

# A Reaction-Diffusion SIR Model with Saturated Incidence Rate and Vaccination

Shaoyi Geng, Xiaolin Lin, Danfeng Pang, Guoqing Li

School of Mathematics and Data Science, Shaanxi University of Science and Technology, Xi'an, China

Email: linxl@sust.edu.cn

**How to cite this paper:** Geng, S.Y., Lin, X.L., Pang, D.F. and Li, G.Q. (2025) A Reaction-Diffusion SIR Model with Saturated Incidence Rate and Vaccination. *Journal of Applied Mathematics and Physics*, 13, 400-418. <https://doi.org/10.4236/jamp.2025.132020>

**Received:** January 5, 2025

**Accepted:** February 11, 2025

**Published:** February 14, 2025

Copyright © 2025 by author(s) and Scientific Research Publishing Inc. This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

## Abstract

In this paper, we establish an SIR reaction-diffusion infectious disease model with saturated incidence rate and vaccination. Firstly, we prove the uniform boundedness of the solution of this model. Secondly, we establish the threshold dynamic behavior of the model based on the basic reproduction number  $R_0$ , specifically, we prove the globally asymptotic stability of the disease-free equilibrium and the uniform persistence of the model. Thirdly, we show the existence and stability of the endemic equilibrium of the homogeneous system and obtain different cases of positive solution. Fourthly, we investigate the effects of vaccination rate and saturated incidence rate on the basic reproduction number. The results indicate that increasing vaccination rate and saturation rate can effectively control the transmission of the disease. Finally, we conduct numerical simulations to verify the aforementioned conclusions.

## Keywords

Reaction-Diffusion Model, Saturated Incidence Rate, Basic Reproduction Number, Threshold Dynamics, Asymptotic Profile

## 1. Introduction

Since infectious diseases can spread widely among people or animals, causing serious impacts on human life and health, infectious diseases have always been a major public threat to all humans and other species. Therefore, it is crucial to study the transmission mechanism, outbreak or extinction patterns, and prevention and control measures of infectious diseases. The dynamics of infectious diseases are usually studied by establishing mathematical models, among which the SIR model is one of the most classic and basic models in infectious disease models. Kermack and McKendrick [1] discussed one of the earliest models, using the SIR (Susceptible-Infectives-Recovery) model to simulate the epidemic in Mumbai, making a

pioneer contribution to the study of infectious disease dynamics. In infectious disease models [2] [3], the heterogeneity of population spatial distribution is usually ignored. To better describe infectious disease models, many scholars consider diffusion terms to the model [4]-[7] and focus on the combined impact of spatial heterogeneity, individual random diffusion, and different infection mechanisms on disease dynamics, resulting in many interesting results.

On the other hand, some papers study infectious disease models with nonlinear incidence rates [8]-[10], hoping to obtain more accurate results on the spread of diseases and predict the trend of disease outbreak. In 1978, Capasso and Serio [11] showed that the number of effective contacts between infectives and susceptible did not always present linearity. Due to the congestion of infected individuals or protective measures taken by susceptible individuals, the contact rate may reach to saturation on high infection level. Therefore, they propose a saturation incidence rate of  $\frac{\beta SI}{1+mI}$ , where  $S$  denotes the susceptible,  $I$  represents the infectives,  $\beta$  is the infection rate,  $m$  is the saturation rate. In 1979, May and Anderson [12] proposed another saturation incidence rate  $\frac{\beta SI}{1+\alpha S}$ , that is, the incidence rate of susceptible approach to saturation, to study the dynamics of host and parasites. Afterward, many works [13]-[16] investigate the saturated incidence rate on the spread of disease.

In 2022, Dong *et al.* [17] established the following reaction-diffusion SI infectious disease model with logistic sources and saturated transmission mechanism:

$$\begin{cases} \frac{\partial S}{\partial t} - d_s \Delta S = a(x)S - b(x)S^2 - \beta(x) \frac{SI}{1+\alpha S} - \mu(x)S, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} - d_I \Delta I = \beta(x) \frac{SI}{1+\alpha S} - [\gamma(x) + \mu(x)]I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega, \end{cases}$$

where  $\Omega$  is a bounded domain with smooth boundaries  $\partial\Omega$ ,  $S(x, t)$  and  $I(x, t)$  represent the density of susceptible and infected individuals at time  $t$  at  $x$ , respectively.  $d_s$  and  $d_I$  are the diffusion coefficients of susceptible and infected individuals,  $a(x)S - b(x)S^2$  represents the logistic growth of susceptible,  $a(x)$  and  $b(x)$  represent the birth rate and density dependent mortality.  $\mu(x)$  denotes the natural mortality rate,  $\gamma(x)$  represents the recovery rate, and  $\alpha$  is the saturation coefficient for susceptible individuals. This article first proves the persistence and extinction of diseases based on the basic reproduction number  $R_0$ . Secondly, the global stability of the endemic equilibrium of disease is established in homogeneous environment. Finally, the asymptotic behavior of the endemic equilibrium in heterogeneous environment is studied when the diffusion rates of susceptible and infected populations go to 0.

Since British doctor Edward Jenner developed a vaccine against smallpox using

cowpox in 1796, the development and administration of vaccine have gone through hundreds of years. Using vaccine for immunization is the great progress in prevention and control of infectious diseases. Many infectious disease models study the effectiveness of vaccination, such as seasonal influenza, HPV and measles. Although vaccine is either lifelong or short time, the corresponding mathematical models [18]-[20] obtain some important results. In 2014, Rahman *et al.* [21] established a model to study the strategies for infectious disease vaccine. Reza *et al.* [22] study the effects of quarantine and vaccination during COVID-19 pandemic. de Carvalho and Rodrigues [23] introduce a nonlinear system of SIR Model with Vaccination:

$$\begin{cases} \frac{dS}{dt} = S(A - S) - \beta SI - pm, \\ \frac{dI}{dt} = \beta SI - (d + \mu)I - gI, \\ \frac{dR}{dt} = pm + gI - \mu R, \end{cases}$$

where  $A$  is the carrying capacity of susceptible individuals,  $\beta$  denotes the transmission rate of the disease,  $m$  represents the birth rate,  $p$  is the proportion of susceptible individuals successfully vaccinated at birth for  $p \in [0, 1]$ ,  $\mu$  is the natural death rate of infected and recovered individuals,  $d$  refers to the death rate of infected individuals due to the disease, and  $g$  is the natural recovery rate. The authors study bifurcation property of the model and evaluate the proportion of vaccinated individuals necessary to eliminate the disease and conclude how the vaccination may affect the outcome of the epidemic.

The above papers investigate the infectious disease from different aspects, such as spatial heterogeneity, diffusion, and vaccination, however, as far as we know, few papers consider the combined effects of their factor in one model. Inspired by this, we propose the following SIR model:

$$\begin{cases} \frac{\partial S}{\partial t} - d_s \Delta S = a(x)S - b(x)S^2 - \beta(x) \frac{SI}{1 + \alpha S} - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} - d_I \Delta I = \beta(x) \frac{SI}{1 + \alpha S} - \sigma(x)I - c(x)I^2, & x \in \Omega, t > 0, \\ \frac{\partial R}{\partial t} - d_R \Delta R = \sigma(x)I + \theta S - \mu(x)R, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = \frac{\partial R}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), R(x, 0) = R_0(x), & x \in \Omega, \end{cases}$$

where  $\theta \in [0, 1]$  is the vaccination rate,  $\sigma(x)$  is the recovery rate of the infectives,  $c(x)$  is the density-dependent death rate of infected individuals due to the limited medical resource, other parameters are same with the above models. We assume all the parameters and the initial value are nonnegative and nontrivial, and  $a(x) > \theta$  for all  $x \in \Omega$ . Since the third equation is decoupled from the other two equations, it is sufficient to consider the following system:

$$\begin{cases} \frac{\partial S}{\partial t} - d_s \Delta S = a(x)S - b(x)S^2 - \beta(x) \frac{SI}{1 + \alpha S} - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} - d_I \Delta I = \beta(x) \frac{SI}{1 + \alpha S} - \sigma(x)I - c(x)I^2, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega. \end{cases} \quad (1)$$

The rest of this paper is organized as follows. In Section 2, we prove the uniform boundedness of the solution. In Section 3, the basic reproduction number  $R_0$  is defined and its asymptotic properties are studied, and the threshold dynamics of the model based on  $R_0$  is obtained. In Section 4, we show the existence and stability of the endemic equilibrium of the homogeneous system. In Section 5, we study the effects of vaccination rate  $\theta$  and saturated incidence rate  $\alpha$  on the basic reproduction number  $R_0$ . In Section 6, we use numerical simulation to present the solution when  $R_0 < 1$  and  $R_0 > 1$ , moreover, we verify the effects of the saturated incidence rate and the vaccination rate on  $R_0$ . Finally, a brief conclusion is given at the end of this paper.

## 2. Well-Posedness

From the standard theory for parabolic equations, we know that system (1) has unique classical solution  $(S, I)$  for nonnegative and nontrivial initial value. Furthermore, by the strong maximum principle and the Hopf boundary lemma, we get that the solution are positive for  $x \in \bar{\Omega}$  and  $t > 0$ .

In this section, we prove the boundedness of the solution of model (1). To this end, we first give the following denotation for simplicity:

$$F^* = \max_{x \in \bar{\Omega}} F(x), \quad F_* = \min_{x \in \bar{\Omega}} F(x),$$

where  $F(x) = \{a(x), b(x), \beta(x), \sigma(x)\}$ .

In addition, the following Young inequality plays a key role in proving the results.

$$ab \leq \varepsilon a^p + C(\varepsilon) b^q,$$

where  $a, b, \varepsilon > 0$ ,  $C(\varepsilon) = \frac{1}{p} (\varepsilon p)^{\frac{q}{p}}$ ,  $1 < p, q < \infty$ ,  $\frac{1}{p} + \frac{1}{q} = 1$ .

**Theorem 1.** There exists a positive constant  $C$ , independent of the initial value, such that for a sufficiently large positive number  $T$ , any solution  $(S, I)$  of system (1) satisfies

$$\|S(\cdot, t)\|_{L^\infty(\Omega)} + \|I(\cdot, t)\|_{L^\infty(\Omega)} \leq C, \quad \forall t \geq T. \quad (2)$$

**Proof.** Let  $H(t) = \int_{\Omega} [S(x, t) + I(x, t)] dx$ , then by Young inequality, we have

$$\begin{aligned} \frac{dH}{dt} &= \int_{\Omega} \frac{\partial S}{\partial t} dx + \int_{\Omega} \frac{\partial I}{\partial t} dx \\ &= \int_{\Omega} [a(x)S - b(x)S^2 - \theta S] dx - \int_{\Omega} \sigma(x)I dx - \int_{\Omega} c(x)I^2 dx \\ &\leq \int_{\Omega} \left[ a^* S - b_* \left( \varepsilon S - \frac{\varepsilon^2}{4} \right) \right] dx - \int_{\Omega} \theta S dx - \int_{\Omega} \sigma(x)I dx - \int_{\Omega} c(x)I^2 dx \end{aligned}$$

$$\begin{aligned} &\leq \int_{\Omega} (a^* - b_* \varepsilon) S + \int_{\Omega} b^* \frac{\varepsilon^2}{4} dx - \int_{\Omega} \theta S dx - \int_{\Omega} \sigma_* I dx \\ &\leq \frac{b_* \varepsilon^2}{4} |\Omega| - mH, \end{aligned}$$

where  $m = \min\{a^* - b_* \varepsilon - \theta, \sigma_*\}$ . Therefore,  $\frac{dH}{dt} + mH \leq \frac{b_* \varepsilon^2}{4} |\Omega|$ . This means

$$H(t) \leq H(0)e^{-mt} + \frac{b_*}{4m} \varepsilon^2 |\Omega| (1 - e^{-mt}),$$

that is,

$$\begin{aligned} &\int_{\Omega} [S(x, t) + I(x, t)] dx \\ &\leq e^{-mt} \int_{\Omega} [S(x, 0) + I(x, 0)] dx + \frac{b_*}{4m} \varepsilon^2 |\Omega| (1 - e^{-mt}), \forall t \geq 0. \end{aligned}$$

According to [24] (Lemma 2.1) with  $\sigma = p_0 = 1$ , and the positiveness of  $S$  and  $I$ , we obtain that

$$\|S(\cdot, t)\|_{L^\infty(\Omega)} + \|I(\cdot, t)\|_{L^\infty(\Omega)} \leq C, \forall t \geq 0.$$

Moreover, we have  $\limsup_{t \rightarrow \infty} \int_{\Omega} [S(x, t) + I(x, t)] dx \leq \frac{b_*}{4m} \varepsilon^2 |\Omega|$ , which is independent of initial value. Then, it follows from [24] (Lemma 2.1) again that (2) holds. This completes the proof.

### 3. Threshold Dynamics

In this section, we give the definition of the basic reproduction number and study the threshold dynamic behavior of system (1). To this end, we consider the following elliptic system:

$$\begin{cases} -d_S \Delta S = a(x)S - b(x)S^2 - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = 0, & x \in \partial\Omega, t > 0. \end{cases} \quad (3)$$

By the standard method of upper and lower solution, we know that (3) admits a positive solution  $\hat{S}$ , and thus  $E_0(\hat{S}, 0)$  is a Disease-Free Equilibrium (DFE) of system (1). Linearizing the system at  $(\hat{S}, 0)$ , and we obtain the following system:

$$\begin{cases} \frac{\partial S}{\partial t} = d_S \Delta S + a(x)S - 2b(x)\hat{S} \cdot S - \frac{\beta(x)\hat{S}I}{1 + \alpha\hat{S}} - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} = d_I \Delta I + \beta(x) \frac{\hat{S}I}{1 + \alpha\hat{S}} - \sigma(x)I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega. \end{cases} \quad (4)$$

Denote the initial infection distribution is represented by  $\varphi(x)$ . Due to the combined effects of individual movement and outflow, the distribution of infected individuals at time  $t$  is denoted by  $T(t)\varphi$ . Therefore, the distribution function of new infections after time  $t$  is denoted by  $F(x)T(t)\varphi(x)$ . Therefore, the

distribution of the total number of new infections is denoted by

$$\int_0^{\infty} F(x)T(t)\varphi(x)dt.$$

Let

$$L(\varphi)(x) := \int_0^{\infty} F(x)T(t)\varphi(x)dt = F(x) \int_0^{\infty} T(t)\varphi(x)dt,$$

where  $F(x) = \frac{\beta(x)\hat{S}}{1+\alpha\hat{S}}$ ,  $T(x)\varphi = \sigma(x)\varphi - d_I\Delta\varphi$ .

Following the results in [5], we define the basic reproduction number  $R_0 = r(L)$ , where  $r(L)$  is the spectral radius of  $L$ . Moreover, the variational expression of  $R_0$  is denoted by

$$R_0 = \sup_{0 \neq \varphi \in H^1(\Omega)} \left\{ \frac{\int_{\Omega} \frac{\beta(x)\hat{S}(x)}{1+\alpha\hat{S}(x)} \varphi^2 dx}{\int_{\Omega} [d_I |\nabla \varphi|^2 + \sigma(x)\varphi^2] dx} \right\}.$$

From the above variational expression, it can be seen that the basic production number depends not only on the diffusion coefficient  $d_s$  and  $d_I$  but also on the saturation rate  $\alpha$  of susceptible.

Substituting  $S(x,t) = e^{(-\lambda t)}\xi(x)$ ,  $I(x,t) = e^{(-\lambda t)}\zeta(x)$  into (4), we obtain the following eigenvalue problem:

$$\begin{cases} d_s \Delta \xi(x) - a(x)\xi(x) - 2b(x)\hat{S}\xi(x) - \frac{\beta(x)\hat{S}\zeta(x)}{1+\alpha\hat{S}} - \theta\xi(x) + \lambda\xi(x) = 0, \\ d_I \Delta \zeta(x) + \beta(x)\frac{\hat{S}}{1+\alpha\hat{S}}\zeta(x) - \sigma(x)\zeta(x) + \lambda\zeta(x) = 0, \\ \frac{\partial \xi}{\partial \nu} = \frac{\partial \zeta}{\partial \nu} = 0. \end{cases}$$

Let  $\lambda^*$  and  $\psi^*$  be the eigenvalue and the corresponding eigenfunction of the following equation:

$$\begin{cases} d_I \Delta \psi(x) + \beta(x)\frac{\hat{S}}{1+\alpha\hat{S}}\psi - \sigma(x)\psi + \lambda\psi = 0, & x \in \Omega, \\ \frac{\partial \psi}{\partial \nu} = 0, & x \in \Omega. \end{cases} \quad (5)$$

Then, we have the following properties:

**Lemma 2.** For  $R_0$ , the following conclusion holds:

- 1) For any  $d_s > 0$ ,  $R_0$  is a decreasing function of  $d_I$ ;
- 2)  $\lambda^*$  has the same sign with  $1 - R_0$ ;
- 3)  $R_0 \rightarrow \max_{x \in \Omega} \left\{ \frac{\beta(x)\hat{S}(x)}{(1+\alpha\hat{S})\sigma(x)} \right\}$  as  $d_I \rightarrow 0$ ;
- 4)  $R_0 \rightarrow \int_{\Omega} \frac{\beta(x)\hat{S}(x)}{1+\alpha\hat{S}(x)} dx \Big/ \int_{\Omega} \sigma(x) dx$  as  $d_I \rightarrow \infty$ ;
- 5) If  $\int_{\Omega} \frac{\beta(x)\hat{S}(x)}{1+\alpha\hat{S}(x)} dx \geq \int_{\Omega} \sigma(x) dx$ , then  $R_0 > 1$  for all  $d_I \geq 0$ ;

6) If  $\int_{\Omega} \frac{\beta(x)\hat{S}(x)}{1+\alpha\hat{S}(x)} dx < \int_{\Omega} \sigma(x) dx$  and  $\frac{\beta(x)\hat{S}}{1+\alpha\hat{S}} - \sigma(x)$  changes the sign for  $x \in \Omega$ , then there exists a threshold  $d_I^* \in (0, \infty)$  such that  $R_0 > 1$  when  $0 \leq d_I < d_I^*$ ,  $R_0 < 1$  when  $d_I > d_I^*$ .

The proof of this lemma is similar with [25] (Lemma 2.3), thus, we omit it here. We also present the local stability of the disease-free equilibrium here without proof.

**Lemma 3.** If  $R_0 < 1$ , then the disease-free equilibrium  $E_0$  is locally asymptotically stable; If  $R_0 > 1$ , then  $E_0$  is unstable.

Now, we establish the threshold dynamics of system (1).

**Theorem 4.** Let  $(S, I)$  be the solution of system (1), then we have:

- 1) If  $R_0 < 1$ , then the disease-free equilibrium  $E_0$  is globally asymptotically stable;
- 2) If  $R_0 > 1$ , then exists a constant  $\eta > 0$  independent of the initial value, such that any solution  $(S, I)$  of (1) satisfies

$$\liminf_{t \rightarrow \infty} S(x, t) \geq \eta, \quad \liminf_{t \rightarrow \infty} I(x, t) \geq \eta, \quad \forall x \in \bar{\Omega}.$$

In addition, system (1) has at least one endemic equilibrium.

**Proof.** According to the first Equation of (1), we have

$$\begin{cases} \frac{\partial S}{\partial t} - d_S \Delta S \leq a(x)S - b(x)S^2 - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x) \geq 0, & x \in \Omega. \end{cases}$$

By the comparison principle, we can get

$$S(x, t) \leq \tilde{S}(x, t), \quad \forall x \in \bar{\Omega}, t \geq 0,$$

where  $\tilde{S}$  is the solution of the following equation:

$$\begin{cases} \frac{\partial S}{\partial t} - d_S \Delta S = a(x)S - b(x)S^2 - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), & x \in \Omega. \end{cases} \quad (6)$$

It then follows from [17] (Lemma 1) that

$$\limsup_{t \rightarrow \infty} S(x, t) \leq \limsup_{t \rightarrow \infty} \tilde{S}(x, t) = \hat{S} \quad \text{uniformly for all } x \in \bar{\Omega}.$$

For any small constant  $\delta \geq 0$ , there exist a  $T_1 > 0$  such that

$$S(x, t) \leq \hat{S} + \delta, \quad \forall x \in \bar{\Omega}, t \geq T_1. \quad (7)$$

If  $R_0 < 1$ . By applying (b) of Lemma (2), we know that  $\lambda^* > 0$ . Let  $\lambda^*(\delta)$  be the principal eigenvalue of (5) by replacing  $\hat{S}(x)$  with  $\hat{S}(x) + \delta$  and  $\psi^*(x) > 0$  be the corresponding eigenfunction. Choose  $\delta_1 > 0$  such that  $\lambda^*(\delta_1) > 0$ . Therefore, according to the second equation of (1) and (7), we can get that  $I$  satisfies

$$\begin{cases} \frac{\partial I}{\partial t} - d_I \Delta I \leq \beta(x) \frac{\hat{S}(x) + \delta_1}{1 + \alpha(\hat{S}(x) + \delta_1)} I - \sigma(x)I, & x \in \Omega, t > T_1, \\ \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > T_1. \end{cases}$$

It is easy to verify that the following system

$$\begin{cases} \frac{\partial \hat{I}}{\partial t} - d_I \Delta \hat{I} = \beta(x) \frac{\hat{S}(x) + \delta}{1 + \alpha(\hat{S}(x) + \delta)} \hat{I} - \sigma(x) \hat{I}, & x \in \Omega, t > T_1, \\ \frac{\partial \hat{I}}{\partial \nu} = 0, & x \in \partial\Omega, t > T_1, \\ \hat{I}(x, 0) = P_* \psi^*(x), & x \in \Omega, \end{cases}$$

has a positive solution  $P_* e^{-\lambda^*(\delta)t} \psi^*(x)$ , where  $P_*$  is a positive constant and satisfies that  $P_* \psi^* \geq I(x, T_1)$  for  $x \in \Omega$ . Then, the comparison principle indicates that  $I(x, t + T_1) \leq P_* e^{-\lambda^*(\delta)t} \psi^*(x)$  for  $x \in \Omega$ ,  $t > 0$ . Thus, we have

$$I(x, t) \rightarrow 0 \quad \text{uniformly on } \bar{\Omega}, \text{ as } t \rightarrow \infty.$$

Therefore,  $S$  satisfies system (6) for all  $t > T_2 > T_1$ . Therefore, we have

$$\lim_{t \rightarrow \infty} S(x, t) = \hat{S} \quad \text{uniformly for all } x \in \bar{\Omega}.$$

By Lemma (3), we know that the disease-free equilibrium  $E_0$  is locally asymptotically stable. As a result, the disease-free equilibrium  $E_0$  is globally asymptotically stable.

If  $R_0 > 1$ . Let  $\mathbf{W} = \mathbf{C}(\bar{\Omega}, R_+^2)$ , and

$$\tilde{\mathbf{W}} = \{\phi = (\phi_1, \phi_2) \in \mathbf{W} : \phi_2(x) \neq 0\},$$

$$\partial\tilde{\mathbf{W}} = \mathbf{W} \setminus \tilde{\mathbf{W}} = \{\phi = (\phi_1, \phi_2) \in \mathbf{W} : \phi_2 \equiv 0\}.$$

For  $\forall \phi \in \mathbf{W}$ , let  $[\Phi(t)\phi](x) = (S(x, t, \phi), I(x, t, \phi))$  is the unique solution of (4) when  $(S_0, I_0) = \phi$ .

If  $\phi \in \partial\tilde{\mathbf{W}}$ , then  $I(x, t, \phi) \equiv 0$  for all  $x \in \Omega$  and  $t \geq 0$ , and thus  $S(x, t, \phi)$  satisfies (6). Then, we know that

$$S(x, t, \phi) \rightarrow \hat{S}(x, t, \phi) \quad \text{uniformly on } \bar{\Omega} \text{ as } t \rightarrow \infty.$$

Next, we show that there exists a positive constant  $\bar{\eta}$  such that

$$\limsup_{t \rightarrow \infty} \|\Phi(t)\phi - E_0\| \geq \bar{\eta}, \quad \forall \phi \in \tilde{\mathbf{W}}. \quad (8)$$

where  $E_0 = (\hat{S}(x), 0)$ . Assume by contradiction that  $\limsup_{t \rightarrow \infty} \|\Phi(t)\phi - E_0\| < \bar{\eta}$ , then there exists a large positive constant  $t_1$ , such that for all  $t > t_1$ , we have

$$\hat{S} - \hat{\eta} < S(x, t) < \hat{S} + \bar{\eta}, \quad 0 \leq I \leq \bar{\eta} \quad \forall t \geq t_1, x \in \bar{\Omega}.$$

If  $R_0 > 1$ , by Lemma (2) (b), we have  $\lambda^* < 0$ . Then, there exists an sufficiently small positive constant  $\bar{\eta}$  such that  $\lambda^*(\bar{\eta}) < 0$ , and  $\lambda^*(\bar{\eta})$  with the corresponding eigenfunction  $\psi_*(x)$  satisfy

$$\begin{cases} -d_I \Delta \psi = \beta \frac{(\hat{S} - \bar{\eta})\psi}{1 + \alpha(\hat{S} - \bar{\eta})} - (\sigma + c\bar{\eta})\psi + \lambda^* \psi, & x \in \Omega, \\ \frac{\partial \psi}{\partial \nu} = 0, & x \in \partial\Omega. \end{cases}$$

Then, we know that the following system

$$\begin{cases} \frac{\partial w}{\partial t} - d_I \Delta w = \beta \frac{(\hat{S} - \bar{\eta})w}{1 + \alpha(\hat{S} - \bar{\eta})} - \sigma w - c\bar{\eta}w, & x \in \Omega, t > t_1, \\ \frac{\partial w}{\partial \nu} = 0, & x \in \partial\Omega, t > t_1, \\ w(x, 0) = \delta\psi_*(x), & x \in \Omega \end{cases}$$

has a solution  $w(x, t) = \delta e^{-\lambda^*(\bar{\eta})t} \psi_*(x)$ , where  $\delta$  is a positive constant. From the second equation of system (1), we see that

$$\begin{cases} \frac{\partial I}{\partial t} - d_I \Delta I \geq \beta \frac{(\hat{S} - \bar{\eta})I}{1 + \alpha(\hat{S} - \bar{\eta})} - \sigma I - c\bar{\eta}I, & x \in \Omega, t > t_1, \\ \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > t_1, \\ I(x, 0) = I_0(x), & x \in \Omega. \end{cases}$$

Therefore, we choose a sufficiently small  $\delta$  such that  $I_0(x) \geq \delta\psi_*(x)$ , and by the comparison principle, we have

$$I(x, t) \geq w(x, t) = \delta e^{-\lambda^*(\bar{\eta})t} \psi_*(x), \quad x \in \Omega, t > t_1.$$

Since  $\lambda^*(\bar{\eta}) < 0$ , we see  $I(x, t) \rightarrow \infty$  as  $t \rightarrow \infty$  uniformly on  $\bar{\Omega}$ . This contradicts Theorem (1), and thus (8) hold. Hence, the conclusions of [26] can prove the results of (2).

#### 4. Existence of the Endemic Equilibrium

Next, we show the existence and stability of the endemic equilibrium in homogeneous case. We consider the following model:

$$\begin{cases} \frac{\partial S}{\partial t} - d_S \Delta S = aS - bS^2 - \beta \frac{SI}{1 + \alpha S} - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} - d_I \Delta I = \beta \frac{SI}{1 + \alpha S} - \sigma I - cI^2, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega. \end{cases} \quad (9)$$

Obviously, we see that  $E_0 = \left( \frac{a - \theta}{b}, 0 \right) \triangleq (S_0, 0)$  is the disease-free equilibrium of system (9). Linearizing system (9) at  $E_0$  yields

$$\begin{cases} \frac{\partial S}{\partial t} = d_S \Delta S + aS - 2bS_0 S - \frac{\beta S_0 I}{1 + \alpha S_0} - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} = d_I \Delta I + \frac{\beta S_0 I}{1 + \alpha S_0} - \sigma I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega. \end{cases}$$

Then, by [5] (Theorem 3.4), we get the basic reproduction number

$$\mathcal{R}_0 = r(FV^{-1}) = \frac{\beta S_0}{\sigma(1 + \alpha S_0)},$$

where  $F = \frac{\beta S_0}{1 + \alpha S_0}$ ,  $V = \sigma$ .

**Theorem 5.** If  $\mathcal{R}_0 \leq 1$ , then  $E_0$  is globally asymptotically stable. If  $\mathcal{R}_0 \geq 1$ , then (9) is uniform persistent.

Next, we explore the existence and stability of positive solutions of system (9). From the second equation of (9), we have

$$S = \frac{\sigma + cI}{\beta - \alpha(\sigma + cI)},$$

then  $I$  satisfies

$$a_0 I^3 + a_1 I^2 + a_2 I + a_3 = 0,$$

where

$$\begin{aligned} a_0 &= -\alpha^2 c^2, \\ a_1 &= 2\alpha c(\beta - \alpha\sigma), \\ a_2 &= 2\sigma\beta\alpha - \alpha^2\sigma^2 - a\alpha c + \alpha\theta c - bc - \beta^2, \\ a_3 &= (a - \theta)(\beta - \alpha\sigma) - b\sigma. \end{aligned}$$

Since  $\mathcal{R}_0 > 1$ , we easily verify that  $a_3 > 0$ . Let

$$f(I) = a_0 I^3 + a_1 I^2 + a_2 I + a_3,$$

then the derivative of  $f(I)$  with respect  $I$  is as follows:

$$f'(I) = 3a_0 I^2 + 2a_1 I + a_2.$$

We can easily know that the roots of  $f'(I) = 0$  are

$$I_* = \frac{-2a_1 + \sqrt{\Delta}}{6a_0}, I_{**} = \frac{-2a_1 - \sqrt{\Delta}}{6a_0},$$

where  $\Delta = 4a_1^2 - 12a_0 a_2$ .

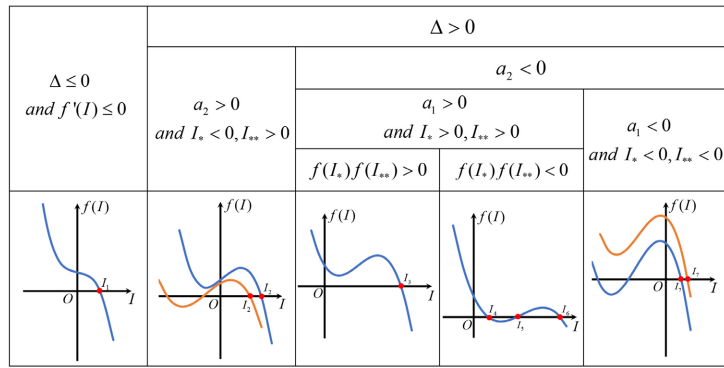
Now, we obtain the following results about the positive solution of the system (9).

**Theorem 6.** The positive solutions of the system (9) are as follows:

- 1) If  $\Delta \leq 0$ , then system (9) has a unique positive solution  $(S_1, I_1)$ ;
- 2) If  $\Delta > 0, a_2 > 0$ , that is  $I_* < 0, I_{**} > 0$ , then system (9) has a unique positive solution  $(S_2, I_2)$ ;
- 3) If  $\Delta > 0, a_2 < 0$ , then there are two cases hold.
  - a) If  $a_1 > 0$  and  $f(I_*)f(I_{**}) > 0$ , then system (9) has a unique positive solution  $(S_3, I_3)$ ; If  $a_1 > 0$  and  $f(I_*)f(I_{**}) < 0$ , then system (9) has three positive solutions denoted as  $(S_4, I_4)$ ,  $(S_5, I_5)$ , and  $(S_6, I_6)$ ;
  - b) If  $a_1 < 0$ , then system (9) has a unique positive solution  $(S_7, I_7)$ .

**Figure 1** shows the intuitive result.

Now, we investigate the stability of the positive solution, without loss of generality, we take  $(S_1, I_1)$  as an example. Linearizing system (9) at the positive solution  $(S_1, I_1)$  for example,



**Figure 1.** The existence of the positive solution  $I$ .

$$\begin{cases} \frac{\partial S}{\partial t} = d_s \Delta S + aS - 2bS_1 S - \frac{\beta I_1}{(1 + \alpha S_1)^2} S - \frac{\beta S_1}{1 + \alpha S_1} I - \theta S, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} = d_I \Delta I + \frac{\beta I_1}{(1 + \alpha S_1)^2} S + \frac{\beta S_1}{1 + \alpha S_1} I - \sigma I - 2cI_1 I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega. \end{cases}$$

The Jacobian matrix of the model (9) at  $(S_1, I_1)$  is

$$J = \begin{pmatrix} a - \theta - 2bS_1 - \frac{\beta I_1}{(1 + \alpha S_1)^2} + k^2 d_s & -\frac{\beta S_1}{1 + \alpha S_1} \\ \frac{\beta I_1}{(1 + \alpha S_1)^2} & \frac{\beta S_1}{1 + \alpha S_1} - \sigma - 2cI_1 + k^2 d_I \end{pmatrix}.$$

Then, the characteristic equation of  $J$  is

$$\begin{aligned} & |J - \lambda E| \\ &= \begin{vmatrix} a - \theta - 2bS_1 - \frac{\beta I_1}{(1 + \alpha S_1)^2} + k^2 d_s - \lambda & -\frac{\beta S_1}{1 + \alpha S_1} \\ \frac{\beta I_1}{(1 + \alpha S_1)^2} & \frac{\beta S_1}{1 + \alpha S_1} - \sigma - 2cI_1 + k^2 d_I - \lambda \end{vmatrix} \\ &= 0. \end{aligned}$$

Let

$$f(\lambda) = |J - \lambda E| = \lambda^2 + A_1 \lambda + A_2 = 0, \tag{10}$$

where

$$\begin{aligned} A_1 &= -(d_s + d_I)k^2 + \theta - a + 2bS_1 + \frac{\beta I_1}{(1 + \alpha S_1)^2} - \frac{\beta S_1}{1 + \alpha S_1} + \sigma + 2cI_1, \\ A_2 &= \left( ad_I - \theta d_I - 2bS_1 d_I + \frac{\beta S_1 d_s}{1 + \alpha S_1} - \frac{\beta I_1 d_I}{(1 + \alpha S_1)^2} - \sigma d_s - 2cd_s I_1 \right) k^2 \\ &\quad + 4bcS_1 I_1 + \frac{a\beta S_1}{1 + \alpha S_1} - (a - \theta)(\sigma + 2cI_1) - \frac{\theta\beta S_1}{1 + \alpha S_1} - \frac{2b\beta S_1^2}{1 + \alpha S_1} \\ &\quad + 2b\sigma S_1 + \frac{\sigma\beta I_1}{(1 + \alpha S_1)^2} + \frac{2c\beta I_1^2}{(1 + \alpha S_1)^2} + d_s d_I k^4. \end{aligned}$$

According to the Routh-Hurwitz criterion, the necessary and sufficient condition for the roots of Equation (10) have negative real parts is

$$A_i > 0 \text{ for all } k^2, i = 1, 2.$$

However, for some large  $k^2$ , we have  $A_1 < 0$ . Therefore, the positive solution  $(S_1, I_1)$  is unstable.

## 5. Influence of Vaccination Rate and the Saturation Rate on $R_0$

In this section, we study the effect of the vaccination rate  $\theta$  and the saturation rate  $\alpha$  on the basic reproduction number.

Since  $\lambda^*$  and  $\psi^*$  be the eigenvalue and the corresponding eigenvalue function of the following equation:

$$\begin{cases} d_I \Delta \psi^*(x) + \beta(x) \frac{\hat{S}}{1 + \alpha \hat{S}} \psi^* - \sigma(x) \psi^* + \lambda \psi^* = 0, & x \in \Omega, \\ \frac{\partial \psi^*}{\partial \nu} = 0, & x \in \Omega, \end{cases}$$

then by [5] (Theorem 3.2)), we have

$$\begin{cases} d_I \Delta \psi^*(x) + \frac{1}{R_0} \beta(x) \frac{\hat{S}}{1 + \alpha \hat{S}} \psi^* - \sigma(x) \psi^* = 0, & x \in \Omega, \\ \frac{\partial \psi^*}{\partial \nu} = 0, & x \in \Omega. \end{cases} \quad (11)$$

**Theorem 7.** Fix  $d_s, d_I, \alpha > 0$ , then  $R_0$  is strictly decreasing function of  $\theta$ .

**Proof.** Taking the derivative of both sides of Equation (11) with respect to  $\theta$ , and denote  $\psi_\theta^*$ ,  $\hat{S}_\theta$  the derivative of  $\psi^*$  and  $\hat{S}$  with respect to  $\theta$ , respectively. Then, we get

$$\begin{cases} d_I (\Delta \psi^*)_\theta - \sigma(x) \psi_\theta^* = \frac{R_0}{R_0^2} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^* - \frac{1}{R_0} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi_\theta^* - \frac{1}{R_0} \frac{\beta(x) \hat{S}_\theta}{(1 + \alpha \hat{S})^2} \psi^*, \\ \frac{\partial \psi_\theta^*}{\partial \nu} = 0, \end{cases} \quad (12)$$

Multiplying both sides of Equation (12) by  $\psi^*$ , and integrating it over  $\Omega$ , then we have

$$\begin{aligned} & \int_{\Omega} d_I (\Delta \psi^*)_\theta \psi^* dx - \int_{\Omega} \sigma(x) \psi^* \psi_\theta^* dx \\ &= \frac{R_0'}{R_0^2} \int_{\Omega} \frac{\beta(x) \hat{S} \psi^{*2}}{1 + \alpha \hat{S}} dx - \frac{1}{R_0} \int_{\Omega} \left[ \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^* \psi_\theta^* - \frac{\beta(x) \hat{S}_\theta \psi^{*2}}{(1 + \alpha \hat{S})^2} \right] dx. \end{aligned} \quad (13)$$

Multiplying both sides of Equation (11) by  $\psi_\theta^*$ , and integrate it over  $\Omega$ , then

$$\int_{\Omega} d_I \Delta \psi^* \psi_\theta^* dx + \frac{1}{R_0} \int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^* \psi_\theta^* dx - \int_{\Omega} \sigma(x) \psi^* \psi_\theta^* dx = 0. \quad (14)$$

Subtracting (14) from (13), then we obtain

$$\frac{R'_0}{R_0^2} \int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^{*2} dx = \frac{1}{R_0} \int_{\Omega} \frac{\beta(x) \hat{S}_{\theta}}{(1 + \alpha \hat{S})^2} \psi^{*2} dx,$$

and thus

$$R'_0 = R_0 \frac{\int_{\Omega} \frac{\beta(x) \hat{S}_{\theta}}{(1 + \alpha \hat{S})^2} \psi^{*2} dx}{\int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^{*2} dx}.$$

Since  $\hat{S}$  satisfies the system (3), we know that it is strictly monotonically decreasing with respect to  $\theta$ . Hence, we have  $\hat{S}_{\theta} < 0$ . Therefore,  $R'_0 < 0$ , that is,  $R_0$  is strictly monotonically decreasing function of  $\theta$ . The result shows that increasing the vaccination rate of susceptible is an effective measure for controlling the disease.

**Theorem 8.** Fix  $d_s, d_I, \theta > 0$ , then  $R_0$  is strictly decreasing function of  $\alpha$ .

**Proof.** Taking the derivative of both sides of Equation (11) with respect to  $\alpha$ , and denote  $\psi'_{\alpha}$ ,  $\hat{S}'_{\alpha}$  the derivative of  $\psi^*$  and  $\hat{S}$  with respect to  $\alpha$ , respectively. Then, we have

$$\begin{cases} d_I (\Delta \psi'_{\alpha}) - \sigma(x) \psi'_{\alpha} = \frac{R'_0}{R_0^2} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^* + \frac{1}{R_0} \frac{\beta(x) \hat{S}^2}{(1 + \alpha \hat{S})^2} \psi^* - \frac{1}{R_0} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi'_{\alpha}, \\ \frac{\partial \psi^*}{\partial \alpha} = 0. \end{cases} \quad (15)$$

Multiplying both sides of Equation (15) by  $\psi^*$ , and integrate it over  $\Omega$ , then we get

$$\begin{aligned} & \int_{\Omega} d_I (\Delta \psi^*)_{\alpha} \psi^* dx - \int_{\Omega} \sigma(x) \psi'_{\alpha} \psi^* dx \\ &= \frac{R'_0}{R_0^2} \int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^{*2} dx + \frac{1}{R_0} \int_{\Omega} \frac{\beta(x) \hat{S}^2}{(1 + \alpha \hat{S})^2} \psi^{*2} dx - \frac{1}{R_0} \int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi'_{\alpha} \psi^* dx. \end{aligned} \quad (16)$$

Multiplying both sides of Equation (11) by  $\psi'_{\alpha}$ , and integrate it over  $\Omega$ , then

$$\int_{\Omega} d_I \Delta \psi^* \psi'_{\alpha} dx + \frac{1}{R_0} \int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^* \psi'_{\alpha} dx - \int_{\Omega} \sigma(x) \psi^* \psi'_{\alpha} dx = 0. \quad (17)$$

Subtracting (17) from (16), we obtain

$$\frac{R'_0}{R_0^2} \int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^{*2} dx = -\frac{1}{R_0} \int_{\Omega} \frac{\beta(x) \hat{S}^2}{(1 + \alpha \hat{S})^2} \psi^{*2} dx.$$

Hence, we get

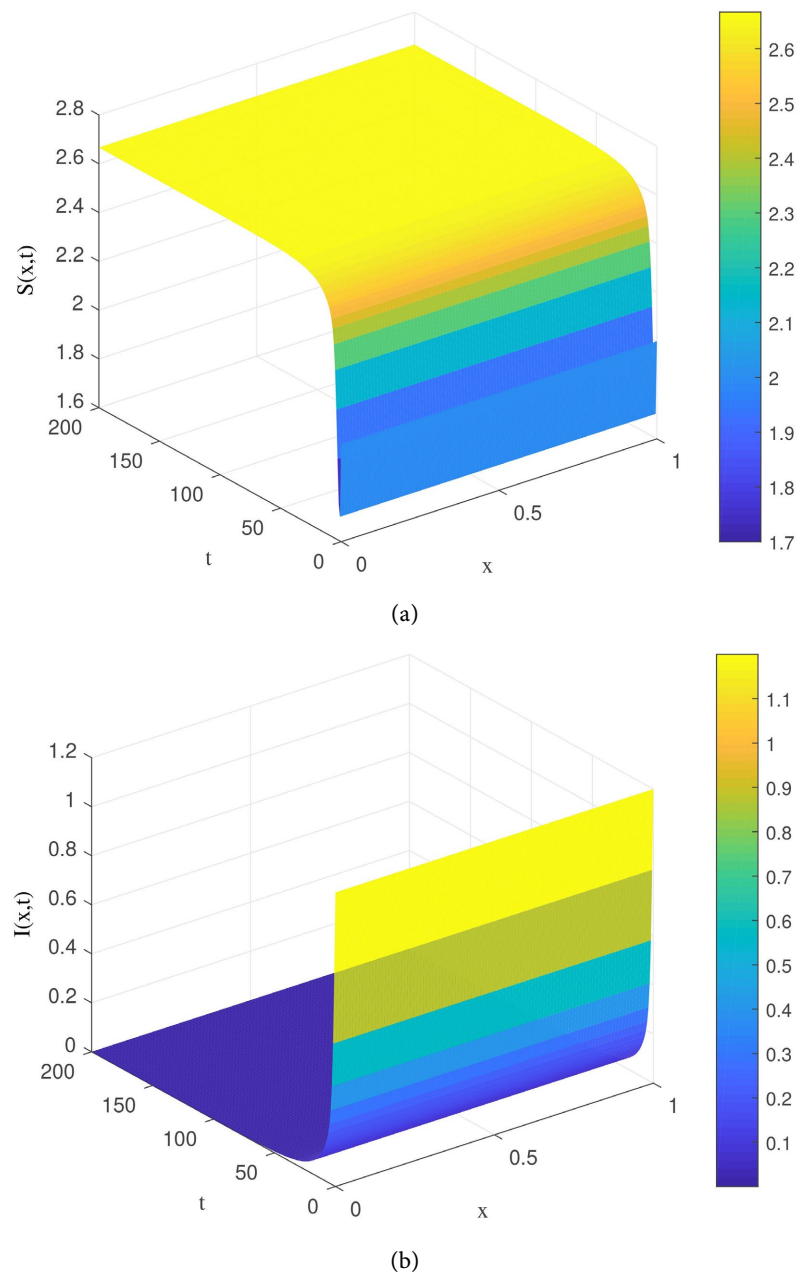
$$R'_0 = -R_0 \frac{\int_{\Omega} \frac{\beta(x) \hat{S}^2}{(1 + \alpha \hat{S})^2} \psi^{*2} dx}{\int_{\Omega} \frac{\beta(x) \hat{S}}{1 + \alpha \hat{S}} \psi^{*2} dx}.$$

Therefore,  $R_0' < 0$ , which implies that  $R_0$  is strictly decreasing with respect to  $\alpha$ .

## 6. Numerical Simulation

In this section, we will show some numerical simulation results to support the above research.

First, we take the parameter as  $a = 0.9$ ,  $b = 0.2$ ,  $d_s = 0.1$ ,  $d_I = 0.1$ ,  $\theta = 0.1$ ,  $\sigma = 0.8$ ,  $c = 0.05$ ,  $\alpha = 0.1$ ,  $\beta = 0.35$ , then we obtain the disease-free equilibrium of the system (1) when  $R_0 = 0.92106 < 1$  (see **Figure 2**).

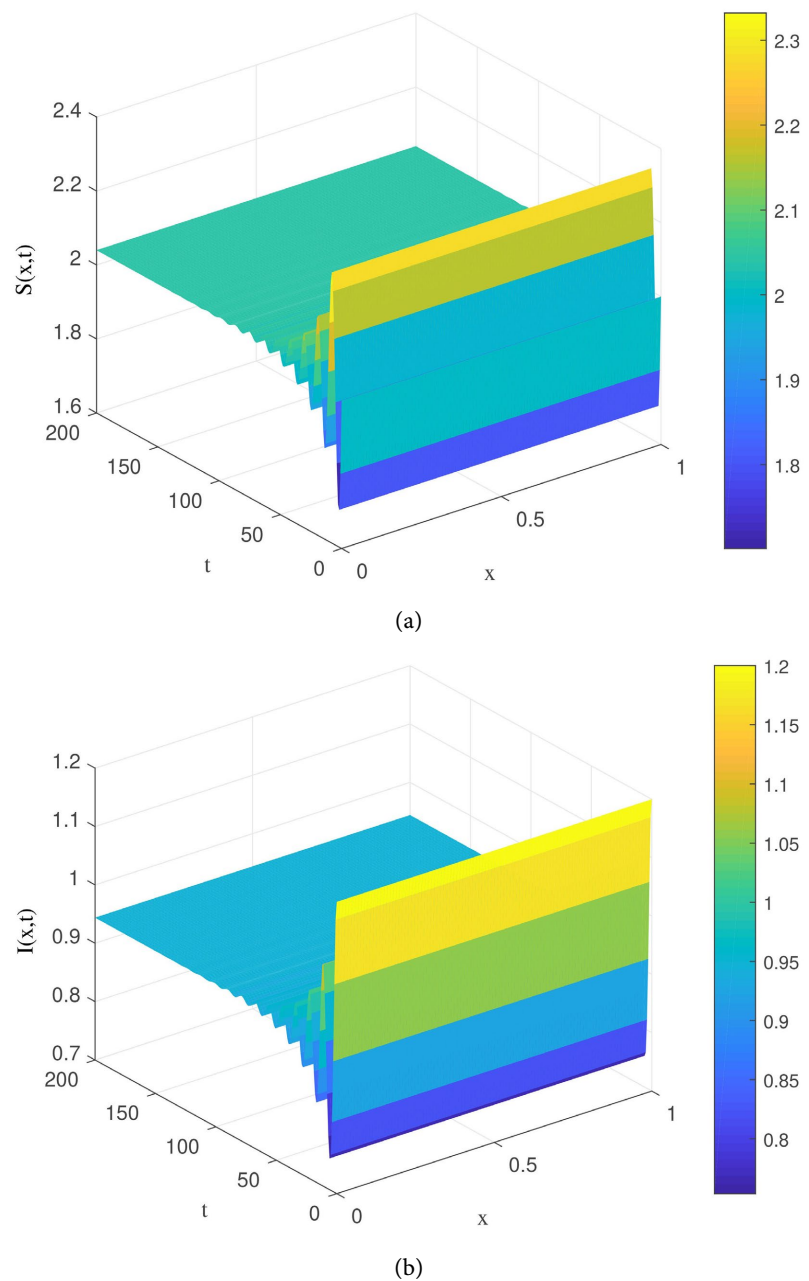


**Figure 2.** Solution  $S$  and  $I$  when  $R_0 < 1$ .

From **Figure 2**, we can find that  $S(x,t)$  tends to be a constant and  $I(x,t)$  approaches 0 as time increases. Thus, the disease-free equilibrium is globally asymptotically stable. From the biological point of view, when the basic reproduction number  $R_0 < 1$ , the disease eventually dies out (see **Figure 2**).

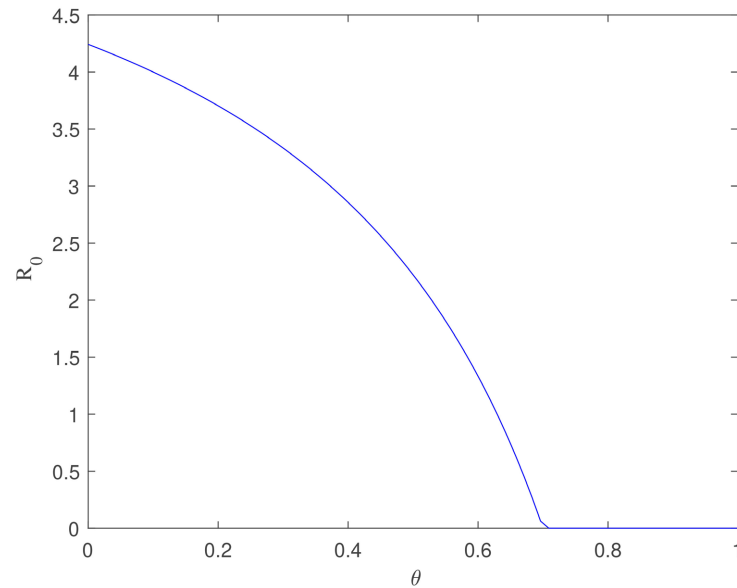
Next, let  $\beta = 0.5$  and other parameters remain unchanged. Then,  $R_0 = 1.3158 > 1$  and system (1) is uniform persistent.

We can see from **Figure 3** that the susceptible individual  $S(x,t)$  and infected  $I(x,t)$  eventually tend to positive value. In other words, the disease will always exist when the basic reproduction number  $R_0 > 1$ .

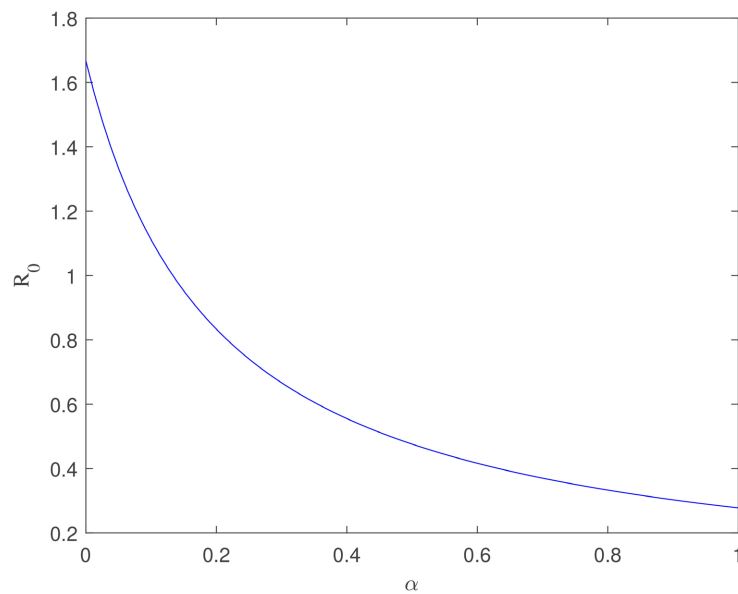


**Figure 3.** Solution  $S$  and  $I$  when  $R_0 > 1$ .

Finally, we study the effects of the vaccination rate  $\theta$  and saturated incidence rate  $\alpha$  on  $R_0$ . **Figure 4** shows that  $R_0$  drop below 1 with  $\theta$  and  $\alpha$  increase, which implies that we can control the disease by increasing the vaccination rate and saturated incidence rate.



(a)



(b)

**Figure 4.** Variation of  $R_0$  with respect to parameters.

## 7. Conclusions

This article investigates an SIR reaction-diffusion infectious disease model with saturation rate and vaccination in heterogeneous environments. Firstly, we prove the well-posedness of the system. Secondly, we define the basic reproduction number

$R_0$  and present its properties when the diffusion rate goes to 0 and infinity. Moreover, we establish the threshold dynamics of the system in terms of  $R_0$ , specifically, the disease-free equilibrium is globally asymptotically stable when  $R_0 < 1$  and the system is uniformly persistent when  $R_0 > 1$ . Thirdly, we study the existence and stability of the homogeneous system and obtain different cases of the positive solution. Fourthly, we discuss the impact of vaccination rate and saturation rate on  $R_0$ . The results of Theorems 7 and 8 indicate that increasing vaccination rate and saturation rate of susceptible individuals can promote disease disappear. Finally, we use numerical simulation to show the disease-free steady state and uniform persistence of the system. We also present the variation of  $R_0$  with respect to the model parameters and obtain the interesting biological conclusion.

The SIR model with vaccination and saturation effects studied in this article can better describe the transmission mechanism of infectious diseases. From the results of this paper, we can give more effective control measures to eradicate infectious diseases, such as increasing vaccination coverage rate or increasing the saturation rate of susceptible. The difficult work of the globally asymptotic stability of endemic equilibrium of homogeneous system will be discussed in further work.

### Acknowledgements

This research was supported by the National Natural Science Foundation of China (NSFC 12301634).

### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

### References

- [1] Kermack, W.O. and McKendrick, A.G. (1927) A Contribution to the Mathematical Theory of Epidemics. *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character*, **115**, 700-721. <https://doi.org/10.1098/rspa.1927.0118>
- [2] Cai, Y., Kang, Y., Banerjee, M. and Wang, W. (2015) A Stochastic SIRS Epidemic Model with Infectious Force under Intervention Strategies. *Journal of Differential Equations*, **259**, 7463-7502. <https://doi.org/10.1016/j.jde.2015.08.024>
- [3] Muroya, Y., Enatsu, Y. and Kuniya, T. (2013) Global Stability for a Multi-Group SIRS Epidemic Model with Varying Population Sizes. *Nonlinear Analysis. Real World Applications*, **14**, 1693-1704. <https://doi.org/10.1016/j.nonrwa.2012.11.005>
- [4] Kambe, T. (2010) Dynamics of an SIS Reaction-Diffusion Epidemic Model for Disease transmission. *Mathematical Biosciences & Engineering*, **7**, 51-66. <https://doi.org/10.3934/mbe.2010.7.51>
- [5] Wang, W. and Zhao, X. (2012) Basic Reproduction Numbers for Reaction-Diffusion Epidemic Models. *SIAM Journal on Applied Dynamical Systems*, **11**, 1652-1673. <https://doi.org/10.1137/120872942>
- [6] Kuniya, T. and Wang, J. (2018) Global Dynamics of an SIR Epidemic Model with Nonlocal Diffusion. *Nonlinear Analysis. Real World Applications*, **43**, 262-282.

- <https://doi.org/10.1016/j.nonrwa.2018.03.001>
- [7] Li, B. and Bie, Q. (2019) Long-time Dynamics of an SIRS Reaction-Diffusion Epidemic Model. *Journal of Mathematical Analysis and Applications*, **475**, 1910-1926. <https://doi.org/10.1016/j.jmaa.2019.03.062>
- [8] Peng, R. and Yi, F. (2013) Asymptotic Profile of the Positive Steady State for an SIS Epidemic Reaction-diffusion Model: Effects of Epidemic Risk and Population Movement. *Physica D: Nonlinear Phenomena*, **259**, 8-25. <https://doi.org/10.1016/j.physd.2013.05.006>
- [9] Chen, Y. and Zhao, W. (2020) Asymptotic Behavior and Threshold of a Stochastic SIQS Epidemic Model with Vertical Transmission and Beddington-DeAngelis Incidence. *Advances in Difference Equations*, **2020**, Article No. 353. <https://doi.org/10.1186/s13662-020-02815-6>
- [10] Pan, Q., Huang, J. and Wang, H. (2022) An SIRS Model with Nonmonotone Incidence and Saturated Treatment in a Changing Environment. *Journal of Mathematical Biology*, **85**, Article No. 23. <https://doi.org/10.1007/s00285-022-01787-3>
- [11] Capasso, V. and Serio, G. (1978) A Generalization of the Kermack-Mckendrick Deterministic Epidemic Model. *Mathematical Biosciences*, **42**, 43-61. [https://doi.org/10.1016/0025-5564\(78\)90006-8](https://doi.org/10.1016/0025-5564(78)90006-8)
- [12] May, R.M. and Anderson, R.M. (1979) Population Biology of Infectious Diseases: Part II. *Nature*, **280**, 455-461. <https://doi.org/10.1038/280455a0>
- [13] Lu, M., Huang, J., Ruan, S. and Yu, P. (2019) Bifurcation Analysis of an SIRS Epidemic Model with a Generalized Nonmonotone and Saturated Incidence Rate. *Journal of Differential Equations*, **267**, 1859-1898. <https://doi.org/10.1016/j.jde.2019.03.005>
- [14] Liu, C. and Cui, R. (2021) Qualitative Analysis on an SIRS Reaction-Diffusion Epidemic Model with Saturation Infection Mechanism. *Nonlinear Analysis: Real World Applications*, **62**, Article ID: 103364. <https://doi.org/10.1016/j.nonrwa.2021.103364>
- [15] Avila-Vales, E., García-Almeida, G.E. and Pérez, Á.G.C. (2021) Qualitative Analysis of a Diffusive SIR Epidemic Model with Saturated Incidence Rate in a Heterogeneous Environment. *Journal of Mathematical Analysis and Applications*, **503**, Article ID: 125295. <https://doi.org/10.1016/j.jmaa.2021.125295>
- [16] Huo, X. and Cui, R. (2020) A Reaction-Diffusion SIS Epidemic Model with Saturated Incidence Rate and Logistic Source. *Applicable Analysis*, **101**, 4492-4511. <https://doi.org/10.1080/00036811.2020.1859495>
- [17] Dong, L., Li, B. and Zhang, G. (2022) Analysis on a Diffusive SI Epidemic Model with Logistic Source and Saturation Infection Mechanism. *Bulletin of the Malaysian Mathematical Sciences Society*, **45**, 1111-1140. <https://doi.org/10.1007/s40840-022-01255-7>
- [18] Mungkasi, S. (2021) Variational Iteration and Successive Approximation Methods for a SIR Epidemic Model with Constant Vaccination Strategy. *Applied Mathematical Modelling*, **90**, 1-10. <https://doi.org/10.1016/j.apm.2020.08.058>
- [19] Ehrhardt, M., Gašper, J. and Kilianová, S. (2019) Sir-Based Mathematical Modeling of Infectious Diseases with Vaccination and Waning Immunity. *Journal of Computational Science*, **37**, Article ID: 101027. <https://doi.org/10.1016/j.jocs.2019.101027>
- [20] Hou, J. and Teng, Z. (2009) Continuous and Impulsive Vaccination of SEIR Epidemic Models with Saturation Incidence Rates. *Mathematics and Computers in Simulation*, **79**, 3038-3054. <https://doi.org/10.1016/j.matcom.2009.02.001>
- [21] Rahman, S.M.A. and Zou, X. (2014) Modelling the Impact of Vaccination on Infectious Diseases Dynamics. *Journal of Biological Dynamics*, **9**, 307-320.

- <https://doi.org/10.1080/17513758.2014.986545>
- [22] Reza, D.S.A.A., Billah, M.N. and Shanta, S.S. (2021) Effect of Quarantine and Vaccination in a Pandemic Situation: A Mathematical Modelling Approach. *Journal of Mathematical Analysis and Modeling*, **2**, 77-87. <https://doi.org/10.48185/jmam.v2i3.318>
- [23] Maurício de Carvalho, J.P.S. and Rodrigues, A.A. (2023) SIR Model with Vaccination: Bifurcation Analysis. *Qualitative Theory of Dynamical Systems*, **22**, Article No. 105. <https://doi.org/10.1007/s12346-023-00802-2>
- [24] Du, Z. and Peng, R. (2015) A Priori  $L^\infty$  Estimates for Solutions of a Class of Reaction-Diffusion Systems. *Journal of Mathematical Biology*, **72**, 1429-1439. <https://doi.org/10.1007/s00285-015-0914-z>
- [25] Allen, L.J., Bolker, B.M., Lou, Y. and Nevai, A.L. (2008) Asymptotic Profiles of the Steady States for an SIS Epidemic Reaction-Diffusion Model. *Discrete & Continuous Dynamical Systems—A*, **21**, 1-20. <https://doi.org/10.3934/dcds.2008.21.1>
- [26] Magal, P. and Zhao, X. (2005) Global Attractors and Steady States for Uniformly Persistent Dynamical Systems. *SIAM Journal on Mathematical Analysis*, **37**, 251-275. <https://doi.org/10.1137/s0036141003439173>