



---

*Research article*

## Dynamics and asymptotic profiles of steady states of an SIRS epidemic model in spatially heterogenous environment

Baoxiang Zhang, Yongli Cai, Bingxian Wang and Weiming Wang\*

School of Mathematics and Statistics, Huaiyin Normal University, Huaian, 223300, China

\* **Correspondence:** Email: [weimingwang2003@163.com](mailto:weimingwang2003@163.com); Tel: +8613852316182;  
Fax: +86051788351540.

**Abstract:** This paper performs qualitative analysis on a reaction–diffusion SIRS epidemic system with ratio–dependent incidence rate in spatially heterogeneous environment. The threshold dynamics in the term of the basic reproduction number  $\mathcal{R}_0$  is established. And the asymptotic profile of endemic equilibrium is determined if the diffusion rate of the susceptible individuals is small. The results show that restricting the movement of susceptible individuals can effectively control the number of infectious individuals.

**Keywords:** ratio–dependent incidence rate; spatial heterogeneity; basic reproduction number; threshold dynamics; asymptotic profile

---

### 1. Introduction

It is now widely believed that the actual spread of many infectious diseases occurs in a diverse or dispersed population [1]. Subpopulations (or compartments) can be determined not only on the basis of disease-related factors such as mode of transmission, latent period, infectious period, and genetic susceptibility or resistance, but also on the basis of social, cultural, economic, demographic, and geographic factors [2]. In the viewpoint of epidemiology above, let  $S(t)$ ,  $I(t)$  and  $R(t)$  be the density of susceptible, infectious and recover individuals at time  $t$ , respectively, and we suppose that the dynamics of the disease transmission is governed by the following equations:

$$\begin{cases} \frac{dS}{dt} = \mu N - g(S, I) - \mu S + \gamma R, \\ \frac{dI}{dt} = g(S, I) - (\mu + \delta)I, \\ \frac{dR}{dt} = \delta I - \mu R - \gamma R, \end{cases} \quad (1.1)$$

where  $\mu, \delta, \gamma$  are all positive constants,  $\mu$  the birth and death rate,  $\delta$  the recovery rate of infectious individuals, and  $\gamma$  the rate of removed individuals who lose immunity and return to susceptible individuals class. Here, we assume that the disease does not have vertical transmission and is generally non-pathogenic and ignores the death induced by the disease. The infectious individuals force  $g(S, I)$  plays a key role in determining disease dynamics [3, 4]. Traditionally, the density-dependent transmission (or the bilinear incidence rate,  $g(S, I) = \beta SI$ , and  $\beta$  is the proportionality constant) and the frequency-dependent transmission (or the standard incidence rate,  $g(S, I) = \frac{\beta SI}{S+I}$ ) are two extreme forms of disease transmission, which have been frequently used in well-known epidemic models [5–7]. There are several different nonlinear transmission functions proposed by researchers, to see more details, we refer to [8–12] and the references therein. Especially, Yuan and Li [13] studied a ratio-dependent nonlinear incident rate which takes the following form (1.2):

$$g(S, I) = f\left(\frac{I}{S}\right)S = \frac{\beta (I/S)^l}{1 + \alpha (I/S)^h}S = \frac{\beta S^{h-l+1} I^l}{S^h + \alpha I^h}, \quad (1.2)$$

where the parameters  $l$  and  $h$  are positive constant,  $\alpha$  is the parameter which measures the psychological or inhibitory effect. It is worthy to note that in the special case of  $\alpha = 1$  and  $h = l = 1$ , (1.2) becomes the well-known frequency-dependent transmission rate  $\frac{\beta SI}{S+I}$ . In this case, the nonlinear incidence rate (1.2) can be seen as an extension form of the frequency-dependent transmission rate.

Before 1970s, ecological population modelers (involving epidemic models) typically used ordinary differential equations (ODE, e.g. model (1.1)), seeking equilibria and analyzing stability. These models provided important insights, such as when species can stably coexist and when susceptible and infectious densities oscillate over time [14]. The ODE models that have been described so far assume that the populations experience the same homogeneous environment. In reality, individual organisms are distributed in space and typically interact with the physical environment and other organisms in their spatial neighborhood [15]. More recently, many studies have shown that the spatial epidemic model is an appropriate tool for investigating the fundamental mechanism of complex spatiotemporal epidemic dynamics. In these studies, reaction-diffusion equations have been intensively used to describe spatiotemporal dynamics [16–25].

Many studies show that spatial heterogeneity generated by species dynamics is mathematically more interesting and also biologically more important [14]. In fact, relationships between individual-level processes and ecological dynamics often depend on population spatial structure, and epidemic dynamics can be governed by localized spatial processes of contact between susceptible and infectious individuals [27]. It has been suggested that spatial heterogeneity may address many of the deficiencies of epidemic models and play an important role in the spread of an epidemic [16, 28–34]. Of them, Grenfell, Bjornstad and Kappey [31] showed that measles waves spreading from large cities to small towns in England and Wales are determined by the spatial hierarchy of the host population structure; Keeling et al. [32] showed that the spatial distribution of farms influences the regional variability of foot-and-mouth outbreaks in UK; Hufnagel et al. [34], Colizza et al. [33] showed that the high degree of predictability of the worldwide spread of infectious diseases is caused by the strong heterogeneity of the transport network; Merler and Ajelli [30] showed that spatial heterogeneity in population density results in a relevant delay in epidemic onset between urban and rural areas. Hence, understanding the role of the spatial heterogeneity in epidemic dynamics is challenging both theoretically and empirically.

In this paper, we mainly focus on the impact of spatial heterogeneity of the disease dynamics of an SIRS epidemic model corresponding to model (1.1). To incorporate the random diffusion, described

by Laplacian operator [26], coming from the random wandering of susceptible, infectious and recover individuals, we add the diffusion terms to model (1.1) for the susceptible, infectious and recover individuals, respectively. To incorporate the spatial heterogeneity, we consider  $\beta := \beta(x)$ , and  $\beta(x)$  is a positive Hölder continuous function on  $\bar{\Omega}$ , which is the space-dependent rate of disease transmission by infectious individuals at position  $x \in \Omega$ ,  $\beta(x)I^l$  measures the infection force of the disease, and  $1/(1 + \alpha I^h)$  describes the psychological or inhibitory effect from the behavioral change of the susceptible individuals when the number of infective individuals is very large. For the sake of convenient analysis, we adopt  $l = 1$ . Then the reaction-diffusion model corresponding to model (1.1) is the following model system which governs the spatial heterogeneity and population mobility:

$$\begin{cases} \frac{\partial S}{\partial t} = d_S \Delta S + \mu N - \frac{\beta(x)S^h I}{S^h + \alpha I^h} - \mu S + \gamma R, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} = d_I \Delta I + \frac{\beta(x)S^h I}{S^h + \alpha I^h} - (\mu + \delta)I, & x \in \Omega, t > 0, \\ \frac{\partial R}{\partial t} = d_R \Delta R + \delta I - \mu R - \gamma R, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \mathbf{n}} = \frac{\partial I}{\partial \mathbf{n}} = \frac{\partial R}{\partial \mathbf{n}} = 0, & x \in \partial\Omega, t > 0, \end{cases} \quad (1.3)$$

where the habitat  $\Omega \subset \mathbb{R}^m (m \geq 1)$  is a bounded domain with smooth boundary  $\partial\Omega$  (when  $m > 1$ ), and  $\mathbf{n}$  is the outward unit normal vector on  $\partial\Omega$ . Moreover  $N = S + I + R$  is the total population,  $d_S$ ,  $d_I$  and  $d_R$  are diffusion coefficients for the susceptible, infectious and recover individuals, respectively.

Assume that the initial values satisfy

(H1)  $S(x, 0)$ ,  $I(x, 0)$  and  $R(x, 0)$  are nonnegative continuous functions in  $\bar{\Omega}$ ,  $\int_{\Omega} I(x, 0) dx > 0$  and

$$\int_{\Omega} (S(x, 0) + I(x, 0) + R(x, 0)) dx = N_0 > 0.$$

Mathematical models later confirm that spatial subdivision is important for the persistence of populations.

The rest of the paper is organized as follows. In Section 2, we give the global existence and uniform boundedness of solution. In Section 3, we investigate the threshold dynamics in terms of the basic reproduction number and study the asymptotic behavior of endemic equilibrium with respect to small diffusion rate of susceptible individuals. Finally, in Section 4, we provide the summary of the main results.

## 2. Global existence and uniform boundedness

The first goal of this paper is to concern with the global existence of classical solutions to model (1.3).

**Theorem 2.1.** *Model (1.3) has a unique global classical solution  $(S(x, t), I(x, t), R(x, t)) \in [C([0, \infty) \times \bar{\Omega}) \cap C^{2,1}((0, \infty) \times \bar{\Omega})]^3$  satisfying  $S(x, t), I(x, t), R(x, t) \geq 0$  for all  $t > 0$  and*

$$\|S(\cdot, t)\|_{L^\infty(\Omega)} + \|I(\cdot, t)\|_{L^\infty(\Omega)} + \|R(\cdot, t)\|_{L^\infty(\Omega)} \leq C(N_0), \quad (2.1)$$

where  $C(N_0) > 0$  is a constant dependent of  $N_0$ .

*Proof.* The local existence and uniqueness of the solutions of model (1.3) follow from a classical result ([35, Theorem 3.3.3]). It follows from the strong maximum principle [36] that  $S(x, t)$ ,  $I(x, t)$  and  $R(x, t)$  are nonnegative for  $x \in \bar{\Omega}$  and  $t \in (0, T_{\max})$ , here  $T_{\max}$  is the maximal existence time for solutions of model (1.3). In what follows, we prove that the local solution can be extended to a global one, that is  $T_{\max} = \infty$ . The method of the proof is similar to ([37, Theorem 2.2]).

Let

$$V(t) := \int_{\Omega} (S(x, t) + I(x, t) + R(x, t)) dx$$

be the total population size at time  $t$ . We can obtain that

$$\frac{\partial}{\partial t} \int_{\Omega} (S(x, t) + I(x, t) + R(x, t)) dx = \int_{\Omega} \Delta(d_S S + d_I I + d_R R) dx = 0, \quad t > 0.$$

The population size  $V$  is a constant, i.e.,

$$\int_{\Omega} (S(x, t) + I(x, t) + R(x, t)) dx = N_0, \quad t \geq 0, \quad (2.2)$$

which shows that  $\|S(\cdot, t)\|_{L^1(\Omega)}$ ,  $\|I(\cdot, t)\|_{L^1(\Omega)}$  and  $\|R(\cdot, t)\|_{L^1(\Omega)}$  are bounded in  $[0, T_{\max})$ .

From model (1.3) we easily deduce that

$$\begin{cases} \frac{\partial S}{\partial t} \leq d_S \Delta S + \mu I + (\mu + \gamma)R, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial t} \leq d_I \Delta I + (\beta(x) - \mu - \delta)I, & x \in \Omega, t > 0, \\ \frac{\partial R}{\partial t} = d_R \Delta R + \delta I - \mu R - \gamma R, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \mathbf{n}} = \frac{\partial I}{\partial \mathbf{n}} = \frac{\partial R}{\partial \mathbf{n}} = 0, & x \in \partial\Omega, t > 0, \end{cases} \quad (2.3)$$

It follows from [38, Lemma 2.1] with  $q = p_0 = 1$  that  $\|S(\cdot, t)\|_{L^\infty(\Omega)}$ ,  $\|I(\cdot, t)\|_{L^\infty(\Omega)}$  and  $\|R(\cdot, t)\|_{L^\infty(\Omega)}$  are also bounded in  $[0, \infty)$ . Thus, we obtain (2.1) and complete the proof.  $\square$

As expected, the steady state will play a central role in the dynamics of model (1.3). A steady state solution of model (1.3) is a time-independent (classical) solution and therefore can be viewed as a function  $(\tilde{S}, \tilde{I}, \tilde{R}) \in [C^2(\Omega) \cap C^1(\bar{\Omega})]^3$  satisfying

$$\begin{cases} d_S \Delta \tilde{S} + \mu \tilde{N} - \frac{\beta(x) \tilde{S}^h \tilde{I}}{\tilde{S}^h + \alpha \tilde{I}^h} - \mu \tilde{S} + \gamma \tilde{R} = 0, & x \in \Omega, \\ d_I \Delta \tilde{I} + \frac{\beta(x) \tilde{S}^h \tilde{I}}{\tilde{S}^h + \alpha \tilde{I}^h} - (\mu + \delta) \tilde{I} = 0, & x \in \Omega, \\ d_R \Delta \tilde{R} + \delta \tilde{I} - (\mu + \gamma) \tilde{R} = 0, & x \in \Omega, \\ \frac{\partial \tilde{S}}{\partial \mathbf{n}} = \frac{\partial \tilde{I}}{\partial \mathbf{n}} = \frac{\partial \tilde{R}}{\partial \mathbf{n}} = 0, & x \in \partial\Omega, \end{cases} \quad (2.4)$$

where  $\tilde{S}(x)$ ,  $\tilde{I}(x)$  and  $\tilde{R}(x)$  denote the density of the steady state solution at location  $x \in \Omega$ . In the view of (2.2), the steady state solutions also satisfy

$$\int_{\Omega} (\tilde{S}(x) + \tilde{I}(x) + \tilde{R}(x)) dx = N_0. \quad (2.5)$$

In epidemiological model, there are two typical constant steady state solutions, namely, disease-free equilibrium and endemic equilibrium. A *disease-free equilibrium* (DFE) is a steady state solution of (2.4) in which  $\tilde{S}(x) > 0$ , both  $\tilde{I}(x)$  and  $\tilde{R}(x)$  vanish at  $x \in \Omega$ , i.e.,  $\tilde{I}(x) = \tilde{R}(x) = 0$ . An *endemic equilibrium* (EE) is a steady state solution in which  $\tilde{S}(x), \tilde{I}(x), \tilde{R}(x) > 0$  for some  $x \in \Omega$ .

### 3. Threshold dynamics

This section aims to establish the threshold dynamics of (1.3) in terms of the basic reproduction number.

#### 3.1. Basic reproduction number

The basic reproductive number, denoted by  $\mathcal{R}_0$ , which is defined as the average number of secondary infections generated by a single infected individual introduced into a completely susceptible population, is one of the important quantities in epidemiology [41, 44].

The DFE of model (1.3) is  $E_0 = \left( \frac{N_0}{|\Omega|}, 0, 0 \right)$ , where  $|\Omega|$  is the Lebesgue measure of  $\Omega$ . Linearizing model (1.3) at  $E_0 = \left( \frac{N_0}{|\Omega|}, 0, 0 \right)$ , we get the following system for the infection related variable  $I$ :

$$\begin{cases} \frac{\partial I}{\partial t} = d_I \Delta I + (\beta - (\mu + \delta))I, & x \in \Omega, t > 0 \\ \frac{\partial I}{\partial \mathbf{n}} = 0, & x \in \partial\Omega, t > 0. \end{cases}$$

Using the next generation approach for spatial heterogeneous populations [44], we characterize the basic reproduction number  $\mathcal{R}_0$  for model (1.3) is

$$\mathcal{R}_0 = \frac{1}{\lambda_0},$$

where  $\lambda_0$  is a unique positive eigenvalue with a positive eigenfunction  $\Psi(x)$  on  $\Omega$  for the elliptic eigenvalues problem

$$\begin{cases} -d_I \Delta \psi + (\mu + \delta)\psi = \lambda \beta \psi, & x \in \Omega, \\ \frac{\partial \psi}{\partial \mathbf{n}} = 0, & x \in \partial\Omega. \end{cases} \quad (3.1)$$

Let  $\lambda^*$  be the principal eigenvalue with a positive eigenfunction  $\psi^*(x)$  on  $\Omega$  for the following eigenvalue problem :

$$\begin{cases} d_I \Delta \psi + (\beta - (\mu + \delta))\psi + \lambda \psi = 0, & x \in \Omega, \\ \frac{\partial \psi}{\partial \mathbf{n}} = 0, & x \in \partial\Omega. \end{cases} \quad (3.2)$$

We have the following result.

**Lemma 3.1.**  $\text{sign}(1 - \mathcal{R}_0) = \text{sign } \lambda^*$ .

*Proof.* Following [42, Lemma 2.3], we consider (3.1) and (3.2),

$$\begin{cases} d_I \Delta \Psi - (\mu + \delta)\psi + \frac{\beta}{\mathcal{R}_0} \Psi = 0, & x \in \Omega, \\ d_I \Delta \psi^* + (\beta - (\mu + \delta))\psi^* + \lambda^* \psi^* = 0, & x \in \Omega, \\ \frac{\partial \Psi}{\partial \mathbf{n}} = \frac{\partial \psi^*}{\partial \mathbf{n}} = 0, & x \in \partial \Omega. \end{cases} \quad (3.3)$$

We multiply the first equation in (3.3) by  $\psi^*$  and the second equation in (3.3) by  $\Psi$ , integrate by parts on  $\Omega$ , and subtract the two resulting equations,

$$\left(1 - \frac{1}{\mathcal{R}_0}\right) \int_{\Omega} \beta \Psi \psi^* dx + \lambda^* \int_{\Omega} \Psi \psi^* dx = 0.$$

Since  $\beta, \Psi, \psi^*$  are positive, we have  $\text{sign}(1 - \mathcal{R}_0) = \text{sign } \lambda^*$ .  $\square$

### 3.2. Stability of the DFE

In this subsection, we show that the stability of the DFE is determined entirely by the magnitude of  $\mathcal{R}_0$ .

**Theorem 3.2.** For model (1.3), if  $\mathcal{R}_0 < 1$ , then  $(S(x, t), I(x, t), R(x, t)) \rightarrow \left(\frac{N_0}{|\Omega|}, 0, 0\right)$  as  $t \rightarrow \infty$ . That is, the DFE is globally asymptotically stable.

*Proof.* Suppose that  $\mathcal{R}_0 < 1$ . By Lemma 3.1, it implies that  $\lambda^* > 0$ . Observe from the second equality of model (1.3) that

$$\begin{cases} \frac{\partial I}{\partial t} \leq d_I \Delta I + (\beta - (\mu + \delta))I, & x \in \Omega, t > 0, \\ \frac{\partial I}{\partial \mathbf{n}} = 0, & x \in \partial \Omega, t > 0. \end{cases} \quad (3.4)$$

Note that the following linear system

$$\begin{cases} \frac{\partial Z}{\partial t} = d_I \Delta Z + (\beta - (\mu + \delta))Z, & x \in \Omega, t > 0, \\ \frac{\partial Z}{\partial \mathbf{n}} = 0, & x \in \partial \Omega, t > 0. \end{cases} \quad (3.5)$$

admits a solution  $ae^{-\lambda^* t} \psi^*(x)$ , and  $a$  is chosen so large that  $I(x, 0) \leq Z(x, 0)$  for every  $x \in \Omega$ . The comparison principle implies that  $I(x, t) \leq ae^{-\lambda^* t} \psi^*(x)$ , and it then follows that  $I(x, t) \rightarrow 0$  as  $t \rightarrow \infty$  for  $x \in \bar{\Omega}$ .

As a result, the equation for  $R(x, t)$  is asymptotic to

$$\frac{\partial R}{\partial t} = d_R \Delta R - (\mu + \gamma)R, \quad x \in \Omega, t > 0.$$

From the comparison principle, we can get that  $R(x, t) \rightarrow 0$  as  $t \rightarrow \infty$  for  $x \in \bar{\Omega}$ . Similarly, we can get that  $S(x, t) \rightarrow \frac{N_0}{|\Omega|}$  as  $t \rightarrow \infty$  for  $x \in \bar{\Omega}$ . This yields the desired result.  $\square$

### 3.3. The existence of the endemic equilibrium

Suppose that  $\mathbb{X} = C(\bar{\Omega}; \mathbb{R}^3)$  have a supremum norm  $\|\cdot\|$ , then  $\mathbb{X}$  is an ordered Banach space with the cone  $\mathbb{P}$  consisting of all nonnegative functions in  $\mathbb{X}$ , and  $\mathbb{X}$  has nonempty interior, denoted by  $\text{int}(\mathbb{P})$ . Set

$$\mathbb{X}_0 = \left\{ W = (S, I, R) \in \mathbb{X} \mid \int_{\Omega} (S + I + R) dx = N_0 \right\}$$

and  $\mathbb{U} = \mathbb{P} \cap \mathbb{X}_0$ . It is easy to verify that model (1.3) defines a dynamic system on  $\mathbb{U}$ . Denote the unique solution of model (1.3) with initial value  $(S_0, I_0, R_0) \in \mathbb{U}$  by  $\Phi_t(S_0, I_0, R_0) = (S(\cdot, t), I(\cdot, t), R(\cdot, t))$  for any  $t > 0$ .  $\Phi_t$  is continuous and compact for  $t > 0$ .  $\Phi_t$  is pointwisely dissipative. Therefore,  $\Phi_t$  has a global attractor [46].

**Theorem 3.3.** *If  $\mathcal{R}_0 > 1$ , model (1.3) admits at least one endemic equilibrium.*

*Proof.* We appeal to the uniform persistence theory developed in [46, 47]. Denote

$$\mathbb{U}_0 := \left\{ (S_0, I_0, R_0) \in \mathbb{U} \mid I_0 \neq 0 \right\}, \quad \partial\mathbb{U}_0 := \left\{ (S_0, I_0, R_0) \in \mathbb{U} \mid I_0 = 0 \right\}.$$

Note that  $\mathbb{U} = \mathbb{U}_0 \cup \partial\mathbb{U}_0$ . Moreover,  $\mathbb{U}_0$  and  $\partial\mathbb{U}_0$  are relatively open and closed subsets of  $\mathbb{U}$ , respectively, and  $\mathbb{U}_0$  is convex. We divide the proof into three steps.

**Step 1.** A direct result of the strong maximum principle for parabolic equations is  $\Phi_t\mathbb{U}_0 \subset \mathbb{U}_0$  for all  $t > 0$ .

**Step 2.** Let  $A_{\partial}$  be the maximal positively invariant set for  $\Phi_t$  in  $\partial\mathbb{U}_0$ , i.e.

$$A_{\partial} := \left\{ W_0 \in \mathbb{U} \mid \Phi_t(W_0) \in \partial\mathbb{U}_0 \right\}.$$

It is easy to verify that  $A_{\partial} := \left\{ W_0 \in \mathbb{U} \mid I_0 = 0 \right\}$ .

Denote  $\omega(W_0)$  as the  $\omega$ -limit set of  $W_0$  in  $U$  and

$$\widehat{A}_{\partial} := \cup_{\{W_0 \in A_{\partial}\}} \omega(W_0).$$

We now prove  $\widehat{A}_{\partial} = \{E_0\}$ . For any  $W_0 \in A_{\partial}$ , i.e.  $I_0 = 0$ , then  $I(x, t) = 0$  for all  $x \in \Omega, t \geq 0$ , and model (1.3) becomes

$$\begin{cases} \frac{\partial S}{\partial t} = d_S \Delta S + (\mu + \gamma)R, & x \in \Omega, t > 0, \\ \frac{\partial R}{\partial t} = d_R \Delta R - \mu R - \gamma R, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \mathbf{n}} = \frac{\partial R}{\partial \mathbf{n}} = 0, & x \in \partial\Omega, t > 0, \end{cases}$$

which implies  $R(\cdot, t) \rightarrow 0, S(\cdot, t) \rightarrow \frac{N_0}{|\Omega|}$  uniformly as  $t \rightarrow \infty$ . Hence,  $\widehat{A}_{\partial} = \{E_0\}$ . Therefore,  $\{E_0\}$  is a compact and isolated invariant set for  $\Phi_t$  restricted in  $A_{\partial}$ .

**Step 3.** We prove that there exists some constant  $\epsilon_0$  independent of initial values such that

$$\lim_{t \rightarrow \infty} \|\Phi_t(W_0) - E_0\| > \epsilon_0.$$

Suppose, on the contrary, that for any  $\epsilon_1 > 0$ , there exists some initial value  $W_0^*$  such that

$$\lim_{t \rightarrow \infty} \|\Phi_t(W_0^*) - E_0\| \leq \frac{\epsilon_1}{2}. \quad (3.6)$$

For any given small  $\epsilon_2 > 0$ , let  $\lambda^*(\epsilon_2)$  be the unique principal eigenvalue of the following eigenvalue problem with a positive eigenfunction  $\phi_I$

$$\begin{cases} -d_I \Delta \phi_I - \left( \frac{\beta \left( \frac{N_0}{|\Omega|} - \epsilon_2 \right)^h}{\left( \frac{N_0}{|\Omega|} - \epsilon_2 \right)^h + \alpha \epsilon_2^h} - (\mu + \delta) \right) \phi_I = \lambda^*(\epsilon_2) \phi_I, & x \in \Omega, \\ \frac{\partial \phi_I}{\partial \mathbf{n}} = 0, & x \in \partial \Omega. \end{cases}$$

Note that  $\lim_{\epsilon_2 \rightarrow 0} \lambda^*(\epsilon_2) = \lambda^* < 0$ , where  $\lambda^*$  is the principal eigenvalue of eigenvalue problem (3.2). Therefore, we can choose  $\epsilon_2$  such that  $\lambda^*(\epsilon_2) < 0$ . Since  $\epsilon_1$  is arbitrary, we choose  $\epsilon_1 = 2\epsilon_2$ . In view of (3.6), there exists  $T > 0$  such that

$$\frac{N_0}{|\Omega|} - \epsilon_2 \leq S^*(x, t) \leq \frac{N_0}{|\Omega|} + \epsilon_2, \quad I^*(x, t), R^*(x, t) \leq \epsilon_2, \quad \forall x \in \bar{\Omega}, \forall t \geq T.$$

By the strong maximum principle of parabolic equations,  $(S^*(\cdot, x), I^*(\cdot, x), R^*(\cdot, x)) \in \text{int}(\mathbb{P})$  for all  $t > 0$ . Then we can choose a sufficiently small number  $c_* > 0$  such that  $I^*(T, x) \geq c_* \phi_I$ . Note that  $c_* e^{-\lambda^*(\epsilon_2)(t-T)} \phi_I$  is a solution of the following linear system

$$\begin{cases} \frac{\partial \hat{I}}{\partial t} = d_I \Delta \hat{I} + \left( \frac{\beta \left( \frac{N_0}{|\Omega|} - \epsilon_2 \right)^h}{\left( \frac{N_0}{|\Omega|} - \epsilon_2 \right)^h + \alpha \epsilon_2^h} - (\mu + \delta) \right) \hat{I}, & x \in \Omega, t > 0, \\ \frac{\partial \hat{I}}{\partial \mathbf{n}} = 0, & x \in \partial \Omega, t > 0, \\ \hat{I}(x, T) = c_* \phi_I, & x \in \Omega. \end{cases} \quad (3.7)$$

It follows from the comparison principle that

$$I^*(x, t) \geq c_* e^{-\lambda^*(\epsilon_2)(t-T)} \phi_I, \quad \forall t \geq T,$$

and, hence,  $I^*(x, t) \rightarrow \infty$  uniformly in  $\bar{\Omega}$  as  $t \rightarrow \infty$ , which contradicts (3.6).

The result of **Step 3** implies that  $\{E_0\}$  is an isolated invariant set for  $\Phi_t$  in  $\mathbb{U}$ , and  $W^S(\{E_0\}) \cap \mathbb{U}_0$  is an empty set, where  $W^S(\{E_0\})$  is the stable set of  $\{E_0\}$  for  $\Phi_t$ .

Finally, Combining **Steps 1–3** and [46, Theorem 1.3.1], we have that  $\Phi_t$  is uniformly persistent with respect to  $(\mathbb{U}, \partial \mathbb{U}_0)$ . Moreover, by [46, Theorem 1.3.7], model (1.3) admits at least one endemic equilibrium.  $\square$

### 3.4. Asymptotic properties of the endemic equilibrium

In this section, we are concerned with the asymptotic behavior of the EE of model (1.3). Our aim is to investigate the effect of the slow movement of susceptible individuals on the spatial distribution of the infectious disease. From now on, unless otherwise specified, we always assume  $\mathcal{R}_0 > 1$  and



(H2)  $\beta(x) > \mu$  for all  $x \in \bar{\Omega}$ .

Consider the linear eigenvalue problem

$$\begin{cases} -d_R \Delta \psi + (\mu + \gamma) \left(1 - \frac{\delta}{\beta - \mu}\right) \psi = \lambda \psi, & x \in \Omega, \\ \frac{\partial \psi}{\partial \mathbf{n}} = 0, & x \in \partial \Omega, \end{cases} \tag{3.8}$$

and denote the smallest eigenvalue of (3.8) by  $\lambda_1 := \lambda_1 \left(-d_R \Delta + (\mu + \gamma) \left(1 - \frac{\delta}{\beta - \mu}\right)\right)$ .

Denote  $\xi := d_S \tilde{S}(x) + d_I \tilde{I}(x) + d_R \tilde{R}(x)$  and set

$$S(x) = \frac{\tilde{S}(x)}{\xi}, \quad I(x) = \frac{\tilde{I}(x)}{\xi}, \quad R(x) = \frac{\tilde{R}(x)}{\xi}.$$

Model (2.4) is equivalent to

$$\begin{cases} d_I \Delta I + \frac{\beta(x) S^h I}{S^h + \alpha I^h} - (\mu + \delta) I = 0, & x \in \Omega, \\ d_R \Delta R + \delta I - \mu R - \gamma R = 0, & x \in \Omega, \\ d_S S + d_I I + d_R R = 1, & x \in \Omega, \\ \frac{\partial S}{\partial \mathbf{n}} = \frac{\partial I}{\partial \mathbf{n}} = \frac{\partial R}{\partial \mathbf{n}} = 0, & x \in \partial \Omega. \end{cases} \tag{3.9}$$

The following results hold:

**Lemma 3.4.**  $(\tilde{S}(x), \tilde{I}(x), \tilde{R}(x))$  is a solution of model (2.4) if and only if  $(S(x), I(x), R(x))$  is a solution of model (3.9). Moreover

$$\xi = \frac{N_0}{\int_{\Omega} (S + I + R) dx}.$$

**Lemma 3.5.** Assume that  $\mathcal{R}_0 > 1$ . For model (3.9),  $I \rightarrow I^*, R \rightarrow R^*$  in  $C^1(\bar{\Omega})$  as  $d_S \rightarrow 0$  for some  $I^*, R^* \in C^1(\bar{\Omega})$  with  $I^* \geq 0, R^* > 0$  on  $\bar{\Omega}$ .

*Proof.* In the view of  $d_S S + d_I I + d_R R = 1$ ,  $\frac{\beta(x) S^h I}{S^h + \alpha I^h}$  is uniformly bounded for any  $d_S > 0$ . It follows from  $L^p$ -estimate that  $\|I\|_{W^{2,p}}$  is bounded for any  $p > 1$ . Thus,  $\|I\|_{C^{1,\tau}}$  is bounded for any  $\tau \in (0, 1)$  by Sobolev embedding theorem. Passing to a subsequence if necessary,  $I \rightarrow I^*$  in  $C^1(\Omega)$  as  $d_S \rightarrow 0$  where  $I^*(x) \geq 0$  for  $x \in \Omega$  and  $\frac{\partial I}{\partial \mathbf{n}} = 0$  for  $x \in \Omega$ . By similar arguments,  $R \rightarrow R^*$  in  $C^1(\Omega)$  as  $d_S \rightarrow 0$  where  $R^*(x) \geq 0$  for  $x \in \Omega$ , which satisfies

$$\begin{cases} d_R \Delta R^* + \delta I^* - (\mu + \gamma) R^* = 0, & x \in \Omega, \\ \frac{\partial R^*}{\partial \mathbf{n}} = 0, & x \in \partial \Omega. \end{cases} \tag{3.10}$$

Now we show that  $I^*(x) \not\equiv 0$  on  $\Omega$  by contradiction argument. If  $I^*(x) = 0$ , then we obtain by (3.10) that  $R^* = 0$ , which implies that  $S \rightarrow \infty$  is almost everywhere (abbreviate a.e.) as  $d_S \rightarrow 0$ . Thus

$$\frac{\beta(x) S^h}{S^h + \alpha I^h} \rightarrow \beta(x) \text{ a.e as } d_S \rightarrow 0. \tag{3.11}$$

Define

$$M = \|I\|_{L^\infty(\Omega)} + \|R\|_{L^\infty(\Omega)}, \hat{I} = \frac{I}{M}, \hat{R} = \frac{R}{M}.$$

Note that  $\hat{I}, \hat{R} > 0$  and  $\|\hat{I}\|_{L^\infty(\Omega)} + \|\hat{R}\|_{L^\infty(\Omega)} = 1$ . Then by a standard compactness argument for elliptic equations, after passing to a further subsequence if necessary,

$$\hat{I} \rightarrow \hat{I}^*, \hat{R} \rightarrow \hat{R}^*, \quad x \in \bar{\Omega}, \quad \text{as } d_S \rightarrow 0,$$

where  $\hat{I}^*, \hat{R}^* \geq 0$  for  $x \in \Omega$  and

$$\begin{cases} \|\hat{I}^*\|_{L^\infty(\Omega)} + \|\hat{R}^*\|_{L^\infty(\Omega)} = 1, \\ \frac{\partial \hat{I}^*}{\partial \mathbf{n}} = \frac{\partial \hat{R}^*}{\partial \mathbf{n}} = 0, \end{cases} \quad x \in \partial\Omega. \tag{3.12}$$

It follows from (3.11) that  $\hat{I}^*$  is a weak solution of

$$\begin{cases} d_I \Delta \hat{I}^* + (\beta(x) - (\mu + \delta))\hat{I}^* = 0, & x \in \Omega, \\ \frac{\partial \hat{I}^*}{\partial \mathbf{n}} = 0, & x \in \partial\Omega. \end{cases} \tag{3.13}$$

By elliptic regularity, we have  $\hat{I}^* \in C^2(\bar{\Omega})$ , which gives

$$\begin{cases} d_I \Delta \hat{I}^* + (\beta(x) - (\mu + \delta))\hat{I}^* = 0, & x \in \Omega, \\ d_R \Delta \hat{R}^* + \delta \hat{I}^* - (\mu + \gamma)\hat{R}^* = 0, & x \in \Omega, \\ \frac{\partial \hat{I}^*}{\partial \mathbf{n}} = \frac{\partial \hat{R}^*}{\partial \mathbf{n}} = 0, & x \in \partial\Omega. \end{cases}$$

It follows from maximum principle together with (3.12) that  $\hat{I}^*(x), \hat{R}^*(x) > 0$ . We conclude that  $(\lambda, \psi) = (0, \hat{I}^*(x))$  is a solution of (3.2). Since  $\hat{I}^*(x) > 0$  on  $\Omega$ , it must be that  $\lambda^* = 0$ , which implies that  $\mathcal{R}_0 = 1$ . This contradiction yields  $I^*(x) \not\equiv 0$ . Therefore, again by maximum principle together with (3.10), we obtain  $R^* > 0$ . □

Note that  $I(x), R(x) > 0$  for any  $x \in \Omega, d_S > 0$ . Denote

$$M(x) = d_I I + d_R R.$$

Let

$$J^+ := \{x \in \bar{\Omega} | M^*(x) = 1\},$$

$$J^- := \{x \in \bar{\Omega} | 0 < M^*(x) < 1\},$$

where  $M^*(x) := d_I I^* + d_R R^*$ . Observe that  $J^- \cup J^+ = \bar{\Omega}$ .

**Lemma 3.6.** *Assume that  $\mathcal{R}_0 > 1$ .*

- (i) *The set  $J^+$  has positive Lebesgue measure.*
- (ii) *If further assume that  $\lambda_1 < 0$ , then the set  $J^-$  has positive Lebesgue measure.*

*Proof.* We prove  $|J^+| > 0$  by contradiction. If  $|J^+| = 0$ , i.e.,  $0 < M^*(x) < 1$  on  $\Omega$  a.e., then it follows from  $d_S S = 1 - d_I I - d_R R$  that  $S \rightarrow \infty$  a.e. as  $d_S \rightarrow 0$  and thus  $\frac{\beta(x)S^h I}{S^h + \alpha I^h} \rightarrow \beta(x)I^*$  a.e. as  $d_S \rightarrow 0$ . Therefore,  $I^*$  is a weak solution of

$$\begin{cases} d_I \Delta I^* + (\beta(x) - (\mu + \delta))I^* = 0, & x \in \Omega, \\ \frac{\partial I^*}{\partial \mathbf{n}} = 0, & x \in \partial\Omega. \end{cases}$$

By elliptic regularity, we have  $I^* \in C^2(\bar{\Omega})$ , which yields

$$\begin{cases} d_I \Delta I^* + (\beta(x) - (\mu + \delta))I^* = 0, & x \in \Omega, \\ d_R \Delta R^* + \delta I^* - (\mu + \gamma)R^* = 0, & x \in \Omega, \\ \frac{\partial I^*}{\partial \mathbf{n}} = \frac{\partial R^*}{\partial \mathbf{n}} = 0, & x \in \partial\Omega. \end{cases} \tag{3.14}$$

In light of (3.14) and  $R^* > 0$ , we have  $I^* > 0$ . We conclude that  $(\lambda, \psi) = (0, I^*(x))$  is a solution of (3.2). Since  $I^*(x) > 0$  on  $\Omega$ , it must be that  $\lambda^* = 0$ , which implies that  $\mathcal{R}_0 = 1$ . This contradiction implies  $|J^+| > 0$ .

We next prove part (ii) by contradiction. Now assume that  $|J^-| = 0$ , i.e.,  $M^*(x) = 1$  on  $\Omega$  a.e.. Denote

$$f(x) = \frac{\beta(x)S^h I}{S^h + \alpha I^h} - \mu I - (\mu + \gamma)R$$

and choose  $\varphi \in C^1(\bar{\Omega})$  such that  $\varphi \geq 0$  on  $\Omega$ . Multiplying the first two equations in (3.9) by  $\varphi$ , adding them together and integrating on  $\Omega$ , we have

$$- \int_{\Omega} \nabla \varphi \cdot \nabla (d_I I + d_R R) dx + \int_{\Omega} \varphi f(x) dx = 0. \tag{3.15}$$

As  $d_S \rightarrow 0$ ,  $M(x) \rightarrow M^*(x) = 1$  a.e on  $\Omega$ . Thus, we obtain

$$\int_{\Omega} \varphi f(x) dx \rightarrow 0 \text{ as } d_S \rightarrow 0 \tag{3.16}$$

for any  $\varphi \in C(\bar{\Omega})$  such that  $\varphi \geq 0$  on  $\Omega$ .

Let  $\psi_0$  be a positive eigenfunction of  $\lambda_1 \left( -d_R \Delta + (\mu + \gamma) \left( 1 - \frac{\delta}{\beta - \mu} \right) \right)$ , i.e.

$$\begin{cases} -d_R \Delta \psi_0 + (\mu + \gamma) \left( 1 - \frac{\delta}{\beta(x) - \mu} \right) \psi_0 = \lambda_1 \psi_0, & x \in \Omega, \\ \frac{\partial \psi_0}{\partial \mathbf{n}} = 0, & x \in \partial\Omega. \end{cases} \tag{3.17}$$

Since  $-d_R \Delta R + (\mu + \gamma)R - \delta I = 0$  and  $S, I, R > 0$  on  $\Omega$ , we have

$$-d_R \Delta R + (\mu + \gamma) \left( 1 - \frac{\delta}{\beta - \mu} \right) R = \frac{\delta}{\beta(x) - \mu} (\beta I - \mu I - (\mu + \gamma)R) \geq \frac{\delta}{\beta(x) - \mu} f(x), \quad x \in \Omega. \tag{3.18}$$

Multiplying (3.18) by  $\psi_0$ , integrating by parts over  $\Omega$  and applying (3.17), we obtain

$$\lambda_1 \int_{\Omega} \psi_0 R dx > \int_{\Omega} \frac{\delta}{\beta(x) - \mu} \psi_0 f(x) dx.$$

Let  $d_S \rightarrow 0$ . It immediately follows from (3.16) that  $\lambda_1 \int_{\Omega} \psi_0 R^* dx \geq 0$ . Since  $\psi_0, R^* > 0$  on  $\Omega$ , we see that  $\lambda_1 \geq 0$ . This contradiction yields (iii).  $\square$

**Theorem 3.7.** *Let (H2) hold. Assume that  $\mathcal{R}_0 > 1$  and  $\lambda_1 \left(-d_R \Delta + (\mu + \gamma) \left(1 - \frac{\delta}{\beta - \mu}\right)\right) < 0$ , then the following assertions hold:*

(i) *As  $d_S \rightarrow 0$ ,  $\tilde{S}$  subject to a sequence,*

$$\tilde{S} \rightarrow \tilde{S}^* = \frac{N_0(1 - M^*(x))}{\int_{\Omega} (1 - M^*(x)) dx}$$

for some  $M^*(x)$  satisfying  $0 < M^*(x) \leq 1$  in  $\Omega$ . Moreover,  $S^* = 0$  on  $J^+ \subset \bar{\Omega}$ ,  $S^* > 0$  on  $J^- \subset \bar{\Omega}$  and  $\int_{\Omega} \tilde{S}^* = N_0$ .

(ii) *There exist positive constants  $C_1, C_2$ , independent of  $d_S$ , such that for sufficiently small  $d_S$*

$$C_1 \leq \frac{\tilde{I}}{d_S}, \frac{\tilde{R}}{d_S} \leq C_2.$$

That is  $\tilde{I}, \tilde{R} \rightarrow 0$  uniformly in  $\Omega$  as  $d_S \rightarrow 0$ .

*Proof.* By (3.9), we have

$$\begin{aligned} N_0 &= \int_{\Omega} (\tilde{S} + \tilde{I} + \tilde{R}) dx \\ &= \xi \int_{\Omega} \left( \frac{1 - d_I I - d_R R}{d_S} + I + R \right) dx, \\ &= \frac{\xi}{d_S} \left( \int_{\Omega} d_S (I + R) dx + \int_{\Omega} (1 - M(x)) dx \right). \end{aligned}$$

It follows from  $S, I, R > 0$  and  $d_S S + d_I I + d_R R = 1$  that  $I, R$  are uniformly bounded with respect to  $d_S$ . Thus,

$$\int_{\Omega} d_S (I + R) dx \rightarrow 0 \text{ as } d_S \rightarrow 0.$$

In view of Lemmas 3.5 and 3.6, we have

$$\int_{\Omega} (1 - M(x)) dx \rightarrow \int_{\Omega} (1 - M^*(x)) dx > 0 \text{ as } d_S \rightarrow 0.$$

Therefore,

$$\frac{\xi}{d_S} \rightarrow \frac{N_0}{\int_{\Omega} (1 - M^*(x)) dx} \text{ as } d_S \rightarrow 0. \tag{3.19}$$

This limit is well-defined because  $J^-$  has positive measure. It follows from  $\tilde{S} = \frac{\xi}{d_S}(1 - M(x))$  that

$$\tilde{S} \rightarrow \tilde{S}^* = \frac{N_0(1 - M^*(x))}{\int_{\Omega} (1 - M^*(x))dx} \quad \text{as } d_S \rightarrow 0, \quad x \in C^1(\bar{\Omega}),$$

and  $\int_{\Omega} \tilde{S}^* = N_0$ .

Now we verify (ii). It follows from  $d_S S + d_I I + d_R R = 1$  and  $\tilde{I} = \frac{\xi}{d_S} d_S I, \tilde{R} = \frac{\xi}{d_S} d_S R$  that

$$0 < \frac{\tilde{I}}{d_S}, \frac{\tilde{R}}{d_S} < \frac{\xi}{d_S} \max \left\{ \frac{1}{d_I}, \frac{1}{d_R} \right\}.$$

Hence (3.19) implies that

$$\limsup_{d_S \rightarrow 0} \sup \frac{\tilde{I}}{d_S}, \limsup_{d_S \rightarrow 0} \sup \frac{\tilde{R}}{d_S} \leq \frac{N_0}{\int_{\Omega} (1 - M^*(x))dx} \max \left\{ \frac{1}{d_I}, \frac{1}{d_R} \right\}. \tag{3.20}$$

Next, by contradiction, we prove

$$\min \left\{ \inf_{\Omega} \tilde{I}, \inf_{\Omega} \tilde{R} \right\} / d_S \rightarrow 0, \quad \text{as } d_S \rightarrow 0.$$

Assume that  $\min \left\{ \inf_{\Omega} \tilde{I}, \inf_{\Omega} \tilde{R} \right\} / d_S = o(d_S)$ . By [48, Lemma 2.3] and (2.4), there exists a positive constant  $\theta$  such that

$$\inf_{\Omega} \tilde{I} \geq \theta \int_{\Omega} \frac{\beta(x)\tilde{S}^h \tilde{I}}{\tilde{S}^h + \alpha \tilde{I}^h} = \theta(\mu + \delta) \int_{\Omega} \tilde{I} dx, \quad \inf_{\Omega} \tilde{R} \geq \theta(\mu + \gamma) \int_{\Omega} \tilde{R} dx = \theta\delta \int_{\Omega} \tilde{I} dx.$$

Hence

$$\int_{\Omega} \tilde{I} dx, \int_{\Omega} \tilde{R} dx = o(d_S),$$

which implies

$$\int_{\Omega} \frac{d_I \tilde{I} + d_R \tilde{R}}{d_S} dx \rightarrow 0 \quad \text{as } d_S \rightarrow 0. \tag{3.21}$$

Noting that

$$N_0 = \int_{\Omega} \frac{\xi}{d_S} dx - \int_{\Omega} \frac{d_I \tilde{I} + d_R \tilde{R}}{d_S} dx + \int_{\Omega} (\tilde{I} + \tilde{R}) dx,$$

and combining (3.19) and (3.21), we can obtain

$$N_0 \rightarrow \frac{N_0 |\Omega|}{\int_{\Omega} (1 - M^*(x)) dx} \quad \text{as } d_S \rightarrow 0,$$

which contradicts Lemma 3.6 (ii) (i.e.,  $|J^-| > 0$ ). We complete the proof of part (ii). □

#### 4. Concluding remarks

It is now widely believed that the mathematical models have been revealed as a powerful tool to understand the mechanism that underlies the spread of the disease. In this paper, we proposed an epidemic model with ratio-dependent incidence rate incorporating both mobility of population and spatial heterogeneity, and focus on how spatial diffusion and environmental heterogeneity affect the basic reproductive number and disease dynamics of the model.

The value of our study lies in two aspects. Mathematically, we prove that the basic reproductive number  $\mathcal{R}_0$  can be used to govern the threshold dynamics of the model: if  $\mathcal{R}_0 < 1$ , the unique DFE is globally asymptotically stable (see Theorem 3.2), while if  $\mathcal{R}_0 > 1$ , there is at least one endemic equilibrium (see Theorem 3.3). Epidemiologically, we find that restricting the movement of susceptible population can effectively control the number of infectious individuals (see Theorem 3.7 (ii)). Simply speaking, our results may provide some potential applications in disease control.

#### Acknowledgements

The authors would like to thank the anonymous referees for very helpful suggestions and comments which led to improvement of our original manuscript. This research was supported by the National Natural Science Foundation of China (Grant numbers 61672013, 11601179 and 61772017), and the Huaian Key Laboratory for Infectious Diseases Control and Prevention (HAP201704).

#### Conflict of interest

The authors declare that they have no competing interests.

#### References

1. R. May and R. Anderson, Spatial heterogeneity and the design of immunization programs, *Math. Biosci.*, **72** (1984), 83–111.
2. H. Hethcote and J. W. Van Ark, Epidemiological models for heterogeneous populations: proportionate mixing, parameter estimation, and immunization programs, *Math. Biosci.*, **84** (1987), 85–118.
3. V. Capasso, *Mathematical structures of epidemic systems*, volume 97. Springer, 1993.
4. M. E. Alexander and S. M. Moghadas. Periodicity in an epidemic model with a generalized nonlinear incidence, *Math. Biosci.*, **189** (2004), 75–96.
5. W. D. Wang, Epidemic models with nonlinear infection forces, *Math. Biosci. Eng.*, **3** (2006), 267–279.
6. D. Xiao and S. Ruan, Global analysis of an epidemic model with nonmonotone incidence rate, *Math. Biosci.*, **208** (2007), 419–429.
7. Y. Cai, Y. Kang and W. M. Wang, A stochastic SIRS epidemic model with nonlinear incidence rate, *Appl. Math. Comp.*, **305** (2017), 221–240.

8. W. Liu, S. A. Levin and Y. Iwasa, Influence of nonlinear incidence rates upon the behavior of sirs epidemiological models, *J. Math. Biol.*, **23** (1986), 187–204.
9. W. Liu, H. W. Hethcote and S. A. Levin, Dynamical behavior of epidemiological models with nonlinear incidence rates, *J. Math. Biol.*, **25** (1987), 359–380.
10. H. W. Hethcote, The mathematics of infectious diseases, *SIAM Rev.*, **42** (2000), 599–653.
11. B. Fred and C.-C. Carlos, *Mathematical models in population biology and epidemiology (Second Edition)*. Springer, 2012.
12. Y. Cai, Y. Kang, M. Banerjee, et al., A stochastic SIRS epidemic model with infectious force under intervention strategies, *J. Differ. Equations*, **259** (2015), 7463–7502.
13. S. Yuan and B. Li, Global dynamics of an epidemic model with a ratio-dependent nonlinear incidence rate, *Discrete Dyn. Nat. Soc.*, **2009** (2009), 609306.
14. C. Neuhauser, Mathematical Challenges in Spatial Ecology, *Notices AMS*, **48** (2001), 1304–1314.
15. S. Ruan, Spatial-Temporal Dynamics in Nonlocal Epidemiological Models, In: Takeuchi Y., Iwasa Y., Sato K. (eds) *Mathematics for Life Science and Medicine*. Springer, Berlin, Heidelberg, 2007.
16. W. E. Fitzgibbon, M. Langlais and J. J. Morgan, A reaction-diffusion system modeling direct and indirect transmission of diseases, *Discrete Cont. Dyn.-B*, **4** (2004), 893–910.
17. Z. Wang and J. Wu, Travelling waves of a diffusive Kermack-McKendrick epidemic model with non-local delayed transmission, *P. Roy. Soc. A-Math. Phy.*, **466** (2010), 237–261.
18. Y. Cai and W. M. Wang, Dynamics of a parasite-host epidemiological model in spatial heterogeneous environment, *Discrete Cont. Dyn.-B*, **20** (2015), 989–1013.
19. Y. Cai and W. M. Wang, Fish-hook bifurcation branch in a spatial heterogeneous epidemic model with cross-diffusion, *Nonl. Anal. Real World Appl.*, **30** (2016), 99–125.
20. W. M. Wang, X. Gao, Y. Cai, et al., Turing patterns in a diffusive epidemic model with saturated infection force, *J. Franklin Inst.*, **355** (2018), 7226–7245.
21. Y. Cai, Y. Kang, M. Banerjee, et al., Complex dynamics of a host-parasite model with both horizontal and vertical transmissions in a spatial heterogeneous environment, *Nonl. Anal. Real World Appl.*, **40** (2018), 444–465.
22. P. Magal, G. Webb and Y. Wu, On a vector-host epidemic model with spatial structure, *Nonlinearity*, **31** (2018), 5589–5614.
23. Y. Cai, Z. Ding, B. Yang, et al., Transmission dynamics of Zika virus with spatial structure—A case study in Rio de Janeiro, Brazil, *Phys. A*, **514** (2019), 729–740.
24. J. Ge, K. Kim, Z. Lin, et al., An SIS reaction-diffusion-advection model in a low-risk and high-risk domain, *J. Differ. Equations*, **259** (2015), 5486–5509.
25. Y. Cai, X. Lian, Z. Peng, et al., Spatiotemporal transmission dynamics for influenza disease in a heterogenous environment, *Nonl. Anal. Real*, **46** (2019), 178–194.
26. E. E. Holmes, M. A. Lewis, J. E. Banks, et al., Partial differential equations in ecology: Spatial interactions and population dynamics, *Ecology*, **75** (1994), 17–29.
27. T. Caraco, M. Duryea, G. Gardner, et al., Host spatial heterogeneity and extinction of an SIS epidemic, *J. Theor. Biol.*, **192** (1998), 351–361.

28. A. L. LLOYD and R. M. May, Spatial heterogeneity in epidemic models, *J. Theor. Biol.*, **179** (1996), 1–11.
29. J. Dushoff and S. Levin, The effects of population heterogeneity on disease invasion, *Math. Biosci.*, **128** (1995), 25–40.
30. S. Merler and M. Ajelli, The role of population heterogeneity and human mobility in the spread of pandemic influenza, *Proc. R. Soc. B*, **277** (2010), 557–565.
31. B. T. Grenfell, O. N. Bjornstad and J. Kappey, Travelling waves and spatial hierarchies in measles epidemics, *Nature*, **414** (2001), 716–723.
32. M. J. Keeling, M. E. Woolhouse; D. J. Shaw, et al., Dynamics of the 2001 UK foot and mouth epidemic: stochastic dispersal in a heterogeneous landscape, *Science*, **294** (2001), 813–817.
33. V. Colizza, A. Barrat, M. Barthélemy, et al., The role of the airline transportation network in the prediction and predictability of global epidemics, *Proc. Natl. Acad. Sci. USA*, **103** (2006), 2015–2020.
34. L. Hufnagel, D. Brockmann and T. Geisel, Forecast and control of epidemics in a globalized world, *Proc. Natl. Acad. Sci. USA*, **101** (2004), 15124–15129.
35. D. Henry and D. B. Henry, *Geometric theory of semilinear parabolic equations*, Springer-Verlag Berlin, 1981.
36. M. H. Protter and H. F. Weinberger, *Maximum principles in differential equations*, Prentice-Hall, New Jersey, 1967.
37. K. Yamazaki and X. Wang, Global well-posedness and asymptotic behavior of solutions to a reaction-convection-diffusion cholera epidemic model, *Discrete Cont. Dyn.-B*, **21** (2017), 1297–1316.
38. Z. Du and R. Peng, A priori  $L^\infty$  estimates for solutions of a class of reaction-diffusion systems, *J. Math. Biol.*, **72** (2016), 1429–1439.
39. N. K. Vaidya, F.-B. Wang and X. Zou, Avian influenza dynamics in wild birds with bird mobility and spatial heterogeneous environment, *Discrete Cont. Dyn.-B*, **17** (2013), 2829–2848.
40. O. Diekmann, J. A. P. Heesterbeek and J. A. J. Metz, On the definition and the computation of the basic reproduction ratio  $R_0$  in models for infectious diseases in heterogeneous populations, *J. Math. Biol.*, **28** (1990), 365–382.
41. P. Van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, **180** (2002), 29–48.
42. L. J. S. Allen, B. M. Bolker, Y. Lou, et al., Asymptotic profiles of the steady states for an SIS epidemic reaction-diffusion model, *Discrete Cont. Dyn.-A*, **21** (2008), 1–20.
43. R. Peng and X. Zhao, A reaction–diffusion SIS epidemic model in a time-periodic environment, *Nonlinearity*, **25** (2012), 1451–1471.
44. W. D. Wang and X.-Q. Zhao, Basic reproduction numbers for reaction-diffusion epidemic models, *SIAM J. Appl. Dyn. Sys.*, **11** (2012), 1652–1673.
45. Y. Lou and T. Nagylaki, Evolution of a semilinear parabolic system for migration and selection without dominance, *J. Differ. Equations*, **225** (2006), 624–665.



- 
46. X. Zhao, J. Borwein and P. Borwein, *Dynamical systems in population biology*, volume 16. Springer, 2003.
  47. P. Magal and X.-Q. Zhao, Global attractors and steady states for uniformly persistent dynamical systems, *SIAM J. Math. Anal.*, **37** (2005), 251–275.
  48. M. A. Pino, A priori estimates and applications to existence-nonexistence for a semilinear elliptic system, *Indiana U. Math. J.*, **43** (1994), 77–129.



AIMS Press

© 2020 the Author(s), licensee AIMS Press. This is an open access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>)