

# ON A DIFFUSIVE SUSCEPTIBLE-INFECTED-SUSCEPTIBLE EPIDEMIC MODEL WITH MASS ACTION MECHANISM AND BIRTH-DEATH EFFECT: ANALYSIS, SIMULATIONS, AND COMPARISON WITH OTHER MECHANISMS\*

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**Abstract.** In the present paper, we are concerned with a susceptible-infected-susceptible epidemic reaction-diffusion model governed by a mass action infection mechanism and linear birth-death growth with no flux boundary condition. By performing qualitative analysis, we study the stability of the disease-free equilibrium, uniform persistence property in terms of the basic reproduction number and the global stability of the endemic equilibrium in a homogeneous environment, and investigate the asymptotic profile of endemic equilibria (when they exist) in a heterogeneous environment when the movement rate of the susceptible and infected populations is small. Our results, together with those in previous works on three other closely related modeling systems, suggest that factors such as infection mechanism, variation of total population, and population movement play vital but subtle roles in the transmission dynamics of diseases and hence provide useful insights into the strategies designed for disease control and prevention.

**Key words.** SIS epidemic reaction-diffusion model, mass action infection mechanism, basic reproduction number, endemic equilibria, small diffusion, asymptotic profile, persistence/extinction

**AMS subject classifications.** 35K57, 35J57, 35B40, 92D25

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**1. Introduction.** The mathematical study of infectious diseases can be traced back to the classic work of Kermack and McKendrick [29] in 1927. In [29], the authors adopted the *mass action infection mechanism* (also called *density-dependent infection mechanism*) to study a deterministic SIR (susceptible-infected-recovered) epidemic model, meaning that the infection (incidence) rate is proportional to the number of encounters between susceptible and infected individuals; mathematically, such an infection rate is characterized by the bilinear function  $\beta SI$ , where  $\beta > 0$  is the disease transmission rate and  $S(t)$  and  $I(t)$  represent the density of susceptible and infected populations respectively. The most significant achievement made in [29] is perhaps the epidemic threshold result that the density of susceptible individuals must exceed a critical value in order for the epidemic outbreak to occur. Due to the seminal importance of the Kermack–McKendrick theory to the field of theoretical epidemiology, their works were republished in 1991; see [30, 31, 32].

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Employing the same infection mechanism and instead considering an SIS (susceptible-infected-susceptible) model, one is led to the following ODE system (see, for instance, [43]):

$$(1.1) \quad \begin{cases} S' = -\beta SI + \gamma I, & t > 0, \\ I' = \beta SI - \gamma I, & t > 0, \end{cases}$$

where  $\gamma > 0$  is the disease recovery rate, together with initial data fulfilling  $S(0) + I(0) = N > 0$  and  $I(0) > 0$ . As one of the simplest models in mathematical epidemiology, (1.1) still demonstrates the threshold result as Kermack and McKendrick [29] observed. In fact, it is clear that  $S(t) + I(t) = N$  for all  $t \geq 0$  and, hence, (1.1) can be reduced to the following logistic-type equation:

$$I' = \beta I \left[ \left( N - \frac{\gamma}{\beta} \right) - I \right].$$

Simple analysis shows that if  $N \leq \gamma/\beta$ , then  $I(t) \rightarrow 0$  and in turn  $S(t) = N - I(t) \rightarrow N$  as  $t \rightarrow \infty$ , while if  $N > \gamma/\beta$ , it holds  $I(t) \rightarrow N - \gamma/\beta > 0$ , and  $S(t) \rightarrow \gamma/\beta > 0$  as  $t \rightarrow \infty$ . Defining the *basic reproduction number*  $\mathcal{R}_0 = N\beta/\gamma$ , then the *disease-free equilibrium* (DFE)  $(N, 0)$  is globally attractive if  $\mathcal{R}_0 \leq 1$ , while the *endemic equilibrium* (EE)  $(\gamma/\beta, N - \gamma/\beta)$  is globally attractive if  $\mathcal{R}_0 > 1$ . We also refer interested readers to the review paper [24] for various ODE models describing infectious diseases.

Nowadays it is widely recognized that spatial spread of an infection is closely related to the heterogeneity of the environment and the spatial-temporal movement of the hosts. This is well supported by numerous studies on diseases including malaria [38, 39], rabies [27, 28, 45], dengue fever [52], West Nile virus [34, 53], hantavirus [1, 2], Asian longhorned beetle [22, 23], etc.; see [51] and references therein. A popular way to incorporate spatial movement of hosts into epidemic models is to assume host random movements, leading to coupled reaction-diffusion equations. Taking into account spatial diffusion and environmental heterogeneity, we obtain the PDE version of (1.1):

$$(1.2) \quad \begin{cases} S_t - d_S \Delta S = -\beta(x)SI + \gamma(x)I, & x \in \Omega, t > 0, \\ I_t - d_I \Delta I = \beta(x)SI - \gamma(x)I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \end{cases}$$

where the spatial domain  $\Omega \subset \mathbb{R}^m$  ( $m \geq 1$ ) is bounded and has smooth boundary  $\partial\Omega$ ; positive constants  $d_S$  and  $d_I$  represent the diffusion rate of susceptible and infected individuals, respectively;  $\beta(x)$  and  $\gamma(x)$  are positive Hölder continuous functions on  $\overline{\Omega}$  accounting for the disease transmission rate and recovery rate, respectively; the Neumann boundary condition means that no population flux crosses the boundary  $\partial\Omega$ . For this model, Deng and Wu [14] studied the global dynamics and existence of EE, while [56, 57] investigated the asymptotic profile of EE (when it exists) as the diffusion rate of susceptible or infected populations is small or large, which consequently suggests interesting implications in terms of epidemiology; see the last section of our paper for further discussion.

System (1.2) does not take into consideration the birth/death effect of susceptible or infected individuals and thus the total population is conserved in the sense that

$$\int_{\Omega} [S(x, t) + I(x, t)] dx = \int_{\Omega} [S_0(x) + I_0(x)] dx =: N \quad \forall t \geq 0.$$

However, it is quite natural to consider the situation that susceptible individuals are subject to a recruitment (source) term modeling their birth and death rate, especially a linear one [6, 24]. Therefore, in this paper we are motivated to study the following reaction-diffusion epidemic system with varying total population and environmental heterogeneity:

$$(1.3) \quad \begin{cases} S_t - d_S \Delta S = \Lambda(x) - S - \beta(x)SI + \gamma(x)I, & x \in \Omega, t > 0, \\ I_t - d_I \Delta I = \beta(x)SI - [\gamma(x) + \mu(x)]I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x) \geq 0, I(x, 0) = I_0(x) \geq 0, & x \in \Omega. \end{cases}$$

The recruitment term  $\Lambda(x) - S$  represents that the susceptible population is subject to linear growth and  $\mu(x)$  accounts for the death rate of the infected, with  $\Lambda$  and  $\mu$  being assumed to be positive Hölder functions on  $\bar{\Omega}$ . All the other parameters have the same interpretation as before. Throughout the paper, the initial data  $S_0$  and  $I_0$  are nonnegative continuous functions on  $\bar{\Omega}$ , and there is a positive number of infected individuals initially, i.e.,  $\int_{\Omega} I_0(x)dx > 0$ .

Another widely accepted type of infection mechanism is the so-called *frequency-dependent transmission* (also called as *standard incidence infection mechanism*) of the form  $\beta SI/(S + I)$ , initiated by de Jong, Diekmann and Heesterbeek [13] in 1995. In this scenario, (1.2) becomes

$$(1.4) \quad \begin{cases} S_t - d_S \Delta S = -\beta(x) \frac{SI}{S + I} + \gamma(x)I, & x \in \Omega, t > 0, \\ I_t - d_I \Delta I = \beta(x) \frac{SI}{S + I} - \gamma(x)I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega, \end{cases}$$

and its counterpart with linear recruitment reads

$$(1.5) \quad \begin{cases} S_t - d_S \Delta S = \Lambda(x) - S - \beta(x) \frac{SI}{S + I} + \gamma(x)I, & x \in \Omega, t > 0, \\ I_t - d_I \Delta I = \beta(x) \frac{SI}{S + I} - \gamma(x)I, & x \in \Omega, t > 0, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, t > 0, \\ S(x, 0) = S_0(x), I(x, 0) = I_0(x), & x \in \Omega. \end{cases}$$

We note that (1.4) was first proposed by Allen et al. [4] and then it (and its variants) was (were) studied extensively by many researchers [11, 12, 16, 18, 19, 20, 25, 33, 46, 47, 49, 50] while (1.5) was analyzed by Li, Peng, and Wang [36]; see also [35] for the case of logistic source instead of the linear one. One also observes that the total population in (1.4) is conserved and that in (1.5) varies.

In [42], by comparing the outcomes of models with density-dependent and frequency-dependent transmission rates to the observed epidemiology of certain diseases, McCallum, Barlow and Hone concluded that both density-dependent and frequency-dependent mechanisms have their own advantages in modeling disease spread, depending on the transmission mode of the disease under consideration. They further pointed out that the transmission mode could be in general decided by estimating the force of infection.

On the other hand, epidemic theory for many ODE models has demonstrated that the *basic reproduction number*, which may be considered as the fitness of a pathogen in a given population, must be greater than unity for the pathogen to invade a susceptible population; see [7, 8, 15, 26, 44, 54] and references therein. For the PDE models (1.2)–(1.5), we can also find their respective basic reproduction number  $\mathcal{R}_0$  and show that  $\mathcal{R}_0$  serves as the threshold value to determine the transmission dynamics of disease, that is, if  $\mathcal{R}_0 > 1$  the disease persists whereas it becomes extinct in the long run if  $\mathcal{R}_0 < 1$ . However, the total population  $N$ , and the movement (migration) rates  $d_I$  and  $d_S$ , may affect  $\mathcal{R}_0$  of (1.2)–(1.5) in different ways. As a result, each of the parameters  $N$ ,  $d_I$ ,  $d_S$  plays a subtle role in disease control; a more detailed description will be made in the last discussion section.

The main goal of the current paper is twofold. The first one is to rigorously investigate qualitative properties of (1.3) and the asymptotic profile of EE (when it exists) with respect to the small movement rate  $d_I$  or  $d_S$ . Theorem 2.4 below tells us that once  $\mathcal{R}_0 > 1$ , the infectious disease will uniformly persist in space. Thus it becomes important to understand how the mobility of population migration affects the spatial distribution of disease, because this will help decision makers to predict the pattern of disease occurrence and, henceforth, to conduct effective/optimal control strategies of disease eradication. Our result in Theorem 3.1 indicates that restricting the motility rate of susceptible individuals cannot eradicate the disease for (1.3), while this strategy works perfectly for (1.2) with small total population size [57, Corollary 2.4]. A similar phenomenon was also observed in models (1.4) and (1.5). Therefore, this suggests that varying total population tends to enhance the persistence of infectious disease. The second goal is to compare our main results on the model (1.3) with those on models (1.2), (1.4), and (1.5), so as to understand the influence of the factors such as infection mechanism, movement rate, and source term on the eradication of epidemics, and to discuss possible applications in disease control. Numerical simulations are also carried out to reinforce the theoretical findings and illustrate possible outcomes for those unknown situations and hence provide clues for further analytical pursuits. We refer to section 4 for a detailed discussion on the implications of analytical results and comparisons between four related SIS epidemic models mentioned above.

The remainder of this paper is organized as follows. In section 2, we first obtain the global existence and boundedness of solutions to the parabolic problem (1.3), then discuss the stability of equilibrium and the uniform persistence property via the basic reproduction number  $\mathcal{R}_0$ , and finally we consider the global attractivity of DFE and EE in spatially homogeneous environments. Section 3 is devoted to the study of the asymptotic profile of EE when the diffusion rate of a susceptible population or an infected population approaches zero. In the last section, we perform numerical simulations, compare our results for (1.3) with those of the other three models, and discuss the implication of our findings in detail from the viewpoint of disease control.

In the rest of the paper, for notational convenience, we denote

$$g^* = \max_{x \in \bar{\Omega}} g(x) \quad \text{and} \quad g_* = \min_{x \in \bar{\Omega}} g(x) \quad \text{for } g = \Lambda, \beta, \gamma, \text{ and } \mu.$$

**2. Properties of solutions to (1.3).** In this section, we consider the parabolic system (1.3) by first establishing the global existence and uniform boundedness of solutions, and then show the local stability of DFE and uniform persistence via the basic reproduction number. Last we investigate the global attractivity of DFE and EE in a homogeneous environment.

**2.1. Global existence and uniform boundedness.** We now establish the global existence and boundedness of solutions to (1.3).

**THEOREM 2.1.** *The solution  $(S(x, t), I(x, t))$  of problem (1.3) exists uniquely and globally. Furthermore, there exist a positive constant  $M$  depending on initial data and the parameters  $d_S, d_I, \Lambda, \beta, \gamma,$  and  $\mu$  such that*

$$(2.1) \quad \|S(\cdot, t)\|_{L^\infty(\Omega)} + \|I(\cdot, t)\|_{L^\infty(\Omega)} \leq M \quad \forall t \geq 0.$$

Moreover, there exists some  $M' > 0$  independent of initial data fulfilling

$$(2.2) \quad \|S(\cdot, t)\|_{L^\infty(\Omega)} + \|I(\cdot, t)\|_{L^\infty(\Omega)} \leq M' \quad \forall t \geq T$$

for some large  $T > 0$ .

*Proof.* From the standard theory for semilinear parabolic systems [5], it follows that (1.3) admits a unique solution  $(S(x, t), I(x, t))$  for  $x \in \bar{\Omega}$  and  $t \in [0, T_{\max})$  with  $T_{\max}$  being the maximal existence time. Moreover, the strong maximum principle for parabolic equations yields that the solution is positive on  $\bar{\Omega} \times (0, T_{\max})$ . Integrating both PDEs of (1.3) and adding the resulting two identities, we are led to

$$(2.3) \quad \begin{aligned} \frac{d}{dt} \int_{\Omega} (S(x, t) + I(x, t)) dx &= \int_{\Omega} \Lambda(x) dx - \int_{\Omega} (S(x, t) + \mu(x)I(x, t)) dx \\ &\leq \int_{\Omega} \Lambda(x) dx - \theta \int_{\Omega} (S(x, t) + I(x, t)) dx, \end{aligned}$$

where  $\theta = \min\{1, \mu_*\} > 0$ . Then the well-known Gronwall's inequality applied to (2.3) asserts that there exists some constant  $M_1 > 0$ , such that

$$(2.4) \quad \int_{\Omega} (S(x, t) + I(x, t)) dx \leq M_1 \quad \forall t \in (0, T_{\max}).$$

We now consider

$$(2.5) \quad \begin{cases} S_t - d_S \Delta S = \Lambda(x) - S + [\gamma(x) - \beta(x)S] I, & x \in \Omega, t \in (0, T_{\max}), \\ \frac{\partial S}{\partial \nu} = 0, & x \in \partial\Omega, t \in (0, T_{\max}), \\ S(x, 0) = S_0(x), & x \in \Omega. \end{cases}$$

For any nonnegative  $I$ , it is straightforward to verify that the positive constant

$$M_2 := \max \left\{ \|\Lambda\|_{L^\infty(\Omega)}, \|S_0\|_{L^\infty(\Omega)}, \left\| \frac{\gamma}{\beta} \right\|_{L^\infty(\Omega)} \right\}$$

is an upper solution of (2.5). The comparison principle for parabolic equations gives

$$S(x, t) \leq M_2 \quad \forall x \in \bar{\Omega}, t \in (0, T_{\max}).$$

Since  $S$  is uniformly bounded and the  $L^1$ -norm of  $I(\cdot, t)$  is also bounded for  $t \in (0, T_{\max})$  thanks to (2.4), in view of [3, Theorem 3.1] or [50, Lemma 3.1] and using the  $I$ -equation, we deduce that  $I$  is also uniformly bounded in  $\bar{\Omega} \times (0, T_{\max})$ . As a result, we must have  $T_{\max} = \infty$  and (2.1) is proved.

We next show (2.2). To this aim, we need to construct a more accurate upper solution of problem (2.5), which is independent of  $S_0$  for all large time. In fact, let  $u(t)$  be the unique solution of the following ODE:

$$u'(t) = \Lambda^* + \|\gamma/\beta\|_{L^\infty(\Omega)} - u(t), \quad t > 0; \quad u(0) = \|S_0\|_{L^\infty(\Omega)} + \|\gamma/\beta\|_{L^\infty(\Omega)}.$$

It is clear that

$$u(t) = \left( \|S_0\|_{L^\infty(\Omega)} + \left\| \frac{\gamma}{\beta} \right\|_{L^\infty(\Omega)} \right) e^{-t} + \left( \Lambda^* + \left\| \frac{\gamma}{\beta} \right\|_{L^\infty(\Omega)} \right) (1 - e^{-t}) \geq \left\| \frac{\gamma}{\beta} \right\|_{L^\infty(\Omega)},$$

which implies  $\gamma(x) - \beta(x)u(t) \leq 0$ ,  $\forall x \in \bar{\Omega}$ ,  $t > 0$ . It can be easily checked that  $u(t)$  is an upper solution of (2.5) and consequently,

$$S(x, t) \leq u(t) \rightarrow \Lambda^* + \left\| \frac{\gamma}{\beta} \right\|_{L^\infty(\Omega)} \quad \text{as } t \rightarrow \infty, \quad \forall x \in \bar{\Omega}.$$

That is, we obtain an upper bound of  $\|S(\cdot, t)\|_{L^\infty(\Omega)}$  which is independent of initial data for all large time. Now applying [50, Lemma 3.1] to the  $I$ -equation, we deduce that  $\|I(\cdot, t)\|_{L^\infty(\Omega)}$  can also be bounded by a positive constant independent of  $(S_0, I_0)$  for large  $t > 0$ .  $\square$

**2.2. Basic reproduction number and uniform persistence.** It is easily seen that the following elliptic problem

$$(2.6) \quad -d_S \Delta S = \Lambda(x) - S, \quad x \in \Omega; \quad \frac{\partial S}{\partial \nu} = 0, \quad x \in \partial\Omega$$

admits a unique positive solution  $\tilde{S}$ , which is globally asymptotically stable for the corresponding parabolic equation with nonnegative initial data. Then  $(\tilde{S}, 0)$  is an equilibrium of (1.3), which we call the DFE. Clearly, it is the unique DFE.

We define the basic reproduction number  $\mathcal{R}_0$  as follows:

$$(2.7) \quad \mathcal{R}_0 = \sup_{0 \neq \varphi \in H^1(\Omega)} \frac{\int_{\Omega} \beta \tilde{S} \varphi^2 dx}{\int_{\Omega} [d_I |\nabla \varphi|^2 + (\gamma + \mu) \varphi^2] dx}.$$

Indeed, one can follow the idea of next generation operators as in [50] to introduce the basic reproduction number, which coincides with the value  $\mathcal{R}_0$ . It is worth mentioning that the basic reproduction number  $\mathcal{R}_0$  defined here is qualitatively different from that in [4] and [14] in that it also depends implicitly on the diffusion rate  $d_S$  of the susceptible individuals.

Let  $(\lambda^*, \psi^*)$  be the principal eigenpair of the eigenvalue problem

$$(2.8) \quad d_I \Delta u + (\beta \tilde{S} - \gamma - \mu)u + \lambda u = 0, \quad x \in \Omega; \quad \frac{\partial u}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

Then, we have the following properties of  $\mathcal{R}_0$ , the proof of which resembles that of [4, Lemma 2.3] and hence is omitted.

**PROPOSITION 2.2.** *The following assertions hold.*

- (a)  $\mathcal{R}_0$  is a monotone decreasing function of  $d_I$  with  $\mathcal{R}_0 \rightarrow \max_{\bar{\Omega}} \beta \tilde{S} / (\gamma + \mu)$  as  $d_I \rightarrow 0$  and  $\mathcal{R}_0 \rightarrow \int_{\Omega} \beta \tilde{S} dx / \int_{\Omega} (\gamma + \mu) dx$  as  $d_I \rightarrow \infty$ .
- (b) If  $\int_{\Omega} \beta(x) \tilde{S}(x) dx < \int_{\Omega} [\gamma(x) + \mu(x)] dx$ , and  $\beta \tilde{S} - (\gamma + \mu)$  changes sign, then there exists a threshold value  $d_I^* \in (0, \infty)$  such that  $\mathcal{R}_0 > 1$  for  $d_I < d_I^*$  and  $\mathcal{R}_0 < 1$  for  $d_I > d_I^*$ .
- (c) If  $\int_{\Omega} \beta(x) \tilde{S}(x) dx > \int_{\Omega} [\gamma(x) + \mu(x)] dx$ , then  $\mathcal{R}_0 > 1$  for all  $d_I > 0$ .
- (d)  $\mathcal{R}_0 > 1$  when  $\lambda^* < 0$ ,  $\mathcal{R}_0 = 1$  when  $\lambda^* = 0$ , and  $\mathcal{R}_0 < 1$  when  $\lambda^* > 0$ .

It turns out that the stability of the DFE  $(\tilde{S}, 0)$  is completely determined by the size of  $\mathcal{R}_0$ .

PROPOSITION 2.3. *The DFE  $(\tilde{S}, 0)$  is linearly stable if  $\mathcal{R}_0 < 1$ , and it is linearly unstable if  $\mathcal{R}_0 > 1$ .*

*Proof.* The linearization of (1.3) around the DFE  $(\tilde{S}, 0)$  reads

$$\begin{cases} \eta_t - d_S \Delta \eta = -\eta + (-\beta \tilde{S} + \gamma)\xi, & x \in \Omega, t > 0, \\ \xi_t - d_I \Delta \xi = (\beta \tilde{S} - \gamma - \mu)\xi, & x \in \Omega, t > 0, \\ \frac{\partial \eta}{\partial \nu} = \frac{\partial \xi}{\partial \nu} = 0, & x \in \partial \Omega, t > 0, \end{cases}$$

with  $\eta(x, t) = S(x, t) - \tilde{S}(x)$  and  $\xi(x, t) = I(x, t)$ . Now suppose that  $(\eta(x, t), \xi(x, t)) = (e^{-\lambda t} \phi(x), e^{-\lambda t} \psi(x))$  is a solution of the above linear system with  $\lambda$  being a complex number. Then simple calculations show that

$$(2.9) \quad \begin{cases} d_S \Delta \phi - \phi + (-\beta \tilde{S} + \gamma)\psi + \lambda \phi = 0, & x \in \Omega, \\ d_I \Delta \psi + (\beta \tilde{S} - \gamma - \mu)\psi + \lambda \psi = 0, & x \in \Omega, \\ \frac{\partial \phi}{\partial \nu} = \frac{\partial \psi}{\partial \nu} = 0, & x \in \partial \Omega. \end{cases}$$

We first assume that  $\mathcal{R}_0 < 1$  and shall show that  $(\tilde{S}, 0)$  is linearly stable, that is, if  $(\lambda, \phi, \psi)$  is any solution of (2.9) with  $\phi$  or  $\psi$  not identically zero, then  $\text{Re}(\lambda) > 0$ . There are two cases to consider:  $\psi \equiv 0$  and  $\phi \neq 0$ ;  $\psi \neq 0$ .

In the former case, clearly  $(\lambda, \phi)$  is an eigenpair of the eigenvalue problem

$$(2.10) \quad d_S \Delta u - u + \lambda u = 0, \quad x \in \Omega; \quad \frac{\partial u}{\partial \nu} = 0, \quad x \in \partial \Omega.$$

It is obvious that  $\lambda$  must be real due to the self-adjoint property of the operator involved in (2.10) and hence  $\lambda \geq 1$ , as we wanted. If the latter case happens, it follows that  $(\lambda, \psi)$  is an eigenpair of the eigenvalue problem (2.8) and hence  $\lambda$  is real and  $\lambda \geq \lambda^* > 0$  due to Proposition 2.2(d). Thus, the linear stability of  $(\tilde{S}, 0)$  is proved.

We now suppose  $\mathcal{R}_0 > 1$  and show the instability of  $(\tilde{S}, 0)$ . Proposition 2.2(d) yields that  $\lambda^* < 0$ . It is well known that the following linear problem

$$d_S \Delta \phi - \phi + \lambda^* \phi = (\beta \tilde{S} - \gamma)\psi^*, \quad x \in \Omega, \quad \frac{\partial \phi}{\partial \nu} = 0, \quad x \in \partial \Omega$$

admits a solution  $\phi^*$ . Consequently,  $(\lambda^*, \phi^*, \psi^*)$  becomes a solution of (2.9) with  $\lambda^* < 0$  and  $\psi^* > 0$  and so  $(\tilde{S}, 0)$  is linearly unstable.  $\square$

Based on the “ultimately uniform boundedness” (2.2), we are able to establish the uniform persistence property of (1.3) when the basic reproduction number  $\mathcal{R}_0 > 1$ . In fact, one can easily adapt the arguments of [50, Theorem 3.3], developed by Magal and Zhao (see [41, Theorem 4.5] and [58, Chapter 13]), to conclude the following assertion.

THEOREM 2.4. *Suppose that  $\mathcal{R}_0 > 1$ . Then system (1.3) is uniformly persistent, i.e., there exists some  $\eta > 0$  independent of the initial data  $(S_0, I_0)$ , such that*

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

Furthermore, (1.3) admits at least one EE provided that  $\mathcal{R}_0 > 1$ .

**2.3. Global stability in a homogeneous environment.** In this subsection, we consider the global stability of the DFE and EE of (1.3) in a homogeneous environment, i.e., all of the parameters  $\Lambda$ ,  $\beta$ ,  $\gamma$ , and  $\mu$  are positive constants. In view of (2.7), we now have an explicit expression for the basic reproduction number  $\mathcal{R}_0 = \frac{\Lambda\beta}{\gamma+\mu}$  and the unique DFE is given by  $(\tilde{S}, 0) = (\Lambda, 0)$ . On the other hand, there exists a unique constant EE  $(\hat{S}, \hat{I})$  if and only if  $\mathcal{R}_0 > 1$ , where

$$\hat{S} = \frac{\gamma + \mu}{\beta} = \frac{\Lambda}{\mathcal{R}_0} \quad \text{and} \quad \hat{I} = \frac{\Lambda}{\mu} \left(1 - \frac{1}{\mathcal{R}_0}\right) = \frac{\gamma + \mu}{\mu\beta} (\mathcal{R}_0 - 1).$$

For later purposes, we recall a simple fact which can be found in [55, Lemma 2.5.1].

**LEMMA 2.1.** *Let  $a$  and  $b$  be positive constants. Assume that  $\varphi, \psi \in C^1([a, \infty))$ ,  $\psi(t) \geq 0$  in  $[a, \infty)$ , and  $\varphi$  is bounded from below. If  $\varphi'(t) \leq -b\psi(t)$  and  $\psi'(t) \leq K$  in  $[a, \infty)$  for some constant  $K$ , then  $\lim_{t \rightarrow \infty} \psi(t) = 0$ .*

By constructing suitable Lyapunov functionals, we can show the following.

**THEOREM 2.5.** *Assume that  $d_S = d_I$ . Then the following assertions hold.*

- (i) *If  $\mathcal{R}_0 \leq 1$ , then the DFE is globally attractive.*
- (ii) *If  $\mathcal{R}_0 > 1$ , then the EE is globally attractive.*

*Proof.* Set  $d_S = d_I = d$ . To verify (i), for any solution  $(S, I)$  of (1.3), we define

$$V(t) = \frac{1}{2} \int_{\Omega} [(S - \Lambda) + I]^2 dx + \frac{\mu + 1}{\beta} \int_{\Omega} I dx.$$

Then, for all  $t > 0$ , direct calculations show that

$$\begin{aligned} V'(t) &= \int_{\Omega} [(S - \Lambda) + I] (S_t + I_t) dx + \frac{\mu + 1}{\beta} \int_{\Omega} I_t dx \\ &= \int_{\Omega} [(S - \Lambda) + I] (d_S \Delta S + \Lambda - S - \mu I + d_I \Delta I) dx \\ &\quad + \frac{\mu + 1}{\beta} \int_{\Omega} (d_I \Delta I + \beta SI - \gamma I - \mu I) dx \\ &= -d \int_{\Omega} |\nabla(S + I)|^2 dx - \int_{\Omega} (S - \Lambda)^2 dx - \mu \int_{\Omega} I(S - \Lambda) dx + \int_{\Omega} I(\Lambda - S) dx \\ &\quad - \mu \int_{\Omega} I^2 dx + \frac{\mu + 1}{\beta} \int_{\Omega} (\beta SI - \gamma I - \mu I) dx \\ &\leq - \int_{\Omega} (S - \Lambda)^2 dx - \mu \int_{\Omega} I^2 dx + \frac{\mu + 1}{\beta} [\beta \Lambda - (\gamma + \mu)] \int_{\Omega} I dx \leq 0, \end{aligned}$$

due to the assumption that  $\mathcal{R}_0 = \beta\Lambda/(\gamma + \mu) \leq 1$ . Define

$$\psi(t) = \int_{\Omega} (S - \Lambda)^2 dx + \mu \int_{\Omega} I^2 dx \geq 0.$$

Recall that Theorem 2.1 tells us that both  $\|S(\cdot, t)\|_{L^\infty(\Omega)}$  and  $\|I(\cdot, t)\|_{L^\infty(\Omega)}$  are bounded. Hence, by [10, Theorem A2], we have

$$(2.11) \quad \|S(\cdot, t)\|_{C^{2+\alpha}(\bar{\Omega})} + \|I(\cdot, t)\|_{C^{2+\alpha}(\bar{\Omega})} \leq C_0 \quad \forall t \geq 1$$



for some positive constant  $C_0$ . Furthermore, using both PDEs of (1.3), one can easily see that  $\psi'(t)$  is bounded from above for  $t \in [1, \infty)$ . We deduce from Lemma 2.1 (by taking  $\varphi(t) = V(t)$ ) that

$$(S(x, t), I(x, t)) \rightarrow (\Lambda, 0) = (\tilde{S}, 0) \text{ in } (L^2(\Omega))^2 \text{ as } t \rightarrow \infty.$$

Furthermore, (2.11) indicates that  $(S(\cdot, t), I(\cdot, t))$  is compact in  $C^2(\bar{\Omega})$  for  $t \geq 1$ . This, together with the above  $L^2$ -convergence, yields that

$$(S(x, t), I(x, t)) \rightarrow (\tilde{S}, 0) \text{ in } (C^2(\bar{\Omega}))^2 \text{ as } t \rightarrow \infty,$$

that is,  $(\tilde{S}, 0)$  attracts all solutions of (1.3).

We next prove (ii). Define

$$W(t) = \frac{1}{2} \int_{\Omega} [(S - \hat{S}) + (I - \hat{I})]^2 dx + \frac{\mu + 1}{\beta} \int_{\Omega} \left( I - \hat{I} - \hat{I} \ln \frac{I}{\hat{I}} \right) dx \geq 0 \quad \forall t > 0.$$

By straightforward computations, we have

$$\begin{aligned} W'(t) &= \int_{\Omega} [(S - \hat{S}) + (I - \hat{I})] (S_t + I_t) dx + \frac{\mu + 1}{\beta} \int_{\Omega} \left( 1 - \frac{\hat{I}}{I} \right) I_t dx \\ &= \int_{\Omega} [(S - \hat{S}) + (I - \hat{I})] (d\Delta S + \Lambda - S - \mu I + d\Delta I) dx \\ &\quad + \frac{\mu + 1}{\beta} \int_{\Omega} \left( 1 - \frac{\hat{I}}{I} \right) (d\Delta I + \beta S I - \gamma I - \mu I) dx \\ &= -d \int_{\Omega} |\nabla(S + I)|^2 dx - \frac{\mu + 1}{\beta} d\hat{I} \int_{\Omega} \frac{|\nabla I|^2}{I^2} dx + \frac{\mu + 1}{\beta} \int_{\Omega} (I - \hat{I})(\beta S - \beta \hat{S}) dx \\ &\quad + \int_{\Omega} [(S - \hat{S}) + (I - \hat{I})] (\hat{S} + \mu \hat{I} - S - \mu I) dx \\ &\leq - \int_{\Omega} (S - \hat{S})^2 dx - \mu \int_{\Omega} (I - \hat{I})^2 dx \leq 0, \end{aligned}$$

where we have used the fact that  $\Lambda = \hat{S} + \mu \hat{I}$  and  $\gamma + \mu = \beta \hat{S}$ .

In Lemma 2.1, let

$$\phi(t) = W(t), \quad \psi(t) = \int_{\Omega} (S - \hat{S})^2 dx + \mu \int_{\Omega} (I - \hat{I})^2 dx \quad \forall t > 0.$$

Then arguing similarly as before, we eventually conclude that

$$(S(x, t), I(x, t)) \rightarrow (\hat{S}, \hat{I}) \text{ in } (C^2(\bar{\Omega}))^2 \text{ as } t \rightarrow \infty.$$

The proof is complete. □

The above theorem tells us that system (1.3) is uniformly persistent in a homogeneous environment provided  $\mathcal{R}_0 > 1$ , at least in the equal diffusion rate case.

*Remark 2.1.* For general positive functions  $\Lambda, \beta, \gamma, \mu$  and constants  $d_S, d_I > 0$ , we suspect that (1.3) has a unique EE which is globally attractive if  $\mathcal{R}_0 > 1$ , and the DFE is globally attractive if  $\mathcal{R}_0 \leq 1$ . However the justification of this suspicion is highly nontrivial and has to be left open in the current paper.

**3. Asymptotic profile of EE.** In this section, we are concerned with the asymptotic behavior of EE of (1.3), which is a positive solution to the elliptic system

$$(3.1) \quad \begin{cases} -d_S \Delta S = \Lambda(x) - S - \beta(x)SI + \gamma(x)I, & x \in \Omega, \\ -d_I \Delta I = \beta(x)SI - [\gamma(x) + \mu(x)]I, & x \in \Omega, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega \end{cases}$$

as one of the diffusion rates  $d_S, d_I$  goes to zero.

**3.1. The case  $d_S \rightarrow 0$ .** Using a singular perturbation argument, one can easily show that  $\tilde{S}$ , being the unique positive solution of (2.6), converges uniformly to  $\Lambda$  as  $d_S \rightarrow 0$  (see [48, Lemma 3.2]). Therefore, according to the continuity of eigenvalues with respect to the potential function, we see that the principal eigenvalue  $\lambda^*$  of (2.8) converges to the principal eigenvalue of the following eigenvalue problem

$$(3.2) \quad d_I \Delta u + (\beta\Lambda - \gamma - \mu)u + \lambda u = 0, \quad x \in \Omega; \quad \frac{\partial u}{\partial \nu} = 0, \quad x \in \partial\Omega,$$

which is denoted by  $\lambda_0$ . To ensure the existence of EE for all small  $d_S$ , one has to assume  $\lambda_0 < 0$ .

Now we are ready to establish the main result of this subsection.

**THEOREM 3.1.** *Assume that  $\lambda_0 < 0$ . Fix  $d_I > 0$ , and let  $d_S \rightarrow 0$ , then every positive solution  $(S, I)$  of (3.1) satisfies (up to a subsequence of  $d_S \rightarrow 0$ )*

$$(S, I) \rightarrow (\underline{S}, \underline{I}) \quad \text{uniformly on } \overline{\Omega},$$

where  $\underline{S}(x) = \frac{\Lambda(x) + \gamma \underline{I}(x)}{1 + \beta \underline{I}(x)}$  and  $\underline{I}$  is a positive solution to

$$(3.3) \quad -d_I \Delta \underline{I} = \beta(x) \underline{S} \underline{I} - (\gamma(x) + \mu(x)) \underline{I}, \quad x \in \Omega; \quad \frac{\partial \underline{I}}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

*Proof.* As was mentioned before, (3.1) has at least one EE for all small  $d_S > 0$  when  $\lambda_0 < 0$ . In the following, we divide our argument into three steps for sake of clarity.

*Step 1: A priori bounds for  $S$  and  $I$ .* Assume  $S(x_0) = \max_{x \in \overline{\Omega}} S(x)$ . We apply the maximum principle [40, Proposition 2.2] to the first equation of (3.1) to derive  $\Lambda(x_0) - S(x_0) - \beta(x_0)S(x_0)I(x_0) + \gamma(x_0)I(x_0) \geq 0$  or

$$(3.4) \quad \Lambda^* \geq \Lambda(x_0) \geq S(x_0) + I(x_0)(\beta(x_0)S(x_0) - \gamma(x_0)).$$

If  $\beta(x_0)S(x_0) - \gamma(x_0) \leq 0$ , then  $\max_{\overline{\Omega}} S = S(x_0) \leq \gamma(x_0)/\beta(x_0) \leq \|\gamma/\beta\|_{L^\infty(\Omega)}$ . If  $\beta(x_0)S(x_0) - \gamma(x_0) > 0$ , it follows from (3.4) that  $\max_{\overline{\Omega}} S = S(x_0) \leq \Lambda^*$ . Thus, for any  $d_S, d_I > 0$ , we have

$$(3.5) \quad \max_{\overline{\Omega}} S \leq \max \left\{ \Lambda^*, \left\| \frac{\gamma}{\beta} \right\|_{L^\infty(\Omega)} \right\}.$$

On the other hand, set  $S(x_1) = \min_{x \in \overline{\Omega}} S(x)$ . Then an application of the maximum principle [40, Proposition 2.2] implies that  $\Lambda(x_1) - S(x_1) - \beta(x_1)S(x_1)I(x_1) + \gamma(x_1)I(x_1) \leq 0$ , equivalently,

$$\frac{\Lambda(x_1) + \gamma(x_1)I(x_1)}{1 + \beta(x_1)I(x_1)} \leq S(x_1).$$

Obviously, there exists a positive constant  $c_*$ , independent of  $d_S, d_I > 0$ , such that

$$c_* \leq \frac{\Lambda(x_1) + \gamma(x_1)I(x_1)}{1 + \beta(x_1)I(x_1)}.$$

Hence, for any  $d_S, d_I > 0$ , it holds

$$(3.6) \quad c_* \leq S(x) \quad \forall x \in \bar{\Omega}.$$

Integrating both PDEs of (3.1) over  $\Omega$  yields

$$\int_{\Omega} \{\Lambda(x) - S - \beta(x)SI + \gamma(x)I\} dx = 0, \quad \int_{\Omega} \{\beta(x)SI - [\gamma(x) + \mu(x)]I\} dx = 0,$$

from which it immediately follows that

$$(3.7) \quad \mu_* \int_{\Omega} Idx \leq \int_{\Omega} \mu Idx + \int_{\Omega} Sdx = \int_{\Omega} \Lambda dx \leq |\Omega|\Lambda^*$$

and

$$(3.8) \quad \beta_* \int_{\Omega} SIdx \leq \int_{\Omega} \beta SIdx \leq (\gamma^* + \mu^*) \int_{\Omega} Idx \leq \frac{|\Omega|\Lambda^*(\gamma^* + \mu^*)}{\mu_*}.$$

We now write the  $I$ -equation as

$$(3.9) \quad -\Delta I = \frac{1}{d_I} [\beta S - (\gamma + \mu)] I, \quad x \in \Omega; \quad \frac{\partial I}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

According to the Harnack-type inequality (see, e.g., [37] or [48, Lemma 2.2]), (3.5) and (3.7), we are led to

$$(3.10) \quad \max_{\bar{\Omega}} I \leq C \min_{\bar{\Omega}} I \leq C \frac{1}{|\Omega|} \int_{\Omega} Idx \leq C.$$

Hereafter,  $C$  represents a positive constant independent of small  $d_S > 0$  which may vary from place to place.

*Step 2: Convergence of  $I$ .* Recall that  $I$  satisfies (3.9). By (3.5) and (3.10), we have

$$\left\| \frac{1}{d_I} [\beta S - (\gamma + \mu)] I \right\|_{L^p(\Omega)} \leq C \quad \forall p > 1.$$

From the standard  $L^p$ -estimate for elliptic equations (see, e.g., [21]), it follows that  $\|I\|_{W^{2,p}(\Omega)} \leq C$  for any given  $p > 1$ . Taking  $p$  to be sufficiently large, we see from the Sobolev embedding that  $\|I\|_{C^{1+\alpha}(\bar{\Omega})} \leq C$  for some  $0 < \alpha < 1$ . As a result, there exists a subsequence of  $d_S \rightarrow 0$ , say  $d_n := d_{S,n}$ , satisfying  $d_n \rightarrow 0$  as  $n \rightarrow \infty$ , and a corresponding positive solution  $(S_n, I_n)$  of (3.1) with  $d_S = d_n$ , such that

$$(3.11) \quad I_n \rightarrow \underline{I} \quad \text{uniformly on } \bar{\Omega} \quad \text{as } n \rightarrow \infty,$$

where  $0 \leq \underline{I} \in C^1(\bar{\Omega})$ . In view of (3.10),

$$(3.12) \quad \text{either } \underline{I} \equiv 0 \text{ on } \bar{\Omega} \text{ or } \underline{I} > 0 \text{ on } \bar{\Omega}.$$

Suppose the former holds in (3.12), that is,

$$(3.13) \quad I_n \rightarrow 0 \quad \text{uniformly on } \overline{\Omega} \quad \text{as } n \rightarrow \infty.$$

Then for sufficiently small  $\epsilon > 0$ , we have  $0 \leq I_n(x) \leq \epsilon \forall x \in \overline{\Omega}$  for all large  $n$ . This fact, together with the first equation of (3.1), implies that for all large  $n$ ,  $S_n$  satisfies

$$-d_n \Delta S_n \leq \Lambda - S_n + \gamma^* \epsilon, \quad x \in \Omega; \quad \frac{\partial S_n}{\partial \nu} = 0, \quad x \in \partial\Omega$$

and

$$-d_n \Delta S_n \geq \Lambda - S_n - \beta^* \epsilon S_n, \quad x \in \Omega; \quad \frac{\partial S_n}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

We consider the following two auxiliary problems:

$$(3.14) \quad -d_n \Delta u = \Lambda - u + \gamma^* \epsilon, \quad x \in \Omega; \quad \frac{\partial u}{\partial \nu} = 0, \quad x \in \partial\Omega,$$

and

$$(3.15) \quad -d_n \Delta v = \Lambda - v - \beta^* \epsilon v, \quad x \in \Omega; \quad \frac{\partial v}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

It is clear that systems (3.14) and (3.15) admit a unique positive solution, denoted by  $u_n$  and  $v_n$ , respectively. A simple subsupersolution argument, combined with the uniqueness, guarantees that  $v_n \leq S_n \leq u_n$  on  $\overline{\Omega}$  for all large  $n$ . Using a singular perturbation argument as in [17, Lemma 2.4], it can be shown that

$$u_n \rightarrow \Lambda + \gamma^* \epsilon, \quad v_n \rightarrow \frac{\Lambda}{1 + \beta^* \epsilon} \quad \text{uniformly on } \overline{\Omega} \quad \text{as } n \rightarrow \infty.$$

Sending  $n \rightarrow \infty$ , we find

$$\frac{\Lambda(x)}{1 + \beta^* \epsilon} \leq \liminf_{n \rightarrow \infty} S_n(x) \leq \limsup_{n \rightarrow \infty} S_n(x) \leq \Lambda(x) + \gamma^* \epsilon.$$

Thanks to the arbitrariness of small  $\epsilon > 0$ , we obtain that

$$(3.16) \quad S_n \rightarrow \Lambda \quad \text{uniformly on } \overline{\Omega} \quad \text{as } n \rightarrow \infty.$$

Observe that  $I_n$  fulfills

$$(3.17) \quad -d_I \Delta I_n = \beta(x) S_n I_n - (\gamma + \mu) I_n, \quad x \in \Omega; \quad \frac{\partial I_n}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

Define  $\tilde{I}_n := \frac{I_n}{\|I_n\|_{L^\infty(\Omega)}}$ . Then  $\|\tilde{I}_n\|_{L^\infty(\Omega)} = 1$  for all  $n \geq 1$ , and  $\tilde{I}_n$  solves

$$(3.18) \quad -d_I \Delta \tilde{I}_n = [\beta(x) S_n - (\gamma + \mu)] \tilde{I}_n, \quad x \in \Omega; \quad \frac{\partial \tilde{I}_n}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

As before, through a standard compactness argument for elliptic equations, after passing to a further subsequence, if necessary, we may assume that

$$\tilde{I}_n \rightarrow \tilde{I} \quad \text{in } C^1(\overline{\Omega}) \quad \text{as } n \rightarrow \infty,$$

where  $0 \leq \tilde{I} \in C^1(\overline{\Omega})$  with  $\|\tilde{I}\|_{L^\infty(\Omega)} = 1$ . By (3.16) and (3.18),  $\tilde{I}$  satisfies

$$(3.19) \quad -d_I \Delta \tilde{I} = [\beta\Lambda - (\gamma + \mu)] \tilde{I}, \quad x \in \Omega; \quad \frac{\partial \tilde{I}}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

The Harnack-type inequality (see, [37] or [48, Lemma 2.2]) applied to (3.19) yields  $\tilde{I} > 0$  on  $\overline{\Omega}$ . However, the positiveness of  $\tilde{I}$  indicates that the principal eigenvalue  $\lambda_0$  of the eigenvalue problem (3.2) must be zero (with  $\tilde{I}$  being a corresponding eigenfunction), contradicting our assumption that  $\lambda_0 < 0$ . Thus, (3.13) cannot occur, and we must have  $\underline{I} > 0$  on  $\overline{\Omega}$ . That is,

$$(3.20) \quad I_n \rightarrow \underline{I} > 0 \quad \text{uniformly on } \overline{\Omega} \text{ as } n \rightarrow \infty.$$

*Step 3: Convergence of S.* Notice that  $S_n$  solves

$$(3.21) \quad -d_n \Delta S_n = \Lambda - S_n - \beta S_n I_n + \gamma I_n, \quad x \in \Omega; \quad \frac{\partial S_n}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

In view of (3.20), we see that for any small  $\epsilon > 0$ , it holds that

$$(3.22) \quad 0 < \underline{I}(x) - \epsilon \leq I_n(x) \leq \underline{I}(x) + \epsilon \quad \forall x \in \overline{\Omega}$$

for all large  $n$ . Thus, for all sufficiently large  $n$ , we have

$$\Lambda - S_n - \beta S_n(\underline{I} + \epsilon) + \gamma(\underline{I} - \epsilon) \leq \Lambda - S_n - \beta S_n I_n + \gamma I_n \leq \Lambda - S_n - \beta S_n(\underline{I} - \epsilon) + \gamma(\underline{I} + \epsilon).$$

Given large  $n$ , we consider the following auxiliary problem

$$(3.23) \quad -d_n \Delta w = \Lambda - w - \beta w(\underline{I} + \epsilon) + \gamma(\underline{I} - \epsilon), \quad x \in \Omega; \quad \frac{\partial w}{\partial \nu} = 0, \quad x \in \partial\Omega.$$

It is clear that (3.23) admits a unique positive solution, denoted by  $\overline{w}_n$ . By similar arguments to those in the proof of [17, Lemma 2.4]), we notice that

$$\overline{w}_n \rightarrow \frac{\Lambda + \gamma(\underline{I} - \epsilon)}{1 + \beta(\underline{I} + \epsilon)} \quad \text{uniformly on } \overline{\Omega} \text{ as } n \rightarrow \infty.$$

Since  $S_n$  is an upper solution of (3.23), it then follows that

$$(3.24) \quad \liminf_{n \rightarrow \infty} S_n(x) \geq \lim_{n \rightarrow \infty} \overline{w}_n(x) = \frac{\Lambda(x) + \gamma(x)(\underline{I}(x) - \epsilon)}{1 + \beta(x)(\underline{I}(x) + \epsilon)} \quad \text{uniformly on } \overline{\Omega}.$$

Similarly, one can further show that

$$(3.25) \quad \limsup_{n \rightarrow \infty} S_n(x) \leq \frac{\Lambda(x) + \gamma(x)(\underline{I}(x) + \epsilon)}{1 + \beta(x)(\underline{I}(x) - \epsilon)} \quad \text{uniformly on } \overline{\Omega}.$$

In view of (3.24) and (3.25), combined with the arbitrariness of small  $\epsilon > 0$ , we have

$$\lim_{n \rightarrow \infty} S_n(x) = \underline{S}(x) := \frac{\Lambda(x) + \gamma(x)\underline{I}(x)}{1 + \beta(x)\underline{I}(x)} \quad \text{uniformly on } \overline{\Omega}.$$

Due to (3.17), it can be easily seen that  $\underline{I}$  satisfies (3.3). The proof is complete.  $\square$

**3.2. The case  $d_I \rightarrow 0$ .** This subsection is devoted to the investigation of the asymptotic behavior of positive solutions of (3.1) with  $d_S > 0$  being fixed and  $d_I \rightarrow 0$ . Because of mathematical difficulty, we can only deal with one space dimension case, that is, the habitat of  $\Omega$  is an interval. Without loss of generality, we take  $\Omega = (0, 1)$ .

In light of 2.2(a) and 2.4, we assume that  $\{\beta(x)\tilde{S}(x) > \gamma(x) + \mu(x) : x \in [0, 1]\}$  is nonempty so that  $\mathcal{R}_0 > 1$  and thus (3.1) admits positive solutions for all small  $d_I > 0$ . Our main result reads as follows.

**THEOREM 3.2.** *Assume that the set  $\{x \in [0, 1] : \beta(x)\tilde{S}(x) > \gamma(x) + \mu(x)\}$  is nonempty. Fix  $d_S > 0$  and let  $d_I \rightarrow 0$ , then every positive solution  $(S, I)$  of (3.1) satisfies (up to a subsequence of  $d_I$ ) that  $S \rightarrow S_0$  uniformly on  $[0, 1]$ , where  $S_0 \in C([0, 1])$  and  $S_0 > 0$  on  $[0, 1]$ , and  $\int_0^1 I dx \rightarrow I_0$  for some positive constant  $I_0$ .*

*Proof.* Notice that (3.5), (3.6), (3.7), and (3.8) remain true in the current situation. Since the spatial domain is one dimensional and  $S$  satisfies

$$(3.26) \quad -d_S S''(x) + S(x) = \Lambda - \beta S(x)I(x) + \gamma I(x), \quad x \in (0, 1); \quad S'(0) = S'(1) = 0,$$

we deduce from the elliptic  $L^1$ -theory in [9] that, for any  $p > 1$ ,  $\|S\|_{W^{1,p}(0,1)} \leq C$ , where  $C$  is a positive constant independent of  $d_I$  but is allowed to vary below. Then for sufficiently large  $p$ , the Sobolev embedding theorem guarantees that  $\|S\|_{C^\alpha([0,1])} \leq C$  for some  $\alpha \in (0, 1)$ . Moreover, up to a sequence of  $d_I \rightarrow 0$ , say  $d_n := d_{I,n} \rightarrow 0$  with  $d_n \rightarrow 0$  as  $n \rightarrow \infty$ , the corresponding positive solution sequence  $(S_n, I_n)$  of (3.1) with  $d_I = d_n$  satisfies  $S_n \rightarrow S_0 > 0$  in  $C([0, 1])$  as  $n \rightarrow \infty$  due to (3.6).

In light of (3.7), by passing a subsequence of  $d_n$  if necessary, we may assume that  $\int_0^1 I_n dx \rightarrow I_0$  as  $n \rightarrow \infty$  for some nonnegative constant  $I_0$ . To show  $I_0 > 0$ , we proceed indirectly and suppose that  $I_0 = 0$ . By integrating (3.26) from 0 to  $x$ , we have

$$S'_n(x) = -\frac{1}{d_S} \int_0^x \{\Lambda(y) - S_n(y) - \beta(y)S_n(y)I_n(y) + \gamma(y)I_n(y)\} dy \quad \forall x \in [0, 1].$$

By sending  $n \rightarrow \infty$  and using  $\int_0^1 I_n dx \rightarrow 0$ , it then follows

$$S'_n(x) \rightarrow -\frac{1}{d_S} \int_0^x [\Lambda(y) - S_0(y)] dy \quad \text{uniformly on } [0, 1].$$

As  $S_n(x) - S_n(0) = \int_0^x S'_n(y) dy$  for any  $n \geq 1$ , we find that  $S_0$  solves

$$S_0(x) - S_0(0) = -\frac{1}{d_S} \int_0^x \left\{ \int_0^y [\Lambda(z) - S_0(z)] dz \right\} dy,$$

which in turn implies that

$$(3.27) \quad -d_S S_0''(x) = \Lambda(x) - S_0(x), \quad x \in (0, 1); \quad S_0'(0) = 0.$$

When integrating (3.26) from  $x$  to 1, one can use an analysis similar to the above to know that  $S_0'(1) = 0$ . Therefore, this and (3.27) give that  $S_0 = \tilde{S}$ , that is,  $S_n \rightarrow \tilde{S}$  uniformly on  $[0, 1]$  as  $n \rightarrow \infty$ .

On the other hand, observe that

$$\lambda_1(d_n, \gamma(x) + \mu(x) - \beta(x)S_n(x)) = 0 \quad \forall n \geq 1,$$

where  $\lambda_1(d_n, \gamma(x) + \mu(x) - \beta(x)S_n(x))$  stands for the principal eigenvalue of the following eigenvalue problem:

$$d_n \Delta u + [\beta(x)S_n(x) - \gamma(x) - \mu(x)]u + \lambda u = 0, \quad x \in (0, 1); \quad u'(0) = u'(1) = 0.$$

Combined with the fact that the principal eigenvalue continuously depends on the parameters, the argument as in [4, Lemma 2.3] yields

$$0 = \lambda_1(d_n, \gamma(x) + \mu(x) - \beta(x)S_n(x)) \rightarrow \min_{x \in [0,1]} \{\gamma(x) + \mu(x) - \beta(x)\tilde{S}(x)\} \quad \text{as } n \rightarrow \infty,$$

contradicting our assumption  $\min_{x \in [0,1]} \{\gamma(x) + \mu(x) - \beta(x)\tilde{S}(x)\} < 0$ . Thus, it is necessary that  $I_0 > 0$ . The proof is complete.  $\square$

#### 4. Summary and discussion.

**4.1. Summary of analytical results.** In this paper, we are concerned with the SIS epidemic model (1.3) with a mass action infection mechanism and linear source. To study the parabolic problem (1.3), our first step is to establish the global existence and uniform boundedness of solutions. Then a basic reproduction number  $\mathcal{R}_0$  is defined via a variational characterization, which determines the local stability of the unique DFE. When the environment is spatially homogeneous and the diffusion rates of the susceptible and infected are equal, by constructing suitable Lyapunov functionals, we further prove the global attractivity of the DFE for  $\mathcal{R}_0 \leq 1$  and that of the EE for  $\mathcal{R}_0 > 1$ . We are mainly interested in the asymptotic behavior of positive steady states  $(S, I)$  of problem (1.3), which exist provided  $\mathcal{R}_0 > 1$  in a general heterogeneous environment, as the diffusion rates of the susceptible or the infected tend to zero. For fixed  $d_I > 0$ , Theorem 3.1 shows that the limiting functions of both  $S$  and  $I$  as  $d_S \rightarrow 0$ , are positive throughout the habitat. In the one dimensional interval, say  $[0, 1]$ , for fixed  $d_S > 0$ , Theorem 3.2 indicates that the limiting function of  $S$  as  $d_I \rightarrow 0$  is positive in  $[0, 1]$  while the total infected population tends to a positive constant.

Since there are four principle models (1.2), (1.3), (1.4), and (1.5) to model the SIS epidemic dynamics based on different infection mechanisms and modeling ideas, it will be helpful to summarize their results and make a comparison so as to understand the influence of the factors such as infection mechanism, movement rate, and source term on the eradication of epidemics. Numerical simulations will be performed to validate theoretical results and to predict possible outcomes for those cases that remain unknown analytically. Then we discuss the implication of these theoretical and numerical findings from the disease control viewpoint. Since the results of the model (1.3) have been summarized above, below we shall briefly recall the results for the SIS models (1.2), (1.4), and (1.5) obtained in the literature.

**4.1.1. Results on (1.4).** The steady state problem corresponding to (1.4) satisfies

$$(4.1) \quad \begin{cases} -d_S \Delta S = -\beta(x) \frac{SI}{S+I} + \gamma(x)I, & x \in \Omega, \\ -d_I \Delta I = \beta(x) \frac{SI}{S+I} - \gamma(x)I, & x \in \Omega, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, \\ \int_{\Omega} [S(x) + I(x)] dx = N. \end{cases}$$

Hereafter,  $N$  is a fixed positive constant, representing the total number of the susceptible and infected populations. That is  $N = \int_{\Omega} (S(x) + I(x)) dx$  is a constant.

As in [4, 49], we introduce the notion of low/high/moderate risk site/domain. We say that  $x$  is a *low* (or *high* or *moderate*)-*risk site* if the local disease transmission rate  $\beta(x)$  is lower than (or higher than or equal to) the local disease recovery rate  $\gamma(x)$ . Let

$$H^- = \{x \in \Omega : \beta(x) < \gamma(x)\} \quad \text{and} \quad H^+ = \{x \in \Omega : \beta(x) > \gamma(x)\}$$

denote the set of low-risk sites and high-risk sites, respectively.

Assume that both  $H^-$  and  $H^+$  are nonempty. The authors in [4] defined the basic reproduction number

$$\hat{\mathcal{R}}_0 = \sup_{\varphi \in H^1(\Omega)} \frac{\int_{\Omega} \beta \varphi^2 dx}{\int_{\Omega} (d_I |\nabla \varphi|^2 + \gamma \varphi^2) dx}$$

and showed that the unique DFE  $(N/|\Omega|, 0)$  is globally stable if  $\hat{\mathcal{R}}_0 < 1$ , while it is unstable and a unique EE exists if  $\hat{\mathcal{R}}_0 > 1$ . Indeed, following the argument similar to [12], one can show that the uniform persistence property holds once  $\hat{\mathcal{R}}_0 > 1$ .

The asymptotic profile of the EE was also investigated in [4] when the diffusivity of the susceptible individuals tends to zero. In particular, the result of [4] shows that as  $d_S \rightarrow 0$ , the unique positive solution  $(S, I)$  (which exists if  $\hat{\mathcal{R}}_0 > 1$ ) of (4.1) fulfills  $(S, I) \rightarrow (\hat{S}, 0)$  uniformly on  $\bar{\Omega}$ , where  $\hat{S}$  satisfies a free boundary problem, is positive at all low-risk sites, and is also positive at some (but not all) high-risk sites. This result indicates that it may be possible to entirely eliminate the infectious disease by restricting the motility rate of the susceptible to be small.

Further asymptotics of the EE in other cases were obtained by Peng [46] wherein it was shown that if  $d_I \rightarrow 0$  and  $d := d_I/d_S \rightarrow d_0 \in [0, \infty]$ , then the unique positive solution  $(S, I)$  of (4.1) satisfies the following:

- If  $d_0 = 0$ , then

$$S \rightarrow \frac{N}{\int_{\Omega} [1 + (\beta - \gamma)_+ \gamma^{-1}]} \quad \text{and} \quad I \rightarrow \frac{N(\beta - \gamma)_+ \gamma^{-1}}{\int_{\Omega} [1 + (\beta - \gamma)_+ \gamma^{-1}]}$$

uniformly on  $\bar{\Omega}$ . In what follows,  $(s)_+ = \max\{s, 0\}$ .

- If  $d_0 \in (0, \infty)$ , then

$$S \rightarrow \frac{Nd_0 [1 - A(d_0; x)]}{\int_{\Omega} [A(d_0; x) + d_0(1 - A(d_0; x))]}, \quad I \rightarrow \frac{NA(d_0; x)}{\int_{\Omega} [A(d_0; x) + d_0(1 - A(d_0; x))]}$$

uniformly on  $\bar{\Omega}$ , where  $A(d_0; x) = \frac{d_0(\beta - \gamma)_+}{d_0(\beta - \gamma)_+ + \gamma}$ .

- If  $d_0 = \infty$ , then  $I \rightarrow 0$  uniformly on  $\bar{\Omega}$ , and  $S \rightarrow \frac{N[1 - A(\infty; x)]}{\int_{\Omega} [1 - A(\infty; x)]}$  uniformly on any compact subset of  $H^-$  and  $H^+$ , respectively, where

$$A(\infty; x) = \begin{cases} 0 & \text{if } x \in \overline{H^-}, \\ 1 & \text{if } x \in H^+. \end{cases}$$

Clearly the limiting function of  $I$  when  $d_I \rightarrow 0$  and  $d \rightarrow d_0 \in [0, \infty)$  is positive on  $H^+$  while zero on  $\overline{H^-}$ . In particular, if  $d_I \rightarrow 0$  and  $d_S > 0$  is fixed, we are in the first scenario above. Thus, for model (1.4), we may conclude that the optimal strategy of eliminating the infectious disease is to restrict the motility rate of the susceptible population, while restricting the motility of infected population can only eradicate the disease in low-risk and moderate-risk sites. Of course, another strategy is to set  $d_I \rightarrow 0$  and  $d_S \rightarrow 0$  while the susceptible moves relatively slower than the infected.



**4.1.2. Result on (1.5).** Now we consider the scenario that the susceptible individuals are allowed to have birth and death, and look at the SIS reaction-diffusion system (1.5) with a linear external source. One of the main results in [36] states that (1.5) admits at least one EE  $(S, I)$  if  $\tilde{\mathcal{R}}_0 > 1$ , which is in fact a positive steady state of (1.5) satisfying

$$(4.2) \quad \begin{cases} -d_S \Delta S = \Lambda(x) - S - \beta(x) \frac{SI}{S+I} + \gamma(x)I, & x \in \Omega, \\ -d_I \Delta I = \beta(x) \frac{SI}{S+I} - \gamma(x)I, & x \in \Omega, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega. \end{cases}$$

Moreover, it was proved in [36] that

- as  $d_S \rightarrow 0$ , both limiting functions of  $S$  and  $I$  are inhomogeneous and positive on the entire habitat  $\bar{\Omega}$ ;
- as  $d_I \rightarrow 0$ , the limiting function of  $S$  is positive on the entire habitat  $\bar{\Omega}$  and that of  $I$  is positive only on high-risk sites.

**4.1.3. Result on (1.2).** In [14, 57, 56], the authors treated the SIS system (1.2) with mass action and its steady state problem:

$$(4.3) \quad \begin{cases} -d_S \Delta S = -\beta(x)SI + \gamma(x)I, & x \in \Omega, \\ -d_I \Delta I = \beta(x)SI - \gamma(x)I, & x \in \Omega, \\ \frac{\partial S}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0, & x \in \partial\Omega, \\ \int_{\Omega} [S(x) + I(x)] dx = N. \end{cases}$$

For the mass action system (1.2), the basic reproduction number depends on the total population size  $N$  and is defined as

$$\tilde{\mathcal{R}}_0 = \sup_{0 \neq \varphi \in H^1(\Omega)} \frac{(N/|\Omega|) \int_{\Omega} \beta \varphi^2}{\int_{\Omega} (d_I |\nabla \varphi|^2 + \gamma \varphi^2)} = \frac{N}{|\Omega|} \hat{\mathcal{R}}_0.$$

It is shown that a positive solution  $(S, I)$  of (4.3) exists whenever  $\tilde{\mathcal{R}}_0 > 1$ . Indeed, following an argument similar to [12], one can show the uniform persistence property holds once  $\tilde{\mathcal{R}}_0 > 1$ . Moreover, one can show that  $\tilde{\mathcal{R}}_0 > 1$  when  $N > \int_{\Omega} \frac{\gamma(x)}{\beta(x)} dx$ , and  $\tilde{\mathcal{R}}_0 > 1$  is also possible when  $N \leq \int_{\Omega} \frac{\gamma(x)}{\beta(x)} dx$  depending on the parameters  $\beta, \gamma$ , and  $d_I$ . Furthermore, for fixed  $d_I > 0$ , the following asymptotics as  $d_S \rightarrow 0$  have been shown in [56, 57]:

- If either  $N - \int_{\Omega} \frac{\gamma}{\beta} > \frac{1}{4} \int_{\Omega} \frac{|\nabla \beta|^2}{\beta^3}$  or  $\frac{N}{|\Omega|} > \frac{\gamma}{\beta}$  on  $\bar{\Omega}$ , then

$$(S, I) \rightarrow \left( \frac{\gamma(x)}{\beta(x)}, \frac{N}{|\Omega|} - \frac{1}{|\Omega|} \int_{\Omega} \frac{\gamma(x)}{\beta(x)} dx \right) \quad \text{uniformly on } \bar{\Omega}.$$

- If  $N \leq \int_{\Omega} \frac{\gamma(x)}{\beta(x)} dx$ , then  $(S, I) \rightarrow (S_*, 0)$  uniformly on  $\bar{\Omega}$ , where  $S_*$  is a positive function.

Under the assumption that  $\Omega^+ = \{x \in \Omega : \frac{N}{|\Omega|} \beta(x) - \gamma(x) > 0\}$  is nonempty, Wu and Zou [57] further proved the following:

- If  $d_I \rightarrow 0$  and  $d_I/d_S \rightarrow d \in (0, \infty)$ , then  $(S, I) \rightarrow (S_{**}, I_{**})$  uniformly on  $\bar{\Omega}$  and  $I_{**}$  is the unique nonnegative solution of

$$\left\{ \frac{N}{|\Omega|} \beta - \gamma - \frac{(1-d)\beta}{|\Omega|} \int_{\Omega} I_{**} \right\}_+ - d\beta I_{**} = 0,$$

and

$$S_{**} = \frac{N}{|\Omega|} - \frac{(1-d)}{|\Omega|} \int_{\Omega} I_{**} - dI_{**}.$$

Therefore, the distribution of  $I_{**}$  depends critically on the magnitude of  $d$ . In fact, if  $d \in (0, 1)$ , then  $\{x \in \Omega : I_{**}(x) > 0\}$  is a proper subset of  $\Omega^+$ ; if  $d \in (1, \infty)$ , then  $\Omega^+$  is a subset of  $\{x \in \Omega : I_{**}(x) > 0\}$ ; if  $d = 1$ , then

$$S_{**} = \frac{N}{|\Omega|} - \left( \frac{N}{|\Omega|} - \frac{\gamma}{\beta} \right)_+ \quad \text{and} \quad I_{**} = \left( \frac{N}{|\Omega|} - \frac{\gamma}{\beta} \right)_+.$$

On the other hand, in the case of a one dimensional domain, say  $\Omega = (0, 1)$ , if  $\gamma < N\beta$  on  $[0, 1]$ , then for fixed  $d_S > 0$ , as  $d_I \rightarrow 0$ , the authors of [56] proved that any EE  $(S, I)$  satisfies  $S \rightarrow \hat{S}$  uniformly on  $[0, 1]$  with a positive function  $\hat{S}$  and  $\int_0^1 I dx$  converges to a positive constant. Biologically, this implies that the infectious disease still persists when the movement of the infected population is small.

## 4.2. Discussion and conclusions.

**4.2.1. Comparison of the basic reproduction number.** For readability, hereafter we call models (1.2), (1.3), (1.4), and (1.5) and their corresponding EE problem (when no confusion is caused) as MO, MW, SO, and SW, respectively, in order that each label of models can bear a meaning (see Table 1). For convenience, we also list the basic reproduction number for each of the models MO, MW, SO, SW in Table 1, where three observations are worth mentioning as follows.

(a) MO is the only one whose basic reproduction number depends on  $N$  via  $N/|\Omega|$  which measures the total population per unit space. This implies that the total population plays a role in the eradication of diseases only for MO, and also explains why a disease is easier to become endemic in a more crowded population than a sparse population as mentioned in [57]. (b) If the birth-death effect is considered, then MO becomes MW whose basic reproduction number no longer depends on total population  $N$ . This indicates that the birth and death effects could be an important factor for the eradication of diseases in SIS models with a mass-action infection mechanism. However, the birth-death effect is not important for models SO and SW anymore, since both have the same basic reproduction number. (c) MW is the only model whose basic reproduction number depends (implicitly) on the diffusivity  $d_S$  of the susceptibles.

TABLE 1

Basic reproduction numbers for SIS epidemic models, where  $\tilde{S}$  in the basic reproduction number for MW is the unique solution of (2.6).

Model	Infection mechanisms	Basic reproduction number
MO=(1.2)	Mass-action incidence without birth-death	$\tilde{\mathcal{R}}_0 = \frac{N}{ \Omega } \sup_{0 \neq \varphi \in H^1(\Omega)} \frac{\int_{\Omega} \beta \varphi^2}{\int_{\Omega} (d_I  \nabla \varphi ^2 + \gamma \varphi^2)}$
MW=(1.3)	Mass-action incidence with birth-death	$\mathcal{R}_0 = \sup_{0 \neq \varphi \in H^1(\Omega)} \frac{\int_{\Omega} \beta \tilde{S} \varphi^2}{\int_{\Omega} (d_I  \nabla \varphi ^2 + (\gamma + \mu) \varphi^2)}$
SO=(1.4)	Standard incidence without birth-death	$\tilde{\mathcal{R}}_0 = \sup_{0 \neq \varphi \in H^1(\Omega)} \frac{\int_{\Omega} \beta \varphi^2}{\int_{\Omega} (d_I  \nabla \varphi ^2 + \gamma \varphi^2)}$
SW=(1.5)	Standard incidence with birth-death	$\hat{\mathcal{R}}_0 = \sup_{0 \neq \varphi \in H^1(\Omega)} \frac{\int_{\Omega} \beta \varphi^2}{\int_{\Omega} (d_I  \nabla \varphi ^2 + \gamma \varphi^2)}$

TABLE 2  
Asymptotic behavior of  $(S(x), I(x))$  as  $d_S \rightarrow 0$  or  $d_I \rightarrow 0$ .

Model	Limit of $(S(x), I(x))$ as $d_S \rightarrow 0$	Limit of $(S(x), I(x))$ as $d_I \rightarrow 0$
MO	$S^*(x) > 0$ and $I^*(x) \equiv 0$ (or $> 0$ ) for small (or large) $N$	$S^*(x) > 0$ and $\int_{\Omega} I^*(x) > 0$
MW	$S^*(x) > 0$ and $I^*(x) > 0$	$S^*(x) > 0$ and $\int_{\Omega} I^*(x) > 0$
SO	$S^*(x) \geq 0$ and $I^*(x) \equiv 0$	$S^*(x) > 0$ and $I^*(x) \equiv 0$ iff $x \in \overline{H^-}$
SW	$S^*(x) > 0$ and $I^*(x) > 0$	$S^*(x) > 0$ and $I^*(x) \equiv 0$ iff $x \in \overline{H^-}$

TABLE 3  
Asymptotic behavior of  $(S(x), I(x))$  as both  $d_S \rightarrow 0$  and  $d_I \rightarrow 0$ .

Model	Limit of $(S(x), I(x))$ as $d_S \rightarrow 0$ and $d_I \rightarrow 0$
MO	For the case $d_I/d_S \rightarrow d \in (0, \infty)$ , $S^*(x) > 0$ , and $I^*(x) \geq 0$ but $I^*(x) \not\equiv 0$
MW	Unknown
SO	$S^*(x) > 0$ and $I^*(x) \equiv 0$ iff $x \in \overline{H^-}$ when $d_I/d_S \rightarrow [0, \infty)$ , $S^*(x) \geq 0$ , and $I^*(x) \equiv 0$ when $d_I/d_S \rightarrow \infty$
SW	Unknown

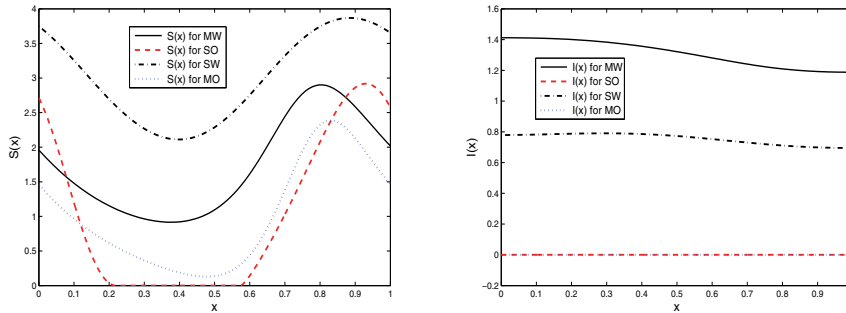


FIG. 1. Numerical simulations of the profile of  $(S(x), I(x))$  as  $d_S \rightarrow 0$  for systems MO, MW, SO, and SW, where parameters are chosen as  $d_S = 10^{-6}$ ,  $d_I = 1$ ,  $\Lambda(x) = 3$ ,  $\mu(x) = 0.5 + x$ , and  $\beta(x)$ , and  $\gamma(x)$  are as plotted in Figure 4(a).

**4.2.2. Asymptotic behavior of EE.** From the disease control point of view, one is mainly concerned with whether the infectious disease can be eradicated (namely, whether  $I(x)$  can go extinct either throughout the entire domain  $\Omega$  or partially). One of the strategies as recalled above is to control the motility of susceptible and/or infected populations. Below in Tables 2 and 3 we capsulize the asymptotic behavior of EE  $(S(x), I(x))$  as  $d_S \rightarrow 0$  or  $d_I \rightarrow 0$  or both. Furthermore we use numerical simulations to illustrate known results and predict possible outcomes for unknown cases. In the following, we shall use  $(S^*, I^*)$  to represent the asymptotic behavior of EE for all models for simplicity. We remark that the parameter values chosen in all simulations are sufficient to guarantee the existence of EE in models under consideration. For example, in Figure 1, for any  $d_S > 0$  and  $d_I > 0$ ,  $\hat{\mathcal{R}}_0 = \tilde{\mathcal{R}}_0 > \int_0^1 \beta(x)dx / \int_0^1 \gamma(x)dx = 1.5/1.2 > 1$  and  $\mathcal{R}_0 > \int_0^1 \beta(x)\tilde{S}(x)dx / \int_0^1 [\gamma(x) + \mu(x)]dx = 4.5/2.2 > 1$ .

When the movement rate  $d_S$  of the susceptibles tends to zero, the asymptotics of solutions have been well understood to a large extent as seen in Table 2, and the asymptotic profiles of EE illustrated in Figure 1 are consistent with analytical results.

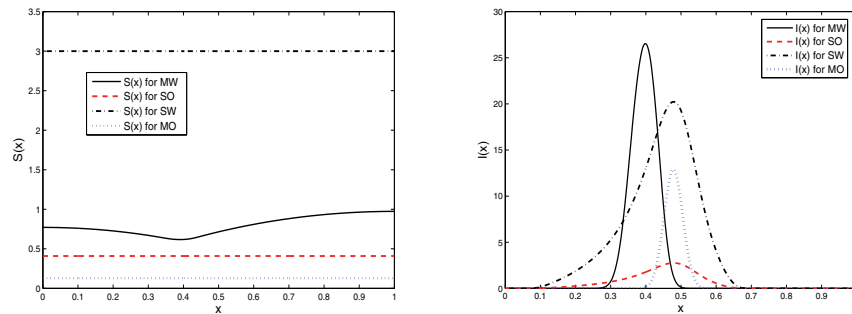


FIG. 2. Numerical simulations of the profile of  $(S(x), I(x))$  as  $d_I \rightarrow 0$  for systems MO, MW, SO, and SW, where  $d_S = 1, d_I = 10^{-5}$ , and other parameters are chosen the same as those in Figure 1.

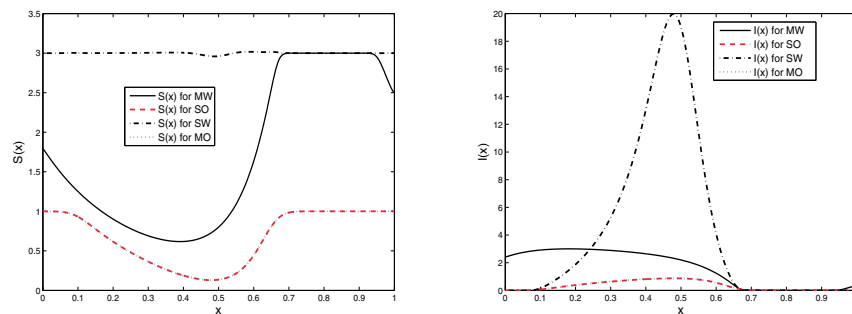


FIG. 3. Numerical simulations of the profile of  $(S(x), I(x))$  as  $d_S, d_I \rightarrow 0$  for systems MO, MW, SO, and SW, where  $d_S = d_I = 10^{-5}$  and other parameters are chosen the same as those in Figure 1.

It is worth mentioning that for MO, since the parameter values are taken so that  $1 = N < \int_0^1 \frac{\gamma(x)}{\beta(x)} dx$ , we have the convergence  $I \rightarrow 0$  as  $d_S \rightarrow 0$  according to the results of [57] which our numerical simulations fit well.

With the same parameter values as in Figure 1, we illustrate the asymptotic profiles of EE as  $d_I \rightarrow 0$  in Figure 2. For the two standard incidence infection models SO and SW, our simulations show that the limiting profile of  $I$  for both models is positive only at high-risk sites which match well with the analytical results. The limiting profile of  $S$  for model SW is constant because of the special choice of  $\Lambda$  (see [36, Theorem 5.2]). For models MW and MO, the exact limiting behavior of  $I(x)$  remains open except knowing that its total population is positive (see Table 2). Our numerical simulations in Figure 2 demonstrate that the infectious disease tends to aggregate in a narrow region and is eradicated outside this region, where model MO has a narrower aggregation region than model MW. We remark that in our simulation the condition  $\gamma < N\beta$  required in [56] is not satisfied on  $[0, 1]$ , and we observe that  $S$  tends to a positive constant though its rigorous proof still remains open.

The asymptotic behavior of EE as  $d_S \rightarrow 0$  and  $d_I \rightarrow 0$  is only partially understood (see results in Table 3). The numerical simulations shown in Figure 3 verify the known results on models SO and MO where the asymptotic profiles of  $(S, I)$  coincide because of our choice of the parameter values. However, the asymptotic behavior of

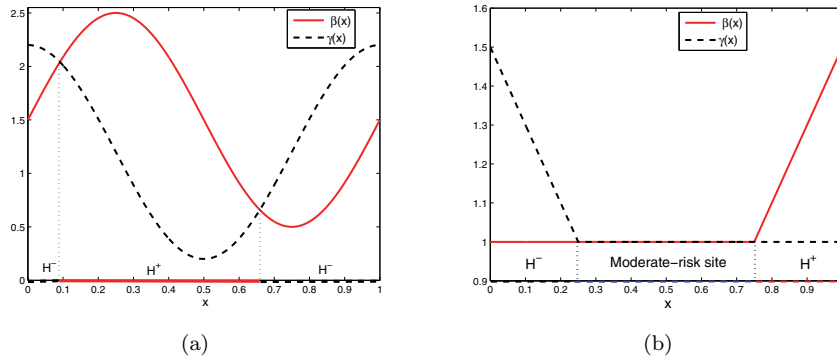


FIG. 4. (a) Graphs of  $\beta(x) = 1.5 + \sin(2\pi x)$ ,  $\gamma(x) = 1.2 + \cos(2\pi x)$  for  $x \in [0, 1]$ , and the set  $H^-$  and  $H^+$  (reproduction of Figure 1 in [4]); (b) graphs of  $\beta(x)$  and  $\gamma(x)$  given by (4.4) with a moderate-risk region in  $[0, 1]$ .

EE as  $d_S \rightarrow 0$  and  $d_I \rightarrow 0$  for models MW and SW entirely remains open and our numerical simulations have the following predictions. First, for MW, the simulation implies that  $S^*(x) > 0$  and  $I^*(x) \geq 0$  but  $I^*(x) \not\equiv 0$  as  $d_S \rightarrow 0$  and  $d_I \rightarrow 0$  with  $d_I/d_S \rightarrow d \in (0, \infty)$ , which is analogous to the asymptotic behavior of EE for MO. In other words, the birth-death effect seems not to be important for SIS models with mass-action infection mechanisms if both diffusion rates of the susceptible and infectious are small with the same order. Second, for model SW, the numerical simulation shows that  $S^*(x)$  is a positive constant and  $I^*(x) \geq 0$ , where  $I^*(x) \equiv 0$  if and only if  $x \in H^-$ . These simulations suggest possible asymptotic behavior of models MW and SW as  $d_S \rightarrow 0$  and  $d_I \rightarrow 0$  for further analytical pursuits.

Finally, to see whether the inclusion of a moderate-risk region will affect the asymptotic profiles of EE as considered in [49], we choose appropriate functions for  $\beta(x)$  and  $\gamma(x)$  as

$$(4.4) \quad \beta(x) = \begin{cases} 1, & x \in [0, 0.75], \\ 2x - 0.5, & x \in [0.75, 1], \end{cases} \quad \gamma(x) = \begin{cases} -2x + 1.5, & x \in [0, 0.25], \\ 1, & x \in [0.25, 1], \end{cases}$$

such that  $\beta(x) = \gamma(x)$  on the interval  $[0.25, 0.75]$  (moderate-risk region) (see a plot in Figure 4(b)), and perform numerical simulations with small  $d_I$ . For model MW, Figure 5(a) indicates that the infected population tends to aggregate on two narrow regions instead of one, compared to the case without a moderate-risk region as illustrated in Figure 2. Moreover, the simulation in Figure 5(b) illustrates that the limiting profile of  $I$  of SO, SW, and MO is positive only at high-risk sites. This is in sharp contrast with Figure 2 where there is no moderate-risk region and the limiting profile of  $I$  for model MO is positive only on a narrow part within the high-risk region.

**4.2.3. Implication on disease control.** We now discuss numerous implications/comments on disease control based on analytical and numerical results summarized in the preceding subsections.

First consider models SO and MO which have conserved total population but are subject to different infection mechanisms. For model SO with any magnitude of total population, it is possible to eliminate the disease entirely by restricting  $d_S$  while the disease cannot be eradicated on high-risk sites by limiting  $d_I$  (see Table 2 and Figures 1 and 2). As for model MO, restricting  $d_S$  can eliminate the disease only if the total

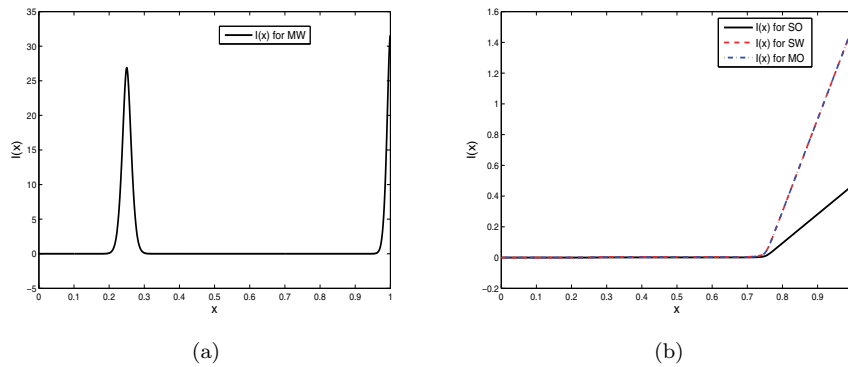


FIG. 5. Numerical simulations of the asymptotic profile of  $I(x)$  as  $d_I \rightarrow 0$  for systems MO, MW, SO, and SW with a moderate-risk site, where  $d_S = 1$ ,  $d_I = 10^{-5}$ ,  $\Lambda(x) = 3$ ,  $\mu(x) = 0.5 + x$ , and  $\beta(x)$  and  $\gamma(x)$  are given by (4.4) as plotted in Figure 4(b).

population is small (see Table 2), whereas the infected individuals tend to aggregate on a narrow region if  $d_I$  is small by the observation from Figure 2. Thus, if the total population remains unchanged, we may conclude that the disease described by the standard incidence infection mechanism modeled by SO is easier to control by limiting the motility  $d_S$  of the susceptible population compared to the mass-action infection mechanism modeled by MO. Nevertheless, the disease subject to mass-action infection mechanism can be eradicated to a larger extent (region) if the motility  $d_I$  of infected individuals is restricted.

Now consider models MW and SW that have the same linear recruitment but different infection mechanisms. From Table 2 and Figures 2 and 3, we see that the infectious disease cannot be eliminated at all by restricting  $d_S$  for either models due to the source term of susceptible population, while restricting  $d_I$  can eliminate the disease partially for both models but the standard incidence infection mechanism seems to be more efficient than the mass-action one.

Let us also consider the effect of linear recruitment on the same infection mechanism, that is, we compare model SO with SW, and MO with MW. Recall that restricting the motility of the susceptible population ( $d_S$  is small) yields the extinction of disease subject to the standard incidence infection mechanism in SO, but this strategy fails for SW with linear recruitment subject to the same infection mechanism. Similar results hold between models MO and MW, but only with small total population. When  $d_I$  is small, the infectious disease modeled by SO and SW is eradicated/persistent at the same region but the latter has a larger total mass, whereas the infectious disease modeled by MW is less condensed compared to its counterpart MO. Thus, if  $d_S$  is small, whichever the infection mechanism is, a varying total population tends to enhance the persistence of disease, while this enhancement induced by standard incidence infection mechanics is not as strong as the mass-action one does. Nevertheless, for small  $d_I$ , the disease subject to mass-action infection mechanism modeled by MO and MW seems to be less endemic since the infected population is more concentrated (see Figure 2).

If the environment is modified to include a moderate-risk region (see Figure 4(b)), then we see that for small  $d_I$ , the disease modeled by SO, SW, and MO can be eradicated precisely at low-risk and moderate-risk sites (see Figure 5(b)). This exhibits quite different behavior than that of model MW for which the infected disease may

also persist in low-risk or moderate-risk sites but can also be eradicated in part of high-risk sites (see Figure 5(a)). Compared to the profiles shown in Figure 2 for the case of small  $d_I$  without a moderate-risk site, from the standing point of disease control, this essentially implies that at least for model MO it is perhaps not a sound strategy to create a moderate-risk domain in the environment and restrict the motility of the infected population at the same time.

We also would like to mention that due to the conservative property of the total population, the steady state problem of SO can be reduced to a single local elliptic equation while that of MO can be reduced to a single nonlocal elliptic equation. Hence, this property makes the corresponding system easier to attack, compared to the case of varying total population. Moreover, it is exactly because of this property that one can consider the asymptotic profiles of the positive solution for small  $d_I$  and  $d_I/d_S \rightarrow d_0$  for some  $d_0$ , as in [46, 57]. This seems to be a rather challenging task for the steady state of models MW and SW due to lack of appropriate a priori estimates.

Finally, it is perhaps worth mentioning that one can also consider the effects of the large motility rate of susceptible or infected populations, as in [35, 36, 46]. In fact, one can easily follow the arguments there and conclude that when the motility of the susceptible population tends to infinity, the density of the susceptibles becomes positive and homogeneous and the density of the infected is also positive but inhomogeneous throughout the habitat; a similar result holds if the movement rate of the infected population becomes large. Since these results are essentially the same as before and they indicate that the large diffusion rate of the susceptibles or infected does not help to eradicate the disease, we do not present these results in this paper.

## REFERENCES

- [1] G. ABRAMSON AND V. M. KENKRE, *Spatiotemporal patterns in hantavirus infection*, Phys. Rev. E (3), 66 (2002), 011912.
- [2] G. ABRAMSON, V. M. KENKRE, T. L. YATES, AND R. R. PARMENTER, *Traveling waves of infection in the hantavirus epidemics*, Bull. Math. Biol., 65 (2003), pp. 519–534.
- [3] N. ALIKAKOS,  *$L^p$  bounds of solutions of reaction-diffusion equation*, Comm. Partial Differential Equations, 4 (1979), pp. 827–868.
- [4] L. J. S. ALLEN, B. M. BOLKER, Y. LOU, AND A. L. NEVAI, *Asymptotic profiles of the steady states for an SIS epidemic reaction-diffusion model*, Discrete Contin. Dyn. Syst., 21 (2008), pp. 1–20.
- [5] H. AMMAN, *Invariant sets and existence theorems for semilinear parabolic and elliptic systems*, J. Math. Anal. Appl., 65 (1978), pp. 432–467.
- [6] R. M. ANDERSON AND R. M. MAY, *Population biology of infectious diseases*, Nature, 280 (1979), pp. 361–367.
- [7] R. M. ANDERSON AND R. M. MAY, *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, Oxford, 1991.
- [8] F. BRAUER AND C. CASTILLO-CHAVEZ, *Mathematical Models in Population Biology and Epidemiology*, Springer, New York, 2000.
- [9] H. BREZIS AND W. A. STRAUSS, *Semi-linear second-order elliptic equations in  $L^1$* , J. Math. Soc. Japan, 25 (1973), pp. 565–590.
- [10] K. J. BROWN, P. C. DUNNE, AND R. A. GARDNER, *A semilinear parabolic system arising in the theory of superconductivity*, J. Differential Equations, 40 (1981), pp. 232–252.
- [11] R. CUI AND Y. LOU, *A spatial SIS model in advective heterogeneous environments*, J. Differential Equations, 261 (2016), pp. 3305–3343.
- [12] R. CUI, K.-Y. LAM, AND Y. LOU, *Dynamics and asymptotic profiles of steady states of an epidemic model in advective environments*, J. Differential Equations, 263 (2017), pp. 2343–2373.
- [13] M. C. M. DE JONG, O. DIEKMANN, AND H. HEESTERBEEK, *How does transmission of infection depend on population size?*, in Epidemic Models: Their Structure and Relation to Data, Cambridge University Press, New York, 1995, pp. 84–94.

- [14] K. DENG AND Y. WU, *Dynamics of a susceptible-infected-susceptible epidemic reaction-diffusion model*, Proc. Roy. Soc. Edinburgh Sect. A, 146 (2016), pp. 929–946.
- [15] O. DIEKMANN AND J. A. P. HEESTERBEEK, *Mathematical Epidemiology of Infective Diseases: Model Building, Analysis and Interpretation*, Wiley, New York, 2000.
- [16] W. DING, W. HUANG, AND S. KANSAKAR, *Traveling wave solutions for a diffusive SIS epidemic model*, Discrete Contin. Dyn. Syst. Ser. B, 18 (2013), pp. 1291–1304.
- [17] Y. DU, R. PENG, AND M. WANG, *Effect of a protection zone in the diffusive Leslie predator-prey model*, J. Differential Equations, 246 (2009), pp. 3932–3956.
- [18] J. FANG, Y. LOU, AND J. WU, *Can pathogen spread keep pace with its host invasion?*, SIAM J. Appl. Math., 76 (2016), pp. 1633–1657.
- [19] J. GE, K. I. KIM, Z. LIN, AND H. ZHU, *A SIS reaction-diffusion-advection model in a low-risk and high-risk domain*, J. Differential Equations, 259 (2015), pp. 5486–5509.
- [20] J. GE, L. LIN, AND L. ZHANG, *A diffusive SIS epidemic model incorporating the media coverage impact in the heterogeneous environment*, Discrete Contin. Dyn. Syst. Ser. B, 22 (2017), pp. 2763–2776.
- [21] D. GILBARG AND N. S. TRUDINGER, *Elliptic Partial Differential Equation of Second Order*, Springer, New York, 2001.
- [22] S. A. GOURLEY AND Y. LOU, *A mathematical model for the spatial spread and biocontrol of the Asian longhorned beetle*, SIAM J. Appl. Math., 74 (2014), pp. 864–884.
- [23] S. A. GOURLEY AND X. ZOU, *A mathematical model for the control and eradication of a wood boring beetle infestation*, SIAM J. Appl. Math., 68 (2008), pp. 1665–1687.
- [24] H. W. HETHCOTE, *The mathematics of infectious diseases*, SIAM Rev., 42 (2000), pp. 599–653.
- [25] W. HUANG, M. HAN, AND K. LIU, *Dynamics of an SIS reaction-diffusion epidemic model for disease transmission*, Math. Biosci. Eng., 7 (2010), pp. 51–66.
- [26] D. S. JONES AND B. D. SLEEMAN, *Differential Equations and Mathematical Biology*, Chapman & Hall/CRC, Boca Raton, FL, 2003.
- [27] A. KÄLLÉN, *Thresholds and travelling waves in an epidemic model for rabies*, Nonlinear Anal., 8 (1984), pp. 851–856.
- [28] A. KÄLLÉN, P. ARCURI, AND J. D. MURRAY, *A simple model for the spatial spread and control of rabies*, J. Theoret. Biol., 116 (1985), pp. 377–393.
- [29] W. O. KERMACK AND A. G. MCKENDRICK, *A contribution to the mathematical theory of epidemics*, R. Soc. Lond. Proc. Ser. A Math. Phys. Eng. Sci., 115 (1927), pp. 700–721.
- [30] W. O. KERMACK AND A. G. MCKENDRICK, *Contributions to the mathematical theory of epidemics—I. 1927*, Bull. Math. Biol., 53 (1991), pp. 33–55.
- [31] W. O. KERMACK AND A. G. MCKENDRICK, *Contributions to the mathematical theory of epidemics—II. The problem of endemicity*, Bull. Math. Biol., 53 (1991), pp. 57–87.
- [32] W. O. KERMACK AND A. G. MCKENDRICK, *Contributions to the mathematical theory of epidemics—III. Further studies of the problem of endemicity*, Bull. Math. Biol., 53 (1991), pp. 89–118.
- [33] K. KOUSUKE, H. MATSUZAWA, AND R. PENG, *Concentration profile of endemic equilibrium of a reaction-diffusion-advection SIS epidemic model*, Calc. Var. Partial Differential Equations, 56 (2017), 112.
- [34] M. A. LEWIS, J. RENCLAWOWICZ, AND P. VAN DEN DRIESSCHE, *Traveling waves and spread rates for a West Nile virus model*, Bull. Math. Biol., 68 (2006), pp. 3–23.
- [35] B. LI, H. LI, AND Y. TONG, *Analysis on a diffusive SIS epidemic model with logistic source*, Z. Angew. Math. Phys., 68 (2017), 96.
- [36] H. LI, R. PENG, AND F.-B. WANG, *Varying total population enhances disease persistence: Qualitative analysis on a diffusive SIS epidemic model*, J. Differential Equations, 262 (2017), pp. 885–913.
- [37] G. M. LIEBERMAN, *Bounds for the steady-state Sel'kov model for arbitrary  $p$  in any number of dimensions*, SIAM J. Math. Anal., 36 (2005), pp. 1400–1406.
- [38] Y. LOU AND X.-Q. ZHAO, *The periodic Ross-Macdonald model with diffusion and advection*, Appl. Anal., 89 (2010), pp. 1067–1089.
- [39] Y. LOU AND X.-Q. ZHAO, *A reaction-diffusion malaria model with incubation period in the vector population*, J. Math. Biol., 62 (2011), pp. 543–568.
- [40] Y. LOU AND W.-M. NI, *Diffusion, self-diffusion and cross-diffusion*, J. Differential Equations, 131 (1996), pp. 79–131.
- [41] P. MAGAL AND X.-Q. ZHAO, *Global attractors and steady states for uniformly persistent dynamical systems*, SIAM J. Math. Anal., 37 (2005), pp. 251–275.
- [42] H. MCCALLUM, N. BARLOW, AND J. HONE, *How should pathogen transmission be modelled?*, Trends Ecol. Evol., 16 (2001), pp. 295–300.
- [43] M. MARTCHEVA, *An Introduction to Mathematical Epidemiology*, Springer, New York, 2015.



- [44] J. D. MURRAY, *Mathematical Biology II: Spatial Models and Biomedical Applications*, Springer, Berlin, 2003.
- [45] J. D. MURRAY, E. A. STANLEY, AND D. L. BROWN, *On the spatial spread of rabies among foxes*, R. Soc. Lond. Proc. Ser. B Biol. Sci., 229 (1986), pp. 111–150.
- [46] R. PENG, *Asymptotic profiles of the positive steady state for an SIS epidemic reaction-diffusion model. Part I*, J. Differential Equations, 247 (2009), pp. 1096–1119.
- [47] R. PENG AND S. LIU, *Global stability of the steady states of an SIS epidemic reaction-diffusion model*, Nonlinear Anal., 71 (2009), pp. 239–247.
- [48] R. PENG, J. SHI, AND M. WANG, *On stationary patterns of a reaction-diffusion model with autocatalysis and saturation law*, Nonlinearity, 21 (2008), pp. 1471–1488.
- [49] R. PENG AND F. YI, *Asymptotic profile of the positive steady state for an SIS epidemic reaction-diffusion model: Effects of epidemic risk and population movement*, Phys. D, 259 (2013), pp. 8–25.
- [50] R. PENG AND X. ZHAO, *A reaction-diffusion SIS epidemic model in a time-periodic environment*, Nonlinearity, 25 (2012), pp. 1451–1471.
- [51] S. RUAN AND J. WU, *Modeling spatial spread of communicable diseases involving animal hosts*, Spatial Ecology, C. Cosner, S. Cantrell, and S. Ruan, eds., Chapman and Hall/CRC, New York, 2009, pp. 293–316.
- [52] L. T. TAKAHASHI, N. A. MAIDANA, W. C. FERREIRA, JR., P. PULINO, AND H. M. YANG, *Mathematical models for the Aedes aegypti dispersal dynamics: Travelling waves by wing and wind*, Bull. Math. Biol., 67 (2005), pp. 509–528.
- [53] A. K. TARBOUSH, Z. LIN, AND M. ZHANG, *Spreading and vanishing in a West Nile virus model with expanding fronts*, Sci. China Math., 60 (2017), pp. 841–860.
- [54] H. R. THIEME, *Mathematics in Population Biology*, Princeton University Press, Princeton, NJ, 2003.
- [55] M. WANG, *Nonlinear Partial Differential Equations of Parabolic Type*, Science Press, Beijing, 1993 (in Chinese).
- [56] X. WEN, J. JI, AND B. LI, *Asymptotic profiles of the endemic equilibrium to a diffusive SIS epidemic model with mass action infection mechanism*, J. Math. Anal. Appl., 458 (2018), pp. 715–729.
- [57] Y. WU AND X. ZOU, *Asymptotic profiles of steady states for a diffusive SIS epidemic model with mass action infection mechanism*, J. Differential Equations, 261 (2016), pp. 4424–4447.
- [58] X.-Q. ZHAO, *Dynamical Systems in Population Biology*, Springer, New York, 2003.