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Dynamics of Nonlinear Stochastic SEIR Infectious Disease Model with Isolation and Latency Period

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Abstract: This article establishes and studies a SEIR infectious disease model with higher-order perturbation. Firstly, we proved the existence and uniqueness of the overall positive solution of the model. Secondly, by constructing a Lyapunov function, we obtained sufficient conditions for the existence and uniqueness of the ergodic stationary distribution of the positive solution of the model. Then, it was proved that infectious diseases would become extinct under certain conditions. Finally, this article verified the theoretical analysis results by numerically simulating the process of infectious diseases from outbreak to extinction, the numerical simulation results are symmetrical with the theoretical analysis.

Keywords: higher-order perturbation; SEIR model; stationary; extinction

1. Introduction

Infectious disease is a medical problem, and its outbreak and prevalence are also a social problem, which will affect economic development and social stability. For example, COVID-19 broke out in China in 2020. In the absence of vaccines or antiviral drugs, physical protection and social isolation are crucial to control the epidemic. Therefore, establishing a reasonable SEIR model to study the epidemic trends of infectious diseases has theoretical and practical significance. Many scholars have studied different SEIR models, for example, Cooke and Driessche et al. [1] proposed and studied a classic SEIR epidemic model, which has become the most important model in disease control. A. Abta et al. [2] studied the delayed SIR model and the corresponding SEIR model from the perspective of global stability, and compared the global behavior of the delayed SIR model and the corresponding SEIR model through numerical simulations. S. Han et al. [3] studied a diffusion SEIR epidemic model with incidence to describe the spread of infection between susceptible and infected individuals. Jiao et al. [4] established a SEIR model with infectious disease latency and home isolation.

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - \mu S(t) - \beta(1-\theta_1)S(t)(I(t)+\theta_2 E(t)), \\ \frac{dE(t)}{dt} = \beta(1-\theta_1)S(t)(I(t)+\theta_2 E(t)) - (\mu+\delta)E(t), \\ \frac{dI(t)}{dt} = \delta E(t) - (\mu+\gamma+\nu)I(t), \\ \frac{dR(t)}{dt} = (\gamma+\theta_3\nu)I(t) - \mu R(t), \end{cases} \tag{1}$$

where $S(t)$, $E(t)$, $I(t)$, and $R(t)$ represent the number of susceptible individuals, latent individuals, infected individuals, and recovered individuals at time t respectively. Λ represents the population input rate, μ represents the natural mortality rate, β, δ, γ represents the

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conversion rate from $S(t)$ to $E(t)$, $E(t)$ to $I(t)$, and $I(t)$ to $R(t)$ respectively, ν represents the hospitalization rate, θ_1 is the home isolation rate of susceptible individuals, θ_2 is the infection rate during the latent period of the disease, θ_3 is the circulation rate of $I(t)$, all parameters are positive, and $\theta_i \in (0,1)(i = 1,2,3)$. Jiao et al. [4] provided the basic reproduction number:

$$R_0 = \frac{\Lambda\beta(1-\theta_1)[\delta + \theta_2(\mu+\gamma+\nu)]}{\mu(\mu+\gamma+\nu)(\mu+\delta)},$$

and it has been proven that if $R_0 < 1$, the disease-free equilibrium point $Q_0 = (S_0, 0, 0, 0)$ is globally asymptotically stable, $S_0 = \frac{\Lambda}{\mu}$, and if $R_0 > 1$, the local equilibrium point $Q^* = (S^*, E^*, I^*, R^*)$ is globally asymptotically stable. R_0 is defined as the average number of secondary infections that occur when one infective is introduced into a completely susceptible host population [5], where

$$\Omega = \left\{(S(t), E(t), I(t), R(t)) \in \mathbb{R}^4_+ \; 0 < S(t) + E(t) + I(t) + R(t) \leq \frac{\Lambda}{\mu}\right\},$$

it means that the disease will spread and persist in the population.

Research shows that infectious disease model is always influenced by environmental changes [6–8], which brings a certain degree of randomness to birth rate, mortality rate, and transmission coefficient [9]. Therefore in epidemic dynamics, compared with the corresponding deterministic models [1–4], stochastic model may be a more appropriate way of modeling epidemics in many circumstances [10,11], epidemic systems with random disturbances are more realistic and symmetrical [12–14]. From a biological and mathematical perspective, there are different methods to establish a stochastic infectious disease model, such as adding Poisson white noise to the epidemic model [15], infectious disease model with Levy noise [16,17], or Markovian switching [18], and incorporating white noise interference into epidemic models [19–21]. This article assumes that model (1) is subject to random interference from environmental white noise, by adding the higher-order perturbation into model (1), we get the following stochastic infectious disease model

$$\begin{cases} \mathrm{d}S(t) = [\Lambda - \mu S(t) - \beta(1-\theta_1)S(t)(I(t) + \theta_2 E(t))]\mathrm{d}t + (\sigma_{11} + \sigma_{12}S(t))S(t)\mathrm{d}B_1(t), \\ \mathrm{d}E(t) = [\beta(1-\theta_1)S(t)(I(t) + \theta_2 E(t)) - (\mu+\delta)E(t)]\mathrm{d}t + (\sigma_{21} + \sigma_{22}E(t))E(t)\mathrm{d}B_2(t), \\ \mathrm{d}I(t) = [\delta E(t) - (\mu+\gamma+\nu)I(t)]\mathrm{d}t + (\sigma_{31} + \sigma_{32}I(t))I(t)\mathrm{d}B_3(t), \\ \mathrm{d}R(t) = [(\gamma + \theta_3\nu)I(t) - \mu R(t)]\mathrm{d}t + (\sigma_{41} + \sigma_{42}R(t))R(t)\mathrm{d}B_4(t), \end{cases} \tag{2}$$

where $B_i(t)(i = 1,2,3,4)$ is the independent standard Brown motion, $\sigma_{ij} > 0(i = 1,2,3,4; j = 1,2)$ is the intensity of random white noise, which is used to describe the volatility of disturbances. Similarly, stochastic model also have the basic reproduction number $\mathcal{R}_0^S$, random disturbances can affect the dynamical system, cause $\mathcal{R}_0^S < R_0$.

The structure of this manuscript is as follows. In Section 2, we prove the existence and uniqueness of global positive solutions for model (2). In Section 3, we present the existence of ergodic stationary distributions for model (2), it is equivalent to $\mathcal{R}_0^S > 1$. In Section 4, we provide sufficient conditions for the extinction of infectious diseases in model (2), it is equivalent to $\mathcal{R}_0^S < 1$. In Section 5, we verify the theoretical results through numerical simulations.

2. The Existence and Uniqueness of the Overall Positive Solution

In this section, we use the Lyapunov function analysis method to prove the uniqueness of the global positive solution for model (2), and obtain the following results:

Theorem 1. *When $t \geq 0$, for any initial value $(S(0), E(0), I(0), R(0)) \in \mathbb{R}^4_+$, there exists a unique positive solution $(S(t), E(t), I(t), R(t)) \in \mathbb{R}^4_+$ for model (2), and this solution has a probability of 1 falling within $\mathbb{R}^4_+$.*

In the actual process of infectious disease transmission, the number of susceptible individuals $S(t)$, latent individuals $E(t)$, infected individuals $I(t)$, and recovered individuals $R(t)$, all of them are positive integers, so the solutions of model (2) should also be overall positive solutions, the solution of the model is symmetrical with the actual number of people, this is a simple and understandable concept. From the perspective of the model, the existence and uniqueness of solutions is a prerequisite for ensuring the meaningfulness of the model, so Theorem 1 is given here to indicate that model (2) has a positive solution, please refer to Appendix A of the Supplementary Materials for the proof process.

3. The Existence of Ergodic Stationary Distribution

Biology suggests that infectious diseases will spread and persist within populations. From a mathematical perspective, it indicates that the solution of the model is a stationary distribution, it is equivalent to $\mathcal{R}_0^S > 1$. So in this section, based on Khasminskii theory and Lyapunov function method, we studied the existence of ergodic stationary distribution for model (2).

Let $X(t)$ be a regular time-homogeneous Markov process in $\mathbb{R}^d$, it is described by the stochastic differential equation

$$\mathrm{d}X(t) = f(X(t))\mathrm{d}t + \sum_{r=1}^{k} g_r(X(t))\mathrm{d}B_r(t),$$

the diffusion matrix of the process $X(t)$ is defined as follows

$$A(x) = (a_{ij}(x)),\ a_{ij}(x) = \sum_{i=1}^{k} g_r^i(x) g_r^j(x).$$

Lemma 1 ([22]). *The Markov process $X(t)$ has a unique ergodic stationary distribution $\pi(\cdot)$ if there exists a bounded open domain $U \subset \mathbb{R}^d$ with regular boundary Γ, having the following properties:*

(i) the diffusion matrix $A(x)$ is strictly positive definite for all $x \in U$,

(ii) there exists a nonnegative C^2-function V such that LV is negative for any $\mathbb{R}^d \setminus U$.

Theorem 2. *For any initial condition $(S(0), E(0), I(0), R(0)) \in \mathbb{R}^4_+$, assume that*

$$\mathcal{R}_0^S := \frac{\Lambda\beta(1-\theta_1)[\delta + \theta_2(\mu+\gamma+\nu+\frac{\sigma_{31}^2}{2})]}{(\mu + \frac{\sigma_{11}^2}{2} + 2\sqrt[3]{\Lambda^2\sigma_{12}^2} + 2\sqrt{\Lambda\sigma_{11}\sigma_{12}})(\mu+\gamma+\nu+\frac{\sigma_{31}^2}{2})(\mu+\delta+\frac{\sigma_{21}^2}{2} + \frac{8}{3}\sqrt[3]{\frac{\Lambda^2\sigma_{22}^2}{2}})} > 1,$$

then model (2) admits a unique stationary distribution $\pi(\cdot)$ and it has the ergodic property.

Remark 1. *In the case of $\mathcal{R}_0^S > 1$, the model (2) can reflect the persistent nature of the disease. According to the expression of $\mathcal{R}_0^S$, if stochastic perturbation $\sigma_{11} = \sigma_{12} = \sigma_{21} = \sigma_{22} = \sigma_{31} = 0$, the expression of $\mathcal{R}_0^S$ is equal to the basic reproduction number R_0 in the deterministic model (1). For $\mathcal{R}_0^S$ and R_0, we conclude that $\mathcal{R}_0^S = \mathcal{R}_0 > 1$ is equivalent to $R_0 > 1$. If stochastic perturbation $\sigma_{11} \neq 0$, $\sigma_{12} \neq 0$, $\sigma_{21} \neq 0$, $\sigma_{22} \neq 0$, $\sigma_{31} \neq 0$, we can prove $\mathcal{R}_0^S/\mathcal{R}_0 < 1$, is equivalent to $\mathcal{R}_0^S < \mathcal{R}_0$.*

Theorem 2 states that under certain conditions, the spread of diseases is sustained and stable, the solution of ergodic stationary distribution is symmetrical with the persistence of

the disease, and this research has practical significance. To prove Theorem 2 according to Lemma 1, we only need to verify that Theorem 2 satisfies the conditions of Lemma 1 (i) (ii), please refer to Appendix B of the supplementary materials for detailed proof.

4. The Extinction of Diseases

In this section, we will provide sufficient conditions for the extinction of this disease, it is divided into two cases, $\mathcal{R}_0 \geq 1 > \mathcal{R}_0^S$ and $\mathcal{R}_0^S < \mathcal{R}_0 < 1$. Then, we establish the following theorem.

Theorem 3. *Let* $(S(t), E(t), I(t), R(t))$ *be the solution to model* (2) *with any initial value* $(S(0), E(0), I(0), R(0)) \in \mathbb{R}_+^4$. *Assume that* $\mu > \sigma_{11}^2/2$.

(1) If $\mathcal{R}_0 \geq 1 > \mathcal{R}_0^S$, *the solution has the following property:*

$$\limsup_{t\to+\infty} \frac{1}{t} \ln[E + \frac{\beta(1-\theta_1)S_0}{\mu+\gamma+\nu} I] \leq v_1, \ a.s.$$

where

$$v_1 := (\mu+\delta)(\mathcal{R}_0 - 1) + [\beta(1-\theta_1)\theta_2 + \tfrac{\mu+\gamma+\nu}{S_0}] \int_0^{+\infty} |x - S_0| \pi(x) \mathrm{d}x - \tfrac{1}{2(\sigma_{21}^{-2}+\sigma_{31}^{-2})},$$
$$\mathcal{R}_0 = \tfrac{\beta(1-\theta_1)S_0[\theta_2(\mu+\gamma+\nu)+\delta]}{(\mu+\gamma+\nu)(\mu+\delta)}, \ S_0 = \tfrac{\Lambda}{\mu}.$$

(2) If $\mathcal{R}_0^S < \mathcal{R}_0 < 1$ *and there is a constant* l $(0 < l < \frac{(\mu+\delta)(1-\mathcal{R}_0)}{\delta})$, *we get*

$$\limsup_{t\to+\infty} \frac{1}{t} \ln[E + (\frac{\beta(1-\theta_1)S_0}{\mu+\gamma+\nu} + l) I] \leq v_2, \ a.s.$$

where

$$v_2 := \tfrac{\max\{\beta(1-\theta_1), \beta(1-\theta_1)\theta_2\}}{\min\left\{1, \frac{\beta(1-\theta_1)S_0}{\mu+\gamma+\nu} + l\right\}} \int_0^{+\infty} |x - S_0| \pi(x) \mathrm{d}x - \tfrac{\min\{(\mu+\delta)(1-\mathcal{R}_0) - l\delta, l(\mu+\gamma+\nu)\}}{\max\left\{1, \frac{\beta(1-\theta_1)S_0}{\mu+\gamma+\nu} + l\right\}} - \tfrac{1}{2(\sigma_{21}^{-2}+\sigma_{31}^{-2})},$$
$$\mathcal{R}_0 = \tfrac{\beta(1-\theta_1)S_0[\theta_2(\mu+\gamma+\nu)+\delta]}{(\mu+\gamma+\nu)(\mu+\delta)}, \ S_0 = \tfrac{\Lambda}{\mu}.$$

Either if $v_1 < 0$ *or* $v_2 < 0$, *then the disease will become extinct exponentially with probability one, i.e.,*

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

In addition, the distribution of $S(t)$ *converges weakly to the measure which has the density*

$$\pi(x) = Q x^{-2-\frac{2(2\Lambda\sigma_{12}+\mu\sigma_{11})}{\sigma_{11}^3}} (\sigma_{11} + \sigma_{12}x)^{-2+\frac{2(2\Lambda\sigma_{12}+\mu\sigma_{11})}{\sigma_{11}^3}} e^{-\frac{2}{\sigma_{11}(\sigma_{11}+\sigma_{12}x)}(\frac{\Lambda}{x} + \frac{2\Lambda\sigma_{12}+\mu\sigma_{11}}{\sigma_{11}})}, \ x \in (0, +\infty),$$

where Q *is a constant such that* $\int_0^{+\infty} \pi(x)\mathrm{d}x = 1$.

Remark 2. *In Case 1* $\mathcal{R}_0 \geq 1 > \mathcal{R}_0^S$, *in order to eradicate the disease, we need* $v_1 < 0$, *so* σ_{21}^2 *and* σ_{31}^2 *must be large enough, this is because stochastic perturbation in model* (2). *Compare with the deterministic model* (1), *if* $\mathcal{R}_0 > 1$ *(i.e.,* $R_0 > 1$ *(see Remark 1)), so the endemic equilibrium is globally asymptotically stable. From a biological perspective, diseases can break out or extinct in populations due to significant external factors such as the environment. Therefore, our conclusion is different from the deterministic model* (1), *we show that higher-order perturbation will lead to the extinction of infectious disease.*

Case 2 $\mathcal{R}_0^S < \mathcal{R}_0 < 1$, *in order to eradicate the disease, we need* $v_2 < 0$, *besides* σ_{21} *and* σ_{31}, *it is also related to other parameters, such as* μ, γ, ν, *etc. From an epidemiological perspective, factors such as isolation rate, rehabilitation rate, and mortality rate of infectious diseases can accelerate their extinction.*

Theorem 3 states that when model (2) satisfies certain conditions, the disease will become extinct, which is theoretically the most ideal circumstances, this is the symmetry between theory and practice that we expect. To prove Theorem 3, we considered two cases and obtained the conditions for disease extinction, please refer to Appendix C of the supplementary materials for detailed proof.

In order to better understand the differences between stochastic model (2) and deterministic model (1), we introduce R_0, $\mathcal{R}_0^S$, $\mathcal{R}_0$ in Table 1.

Table 1. Parameter description.

Basic Reproduction Number	Condition	Instructions
Deterministic model R_0	$R_0 > 1$	Disease persists
	$R_0 < 1$	Disease extinction
Stochastic model $\mathcal{R}_0^S$	$\sigma_{ij} = 0$	$\mathcal{R}_0^S = \mathcal{R}_0$
	$\sigma_{ij} \neq 0$	$\mathcal{R}_0^S < \mathcal{R}_0$
	$\mathcal{R}_0^S > 1$	Disease persists
	$\mathcal{R}_0 \geq 1 > \mathcal{R}_0^S$	Disease extinction
	$\mathcal{R}_0^S < \mathcal{R}_0 < 1$	Disease extinction

5. Numerical Simulation

By introducing and proving the theorems in Sections 3 and 4, we used numerical simulations to verify the conclusions in Sections 3 and 4. Applying Milstein's high-order method [23], we obtained the discrete equation of the model (2)

$$\begin{cases} S_{k+1} = S_k + [\Lambda - \mu S_k - \beta(1-\theta_1)S_k(I_k + \theta_2 E_k)]\triangle t + (\sigma_{11} + \sigma_{12}S_k)S_k\xi_{1k}\sqrt{\triangle t} \\ \quad + (\sigma_{11} + \sigma_{12}S_k)(\sigma_{11} + 2\sigma_{12}S_k)S_k(\xi_{1k}^2 - 1)\frac{\triangle t}{2}, \\ E_{k+1} = E_k + [\beta(1-\theta_1)S_k(I_k + \theta_2 E_k) - (\mu + \delta)E_k]\triangle t + (\sigma_{21} + \sigma_{22}E_k)E_k\xi_{2k}\sqrt{\triangle t} \\ \quad + (\sigma_{21} + \sigma_{22}E_k)(\sigma_{21} + 2\sigma_{22}E_k)E_k(\xi_{2k}^2 - 1)\frac{\triangle t}{2}, \\ I_{k+1} = I_k + [\delta E_k - (\mu + \gamma + \nu)I_k]\triangle t + (\sigma_{31} + \sigma_{32}I_k)I_k\xi_{3k}\sqrt{\triangle t} \\ \quad + (\sigma_{31} + \sigma_{32}I_k)(\sigma_{31} + 2\sigma_{32}I_k)I_k(\xi_{3k}^2 - 1)\frac{\triangle t}{2}, \\ R_{k+1} = R_k + [(\gamma + \theta_3\nu)I_k - \mu R_k]\triangle t + (\sigma_{41} + \sigma_{42}R_k)R_k\xi_{4k}\sqrt{\triangle t} \\ \quad + (\sigma_{41} + \sigma_{42}R_k)(\sigma_{41} + 2\sigma_{42}R_k)R_k(\xi_{4k}^2 - 1)\frac{\triangle t}{2}, \end{cases}$$

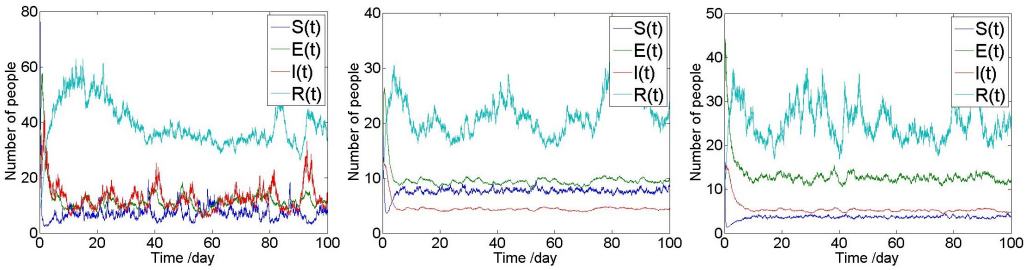
where $\xi_{ik}(i = 1,2,3,4;\ k = 1,2,\cdots,n)$ are independent random variables that follow a standard normal distribution. In the numerical simulation process, we assumed the initial value $S(0) = 40$, $E(0) = 8$, $I(0) = 16$, $R(0) = 8$, and $n = 10{,}000$, step size $\triangle t = 0.01$.

Firstly, we simulated that the model (2) is ergodic stationary distribution in Section 3, it is equivalent to $\mathcal{R}_0^S > 1$. It indicated that if treatment measures are not effective, infectious diseases will persist in the population. We have set up three different sets of data, as shown in the Table 2.

Table 2. The parameters of ergodic stationary distribution.

Sets	Λ	μ	β	δ	γ	ν	θ_1	θ_2	θ_3
1	10	0.1	0.1	0.7	0.3	0.2	0.1	0.2	0.3
2	10	0.2	0.3	0.7	0.8	0.5	0.6	0.5	0.4
3	10	0.15	0.4	0.6	0.9	0.4	0.5	0.6	0.2
Sets	σ_{11}	σ_{12}	σ_{21}	σ_{22}	σ_{31}	σ_{32}	σ_{41}	σ_{42}	$\mathcal{R}_0^S$
1	0.25	0.02	0.1	0.001	0.15	0.01	0.01	0.002	1.0617
2	0.01	0.01	0.02	0.001	0.02	0.001	0.03	0.003	1.6735
3	0.03	0.02	0.04	0.002	0.01	0.003	0.04	0.004	2.2601

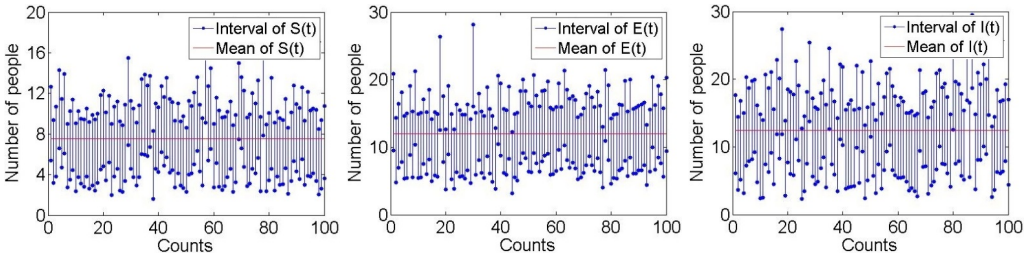
In the three sets of data, we calculated that $\mathcal{R}_0^S > 1$, as shown in the Table 2, it satisfies the conditions of Theorem 2, and from the conclusion of the theorem, it can be concluded that model (2) has a unique ergodic stationary distribution, infectious diseases will persist in the population, and as time t passes, $S(t)$, $E(t)$, $I(t)$, $R(t)$ fluctuate up and down around a certain value, respectively, the numerical simulation results are symmetrical with the persistence of the disease, as shown in the Figure 1.



(**a**) $\mathcal{R}_0^S = 1.0617 > 1$ (**b**) $\mathcal{R}_0^S = 1.6735 > 1$ (**c**) $\mathcal{R}_0^S = 2.2601 > 1$

Figure 1. Time sequence diagram of persistent infectious diseases.

To demonstrate the robustness of numerical simulation, we conducted multiple repeated simulations and established confidence intervals for $S(t)$, $E(t)$, $I(t)$, such as $\mathcal{R}_0^S = 1.0617 > 1$, $t \in [3500, 4000]$, randomly selected 100 samples from $S(t)$, $E(t)$, $I(t)$, plotted their average values over time, and established a 95% empirical confidence interval, as shown in the Figure 2.



(**a**) Confidence interval of $S(t)$ (**b**) Confidence interval of $E(t)$ (**c**) Confidence interval of $I(t)$

Figure 2. The persistent existence of infectious diseases.

From Figure 2, it can be seen that among these 100 intervals. The mean of $S(t)$ is about 7.2918, 98 of 100 contain parameter truth values; the mean of $E(t)$ is about 11.2985, 98 of 100 contain parameter truth values; the mean of $I(t)$ is about 12.6265, 94 of 100 contain parameter truth values, so the results are reliable.

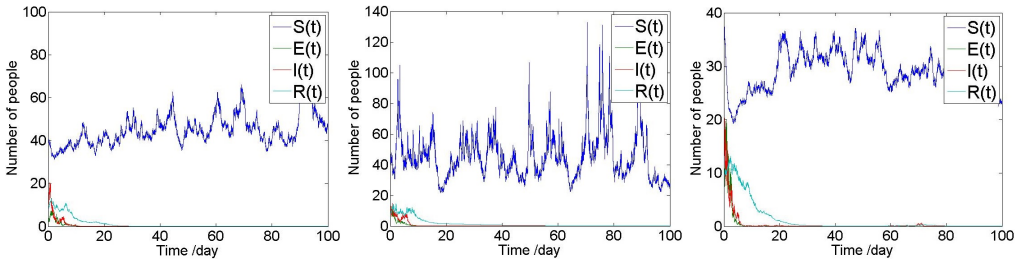
Secondly, we simulated the extinction of diseases in Section 4, which is equivalent to $\mathcal{R}_0^S < 1$. For Case 1, $\mathcal{R}_0 > 1 > \mathcal{R}_0^S$, that is to say, when the interference intensity of external random factors σ_{21}, σ_{31} are high, infectious diseases will become extinct. We have set up three different sets of data, as shown in the Table 3.

Table 3. For Case 1, the parameters of diseases extinction.

Sets	Λ	μ	β	δ	γ	ν	θ_1	θ_2	θ_3
4	8	0.15	0.1	0.8	0.2	0.2	0.9	0.4	0.3
5	6	0.05	0.04	0.65	0.28	0.27	0.9	0.5	0.4
6	6	0.2	0.1	0.8	0.2	0.2	0.8	0.5	0.6

Sets	σ_{11}	σ_{12}	σ_{21}	σ_{22}	σ_{31}	σ_{32}	σ_{41}	σ_{42}	$\mathcal{R}_0$	$\mathcal{R}_0^S$
4	0.01	0.002	0.8	0.007	0.8	0.003	0.1	0.005	1.0411	0.2210
5	0.03	0.006	0.6	0.005	0.6	0.003	0.004	0.03	1.0857	0.0882
6	0.01	0.002	0.75	0.009	0.75	0.008	0.1	0.005	1.1000	0.3264

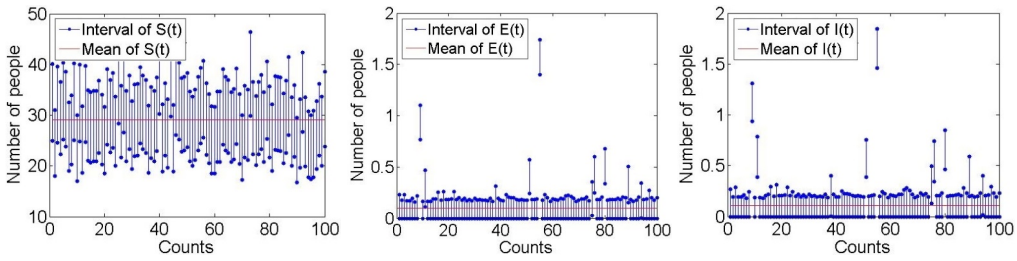
In the three sets of data, according to Theorem 3(1), we calculated that $\mathcal{R}_0 > 1 > \mathcal{R}_0^S$, as shown in the Table 3, it satisfies the conditions of Theorem 3(1), and from the conclusion of the theorem, it can be concluded that diseases are bound to become extinct, and $\lim_{t\to+\infty} \frac{1}{t}\int_0^t S(u)\mathrm{d}u = \frac{\Lambda}{\mu}$, $\lim_{t\to+\infty} E(t) = 0$, $\lim_{t\to+\infty} I(t) = 0$, $\lim_{t\to+\infty} R(t) = 0$, as shown in the Figure 3.



(**a**) $\mathcal{R}_0^S = 0.2210 < 1$ (**b**) $\mathcal{R}_0^S = 0.0882 < 1$ (**c**) $\mathcal{R}_0^S = 0.3264 < 1$

Figure 3. For Case 1, time sequence diagram of diseases extinction.

Similarly, we conducted repeated simulations of disease extinction. For Case 1, $\mathcal{R}_0^S = 0.3264 < 1$, $t \in [3500, 4000]$, randomly selected 100 samples from $S(t)$, $E(t)$, $I(t)$, and established a 95% empirical confidence interval for $S(t)$, $E(t)$, $I(t)$, as shown in the Figure 4.



(**a**) Confidence interval of $S(t)$ (**b**) Confidence interval of $E(t)$ (**c**) Confidence interval of $I(t)$

Figure 4. Case 1 of disease extinction.

From Figure 4, it can be seen that among these 100 intervals. The mean of $S(t)$ is about 29.3913, 97 of 100 contain parameter truth values; the mean of $E(t)$ is about 0.0728, 93 of 100 contain parameter truth values; the mean of $I(t)$ is about 0.0983, 92 of 100 contain parameter truth values, so the results are reliable.

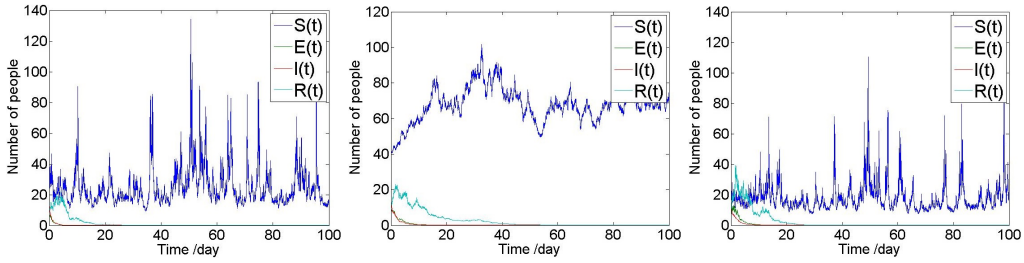
Thirdly, we simulated the Case 2 of diseases extinction, which is equivalent to $\mathcal{R}_0^S < \mathcal{R}_0 < 1$, that is to say, the hospitalization rate ν, the home isolation rate θ_1, their values are relatively high, the infection rate during the latent period of the disease θ_2 is low which are equivalent to playing a positive role, infectious diseases will become extinct. We have set up three different sets of data, as shown in the Table 4.

Table 4. For Case 2, the parameters of diseases extinction.

Sets	Λ	μ	β	δ	γ	ν	θ_1	θ_2	θ_3
7	8	0.1	0.05	0.85	0.75	0.65	0.8	0.5	0.4
8	8	0.1	0.1	0.8	0.7	0.7	0.9	0.4	0.3
9	7	0.2	0.1	0.4	0.6	0.5	0.85	0.55	0.45

Sets	σ_{11}	σ_{12}	σ_{21}	σ_{22}	σ_{31}	σ_{32}	σ_{41}	σ_{42}	$\mathcal{R}_0$	$\mathcal{R}_0^S$
7	0.05	0.02	0.2	0.02	0.1	0.01	0.2	0.01	0.8982	0.0614
8	0.01	0.001	0.1	0.007	0.1	0.003	0.1	0.005	0.8296	0.2431
9	0.05	0.03	0.05	0.04	0.05	0.02	0.2	0.01	0.7504	0.0536

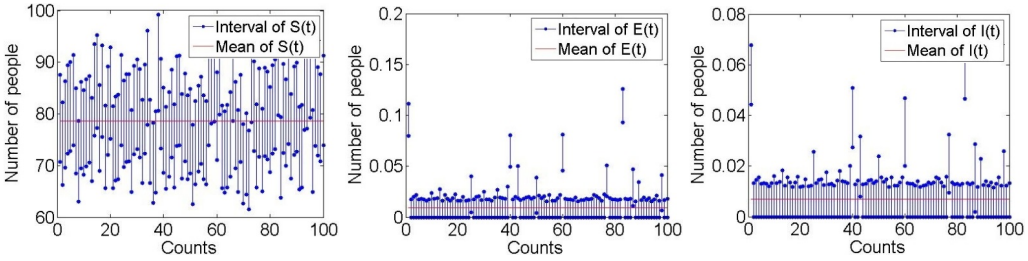
In the three sets of data, according to Theorem 3(2), we calculated that $\mathcal{R}_0^S < \mathcal{R}_0 < 1$, as shown in the Table 4, it satisfies the conditions of Theorem 3(2), and from the conclusion of the theorem, it can be concluded that diseases are bound to become extinct, and $\lim_{t\to+\infty} \frac{1}{t}\int_0^t S(u)\mathrm{d}u = \frac{\Lambda}{\mu}$, $\lim_{t\to+\infty} E(t) = 0$, $\lim_{t\to+\infty} I(t) = 0$, $\lim_{t\to+\infty} R(t) = 0$, the numerical simulation results are symmetrical with the extinction of diseases, as shown in the Figure 5.



(**a**) $\mathcal{R}_0^S = 0.0614 < 1$ (**b**) $\mathcal{R}_0^S = 0.2431 < 1$ (**c**) $\mathcal{R}_0^S = 0.0536 < 1$

Figure 5. For Case 2, time sequence diagram of diseases extinction.

Similarly, we conducted repeated simulations of disease extinction. For Case 2, $\mathcal{R}_0^S = 0.2431 < 1$, $t \in [4500, 5000]$, randomly selected 100 samples from $S(t)$, $E(t)$, $I(t)$, and established a 95% empirical confidence interval for $S(t)$, $E(t)$, $I(t)$, as shown in the Figure 6.



(**a**) Confidence interval of $S(t)$ (**b**) Confidence interval of $E(t)$ (**c**) Confidence interval of $I(t)$

Figure 6. Case 2 of disease extinction.

From Figure 6, it can be seen that among these 100 intervals. The mean of $S(t)$ is about 78.8636, 91 of 100 contain parameter truth values; the mean of $E(t)$ is about 0.0069, 93 of 100 contain parameter truth values; the mean of $I(t)$ is about 0.0039, 94 of 100 contain parameter truth values, so the results are reliable.

Last, we can adjust different parameters of the model to simulate the three stages of disease transmission in the population, t_1: disease outbreak, t_2: the disease is under certain control, t_3: disease extinction, specific parameters are shown in Table 5.

Table 5. Parameters of diseases from outbreak to extinction.

Stage	Λ	μ	β	δ	γ	ν	θ_1	θ_2	θ_3
t_1	10	0.4	0.5	0.8	0.2	0.1	0.2	0.8	0.2
t_2	10	0.2	0.2	0.6	0.5	0.5	0.4	0.4	0.3
t_3	10	0.1	0.1	0.5	0.7	0.7	0.9	0.1	0.5
Stage	σ_{11}	σ_{12}	σ_{21}	σ_{22}	σ_{31}	σ_{32}	σ_{41}	σ_{42}	$\mathcal{R}_0^S$
t_1	0.02	0.009	0.03	0.001	0.03	0.01	0.02	0.002	6.7460
t_2	0.02	0.009	0.03	0.001	0.03	0.01	0.02	0.002	1.7496
t_3	0.01	0.002	0.01	0.001	0.03	0.01	0.01	0.002	0.2250

In the first stage t_1, when the disease breaks out, people have a higher mortality rate μ, and the conversion rate β, δ, from $S(t)$ to $E(t)$ and $E(t)$ to $I(t)$ are also higher, due to the

sudden outbreak of the disease, hospitals are unable to provide timely treatment plans, so the recovery rate γ is low, in addition, the isolation measures for diseases in society are insufficient, so the home isolation rate θ_1 is low, and the infection rate during the latent period of disease θ_2 is high.

In the second stage t_2, after a period of time, the hospital provided an effective treatment plan, and society began to adopt isolation measures. The disease was controlled to a certain extent, forming a local epidemic disease. The mortality rate μ decreased, the conversion rate β from $S(t)$ to $E(t)$ decreased, and the conversion rate δ from $E(t)$ to $I(t)$ decreased, and the recovery rate of patients γ increased, in addition, the home isolation rate θ_1 increased, the infection rate during the latent period of disease θ_2 decreased.

In the third stage t_3, hospitals improved treatment effectiveness, society strengthened isolation measures, just like society continued to strengthen home isolation θ_1 and reduced latent infection rates θ_2, fewer people got sick, and more and more people recovered. After a period of time, the disease will become extinct. The numerical simulation results are shown in Figure 7.

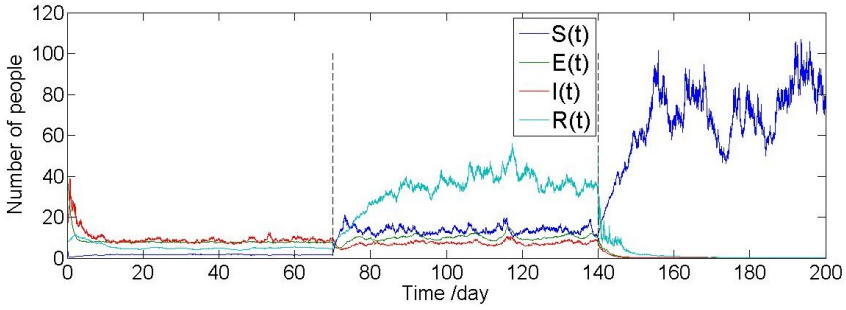


Figure 7. Time series chart of diseases from outbreak to extinction.

Through numerical simulation, we can see that in the first stage t_1, $\mathcal{R}_0^S = 6.7460 > 1$, the disease outbreak has a high mortality rate, the number of susceptible individuals $S(t)$ and the number recovered individuals $R(t)$ are low, the number of latent individuals $E(t)$ and the number of infected individuals $I(t)$ are relatively high; in the second stage t_2, $\mathcal{R}_0^S = 1.7496 > 1$, the disease is under certain control, forming a local epidemic disease, the number of susceptible individuals $S(t)$ and the number of recovered individuals $R(t)$ are relatively high, the number of latent individuals $E(t)$ and the number of infected individuals $I(t)$ are low; in the third stage t_3, $\mathcal{R}_0^S = 0.2250 > 1$, the disease becomes extinct, and the number of susceptible individuals $S(t)$ increases, the latent individuals $E(t)$, infected individuals $I(t)$ and recovered individuals $R(t)$ become 0, the numerical simulation process is symmetrical with the control process of infectious diseases.

6. Conclusions

In this article, we added higher-order perturbations to the SEIR infectious disease model with isolation and latency period, we provided sufficient conditions for the existence and uniqueness of the ergodic stationary distribution of the positive solution, as well as sufficient conditions for the extinction of infectious diseases. Through analysis and numerical simulation, by Theorem 3, $\mathcal{R}_0^S < 1$, infectious diseases will become extinct. From the expression $\mathcal{R}_0^S$, it can be concluded that increasing home isolation rate θ_1, and reducing the infection rate during the latent period of disease θ_2, they can reduce $\mathcal{R}_0^S$, infectious diseases will become extinct, this is symmetrical with the actual measures of infectious disease control. To some extent, it is beneficial for controlling the spread of diseases and accelerating their extinction. Otherwise, infectious diseases will persist in the population, affecting people's health, social stability, and causing economic losses.

In addition, media reports on epidemic prevention measures can also affect the spread of COVID-19. However, for simplicity, we have not taken into account other intervention factors, such as the economy, education, policies, and so on, we will continue to research and improve the model, and make the infectious disease model more symmetrical with practical problems. Hepatitis B and C are similar to COVID-19 in transmission. Hence, the results of this theoretical study also are instructive to the control of hepatitis B and C.

Last, we verified the theoretical results through numerical simulations, we did not consider parameter inference, but rather considered the parameters to be know at priori. Parameter inference is fundamental in epidemiological modelling, it is very important, so the parameter estimation in the model (2) can be obtained through approximate Bayesian computation, as explained by Theodore Kypraios et al. [24], or finding maximum likelihood estimation through iterative filtering method, as explained by Theresa Stocks et al. [25], or Poisson approximate likelihood, as explained by Michael Whitehouse et al. [26], we will complete the theoretical analysis and verification of parameter inference in the future.

Supplementary Materials: Supplementary material is available at https://www.mdpi.com/article/10.3390/sym17020155/s1. Appendix A: the proof of Theorem 1; Appendix B: the proof of Theorem 2, reference [27] was cited during the proof process; Appendix C: the proof of Theorem 3, references [21,28–30] were cited during the proof process.

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