta + \delta}\frac{I}{Q} = 1,$$

which implies

$$\frac{I}{Q} < \frac{\eta + \delta}{\omega_2}. \tag{3.4}$$

Define the matrix M_0 as

$$M_0 = \begin{pmatrix} 0 & \dfrac{\bar{\xi}\Lambda}{\eta(\eta + k)} \\ \dfrac{k}{\eta + \delta} & 0 \end{pmatrix}.$$

Direct calculation yields

$$(\omega_1, \omega_2)\,(M_0 - \mathbb{I}_2)\begin{pmatrix} E \\ I \end{pmatrix} = \left(\sqrt{R_0} - 1\right)(\omega_1 E + \omega_2 I), \tag{3.5}$$

where $\mathbb{I}_2$ denotes the 2×2 identity matrix. Applying Itô's calculus directly to $Q(E, I)$ and combining (3.4) with (3.5) gives

$$\begin{aligned}
\mathcal{L}(\ln Q) &= \frac{1}{Q}\left(\frac{\omega_1}{\eta + k}\frac{\xi^+(t) S I}{1 + \alpha I^2} - \omega_1 E + \frac{\omega_2}{\eta + \delta} k E - \omega_2 I\right) \\
&\le \frac{1}{Q}\left(\frac{\omega_1 \Lambda}{\eta(\eta + k)}\bar{\xi} I - \omega_1 E + \frac{\omega_2}{\eta + \delta} k E - \omega_2 I\right) + \frac{1}{Q}\frac{\omega_1 \Lambda}{\eta(\eta + k)}\left|\xi^+(t) - \bar{\xi}\right| I \\
&= \frac{1}{Q}(\omega_1, \omega_2)\,(M_0 - \mathbb{I}_2)\begin{pmatrix} E \\ I \end{pmatrix} + \frac{1}{Q}\frac{\omega_1 \Lambda}{\eta(\eta + k)}\left|\xi^+(t) - \bar{\xi}\right| I \\
&= \frac{1}{Q}\left(\sqrt{R_0} - 1\right)(\omega_1 E + \omega_2 I) + \frac{I}{Q}\frac{\omega_1 \Lambda}{\eta(\eta + k)}\left|\xi(t) - \bar{\xi}\right| \\
&\le \frac{\omega_1 E + \omega_2 I}{\frac{\omega_1}{\eta + k} E + \frac{\omega_2}{\eta + \delta} I}\left(\sqrt{R_0} - 1\right) + \frac{\eta + \delta}{\omega_2}\frac{\omega_1 \Lambda}{\eta(\eta + k)}\left|\xi(t) - \bar{\xi}\right| \\
&\le \min\{\eta + k, \eta + \delta\}\left(\sqrt{R_0} - 1\right) + \frac{\eta + \delta}{\omega_2}\frac{\omega_1 \Lambda}{\eta(\eta + k)}\left|\xi(t) - \bar{\xi}\right| \\
&= \min\{\eta + k, \eta + \delta\}\left(\sqrt{R_0} - 1\right) + \frac{\Lambda k}{\eta(\eta + k)\sqrt{R_0}}\left|\xi(t) - \bar{\xi}\right|.
\end{aligned}$$

Integrating the above expression gives

$$\frac{\ln Q(t) - \ln Q(0)}{t} \le \min\{\eta + k, \eta + \delta\}\left(\sqrt{R_0} - 1\right) + \frac{\Lambda k}{\eta(\eta + k)\sqrt{R_0}} \cdot \frac{1}{t}\int_0^t |\xi(s) - \bar{\xi}| ds. \tag{3.6}$$

Combining (3.2) and letting $t \to +\infty$ yields

$$\begin{aligned}
\limsup_{t \to +\infty} \frac{\ln Q(t)}{t} &\le \min\{\eta + k, \eta + \delta\}\left(\sqrt{R_0} - 1\right) + \frac{k\Lambda}{\eta(\eta + k)\sqrt{R_0}} \cdot \frac{\sigma}{\sqrt{\pi\theta}} \\
&= \min\{\eta + k, \eta + \delta\}\left(\sqrt{R_0} + \frac{k\Lambda\sigma}{\eta(\eta + k)\sqrt{R_0 \pi\theta}\min\{\eta + k, \eta + \delta\}} - 1\right) \\
&= \min\{\eta + k, \eta + \delta\}\left(R_0^E - 1\right).
\end{aligned}$$

Clearly, when $R_0^E < 1$,

$$\limsup_{t \to \infty} \frac{\ln Q(t)}{t} \le \min\{\eta + k, \eta + \delta\}(R_0^E - 1) < 0.$$

This implies that, under the condition $R_0^E < 1$,

$$\lim_{t \to +\infty} E(t) = \lim_{t \to +\infty} I(t) = 0.$$

Hence the disease goes extinct. Theorem 3 is proved.

4. Existence of a stationary distribution

This section examines the long-term dynamical behavior of model (3.1), with particular focus on the persistence of the disease.

Define the parameter

$$R_0^s = \frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)\left(\eta + k + c_1\frac{\Lambda}{\eta}\frac{\sigma}{\sqrt{\pi\theta}}\right)},$$

where

$$\tilde{\xi} = \left(\int_0^{+\infty} x^{\frac{1}{4}}\pi(x)dx\right)^4, \quad c_1 = \frac{\tilde{\xi}\Lambda k}{\eta^2(\eta+\delta)}.$$

Theorem 4. *When $R_0^s > 1$, system (3.1) admits a stationary distribution.*

Proof. Define the function

$$W_1 = -\ln E - c_1 \ln S - c_2 \ln I,$$

where c_2 will be presented in the subsequent proof. Using the Itô formula, we obtain

$$\begin{aligned}
\mathcal{L}W_1 &= \eta + k - \frac{\xi^+ S I}{E(1+\alpha I^2)} + c_1\left(\eta + \frac{\xi^+ I}{1+\alpha I^2} - \frac{\Lambda}{S}\right) + c_2\left(\eta + \delta - \frac{kE}{I}\right) \\
&= -\frac{\xi^+ S I}{E(1+\alpha I^2)} - \frac{c_1\Lambda}{S} - \frac{c_2 kE}{I} + \eta + k + c_1\eta + c_2(\eta+\delta) + \frac{c_1\xi^+ I}{1+\alpha I^2} \\
&= -\frac{\xi^+ S I}{E(1+\alpha I^2)} - \frac{c_1\Lambda}{S} - \frac{c_2 kE}{I} - c_3(1+\alpha I^2) + c_3(1+\alpha I^2) + \eta + k + c_1\eta \\
&\quad + c_2(\eta+\delta) + \frac{c_1\xi^+ I}{1+\alpha I^2} \\
&\le -4\sqrt[4]{\xi^+ c_1 c_2 c_3 \Lambda k} + c_3\alpha I^2 + \eta + k + c_1\eta + c_2(\eta+\delta) + c_3 + c_1\xi^+ I \\
&= -4\sqrt[4]{\tilde{\xi} c_1 c_2 c_3 \Lambda k} + \eta + k + c_1\eta + c_2(\eta+\delta) + c_3 + c_3\alpha I^2 + c_1\bar{\xi} I \\
&\quad + 4\left(\sqrt[4]{\tilde{\xi} c_1 c_2 c_3 \Lambda k} - \sqrt[4]{\xi^+ c_1 c_2 c_3 \Lambda k}\right) + c_1 I\left(\xi^+ - \bar{\xi}\right).
\end{aligned}$$

Let c_1, c_2, c_3 satisfy the following equations:

$$c_1\eta = c_2(\eta+\delta) = c_3 = \frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)},$$

and then

$$\begin{aligned}\mathcal{L}W_1 \leq & -\frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)} + \eta + k + c_3\alpha I^2 + c_1\bar{\xi}I + 4\left(\sqrt[4]{\tilde{\xi}c_1c_2c_3\Lambda k} - \sqrt[4]{\xi^+c_1c_2c_3\Lambda k}\right) \\ & + c_1 I\left(\xi^+ - \bar{\xi}\right) \\ \leq & -\frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)} + \left(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi}\right)I + \eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}} + c_1\frac{\Lambda}{\eta}\left|\xi - \bar{\xi}\right| \\ & - c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}} + 4\left(\sqrt[4]{\tilde{\xi}c_1c_2c_3\Lambda k} - \sqrt[4]{\xi^+c_1c_2c_3\Lambda k}\right) \\ = & -\frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)} + \eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}} + \left(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi}\right)I + c_1\frac{\Lambda}{\eta}\left(\left|\xi - \bar{\xi}\right| - \frac{\sigma}{\sqrt{\pi\theta}}\right) \\ & + 4\left(\sqrt[4]{\tilde{\xi}c_1c_2c_3\Lambda k} - \sqrt[4]{\xi^+c_1c_2c_3\Lambda k}\right) \\ = & -\frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)} + \eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}} + \left(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi}\right)I + G(\xi(t)),\end{aligned}$$

where

$$G(\xi(t)) = 4\left(\sqrt[4]{\tilde{\xi}c_1c_2c_3\Lambda k} - \sqrt[4]{\xi^+c_1c_2c_3\Lambda k}\right) + c_1\frac{\Lambda}{\eta}\left(\left|\xi - \bar{\xi}\right| - \frac{\sigma}{\sqrt{\pi\theta}}\right).$$

Let

$$W_2 = W_1 + \frac{c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi}}{\eta+\delta}I,$$

and then

$$\begin{aligned}\mathcal{L}W_2 \leq & -\frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)} + \eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}} + \frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k}{\eta+\delta}E + G(\xi(t)) \\ = & -\left(R_0^s - 1\right)\left(\eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}}\right) + \frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k}{\eta+\delta}E + G(\xi(t)),\end{aligned}$$

where

$$R_0^s = \frac{\tilde{\xi}\Lambda k}{\eta(\eta+\delta)\left(\eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}}\right)}.$$

Denote

$$W_3 = -\ln S - \ln I - \ln\left(\frac{\Lambda}{\eta} - S - E - I\right) + \frac{\xi^2}{2},$$

$$\begin{aligned}\mathcal{L}W_3 = & -\frac{\Lambda}{S} + \eta + \frac{\xi^+ I}{1+\alpha I^2} + \eta + \delta - \frac{kE}{I} + \eta - \frac{\delta I}{\frac{\Lambda}{\eta} - (S+E+I)} + \theta\xi(\bar{\xi} - \xi) + \frac{\sigma^2}{2} \\ \leq & -\frac{\Lambda}{S} + 3\eta + \xi^+\frac{\Lambda}{\eta} - \frac{kE}{I} + \delta + \theta\xi(\bar{\xi} - \xi) + \frac{\sigma^2}{2} - \frac{\delta I}{\frac{\Lambda}{\eta} - (S+E+I)} \\ \leq & -\frac{\Lambda}{S} - \frac{kE}{I} - \frac{\delta I}{\frac{\Lambda}{\eta} - (S+E+I)} - \frac{\theta\xi^2}{2} + B,\end{aligned}$$

where

$$B = \sup_{\xi\in\mathbb{R}}\left\{-\frac{\theta|\xi|^2}{2} + (\theta\bar{\xi} + \frac{\Lambda}{\eta})|\xi| + 3\eta + \delta + \frac{\sigma^2}{2}\right\}.$$

Define

$$\overline{W} = MW_2 + W_3,$$

where M is a sufficiently large positive constant satisfying

$$-M\left(R_0^s - 1\right)\left(\eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}}\right) + B \le -2.$$

Since $\overline{W}(S, E, I, \xi)$ tends to $+\infty$ as (S, E, I, ξ) approaches the boundary of Ω^*, it attains its minimum in the interior of Ω^*. This yields a non-negative function

$$W(S, E, I, \xi) = \overline{W}(S, E, I, \xi) - \overline{W}_{\min}.$$

Applying Itô's formula gives

$$\begin{aligned}\mathcal{L}W \le\ & -M\left(R_0^s - 1\right)\left(\eta + k + c_1\frac{\Lambda}{\eta}\cdot\frac{\sigma}{\sqrt{\pi\theta}}\right) + \frac{M(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k}{\eta+\delta}E + B - \frac{\delta I}{\frac{\Lambda}{\eta} - (S + E + I)} \\ & - \frac{kE}{I} - \frac{\Lambda}{S} - \frac{1}{2}\theta|\xi|^2 + MG(\xi(t)) \\ :=\ & F(S, E, I, \xi) + MG(\xi(t)).\end{aligned} \tag{4.1}$$

Denote by D_ε the compact set such that

$$D_\varepsilon = \left\{(S, E, I, \xi) \in \Omega^* \mid S \ge \varepsilon,\ E \ge \varepsilon,\ I \ge \varepsilon^2,\ \frac{\Lambda}{\eta} - (S + E + I) \ge \varepsilon^3,\ |\xi| \le \frac{1}{\varepsilon}\right\}.$$

The complement of D_ε can be divided into five subsets:

$$\begin{aligned}D_{1,\varepsilon}^c &= \{(S, E, I, \xi) \in \Omega^* \mid E < \varepsilon\},\\ D_{2,\varepsilon}^c &= \{(S, E, I, \xi) \in \Omega^* \mid E > \varepsilon,\ I < \varepsilon^2\},\\ D_{3,\varepsilon}^c &= \{(S, E, I, \xi) \in \Omega^* \mid S < \varepsilon\},\\ D_{4,\varepsilon}^c &= \{(S, E, I, \xi) \in \Omega^* \mid I < \varepsilon^2,\ \tfrac{\Lambda}{\eta} - (S + E + I) < \varepsilon^3\},\\ D_{5,\varepsilon}^c &= \{(S, E, I, \xi) \in \Omega^* \mid |\xi| > \frac{1}{\varepsilon}\},\end{aligned}$$

where $\varepsilon > 0$ is taken sufficiently small to satisfy the following conditions:

$$M\frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k}{\eta+\delta}\varepsilon \le 1,$$

$$-\min\left\{\frac{k}{\varepsilon}, \frac{\Lambda}{\varepsilon}, \frac{\delta}{\varepsilon}, \frac{\theta}{2\varepsilon^2}\right\} + M\frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k\Lambda}{\eta(\eta+\delta)} \le 1.$$

It remains to verify that for any $(S, E, I, \xi) \in D_\varepsilon^c$, the inequality $F(S, E, I, \xi) \le -1$ holds.

Case 1: For $(S, E, I, \xi) \in D^c_{1,\varepsilon}$, it follows that

$$F(S, E, I, \xi) \leq -2 + M\frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k}{\eta + \delta}\varepsilon \leq -1.$$

Case 2: For $(S, E, I, \xi) \in D^c_{2,\varepsilon}$, it follows that

$$F(S, E, I, \xi) \leq -2 + M\frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k\Lambda}{\eta(\eta + \delta)} - \frac{k}{\varepsilon} \leq -1.$$

Case 3: For $(S, E, I, \xi) \in D^c_{3,\varepsilon}$, it follows that

$$F(S, E, I, \xi) \leq -2 + M\frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k\Lambda}{\eta(\eta + \delta)} - \frac{\Lambda}{\varepsilon} \leq -1.$$

Case 4: For $(S, E, I, \xi) \in D^c_{4,\varepsilon}$, it follows that

$$F(S, E, I, \xi) \leq -2 + M\frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k\Lambda}{\eta(\eta + \delta)} - \frac{\delta}{\varepsilon} \leq -1.$$

Case 5: For $(S, E, I, \xi) \in D^c_{5,\varepsilon}$, it follows that

$$F(S, E, I, \xi) \leq -2 + M\frac{(c_3\alpha\frac{\Lambda}{\eta} + c_1\bar{\xi})k\Lambda}{\eta(\eta + \delta)} - \frac{\theta}{2\varepsilon^2} \leq -1.$$

From the five cases above, it follows that there exist a constant ε and a closed set D_ε such that

$$F(S, E, I, \xi) \leq -1, \quad \forall\, (S, E, I, \xi) \in \Omega^* \setminus D_\varepsilon.$$

On the other hand, there exists a positive constant H such that

$$F(S, E, I, \xi) \leq H < +\infty, \quad \forall\, (S, E, I, \xi) \in \Omega^*. \tag{4.2}$$

One first integrates both sides of (4.1) over $[0, t]$ and then divides the resulting expression by t, assuming the given initial value $(S(0), E(0), I(0), \xi(0)) \in \Omega^*$, followed by evaluating the corresponding mathematical expectation, which yields

$$\begin{aligned}
0 \leq\ & \frac{\mathbb{E}\left[W(S, E, I, \xi)\right]}{t} \\
=\ & \frac{\mathbb{E}\left[W(S(0), E(0), I(0), \xi(0))\right]}{t} + \frac{1}{t}\int_0^t \mathbb{E}\left[\mathcal{L}W\left(S(\tau), E(\tau), I(\tau), \xi(\tau)\right)\right] d\tau \\
\leq\ & \frac{\mathbb{E}\left[W(S(0), E(0), I(0), \xi(0))\right]}{t} + \frac{1}{t}\int_0^t \mathbb{E}\left[F\left(S(\tau), E(\tau), I(\tau), \xi(\tau)\right)\right] d\tau \\
& + 4M\sqrt[4]{c_1c_2c_3\Lambda k}\cdot\frac{1}{t}\int_0^t \mathbb{E}\left[\left(\sqrt[4]{\bar{\xi}} - \sqrt[4]{\xi^+(\tau)}\right)\right] d\tau \\
& + Mc_1\frac{\Lambda}{\eta}\cdot\frac{1}{t}\int_0^t \mathbb{E}\left[\left(|\xi(\tau) - \bar{\xi}| - \frac{\sigma}{\sqrt{\pi\theta}}\right)\right] d\tau.
\end{aligned} \tag{4.3}$$

Utilizing the ergodicity of $\xi(t)$ as described in (3.2) and (3.3), together with inequations (4.2) and (4.3), and taking the infimum on both sides of the above expression gives

$$\begin{aligned}
0 &\leq \liminf_{t\to+\infty} \frac{1}{t}\int_0^t \mathbb{E}\left[F\left(S(\tau),E(\tau),I(\tau),\xi(\tau)\right)\right]d\tau \\
&= \liminf_{t\to+\infty} \frac{1}{t}\int_0^t \mathbb{E}\left[F\left(S(\tau),E(\tau),I(\tau),\xi(\tau)\right)\mathbf{1}_{\{(S(\tau),E(\tau),I(\tau),\xi(\tau))\in D_\varepsilon\}}\right]d\tau \\
&\quad + \liminf_{t\to+\infty} \frac{1}{t}\int_0^t \mathbb{E}\left[F\left(S(\tau),E(\tau),I(\tau),\xi(\tau)\right)\mathbf{1}_{\{(S(\tau),E(\tau),I(\tau),\xi(\tau))\in \Omega^*\setminus D_\varepsilon\}}\right]d\tau \\
&\leq H\liminf_{t\to+\infty} \frac{1}{t}\int_0^t \mathbf{1}_{\{(S(\tau),E(\tau),I(\tau),\xi(\tau))\in D_\varepsilon\}}d\tau \\
&- \liminf_{t\to+\infty} \frac{1}{t}\int_0^t \mathbf{1}_{\{(S(\tau),E(\tau),I(\tau),\xi(\tau))\in \Omega^*\setminus D_\varepsilon\}}d\tau \\
&= -1 + (H+1)\liminf_{t\to+\infty} \frac{1}{t}\int_0^t \mathbf{1}_{\{(S(\tau),E(\tau),I(\tau),\xi(\tau))\in D_\varepsilon\}}d\tau,
\end{aligned}$$

where $\mathbf{1}$ denotes the indicator function. This leads to the following result:

$$\liminf_{t\to+\infty} \frac{1}{t}\int_0^t \mathbf{1}_{\left\{(S(\tau),E(\tau),I(\tau),\xi(\tau))\in D_\varepsilon\right\}}d\tau \geq \frac{1}{H+1} > 0 \quad \text{a.s.} \tag{4.4}$$

Utilizing the fundamental definition of event probability together with Fatou's lemma [25], Eq (4.4) implies the following equivalent form:

$$\liminf_{t\to+\infty} \frac{1}{t}\int_0^t P(\tau,(S(\tau),E(\tau),I(\tau)),\xi(\tau),D_\varepsilon)\,d\tau \geq \frac{1}{H+1} > 0,$$

where $P(t,(S(t),E(t),I(t),\xi(t)),D_\varepsilon)$ denotes the transition probability of $(S(t),E(t),I(t),\xi(t))$ belonging to the set D_ε. Consequently, according to the achievement in [25,26], when $R_0^s > 1$, model (3.1) admits a stationary distribution on $\mathbb{R}^3_+ \times \mathbb{R}$.

5. Probability density function of the stationary distribution

The previous section established conditions under which system (3.1) admits a stationary distribution. A natural follow-up question concerns the derivation of the probability density function for this stationary distribution. In order to resolve this inquiry, a preliminary auxiliary result must be introduced:

Lemma 1. *[27] Let us examine the subsequent matrix equality* $\Theta^2 + B\Sigma + \Sigma B^T = 0$, *with* $\Theta = \mathrm{diag}(1,0,0,0)$ *and B is a standard matrix of the form*

$$B = \begin{pmatrix} -\varrho_1 & -\varrho_2 & -\varrho_3 & -\varrho_4 \\ 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \end{pmatrix}.$$

If $\varrho_1 > 0$, $\varrho_3 > 0$, $\varrho_4 > 0$, *and* $\varrho_1\varrho_2\varrho_3 - \varrho_3^2 - \varrho_1^2\varrho_4 > 0$, *then* Σ *is a positive definite matrix, which can be written explicitly as*

$$\Sigma = \begin{pmatrix} \frac{\varrho_2\varrho_3-\varrho_1\varrho_4}{2(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} & 0 & -\frac{\varrho_3}{2(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} & 0 \\ 0 & \frac{\varrho_3}{2(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} & 0 & -\frac{\varrho_1}{2(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} \\ -\frac{\varrho_3}{2(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} & 0 & \frac{\varrho_1}{2(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} & 0 \\ 0 & -\frac{\varrho_1}{2(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} & 0 & \frac{\varrho_1\varrho_2-\varrho_3}{2\varrho_4(\varrho_1\varrho_2\varrho_3-\varrho_3^2-\varrho_1^2\varrho_4)} \end{pmatrix}.$$

Let $P^* = (S^*, E^*, I^*, \xi^*)$ represent the equilibrium point of the deterministic system corresponding to model (3.1). The coordinates of P^* satisfy

$$\begin{cases} \Lambda - \frac{\xi^* S^* I^*}{1+\alpha I^{*2}} - \eta S^* = 0, \\ \frac{\xi^* S^* I^*}{1+\alpha I^{*2}} - (\eta + k)E^* = 0, \\ kE^* - (\eta + \delta)I^* = 0, \\ \theta(\bar{\xi} - \xi^*) = 0. \end{cases}$$

Define $Z(t) = (Z_1, Z_2, Z_3, Z_4)^{\mathrm{T}} = (S - S^*, E - E^*, I - I^*, \xi - \xi^*)^{\mathrm{T}}$. Then the linearized form corresponding to model (3.1) can be expressed as

$$\begin{cases} dZ_1 = [-a_{11}Z_1 - a_{13}Z_3 - a_{14}Z_4]\, dt, \\ dZ_2 = [a_{21}Z_1 - a_{22}Z_2 + a_{23}Z_3 + a_{24}Z_4]\, dt, \\ dZ_3 = [a_{32}Z_2 - a_{33}Z_3]\, dt, \\ dZ_4 = [-a_{44}Z_4]dt + \sigma dB(t), \end{cases} \tag{5.1}$$

where

$$a_{11} = \eta + \frac{\xi^* I^*}{1 + \alpha I^{*2}}, \quad a_{13} = \frac{\xi^* S^*(1 - \alpha I^{*2})}{(1 + \alpha I^{*2})^2}, \quad a_{14} = \frac{S^* I^*}{1 + \alpha I^{*2}}, \quad a_{21} = \frac{\xi^* I^*}{1 + \alpha I^{*2}},$$

$$a_{22} = \eta + k, \qquad a_{23} = \frac{\xi^* S^*(1 - \alpha I^{*2})}{(1 + \alpha I^{*2})^2}, \quad a_{24} = \frac{S^* I^*}{1 + \alpha I^{*2}}, \quad a_{32} = k,$$

$$a_{33} = \eta + \delta, \qquad a_{44} = \theta.$$

Consequently, model (5.1) admits an equivalent representation in matrix notation, given by

$$dZ(t) = AZ(t)dt + GdB(t), \tag{5.2}$$

where

$$A = \begin{pmatrix} -a_{11} & 0 & -a_{13} & -a_{14} \\ a_{21} & -a_{22} & a_{23} & a_{24} \\ 0 & a_{32} & -a_{33} & 0 \\ 0 & 0 & 0 & -a_{44} \end{pmatrix},$$

$$G = \mathrm{diag}(0, 0, 0, \sigma), B(t) = (0, 0, 0, B(t))^{\mathrm{T}}.$$

According to the theory presented in [28], if A is a Hurwitz matrix, then the solution of model (5.2) possesses a unique probability density function $\Phi(z_1, z_2, z_3, z_4)$ near the zero equilibrium (0,0,0,0), and its probability density function satisfies the following Fokker–Planck equation [29]:

$$\frac{\partial \Phi(z(t), t)}{\partial t} + \frac{\partial}{\partial z(t)} [Az(t)\Phi(z(t), t)] - \frac{\sigma^2}{2} \frac{\partial^2 \Phi(z(t), t)}{\partial z_4^2} = 0.$$

Since the matrix G in model (5.2) is constant, Gaussian distribution theory [30] implies that $\Phi(z_1, z_2, z_3, z_4)$ can be expressed as

$$\Phi(z_1, z_2, z_3, z_4) = C \exp\left[-\frac{1}{2}(z_1, z_2, z_3, z_4)Q(z_1, z_2, z_3, z_4)^{\mathrm{T}}\right],$$

where C is a constant chosen to satisfy $\int_{\mathbb{R}^4} \Phi(z_1, z_2, z_3, z_4)\, dz_1 dz_2 dz_3 dz_4 = 1$, and Q is a real symmetric matrix satisfying

$$QG^2Q + A^{\mathrm{T}}Q + QA = 0. \tag{5.3}$$

If Q is positive definite, setting $\Sigma = Q^{-1}$ transforms Eq (5.3) into

$$G^2 + A\Sigma + \Sigma A^{\mathrm{T}} = 0. \tag{5.4}$$

Theorem 5. *If $R_0^s > 1$, then the solution of system* (5.2) *follows a unique normal probability density function $\Phi(z_1, z_2, z_3, z_4)$ near (0,0,0,0), given by*

$$\Phi(z_1, z_2, z_3, z_4) = (2\pi)^{-2}|\Sigma|^{-\frac{1}{2}} \exp\left[-\tfrac{1}{2}(z_1, z_2, z_3, z_4)\Sigma^{-1}(z_1, z_2, z_3, z_4)^{\mathrm{T}}\right], \tag{5.5}$$

where

$$\Sigma = \rho^2(J_4J_3J_2J_1)^{-1}\Sigma_2\left((J_4J_3J_2J_1)^{-1}\right)^{\mathrm{T}},$$

and ρ, J_1, J_2, J_3, J_4 together with Σ_2 are presented in the proof below.

Proof. First, a critical prerequisite is to confirm the Hurwitz stability of A. Let $\varphi_A(\lambda)$ stand for the characteristic polynomial corresponding to A, given by

$$\begin{aligned}
\varphi_A(\lambda) &= \begin{vmatrix} \lambda + a_{11} & 0 & a_{13} & a_{14} \\ -a_{21} & \lambda + a_{22} & -a_{23} & -a_{24} \\ 0 & -a_{32} & \lambda + a_{33} & 0 \\ 0 & 0 & 0 & \lambda + a_{44} \end{vmatrix} \\
&= (\lambda + a_{44}) \begin{vmatrix} \lambda + a_{11} & 0 & a_{13} \\ -a_{21} & \lambda + a_{22} & -a_{23} \\ 0 & -a_{32} & \lambda + a_{33} \end{vmatrix} \\
&= (\lambda + a_{44})\left(\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3\right).
\end{aligned}$$

It can be easily deduced from the proof process of Theorem 1 that A is a Hurwitz matrix.

The next step is to solve the equation $G^2 + A\Sigma + \Sigma A^{\mathrm{T}} = 0$ for Σ, and to demonstrate that the resulting Σ is positive definite.

Let $A_1 = J_1 A J_1^{-1}$ and $G_1 = J_1 G J_1^{-1}$, where

$$J_1 = \begin{pmatrix} 0 & 0 & 0 & 1 \\ 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \end{pmatrix},$$

hence,

$$A_1 = \begin{pmatrix} -a_{44} & 0 & 0 & 0 \\ -a_{14} & -a_{11} & 0 & -a_{13} \\ a_{24} & a_{21} & -a_{22} & a_{23} \\ 0 & 0 & a_{32} & -a_{33} \end{pmatrix}, G_1 = \begin{pmatrix} \sigma & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

Let $A_2 = J_2 A_1 J_2^{-1}$, where

$$J_2 = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 1 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix},$$

and consequently,

$$A_2 = \begin{pmatrix} -a_{44} & 0 & 0 & 0 \\ -a_{14} & -a_{11} & 0 & -a_{13} \\ 0 & a_{32} & -a_{22} & 0 \\ 0 & -a_{32} & a_{32} & -a_{33} \end{pmatrix}.$$

Similarly, let $A_3 = J_3 A_2 J_3^{-1}$, where

$$J_3 = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 1 & 1 \end{pmatrix},$$

and then we have

$$A_3 = \begin{pmatrix} -a_{44} & 0 & 0 & 0 \\ -a_{14} & -a_{11} & a_{13} & -a_{13} \\ 0 & a_{32} & -a_{22} & 0 \\ 0 & 0 & a_{32} - a_{22} + a_{33} & -a_{33} \end{pmatrix}.$$

Denoting the matrix $J = J_3 J_2 J_1$, Eq (5.4) can be equivalently expressed as

$$JG^2J^T + (JAJ^{-1})(J\Sigma J^T) + (J\Sigma J^T)(JAJ^{-1})^T = 0.$$

By examining the specific algebraic layouts characterizing J_3, alongside J_2 as well as J_1, it follows that $JG^2J^{\mathrm{T}} = \mathrm{diag}(\sigma^2, 0, 0, 0) \triangleq G_1^2$. Setting

$$J\Sigma J^{\mathrm{T}} = \Sigma_1,$$

the above expression can be written as

$$G_1^2 + A_3\Sigma_1 + \Sigma_1 A_3^T = 0. \tag{5.6}$$

Following the approach in [31], the standard transformation matrix J_4 corresponding to A_3 is given by

$$J_4 = \begin{pmatrix} n_1 & n_2 & n_3 & n_4 \\ 0 & a_{32}(a_{32} - a_{22} + a_{33}) & -(a_{32} + a_{22})(a_{32} - a_{22} + a_{33}) & a_{33}^2 \\ 0 & 0 & a_{32} - a_{22} + a_{33} & -a_{33} \\ 0 & 0 & 0 & 1 \end{pmatrix},$$

where

$$\begin{aligned} n_1 &= -a_{14}a_{32}\left(a_{32} - a_{22} + a_{33}\right), \\ n_2 &= -a_{32}\left(a_{11} + a_{22} + a_{33}\right)\left(a_{32} - a_{22} + a_{33}\right), \\ n_3 &= \left[a_{13}a_{32} + a_{22}\left(a_{32} + a_{22}\right) + a_{33}^2\right]\left(a_{32} - a_{22} + a_{33}\right), \\ n_4 &= -a_{13}a_{32}\left(a_{32} - a_{22} + a_{33}\right) - a_{33}^3. \end{aligned}$$

Thus, we obtain

$$A_4 = J_4 A_3 J_4^{-1} = \begin{pmatrix} -a_1 & -a_2 & -a_3 & -a_4 \\ 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \end{pmatrix},$$

where $a_1 = p_1 + a_{44}$, $a_2 = p_2 + p_1 a_{44}$, $a_3 = p_3 + p_2 a_{44}$, and $a_4 = p_3 a_{44}$. Notably, the positivity conditions $p_i > 0$ for $i = 1, 2, 3$ together with $a_{44} > 0$ imply that $a_i > 0$ for all $i = 1, 2, 3, 4$. Moreover, one obtains

$$a_1(a_2a_3 - a_1a_4) - a_3^2 = (a_{44}^3 + p_1a_{44}^2 + p_2a_{44} + p_3)(p_1p_2 - p_3) > 0.$$

Based on the preceding discussion, Eq (5.6) can be transformed into

$$J_4 G_1^2 J_4^{\mathsf{T}} + (J_4 A_3 J_4^{-1})(J_4 \Sigma_1 J_4^{\mathsf{T}}) + (J_4 \Sigma_1 J_4^{\mathsf{T}})(J_4 A_3 J_4^{-1})^{\mathsf{T}} = 0. \tag{5.7}$$

Furthermore, it is known that $J_4 G_1^2 J_4^{\mathrm{T}} = \mathrm{diag}(\sigma^2 n_1^2, 0, 0, 0)$. Let $\rho = \sigma n_1$, $\Sigma_2 = \rho^{-2} J_4 \Sigma_1 J_4^{\mathrm{T}}$, and $G_0 = \mathrm{diag}(1, 0, 0, 0)$. Then Eq (5.7) becomes

$$G_0^2 + A_4 \Sigma_2 + \Sigma_2 A_4^{\mathrm{T}} = 0. \tag{5.8}$$

By Lemma 1, Σ_2 is a positive definite matrix. The explicit form of Σ_2 can be expressed as

$$\Sigma_2 = \begin{pmatrix} \sigma_{11} & 0 & \sigma_{13} & 0 \\ 0 & \sigma_{22} & 0 & \sigma_{24} \\ \sigma_{13} & 0 & \sigma_{33} & 0 \\ 0 & \sigma_{24} & 0 & \sigma_{44} \end{pmatrix},$$

where

$$\begin{aligned} \sigma_{11} &= \frac{a_2a_3 - a_1a_4}{2\left(a_1a_2a_3 - a_3^2 - a_1^2a_4\right)}, \\ \sigma_{13} &= -\sigma_{22} = -\frac{a_3}{2\left(a_1a_2a_3 - a_3^2 - a_1^2a_4\right)}, \\ \sigma_{24} &= -\sigma_{33} = -\frac{a_1}{2\left(a_1a_2a_3 - a_3^2 - a_1^2a_4\right)}, \\ \sigma_{44} &= \frac{a_1a_2 - a_3}{2a_4\left(a_1a_2a_3 - a_3^2 - a_1^2a_4\right)}. \end{aligned}$$

From the relations $J\Sigma J^{\mathrm{T}} = \Sigma_1$ and $\Sigma_1 = \rho^2 J_4^{-1}\Sigma_2 (J_4^{-1})^{\mathrm{T}}$, the explicit form of Σ can be derived.

$$\Sigma = J^{-1}\Sigma_1 (J^{-1})^{\mathrm{T}} = \rho^2 (J_4 J)^{-1}\Sigma_2 \Big[(J_4 J)^{-1}\Big]^{\mathrm{T}}.$$

Moreover, this matrix is verified to be positive definite.

In summary, the probability density function near $(0, 0, 0, 0)$ is

$$\Phi(z_1, z_2, z_3, z_4) = (2\pi)^{-2}|\Sigma|^{-\frac{1}{2}} \exp\Big[-\tfrac{1}{2}(z_1, z_2, z_3, z_4)\Sigma^{-1}(z_1, z_2, z_3, z_4)^{\mathrm{T}}\Big].$$

Corollary 1. *When $R_0^s > 1$, the solution to system* (3.1) *follows a normal probability density distribution $\Phi(S, E, I, \xi)$ near $(S^*, E^*, I^*, \bar{\xi})$. This distribution is explicitly defined as*

$$\Phi(S, E, I, \xi) = (2\pi)^{-2}|\Sigma|^{-\frac{1}{2}} \exp\Big[-\frac{1}{2}(S - S^*, E - E^*, I - I^*, \xi - \bar{\xi})\Sigma^{-1} (S - S^*, E - E^*, I - I^*, \xi - \bar{\xi})^T\Big].$$

6. Numerical simulation

Next, we use the numerical simulation method to discuss the dynamical properties of model (3.1). First, discretize model (3.1). Let $x_1(t) = \xi(t) - \bar{\xi}$. The corresponding discretized system of (3.1) is given by [32]

$$\begin{cases} S^{j+1} = S^j + \Big[\Lambda - (\bar{\xi} + x_1^j)\dfrac{S^j I^j}{1 + \alpha(I^j)^2} - \eta S^j\Big]\Delta t, \\ E^{j+1} = E^j + \Big[(\bar{\xi} + x_1^j)\dfrac{S^j I^j}{1 + \alpha(I^j)^2} - (\eta + k)E^j\Big]\Delta t, \\ I^{j+1} = I^j + [kE^j - (\eta + \delta)I^j]\Delta t, \\ x_1^{j+1} = x_1^j - \theta x_1^j \Delta t + \sigma\sqrt{\Delta t}\, y_{1,j} + \frac{\sigma^2}{2}(y_{1,j}^2 - 1)\Delta t. \end{cases}$$

Here, $\Delta t > 0$ denotes the time-step size, and $y_{1,j}$ are random variables following a Gaussian distribution $\mathcal{N}(0, 1)$ for $j = 1, 2, \ldots, n$. Moreover, (S^j, E^j, I^j, x_1^j) represents the result of the j-th iteration of the discrete equations. In the following numerical experiments, all stochastic simulations are conducted using the Euler–Maruyama discretization scheme with step size $\Delta t = 0.01$ over a total time horizon $T = 100$. For each parameter set, we generate $M = 10,000$ independent sample paths to ensure statistical reliability.

Let us establish the starting state as $(S(0), E(0), I(0), \xi(0)) = (2, 1, 1.1, 0.4)$. The parameters used in the following numerical examples are summarized in Table 1, along with their values and sources.

Table 1. Parameter values used in numerical simulations.

Parameter	Example 6.1	Example 6.2	Source
Λ	0.6	1	Assumed
η	0.35	0.2	Assumed
$\bar{\xi}$	0.4	0.4	[21]
θ	0.6	0.6	Assumed
σ	0.08	0.01	[21]
k	1/3	1/3	[21]
δ	1/9	1/9	[21]
α	0.1	0.1	Assumed (see [5])

Example 6.1. Select parameters from Table 1. Through a simple calculation, we obtain

$$R_0 = 0.7254 < 1, R_0^E = 0.9354 < 1.$$

This thus meets all the requirements stipulated in Theorem 3. Figure 1 presents the corresponding simulation results. Clearly, both the infectious and exposed classes approach zero, indicating that the disease will eventually become extinct in the long term. The trajectories of $E(t)$ and $I(t)$ exhibit a clear exponential decay, falling below a sufficiently small condition within a finite time (approximately within $t < 50$), as shown in Figure 1. Moreover, the susceptible population $S(t)$ converges to $\frac{\Lambda}{\eta} = 1.714$, consistent with the disease-free equilibrium of the deterministic system. From the expression of R_0^E, it is observed that as $\sigma \to 0$, R_0^E tends to $\sqrt{R_0}$. Hence, when environmental noise is sufficiently small, the condition $R_0^E < 1$ aligns with the deterministic extinction condition $R_0 < 1$, providing a suffcient condition for disease extinction across both modeling frameworks.

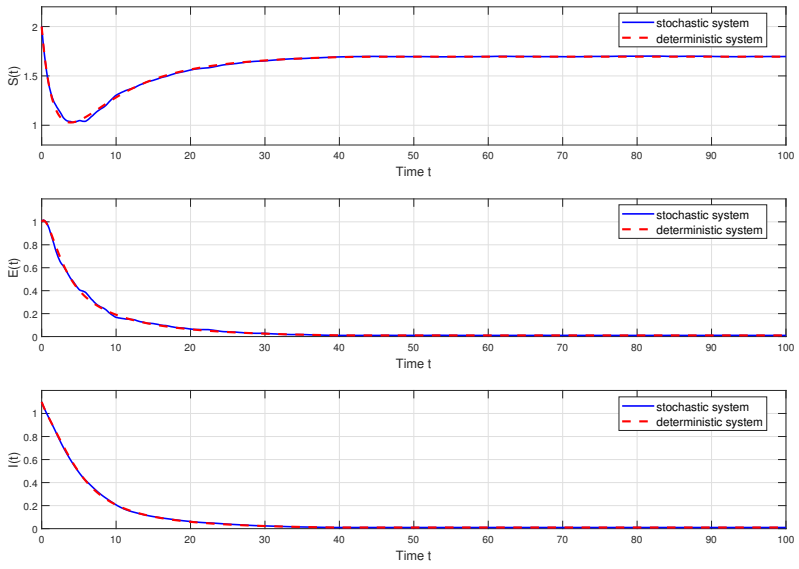

Figure 1. The solution trajectories of stochastic and deterministic systems under the condition $R_0^E < 1$.

Example 6.2. Select parameters from Table 1, and direct calculation via MATLAB yields

$$R_0 = 4.0179 > 1, R_0^s = 2.3760 > 1,$$

and the equilibrium $(S^*, E^*, I^*, \xi^*) = (1.4916, 1.3165, 1.4096, 0.4)$. These parameter values satisfy the conditions of Theorem 4. Moreover, the covariance matrix is obtained as

$$\Sigma = \begin{pmatrix} 0.3983 & -0.2109 & -0.1519 & -0.1234 \\ -0.2109 & 0.1406 & 0.0642 & 0.0871 \\ -0.1519 & 0.0642 & 0.0688 & 0.0319 \\ -0.1234 & 0.0871 & 0.0319 & 0.0833 \end{pmatrix}.$$

Consequently, the process $(S(t), E(t), I(t), \xi(t))$ obeys a normal (Gaussian) probability density in its stationary state

$$(S, E, I, \xi) \sim \mathcal{N}\Big((1.4916, 1.3165, 1.4096, 0.4)^{\mathrm{T}}, \Sigma\Big).$$

Furthermore, three marginal density functions can be derived:

$$\Phi_S = 0.6321e^{-1.2553(S-1.4916)^2}, \quad \Phi_E = 1.0639e^{-3.5562(E-1.3165)^2}, \quad \Phi_I = 1.5210e^{-7.2674(I-1.4096)^2}.$$

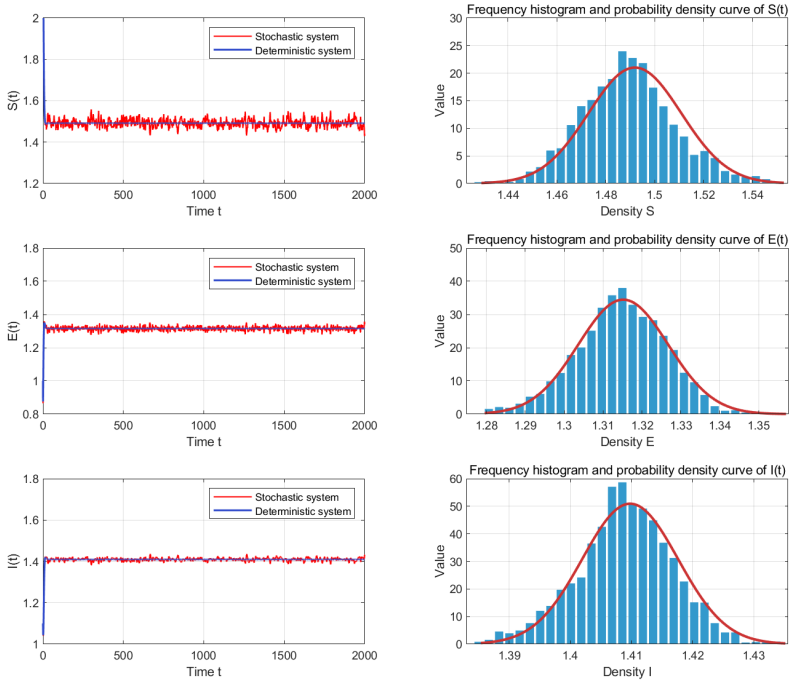

Figure 2. The left column presents the $(S(t), E(t), I(t))$ trajectories of the stochastic and deterministic systems under $\sigma = 0.01$; the right columns display histograms and marginal densities of the solution.

Figure 2 reveals the existence of a stationary distribution for system (3.1), which suggests that the disease persists in a stochastic sense. As uncertainty is inherent in transmission dynamics, the number

of infected individuals exhibits dynamic fluctuations around a mean level, rather than remaining at a fixed equilibrium. The amplitude of these fluctuations is proportional to the strength of the environmental noise. Regarding the histogram on the right, the empirical distributions of S, E, I, ξ all exhibit bell-shaped curves that closely resemble normal distributions, which is consistent with the theoretical prediction that the stationary distribution near the endemic equilibrium is Gaussian. Moreover, the peaks of the histograms are located at $S \approx 1.49$, $E \approx 1.32$, $I \approx 1.41$, and $\bar{\xi} \approx 0.4$, which are in excellent agreement with the theoretical means $(S^*, E^*, I^*, \bar{\xi}) = (1.4916, 1.3165, 1.4096, 0.4)$. The widths of the histograms, representing the dispersion of each variable, are consistent with the theoretical standard deviations derived from Σ. Changing the noise intensity to $\sigma = 0.02$ and $\sigma = 0.04$ yields Figures 3 and 4, respectively. Comparing Figures 2–4, it can be observed that as the perturbation intensity increases, the stochastic system exhibits sustained oscillations with elevated amplitude about the deterministic equilibrium. The right panels of Figures 2–4 show that a smaller noise intensity leads to a narrower distribution range of data points and a sharper marginal density curve. As the noise intensity approaches zero, the behavior of the stochastic system converges to that of its deterministic counterpart.

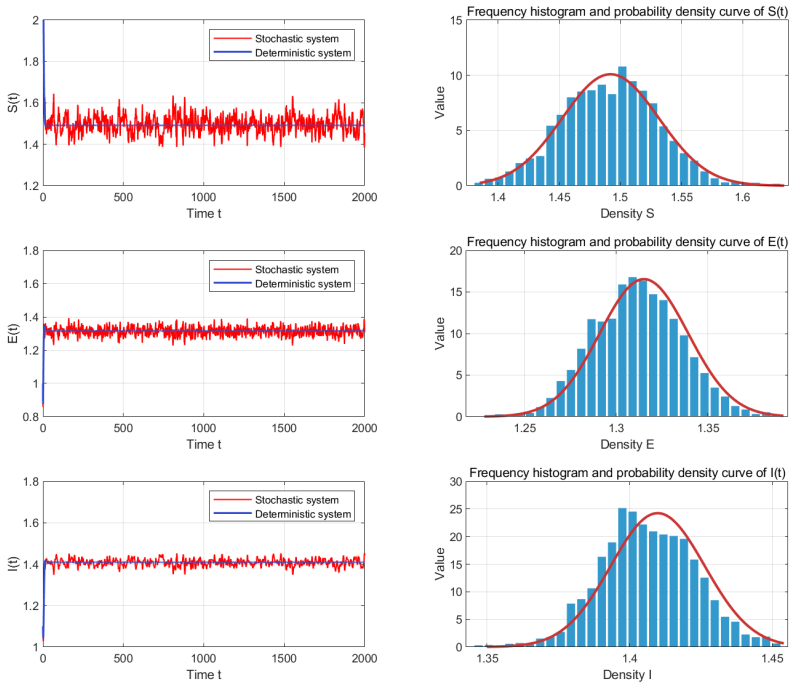

Figure 3. The left column presents the $(S(t), E(t), I(t))$ trajectories of the stochastic and deterministic systems under $\sigma = 0.02$; the right columns display histograms and marginal densities of the solution.

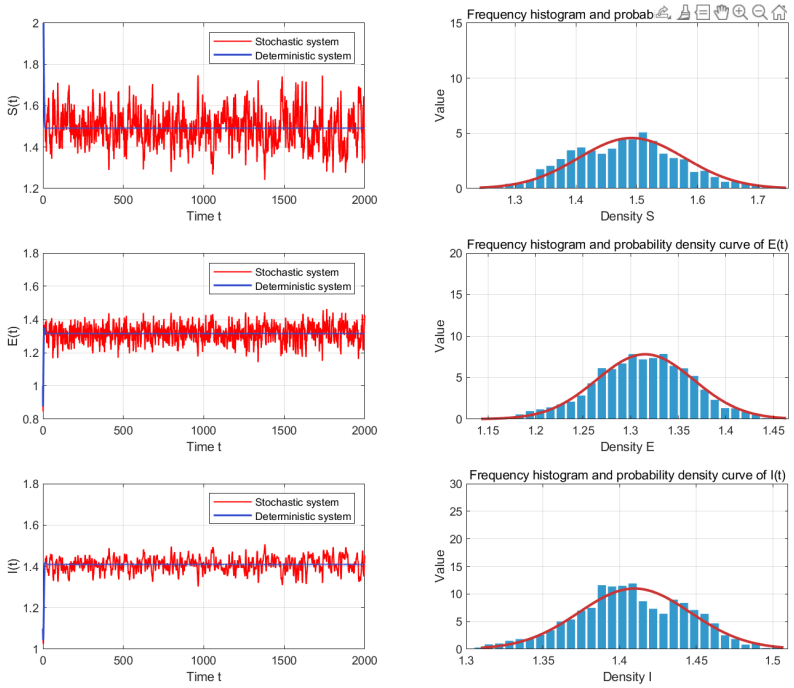

Figure 4. The left column presents the $(S(t), E(t), I(t))$ trajectories of the stochastic and deterministic systems under $\sigma = 0.04$; the right columns display histograms and marginal densities of the solution.

Example 6.3. Keeping all other parameters from Example 6.2 unchanged, the mean-reversion speed θ is varied as follows:

$$\theta = 0.2, \quad \theta = 0.4, \quad \theta = 0.6.$$

The evolution of $S(t), E(t), I(t)$ under these three choices is shown in Figure 5. Clearly, as θ increases, the solution of the stochastic system gradually approaches that of deterministic model (2.1).

Example 6.4 Retaining all other parameters from Example 6.2, the psychological-effect parameter α is varied to analyze its influence on the number of infected individuals I. The following three values are considered:

$$\alpha = 0.1, \quad \alpha = 0.2, \quad \alpha = 0.4.$$

Figure 6 displays the evolution of the infected population $I(t)$. It can be observed that the strength of the psychological effect contributes positively to suppressing the number of infections: as α increases, the magnitude of $I(t)$ is reduced to a certain extent.

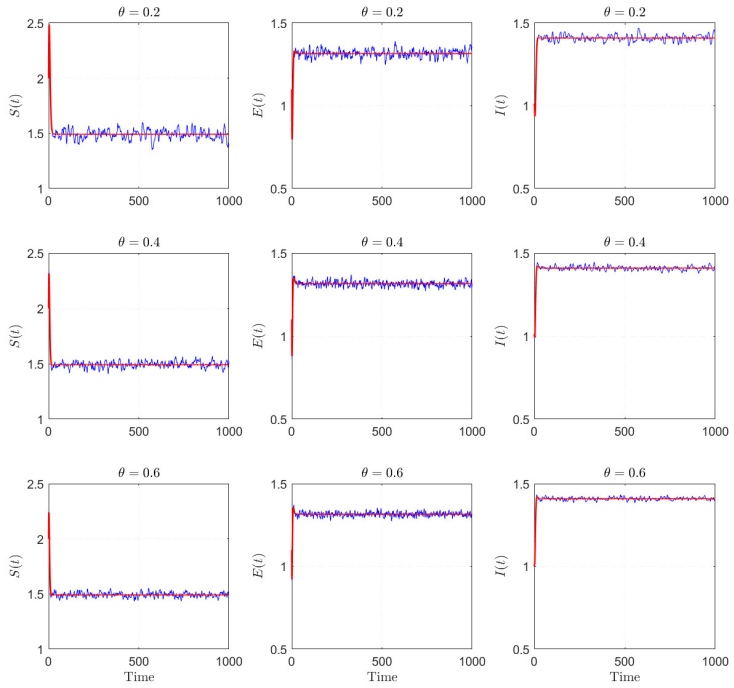

Figure 5. Solution of system (3.1) and its corresponding deterministic system with different θ.

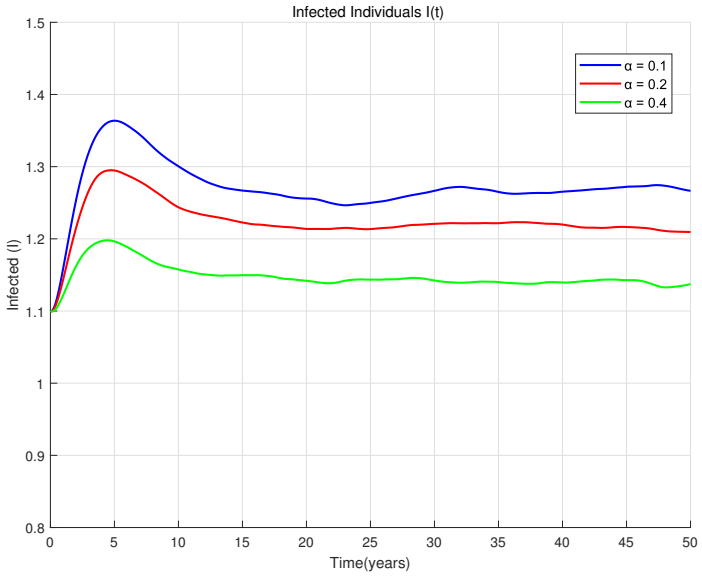

Figure 6. The stochastic solutions of I under $\alpha = 0.1$, 0.2, and 0.4.

7. Discussion and conclusions

For emerging and severe infectious diseases such as COVID-19 and SARS, this study employs a nonlinear, non-monotone incidence rate $\frac{\xi SI}{1+\alpha I^2}$ to capture the psychological effect on the public induced

by large-scale epidemic spread. Meanwhile, the OU process is introduced to describe the random fluctuations of the infection rate. The proposed model provides a reliable theoretical framework for revealing the transmission patterns of such diseases. For the corresponding deterministic SEIR model, a global qualitative analysis is conducted concerning the existence and stability of the disease-free equilibrium and the endemic equilibrium. The basic reproduction number has been obtained as

$$R_0 = \frac{\bar{\xi}\Lambda k}{\eta(\eta+\delta)(\eta+k)}.$$

It reveals that the disease-free equilibrium attains global asymptotic stability under the condition $R_0 < 1$, while the endemic equilibrium is locally asymptotically stable when $R_0 > 1$. For the stochastic SEIR model, the long-term dynamical behavior, including disease extinction and persistence, is investigated using Itô's formula, suitably constructed Lyapunov functions, and inequality techniques. When $R_0^E < 1$, the disease undergoes exponential extinction with probability one; when $R_0^s > 1$, the system admits a stationary distribution, indicating stochastic persistence of the disease. By linearizing the stochastic system around the endemic equilibrium and solving the corresponding Lyapunov equation, the structure of the stationary probability density function is shown to be Gaussian, and its covariance matrix is derived explicitly. Finally, numerical simulations verify the theoretical results and reveal the influence of key parameters. The simulation outcomes demonstrate that the noise intensity σ and the mean-reversion speed θ directly affect the magnitude of fluctuations in the system's solution: when the noise intensity is reduced or the mean-reversion speed is increased, the stochastic solution tends to approach the deterministic equilibrium more closely. The influence of the psychological effect parameter α on the number of infected individuals I is also examined. The psychological effect contributes positively to suppressing infections, although it cannot ultimately prevent a large-scale outbreak. As α increases, the number of infected individuals is reduced and better controlled. These findings offer more comprehensive theoretical support for optimizing epidemic control strategies and enhancing the precision of emergency response measures.

Use of AI tools declaration

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

Conflict of interest

The authors declare there are no conflicts of interest.

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