

SPATIAL SEGREGATION IN REACTION-DIFFUSION EPIDEMIC MODELS*

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Abstract. In this paper, we formulate susceptible-infected-susceptible reaction-diffusion epidemic models with cognition and show the impact of movement strategies on disease outbreak and mitigation under a spatially heterogeneous environment. The cognitive diffusion either takes a Fokker–Planck type diffusion obtained by Chapman’s diffusion law (called random diffusion) or follows Fick’s diffusion law (called symmetric diffusion). We derive a variational expression of the basic reproduction number \mathcal{R}_0 for both models and prove that the disease-free equilibrium is unique and globally asymptotically stable if $\mathcal{R}_0 < 1$. Furthermore, if $\mathcal{R}_0 > 1$, the model following Fick’s diffusion law admits at least one endemic equilibrium and the model following Chapman’s diffusion law has a unique endemic equilibrium. The theoretical results are illustrated by numerical simulations, which additionally show the segregation phenomenon between susceptible and infected populations regulated by different movement strategies. Spatial segregation here is natural, not caused by an isolation policy, and thus is the most important indicator for an infectious disease to spread or wane in the absence of intervention. The first example shows that a heterogeneous random diffusion segregates infected and susceptible populations further than an ODE model and thus reduces the infection size. However, symmetric diffusion never does that. The second example shows that a heterogeneous random diffusion detriments segregation but still reduces the infection severity by moving infected individuals to a disease-free region. In a certain situation, a heterogeneous random diffusion may increase the infection severity as shown in the last example.

Key words. spatial heterogeneity, cognitive movement, basic reproduction number, disease-free equilibrium, endemic equilibrium, segregation

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1. Introduction. Mathematical models are powerful tools to study disease transmission and control. The most widely used models are ordinary differential equation (ODE) compartment models that determine whether an infectious disease will surge or not by the basic reproduction number \mathcal{R}_0 compared with one. Population movement and spatial heterogeneity are obviously significant in disease spread and mitigation.

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Toward this aspect, various partial differential equation (PDE) models have been proposed in the literature (see [1, 7, 23, 30, 31]).

To study the effect of spatial heterogeneity and diffusive population on the disease dynamics, Allen et al. [1] developed an SIS (susceptible-infected-susceptible) reaction-diffusion epidemic model,

$$\begin{aligned}
 (1.1) \quad & S_t = d_S \Delta S - \left(\frac{\beta(x)S}{S+I} - r(x) \right) I, \quad t > 0, x \in \Omega, \\
 & I_t = d_I \Delta I + \left(\frac{\beta(x)S}{S+I} - r(x) \right) I, \quad t > 0, x \in \Omega, \\
 & \nabla S \cdot \mathbf{n} = \nabla I \cdot \mathbf{n} = 0, \quad t > 0, x \in \partial\Omega, \\
 & S(x, 0) = S_0(x), I(x, 0) = I_0(x), \quad x \in \Omega,
 \end{aligned}$$

where $\Omega \in \mathbb{R}^m$ ($m \geq 1$) is a bounded domain with smooth boundary $\partial\Omega$ ($m > 1$) and \mathbf{n} is the outward unit normal vector on $\partial\Omega$. The variables $S(t, x)$ and $I(t, x)$ represent the population densities of susceptible and infected individuals, respectively, at time $t > 0$ and location $x \in \Omega$. The diffusion rates d_S and d_I are positive constants. The spatial functions $\beta(x)$ and $r(x)$ represent disease transmission rate and recovery rate at x , and they are Hölder continuous functions on $\bar{\Omega}$.

Allen et al. derived a variational expression of the basic reproduction number for the PDE system (1.1) as follows:

$$\mathcal{R}_0 = \sup_{\phi \in W^{1,2}(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} \beta \phi^2 dx}{\int_{\Omega} d_I |\nabla \phi|^2 dx + \int_{\Omega} r \phi^2 dx} \right\}.$$

Moreover, they showed the monotone and asymptotic behavior of \mathcal{R}_0 on the diffusion rate d_I . In particular, the existence and stability of the disease-free equilibrium and the asymptotic behavior of an endemic equilibrium were investigated mathematically. One can find many followup papers [21, 22, 4, 3, 15, 32, 5, 14]. However, all these studies assumed the simplest diffusion terms with constant diffusion rates.

Although the spatial heterogeneity of the model (1.1) was considered in the transmissibility $\beta(x)$ and the recovery $r(x)$, the simplest diffusion terms were assumed to be mimicking particle movement. However, humans have cognition that is crucial in the mechanistic modeling of any organisms with perception, memory, and learning. To model the cognitive movement, we start with dispersal on a patch system. Let u_i be the population in patch i . Denote c_{ij} ($c_{i \leftarrow j}$) as the migration or departing rate from patch j to patch i (see Figure 1). Then, the rate of change of population u_i satisfies

$$(1.2) \quad \dot{u}_i = c_{i-1} u_{i-1} + c_{i+1} u_{i+1} - c_{i-1} u_i - c_{i+1} u_i.$$

If $c_{ii+1} = c_{i+1i}$, i.e., the dispersal rate of population from patch i to $i + 1$ is equal to that from patch $i + 1$ to i , the dispersal is called *symmetric* (see [12]). If we denote $\gamma_{i+1/2} := c_{ii+1} = c_{i+1i}$, then model (1.2) is rewritten as



FIG. 1. Scheme of dispersal between patches.

$$\dot{u}_i = \gamma_{i+1/2}(u_{i+1} - u_i) - \gamma_{i-1/2}(u_i - u_{i-1}) \cong \gamma_{i+1/2}u'_{i+1/2} - \gamma_{i-1/2}u'_{i-1/2} \cong (\gamma u')'_i.$$

In this approximation, the distance between two adjacent patches is one, which is treated as sufficiently small. In a space with any dimension, this relation is written as a PDE,

$$(1.3) \quad u_t = \nabla \cdot (\gamma(x)\nabla u),$$

which follows Fick's diffusion law. The physical meaning of this symmetric diffusion is unclear. We are more interested in the case when $c_{i-1i} = c_{i+1i}$, i.e., when the dispersal rate from patch i to $i-1$ is equal to the one from patch i to $i+1$. In this case, the probabilities of moving to the right patch or to the left patch are identical and hence, the dispersal is called *random*. If we denote $\gamma_i := c_{i+1i} = c_{i-1i}$, then model (1.2) is written as

$$\dot{u}_i = \gamma_{i-1}u_{i-1} + \gamma_{i+1}u_{i+1} - 2\gamma_i u_i \cong (\gamma u)''.$$

The corresponding PDE model in any spatial dimension is

$$(1.4) \quad u_t = \Delta(\gamma(x)u),$$

which follows Chapman's diffusion law. The physical meaning of this random diffusion is clear. Such a random diffusion is also called Fokker-Planck type or Ito type. If the dispersal is random and symmetric at the same time, all of the c_{ij} coefficients are equal, which leads to the spatially homogeneous diffusion. For more model formulations on diffusion, readers can refer to [24, 19] and references therein.

By considering spatially heterogeneous movements, we propose the following SIS reaction-diffusion epidemic model with random diffusion (or Fokker-Planck type),

$$(1.5) \quad \begin{aligned} S_t &= \Delta(f(x)S) - \left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, x \in \Omega, \\ I_t &= \Delta(g(x)I) + \left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, x \in \Omega, \\ \nabla(f(x)S) \cdot \mathbf{n} &= \nabla(g(x)I) \cdot \mathbf{n} = 0, & t > 0, x \in \partial\Omega, \\ S(0, x) &= S_0(x) \geq 0, I(0, x) = I_0(x) \geq, \neq 0, & x \in \Omega, \end{aligned}$$

where $f(x)$ and $g(x)$ are dispersal rates of susceptible and infected groups at x , respectively. For comparison, we investigate the properties of a corresponding model with symmetric diffusion (or Fickian type):

$$(1.6) \quad \begin{aligned} S_t &= \nabla \cdot (f(x)\nabla S) - \left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, x \in \Omega, \\ I_t &= \nabla \cdot (g(x)\nabla I) + \left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, x \in \Omega, \\ \nabla S \cdot \mathbf{n} &= \nabla I \cdot \mathbf{n} = 0, & t > 0, x \in \partial\Omega, \\ S(0, x) &= S_0(x) \geq 0, I(0, x) = I_0(x) \geq, \neq 0, & x \in \Omega. \end{aligned}$$

However, the symmetric diffusion shows similar dynamical behaviors as a homogeneous diffusion (see section 5). Note that the Fokker-Planck type diffusion is written as

$$\Delta(f(x)S) = \nabla \cdot (f(x)\nabla S) + \nabla \cdot (S\nabla f(x)).$$

Hence, the advection term $\nabla \cdot (S\nabla f(x))$ is the difference between the two diffusion laws.

It is natural to expect that susceptible and infected individuals disperse differently. We assume that the dispersal rate f of the susceptible population S is an increasing function of the transmission rate β since the infection probability is the primary concern of healthy people. In other words, when susceptible people are located in a region with a high transmission rate $\beta(x)$ (such as indoor playground), they will leave to avoid infection. On the other hand, we assume that the dispersal rate g of the infected population I is an increasing function of the reciprocal of the recovery rate r . In fact, infected people only care about recovery (or treatment). For example, if infected people are located in a region with a high recovery rate $r(x)$ (such as a hospital), they will prefer to stay there for faster recovery. As a summary, these two groups of people have their own dispersal strategies based on their cognition (see [2, 10, 11]). This model formulation is an application of our cognitive movement modeling efforts [28] in epidemiological modeling.

In the proposed models, we have four functions f, g, β and r . We assume these functions satisfy the following hypotheses:

(H1) There exist constants m_0 and M_0 such that

$$0 < m_0 < f, g < M_0 < \infty,$$

which is used for the uniform parabolicity of the problem.

(H2) f and g are increasing functions of β and r^{-1} , respectively, i.e.,

$$f = f(\beta) \text{ and } g = g(r^{-1})$$

are monotone increasing.

(H3) The set $\Omega^+ := \{x \in \Omega : \beta(x) > r(x)\}$ is nonempty.

Most studies on infectious disease PDE models mainly focused on asymptotic behaviors for sufficiently large time. However, few studies have been done on the transient dynamics of epidemiological models due to lack of appropriate mathematical tools. In this work, we study the transient dynamics of systems (1.5), (1.6), and (1.1) through numerical simulations. We will explore the spatial segregation induced by cognitive diffusion, the use of a disease-free region, and negative effects of mixing by diffusion. Biological segregation is a phenomenon where population groups are separated in certain areas [26]. To measure the degree of segregation, we define the segregation indices

$$(1.7) \quad \kappa(v_1, v_2) = \frac{\|v_1 - v_2\|_{L^1(\Omega)}}{\|v_1\|_{L^1(\Omega)} + \|v_2\|_{L^1(\Omega)}}$$

and

$$(1.8) \quad \chi(v_1, v_2) = \max_{x \in \Omega} \{v_1(x) - v_2(x)\} \cdot \min_{x \in \Omega} \{v_1(x) - v_2(x)\}$$

for $v_i(x) \in L^1(\Omega)$ and $\|v_i\|_{L^1(\Omega)} = \int_{\Omega} |v_i(x)| dx, i = 1, 2$. Obviously $0 \leq \kappa(v_1, v_2) \leq 1$. If $\kappa(v_1, v_2)$ is large and $\chi(v_1, v_2) < 0$, then the segregation phenomenon between v_1 and v_2 is more obvious (so-called perfect/strong segregation). If $\kappa(v_1, v_2)$ is small and $\chi(v_1, v_2) < 0$, then the segregation phenomenon is less obvious (so-called weak segregation). If $\kappa(v_1, v_2) = 0$ or $\chi(v_1, v_2) \geq 0$, then the segregation phenomenon disappears.

The main goals of the paper are as follows: (i) The variational characterizations of the basic reproduction number \mathcal{R}_0 for systems (1.5) and (1.6) are defined, respectively, and then the monotonic and asymptotic behaviors of \mathcal{R}_0 on the diffusion rate g are discussed; (ii) We prove the existence and stability of disease-free equilibrium (DFE) and endemic equilibrium (EE) of (1.5) and (1.6), respectively, and verify the theoretical results by numerical simulations; More importantly, (iii) we investigate the segregation phenomena of (1.5), (1.6), and (1.1) numerically. It is worth mentioning that there are several significant improvements. First, in modeling we propose two new models with different diffusion mechanisms and assume that the dispersal rates of susceptible and infected individuals depend on the transmission and recovery rates, respectively. Second, in terms of theory we generalize the results of [1, Lemmas 2.2–2.3] on the monotonicity of \mathcal{R}_0 for models (1.6) and (1.5) (see Lemmas 2.2, 2.3, 3.3, and 3.4). Finally, spatial segregation occurs naturally here (not caused by an isolation policy) and thus is the most important indicator for an infectious disease to spread or wane in the absence of intervention.

The remainder of the paper is organized as follows. In sections 2 and 3, we show the existence and stability of DFE and EE for the models (1.6) and (1.5). In section 4, our theoretical results are verified by numerical simulations. In section 5, we consider three numerical examples to illustrate the segregation phenomena of epidemic PDE models and their impact on the disease spread. Section 6 summarizes this paper with a brief discussion.

2. The epidemic model with symmetric diffusion. In this section, we prove the existence and stability of DFE and EE for model (1.6) with symmetric diffusion. Let $\mathbb{X} := C(\bar{\Omega}) \times C(\bar{\Omega})$. We start with the well-posedness result.

PROPOSITION 2.1. *For any $(S_0(x), I_0(x)) \in \mathbb{X}$, system (1.6) has a unique positive solution $(S(t, x), I(t, x))$ satisfying*

$$(S(t, x), I(t, x)) \in C^{1,2}((0, \infty) \times \bar{\Omega}) \times C^{1,2}((0, \infty) \times \bar{\Omega}).$$

Furthermore, there is a constant $C > 0$ independent of the initial values, and $T_0 > 0$ such that the solution $(S(t, x), I(t, x))$ meets

$$\|S(t, x)\|_{L^\infty(\Omega)} + \|I(t, x)\|_{L^\infty(\Omega)} \leq C \quad \text{for all } t > T_0.$$

Proof. With the help of the regularity theory of parabolic equations [20], system (1.6) admits a unique nonnegative classical solution $(S(t, x), I(t, x)) \in C^{1,2}((0, T_m) \times \bar{\Omega}) \times C^{1,2}((0, T_m) \times \bar{\Omega})$, where T_m represents the maximal existence time of the solution. In addition, applying strong maximum principle [25] yields that $S(\cdot, \cdot)$ and $I(\cdot, \cdot)$ are positive in $(0, T_m) \times \bar{\Omega}$.

Adding two equations of (1.6) and then integrating over Ω , we get

$$\partial_t \int_{\Omega} (S(t, x) + I(t, x)) dx = (f(\beta(x)) \nabla S \cdot \mathbf{n} + g(r^{-1}(x)) \nabla I \cdot \mathbf{n})|_{\partial\Omega} = 0.$$

which is owing to $\nabla S \cdot \mathbf{n} = \nabla I \cdot \mathbf{n} = 0$ on $\partial\Omega$. Thus, we obtain

$$(2.1) \quad \int_{\Omega} ((S(t, x) + I(t, x))) dx = N_0, \quad t > 0,$$

for some constant $N_0 > 0$. In particular, $N_0 = \int_{\Omega} (S_0(x) + I_0(x)) dx$. This means that the $L^1(\Omega)$ norms of $S(t, \cdot)$ and $I(t, \cdot)$ are bounded for any $0 < t < T_m$. Hence,

by Theorem 1 and Corollary 1 of [6] ($p_0 = 1, \sigma = 1$) and the positivity of $S(t, \cdot)$ and $I(t, \cdot)$, the solution $(S(t, \cdot), I(t, \cdot))$ exists for all time and there exists a constant $C > 0$, independent of $S_0(\cdot)$ and $I_0(\cdot)$, and $T_0 > 0$ such that

$$\|S(t, x)\|_{L^\infty(\Omega)} + \|I(t, x)\|_{L^\infty(\Omega)} \leq C$$

for any $t > T_0$. The proof is completed. □

2.1. DFE. In this subsection, we derive the basic reproduction number of (1.6) and discuss the stability of DFE.

By direct calculations, model (1.6) has a unique DFE $E_0 = (\tilde{S}_0, 0) := (N_0/|\Omega|, 0)$. Linearizing system (1.6) at E_0 gives

$$\begin{aligned} \bar{S}_t &= \nabla \cdot (f(\beta(x))\nabla \bar{S}) - (\beta(x) - r(x))\bar{I}, & t > 0, x \in \Omega, \\ \bar{I}_t &= \nabla \cdot (g(r^{-1}(x))\nabla \bar{I}) + (\beta(x) - r(x))\bar{I}, & t > 0, x \in \Omega, \\ \nabla \bar{S} \cdot \mathbf{n} &= \nabla \bar{I} \cdot \mathbf{n} = 0, & t > 0, x \in \partial\Omega, \end{aligned} \tag{2.2}$$

where

$$\bar{S}(t, x) = S(t, x) - \tilde{S}_0 \text{ and } \bar{I}(t, x) = I(t, x). \tag{2.3}$$

Since \bar{S} is decoupled, it suffices to consider the following system:

$$\begin{aligned} \bar{I}_t &= \nabla \cdot (g(r^{-1}(x))\nabla \bar{I}) + (\beta(x) - r(x))\bar{I}, & t > 0, x \in \Omega, \\ \nabla \bar{I} \cdot \mathbf{n} &= 0, & t > 0, x \in \partial\Omega. \end{aligned} \tag{2.4}$$

Letting $\bar{I}(t, x) = e^{-\zeta t} \phi(x)$ and substituting it into (2.4) results in

$$\begin{aligned} \nabla \cdot (g(r^{-1}(x))\nabla \phi) + (\beta(x) - r(x))\phi + \zeta \phi &= 0, & x \in \Omega, \\ \nabla \phi \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned} \tag{2.5}$$

Similarly to the analysis of [1, (2.2)], the eigenvalue problem (2.5) has at least one eigenvalue ζ^* corresponding to the positive eigenfunction ϕ^* , while other eigenvalues do not have a positive eigenfunction. Then (ζ^*, ϕ^*) satisfies

$$\begin{aligned} \nabla \cdot (g(r^{-1}(x))\nabla \phi^*) + (\beta(x) - r(x))\phi^* + \zeta^* \phi^* &= 0, & x \in \Omega, \\ \nabla \phi^* \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned} \tag{2.6}$$

Moreover, ζ^* can be expressed by the following variational characterization,

$$\zeta^* = \inf_{\phi \in W^{1,2}(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} g((r^{-1}))|\nabla \phi|^2 dx + \int_{\Omega} (r - \beta)\phi^2 dx}{\int_{\Omega} \phi^2 dx} \right\}, \tag{2.7}$$

where ζ^* and ϕ^* are differentiable with respect to (w.r.t.) g .

Define $\bar{g}(\cdot) := d\nabla g(\cdot)/dg(\cdot)$ where d/dg represents the derivative w.r.t. g . More precisely,

$$\nabla g(\cdot) = \left(\frac{\partial g}{\partial x_1}, \dots, \frac{\partial g}{\partial x_m} \right) \text{ and } \frac{d\nabla g}{dg} = \left(\frac{\partial^2 g / \partial x_1^2}{\partial g / \partial x_1}, \dots, \frac{\partial^2 g / \partial x_m^2}{\partial g / \partial x_m} \right) := (\bar{g}_{x_1}, \dots, \bar{g}_{x_m}),$$

where $x := (x_1, \dots, x_m) \in \Omega \subset \mathbb{R}^m$. Let $\Omega_1, \dots, \Omega_m$ be the disjoint subsets of Ω with $\Omega_1 \cup \dots \cup \Omega_m = \Omega$, and $\Gamma_1, \dots, \Gamma_m$ be the disjoint subsets of $\partial\Omega$ with $\Gamma_i = \partial\Omega_i$ and thus $\Gamma_1 \cup \dots \cup \Gamma_m = \partial\Omega$.

LEMMA 2.2. *Define ζ^* by (2.7) and assume $g(r^{-1}(\cdot)) = \eta g_1(r^{-1}(\cdot))$, where η and g_1 are a positive constant and $C^3(\bar{\Omega})$ function, respectively. If $\beta(\cdot) - r(\cdot)$ changes sign on Ω , then the following properties hold:*

(i) If

$$\bar{g}_{x_i}(x)(\phi^*)^2(x) \leq 0 \text{ on } \Gamma_i, \quad i = 1, \dots, m, \text{ and } \nabla \bar{g}(x) \geq 0 \text{ on } \Omega,$$

then ζ^* is a strictly monotone increasing function w.r.t. g .

- (ii) $\zeta^* \rightarrow \min_{x \in \bar{\Omega}} \{r(x) - \beta(x)\} < 0$, as $\eta \rightarrow 0$ for fixed g_1 .
- (iii) $\zeta^* \rightarrow \frac{1}{|\Omega|} \int_{\Omega} (r(x) - \beta(x)) dx$, as $\eta \rightarrow \infty$ for fixed g_1 .
- (iv) If $\int_{\Omega} r(x) dx \leq \int_{\Omega} \beta(x) dx$, then $\zeta^* < 0$ for any $g > 0$.
- (v) If $\int_{\Omega} r(x) dx > \int_{\Omega} \beta(x) dx$, then for fixed g_1 , $\zeta^*(\eta) = 0$ admits a unique root $\eta^* > 0$ such that $\zeta^* < 0$ when $\eta < \eta^*$, and $\zeta^* > 0$ when $\eta > \eta^*$.

Proof. (i) Thanks to (2.7), it is straightforward that ζ^* is an increasing function of g . In what follows, it is only necessary to prove that the monotonicity is strict. Differentiating the first equation of (2.6) w.r.t. g , we obtain

$$(2.8) \quad \begin{aligned} \Delta \phi^* + g \Delta \phi_g^* + \bar{g} \nabla \phi^* + \nabla g \nabla \phi_g^* + (\beta(x) - r(x)) \phi_g^* + \zeta_g^* \phi^* + \zeta^* \phi_g^* &= 0, & x \in \Omega, \\ \nabla \phi^* \cdot \mathbf{n} = \nabla \phi_g^* \cdot \mathbf{n} &= 0, & x \in \partial \Omega, \end{aligned}$$

where $\phi_g^* := d\phi^*/dg$. Multiplying the first equations of (2.8) and (2.6) by ϕ^* and ϕ_g^* , respectively, and then integrating by parts over Ω leads to

$$\begin{aligned} - \int_{\Omega} |\nabla \phi^*|^2 - \int_{\Omega} g \nabla \phi^* \nabla \phi_g^* dx + \frac{1}{2} \sum_{i=1}^m \int_{\Omega \setminus \Omega_i} (\bar{g}_{x_i}(\phi^*)^2)|_{\Gamma_i} - \frac{1}{2} \int_{\Omega} (\phi^*)^2 \nabla \bar{g} \\ + \int_{\Omega} (\beta(x) - r(x)) \phi^* \phi_g^* + \zeta_g^* \int_{\Omega} (\phi^*)^2 + \zeta^* \int_{\Omega} \phi^* \phi_g^* = 0 \end{aligned}$$

and

$$- \int_{\Omega} g \nabla \phi_g^* \nabla \phi^* + \int_{\Omega} (\beta(x) - r(x)) \phi_g^* \phi^* + \zeta^* \int_{\Omega} \phi^* \phi_g^* = 0.$$

Subtracting the above two equations leads to

$$(2.9) \quad \zeta_g^* \int_{\Omega} (\phi^*)^2 = \int_{\Omega} |\nabla \phi^*|^2 - \frac{1}{2} \sum_{i=1}^m \int_{\Omega \setminus \Omega_i} (\bar{g}_{x_i}(\phi^*)^2)|_{\Gamma_i} + \frac{1}{2} \int_{\Omega} (\phi^*)^2 \nabla \bar{g}.$$

Case 1. If

$$\bar{g}_{x_i}(x)(\phi^*)^2(x) \leq 0 \text{ on } \Gamma_i, \quad i = 1, \dots, m, \text{ and } \nabla \bar{g}(x) > 0 \text{ on } \Omega$$

or

$$\bar{g}_{x_i}(x)(\phi^*)^2(x) < 0 \text{ on } \Gamma_i, \quad i = 1, \dots, m, \text{ and } \nabla \bar{g}(x) \geq 0 \text{ on } \Omega,$$

then $\zeta_g^* > 0$ since ϕ^* is a positive function on Ω .

Case 2. If

$$\bar{g}_{x_i}(x) \equiv 0 \text{ on } \Gamma_i, \quad i = 1, \dots, m, \text{ and } \nabla \bar{g}(x) \equiv 0 \text{ on } \Omega,$$

then the formula (2.9) is rewritten as

$$\zeta_g^* \int_{\Omega} (\phi^*)^2 = \int_{\Omega} |\nabla \phi^*|^2,$$

which indicates that $\zeta_g^* \geq 0$ for any ϕ^* , and $\zeta_g^* = 0$ if and only if ϕ^* is a positive constant. Assuming ϕ^* is a positive constant, it follows from (2.6) that

$$(\beta(x) - r(x)) + \zeta^* = 0, \quad x \in \Omega.$$

This is a contradiction with the fact that $\beta(x) - r(x)$ changes sign in Ω . Therefore, $\zeta_g^* > 0$.

(ii) The conclusion is the direct result of [17, Lemma 3.1].

(iii) In (2.7), we choose $\phi = 1/|\Omega|$. Then

$$\zeta^* = \frac{1}{|\Omega|} \int_{\Omega} (r(x) - \beta(x)) dx \leq \max_{x \in \Omega} \{r(x) - \beta(x)\}.$$

According to the definition of ζ^* , one has

$$\zeta^* \leq \max_{x \in \Omega} \{r(x) - \beta(x)\} \text{ for any } \phi(\cdot) \in W^{1,2}(\Omega) \text{ and } \eta > 0.$$

Thus, by (i) and utilizing the monotone bounded convergence theorem, there exists a constant $\zeta_{\infty}^* < \infty$ such that $\zeta^* \rightarrow \zeta_{\infty}^*$ when η tends to ∞ . Dividing both sides of the first equation for (2.6) by η yields

$$\nabla \cdot (g_1(r^{-1})\nabla\phi^*) + \frac{\beta(x) - r(x) + \zeta^*}{\eta} = 0.$$

Through the elliptic regularity, there is a positive constant ϕ_{∞}^* such that ϕ^* converges to ϕ_{∞}^* in $C(\bar{\Omega})$ as η tends to ∞ . Consequently, integrating (2.6) over Ω , we have

$$\int_{\Omega} (\beta(x) - r(x))\phi^* dx + \zeta^* \int_{\Omega} \phi^* dx = 0$$

which implies that $\zeta^* \rightarrow \frac{1}{|\Omega|} \int_{\Omega} (r(x) - \beta(x)) dx$ as $\eta \rightarrow \infty$.

(iv) From (iii), as $\eta \rightarrow \infty$, for fixed g_1 , we have

$$\zeta^* \rightarrow \frac{1}{|\Omega|} \int_{\Omega} (r(x) - \beta(x)) dx \leq 0.$$

Hence, $\zeta^* < 0$ for any $g > 0$ since ζ^* is a strictly increasing function of g . Moreover, by inspiration of (i)-(iv), the proof of (v) is obvious and so we omit it. \square

Remark 2.1. The conditions in (i) of Lemma 2.2 include the case that the diffusion coefficient is constant [1]. In fact, when $g(r^{-1}) \equiv \text{constant}$, then

$$\bar{g}_{x_i}(x) \equiv 0 \text{ on } \Gamma_i, \quad i = 1, \dots, m, \text{ and } \nabla \bar{g}(x) \equiv 0 \text{ on } \Omega.$$

Equivalently, Case 2 holds.

In order to explore the stability of DFE, it is necessary to derive the basic reproduction number \mathcal{R}_0 of system (1.6). To this end, we analyze the following eigenvalue problem:

$$(2.10) \quad \begin{aligned} \nabla \cdot (g(r^{-1}(x))\nabla\phi) - r(x)\phi + \zeta\beta(x)\phi &= 0, & x \in \Omega, \\ \nabla\phi \cdot \mathbf{n} &= 0, & x \in \partial\Omega, \end{aligned}$$

and obtain the following conclusions.

LEMMA 2.3. *Let ζ_0 be a positive eigenvalue of (2.10) with a positive eigenfunction and assume $g(r^{-1}(\cdot)) = \eta g_1(r^{-1}(\cdot))$. Then ζ_0 is unique and*

$$(2.11) \quad \zeta_0 = \inf_{\phi \in W^{1,2}(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} g(r^{-1}(\cdot)) |\nabla \phi|^2 dx + \int_{\Omega} r \phi^2 dx}{\int_{\Omega} \beta \phi^2 dx} \right\}.$$

Furthermore, the basic reproduction number \mathcal{R}_0 of (1.6) is defined by

$$(2.12) \quad \mathcal{R}_0 = \sup_{\phi \in W^{1,2}(\Omega), \phi \neq 0} \left\{ \frac{\int_{\Omega} \beta \phi^2 dx}{\int_{\Omega} g(r^{-1}(\cdot)) |\nabla \phi|^2 dx + \int_{\Omega} r \phi^2 dx} \right\}.$$

Moreover, if $\beta(\cdot) - r(\cdot)$ changes sign on Ω , then \mathcal{R}_0 fulfills the following properties:

- (i) $\mathcal{R}_0 = 1/\zeta_0$ is monotone decreasing w.r.t. g .
- (ii) $\mathcal{R}_0 \rightarrow \max_{x \in \bar{\Omega}} \{\beta(x)/r(x)\}$ as $\eta \rightarrow 0$ for fixed $g_1(r^{-1}) > 0$.
- (iii) $\mathcal{R}_0 \rightarrow \int_{\Omega} \beta(x) dx / \int_{\Omega} r(x) dx$ as $\eta \rightarrow \infty$ for fixed $g_1(r^{-1}) > 0$.
- (iv) $\text{sign}(1 - \mathcal{R}_0) = \text{sign} \zeta^*$.
- (v) $\min_{x \in \bar{\Omega}} \{\beta(x)/r(x)\} \leq \mathcal{R}_0 \leq \max_{x \in \bar{\Omega}} \{\beta(x)/r(x)\}$.

Proof. Multiplying the first equation of (2.10) by ϕ and then integrating by parts over Ω yields

$$\zeta \int_{\Omega} \beta(x) \phi^2 dx = \int_{\Omega} g(r^{-1}(\cdot)) |\nabla \phi|^2 dx + \int_{\Omega} r(x) \phi^2 dx.$$

Thus, we can obtain (2.11) by the classical Krein–Rutman theorem [13]. Let (ζ_0, ϕ) be a solution of (2.10), where ϕ is the positive eigenfunction corresponding to ζ_0 . To prove the uniqueness, assume $(\hat{\zeta}_0, \hat{\phi})$ is another solution of (2.10). Then one has

$$(2.13) \quad \begin{aligned} \nabla \cdot (g(r^{-1}(x)) \nabla \phi) - r(x) \phi + \zeta_0 \beta(x) \phi &= 0, & x \in \Omega, \\ \nabla \phi \cdot \mathbf{n} &= 0, & x \in \partial\Omega, \end{aligned}$$

and

$$(2.14) \quad \begin{aligned} \nabla \cdot (g(r^{-1}(x)) \nabla \hat{\phi}) - r(x) \hat{\phi} + \hat{\zeta}_0 \beta(x) \hat{\phi} &= 0, & x \in \Omega, \\ \nabla \hat{\phi} \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned}$$

Multiplying the first equations of (2.13) and (2.14) by $\hat{\phi}$, and ϕ , respectively, and then integrating by parts over Ω leads to

$$(\zeta_0 - \hat{\zeta}_0) \int_{\Omega} \beta(x) \phi \hat{\phi} dx = 0.$$

Hence, we have $\zeta_0 = \hat{\zeta}_0$ because β , ϕ , and $\hat{\phi}$ are positive functions. Moreover, it follows from [30, Theorem 3.2] that $\mathcal{R}_0 = 1/\zeta_0$. Similarly to the proof of Lemma 2.2 and [33, Theorem 3], we can prove (i)–(iii).

(iv) By the definition of \mathcal{R}_0 , there exists a positive function $\phi(x) \in C^2(\bar{\Omega})$ such that

$$(2.15) \quad \begin{aligned} \nabla \cdot (g(r^{-1}(x)) \nabla \phi) - r(x) \phi + \frac{1}{\mathcal{R}_0} \beta(x) \phi &= 0, & x \in \Omega, \\ \nabla \phi \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned}$$

Multiplying the first equations of (2.6) and (2.15) by ϕ and ϕ^* , respectively, and integrating by parts over Ω yields

$$\zeta^* \int_{\Omega} \phi^* \phi dx = \frac{1 - \mathcal{R}_0}{\mathcal{R}_0} \int_{\Omega} \beta(x) \phi^* \phi dx.$$

Note that ϕ^* and ϕ are positive in Ω . Hence, $\mathcal{R}_0 > 1$ if $\zeta^* < 0$, $\mathcal{R}_0 = 1$ if $\zeta^* = 0$, and $\mathcal{R}_0 < 1$ if $\zeta^* > 0$.

(v) Integrating the first equation of (2.15) over Ω gives

$$\int_{\Omega} r(x) \left[\mathcal{R}_0 - \frac{\beta(x)}{r(x)} \right] \phi dx = 0.$$

Then $\min\{\beta(\cdot)/r(\cdot)\} \leq \mathcal{R}_0 \leq \max\{\beta(\cdot)/r(\cdot)\}$ thanks to the positivity of r and ϕ . This ends the proof. \square

LEMMA 2.4. *If $\mathcal{R}_0 < 1$, then the DEF E_0 is stable; if $\mathcal{R}_0 > 1$, then the DEF E_0 is unstable.*

Proof. (i) For $\mathcal{R}_0 < 1$. Letting $(\bar{S}(t, \cdot), \bar{I}(t, \cdot)) = e^{-\zeta t}(\varphi(\cdot), \phi(\cdot))$, and substituting it into (2.2), one gets

$$\begin{aligned} (2.16) \quad & \nabla \cdot (f(\beta(x))\nabla\varphi) - (\beta(x) - r(x))\phi + \zeta\varphi = 0, & x \in \Omega, \\ & \nabla \cdot (g(r^{-1}(x))\nabla\phi) + (\beta(x) - r(x))\phi + \zeta\phi = 0, & x \in \Omega, \\ & \nabla\varphi \cdot \mathbf{n} = \nabla\phi \cdot \mathbf{n} = 0, & x \in \partial\Omega. \end{aligned}$$

To prove the linear stability of E_0 , it suffices to show (2.16) possesses a solution (ζ, φ, ϕ) for which the real part of ζ is positive, and φ or ϕ is not identically zero on Ω . Arguing by contradiction, we assume it has a (ζ, φ, ϕ) satisfying the real part of ζ is nonpositive, and φ or ϕ is not identically zero on Ω . We claim $\phi(x) \not\equiv 0$ for all $x \in \Omega$. If not, then one has $\varphi(\cdot) \not\equiv 0$ on Ω . From the first equation of (2.16), we have

$$\begin{aligned} (2.17) \quad & \nabla \cdot (f(\beta(x))\nabla\varphi) + \zeta\varphi = 0, & x \in \Omega, \\ & \nabla\varphi \cdot \mathbf{n} = 0, & x \in \partial\Omega. \end{aligned}$$

Thus, $\zeta \int_{\Omega} \varphi dx = 0$ by integrating the above equation over Ω . This implies that $\zeta = 0$ because of $\varphi \not\equiv 0$ on Ω . By (2.17), we see that φ is a constant, denoted by $\bar{\varphi}$. Combining with (2.1) and (2.3), one gets

$$\begin{aligned} (2.18) \quad & \int_{\Omega} (\varphi + \phi) dx = e^{\zeta t} \int_{\Omega} (\bar{S}(t, x) + \bar{I}(t, x)) dx \\ & = e^{\zeta t} \int_{\Omega} (S(t, x) - \tilde{S}_0 + I(t, x)) dx = 0. \end{aligned}$$

Since $\phi(\cdot) \equiv 0$ on Ω , $\bar{\varphi} \equiv 0$ which contradicts $\varphi(\cdot) \not\equiv 0$ on Ω . Hence, the above claim is true. Observing that the operator $\nabla \cdot (g(r^{-1})\nabla) + \beta - r$ is self-adjoint, it follows from the second equation of (2.16) that ζ is real and nonpositive. By the definition of ζ^* , we obtain $\zeta^* \leq \zeta \leq 0$. However, from Lemma 2.3(iv), $\mathcal{R}_0 \geq 1$ which is a contradiction. Therefore, ζ has a positive real part which means that the DFE is linearly stable. Furthermore, the DFE is stable by applying the theories in [9].

(ii) For $\mathcal{R}_0 > 1$. Similarly to the case $\mathcal{R}_0 < 1$, to cope with the linear instability of DFE, it is only necessary to prove that there exists a solution (ζ, φ, ϕ) for (2.16) satisfying (2.18), where ζ has a negative real part and $\phi(x)$ is positive for all $x \in \Omega$. Note that ζ^* and ϕ^* meet (2.6) and ϕ^* is positive on Ω . Substituting (ζ^*, ϕ^*) into the first equation of (2.16), we obtain

$$\begin{aligned} & \nabla \cdot (f(\beta(x))\nabla\varphi) - (\beta(x) - r(x))\phi^* + \zeta^*\varphi = 0, & x \in \Omega, \\ & \nabla\varphi \cdot \mathbf{n} = \nabla\phi^* \cdot \mathbf{n} = 0, & x \in \partial\Omega. \end{aligned}$$

Owing to $\zeta^* < 0$ from Lemma 2.3(iv), the above system admits a unique solution φ^* . Adding the two equations of (2.16) with $(\zeta^*, \phi^*, \varphi^*)$ and integrating the resulting equation over Ω yields

$$\zeta^* \int_{\Omega} (\varphi^* + \phi^*) dx = 0,$$

which indicates that $\int_{\Omega} (\varphi^* + \phi^*) dx = 0$. To sum up, system (2.16) has a solution $(\zeta^*, \phi^*, \varphi^*)$, where $\zeta^* < 0$ and $\phi^* > 0$ on Ω . Thus, the DFE is linearly unstable. According to [9], the DFE is unstable. This finishes the proof. \square

LEMMA 2.5. *If $\mathcal{R}_0 < 1$, then the solution $(S(t, \cdot), I(t, \cdot)) \rightarrow (\tilde{S}_0, 0)$ in $C(\Omega)$ as $t \rightarrow \infty$.*

Proof. By the second equation of system (1.6), we obtain

$$\begin{aligned} I_t &\leq \nabla \cdot (g(r^{-1}(x))\nabla I) + (\beta(x) - r(x))I, & t > 0, x \in \Omega, \\ \nabla I \cdot \mathbf{n} &= 0, & t > 0, x \in \partial\Omega, \\ I(x, 0) &= I_0(x) \geq 0, & x \in \Omega. \end{aligned}$$

Define an auxiliary function $\psi(t, \cdot) := Pe^{-\zeta^*t}\phi^*(\cdot)$, wherein $\zeta^*, \phi^* > 0$ are determined by Lemma 2.2 and P is a sufficiently large constant satisfying $\psi(0, x) \geq I(0, x)$ for any $x \in \Omega$. Through simple calculations, we obtain

$$\begin{aligned} \psi_t &= \nabla \cdot (g(r^{-1}(x))\nabla \psi) + (\beta(x) - r(x))\psi, & t > 0, x \in \Omega, \\ \nabla \psi \cdot \mathbf{n} &= 0, & t > 0, x \in \partial\Omega, \\ \psi(0, x) &\geq I(0, x) \geq 0, & x \in \Omega. \end{aligned}$$

Utilizing the comparison principle gives $I(t, x) \leq \psi(t, x)$ for all $t > 0$ and $x \in \Omega$. Then $I(t, x)$ converges to zero as t tends to ∞ owing to the fact that $\psi(t, \cdot) \rightarrow 0$ when $t \rightarrow \infty$ in Ω .

Next, we show $S(t, x) \rightarrow \tilde{S}_0$ as $t \rightarrow \infty$, $x \in \Omega$. Combining the above analysis and the assumptions that β and r are continuous functions, from the first equation of (1.6), there is a constant $M_1 > 0$ such that

$$|S_t - \nabla \cdot (f(\beta(x))\nabla S)| \leq M_1 e^{-\zeta^*t}$$

for $t > 0$, $x \in \Omega$. Then $|S_t - \nabla \cdot (f(\beta(x))\nabla S)| \rightarrow 0$ when $t \rightarrow \infty$, $x \in \Omega$. Thus, via using the boundary condition $\nabla S \cdot \mathbf{n} = 0$ on $\partial\Omega$, one can see that $S(t, \cdot)$ converges to a positive constant as $t \rightarrow \infty$ in Ω . Set

$$S(t, x) = u_1(t) + u_2(t, x),$$

where $u_1(t) = \frac{1}{|\Omega|} \int_{\Omega} S(t, x) dx$. It is straightforward to verify that

$$\frac{du_1(t)}{dt} = \frac{1}{|\Omega|} \int_{\Omega} (S_t(t, x) - \nabla \cdot (f(\beta(x))\nabla S)) dx$$

which implies that there is a constant $M_2 > 0$ such that $|du_1(t)/dt| \leq M_2 e^{-\zeta^*t}$, $t > 0$. Moreover, one has

$$\begin{aligned} \partial_t u_2 &= \nabla \cdot (f(\beta(x))\nabla u_2) + k(t, x), & t > 0, x \in \Omega, \\ \nabla u_2 \cdot \mathbf{n} &= 0, & t > 0, x \in \partial\Omega, \end{aligned}$$

where

$$k(t, x) = \left(-\frac{\beta(x)S}{S+I} + r(x) \right) I - \frac{du_1(t)}{dt}.$$

With the aid of the above discussions, we obtain

$$|k(t, x)| \leq M_3 e^{-\zeta^* t}, \quad x \in \Omega, \text{ for some constant } M_3 > 0.$$

Since $\int_{\Omega} (S(t, x) + I(t, x)) dx = N_0$ and $I(t, \cdot) \rightarrow 0, t \rightarrow \infty$, we have

$$u_1(t) = \frac{1}{|\Omega|} \int_{\Omega} S(t, x) dx \rightarrow \frac{N_0}{|\Omega|} = \tilde{S}_0 \text{ as } t \rightarrow \infty.$$

Choose $0 = \zeta^0 \leq \zeta^1 \leq \dots$ representing the eigenvalues of the operator $-\nabla \cdot (f \nabla)$ with homogeneous Neumann boundary condition, and the corresponding normalized eigenfunctions $\{\omega_l(x) : l = 0, 1, \dots, x \in \Omega\}$. Then there exist two sequences of functions $b_l(t)$ and $k_l(t), l = 0, 1, \dots$, such that

$$u_2(t, x) = \sum_{l=0}^{\infty} b_l(t) \omega_l(x) \text{ and } k(t, x) = \sum_{l=0}^{\infty} k_l(t) \omega_l(x).$$

Since $|k_l(t)| \leq M_4 e^{-\zeta^{**} t}, t > 0$, for some $M_4 > 0$ and $\int_{\Omega} u_2(t, x) dx \equiv 0$, it follows there exists a constant $M_5 > 0$ such that $|b_l(t)| \leq M_5 e^{-\zeta^{**} t}, t > 0$, where $\zeta^{**} := \min\{\zeta^*, \zeta^1\}$. Hence, $u_2(t, \cdot) \rightarrow 0$ as $t \rightarrow \infty$ on Ω . Thus, $S(t, x) \rightarrow \tilde{S}_0$ as $t \rightarrow \infty, x \in \Omega$. This ends the proof. \square

Combining with Lemmas 2.3–2.5, we have the following main conclusions.

THEOREM 2.6. *Assume (H1)–(H3) hold and $\beta(\cdot) - r(\cdot)$ changes sign on Ω . Then system (1.6) has a unique DFE $E_0 = (\tilde{S}_0, 0)$ with fixed N_0 . Moreover, if $\mathcal{R}_0 < 1$, then the DFE is globally asymptotically stable, but if $\mathcal{R}_0 > 1$, then it is unstable.*

2.2. Endemic equilibrium. In this subsection, we show the existence of EE for system (1.6) when $\mathcal{R}_0 > 1$. To prove this, the following lemma is needed.

LEMMA 2.7. *Suppose $\mathcal{R}_0 > 1$. Then there exists an $\varepsilon_0 > 0$ such that the solution of (1.6) satisfies*

$$(2.19) \quad \liminf_{t \rightarrow \infty} \|(S(t, x), I(t, x)) - (\tilde{S}_0, 0)\|_{L^\infty(\Omega)} > \varepsilon_0$$

uniformly for $x \in \bar{\Omega}$.

Proof. We apply the persistence theory developed by [18] and [34] to prove this result. Denote

$$Z_0 := \{\mathbf{u}_0 \in C(\bar{\Omega}) | I_0(x) \not\equiv 0\} \text{ and } \partial Z_0 := \{\mathbf{u}_0 \in C(\bar{\Omega}) | I_0(x) \equiv 0\},$$

where $\mathbf{u}_0 = (S_0, I_0)$. Let $\Phi(t)\mathbf{u}_0 := (S(t, \cdot), I(t, \cdot)), t > 0$, be the unique solution of (1.6). It is obvious that $\Phi(t)$ is continuous and compact. According to Proposition 2.1, the map $\Phi(t)$ is pointwisely dissipative. By utilizing [8, Theorem 3.4.8] (or [34, Theorem 1.3.7]), $\Phi(t)$ admits a compact global attractor. To end the proof, one needs to examine the following claims with the help of ideas from [33].

Claim 1. $\Phi(t)Z_0 \subset Z_0$. One can easily obtain this via the strong maximum principle.

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Let U_∂ be the maximum positive invariant set of $\Phi(t)$ in ∂Z_0 , i.e., $U_\partial := \{\mathbf{u}_0 \in C(\bar{\Omega}) \mid \Phi(t)\mathbf{u}_0 \in \partial Z_0\}$. Obviously, $U_\partial = \partial Z_0$. Denote $\omega(\mathbf{u}_0)$ as the omega limit set of \mathbf{u}_0 . Set

$$\bar{U}_\partial := \bigcup_{\{\mathbf{u}_0 \in U_\partial\}} \omega(\mathbf{u}_0).$$

Claim 2. $\bar{U}_\partial = \{E_0\}$. In fact, for any $\mathbf{u}_0 \in U_\partial$, by the definition of U_∂ , one has $I(t, x) \equiv 0$ for all $x \in \Omega$, $t \geq 0$. Thus, substituting it into system (1.6) gives

$$\begin{aligned} S_t &= \nabla \cdot (f(\beta(x))\nabla S), & t > 0, & x \in \Omega, \\ \nabla S \cdot \mathbf{n} &= 0, & t > 0, & x \in \partial\Omega, \\ S(x, 0) &= S_0(x) \geq 0, & x &\in \Omega. \end{aligned}$$

Since $\int_\Omega ((S(t, x) + I(t, x))) dx = N_0$, it follows $S(t, \cdot) \rightarrow \tilde{S}_0$ uniformly in Ω as $t \rightarrow \infty$. Hence, $\bar{U}_\partial = \{E_0\}$, and then $\{E_0\}$ is an isolated and compact invariant set for $\Phi(t)$ restricted in U_∂ .

Claim 3. There is an $\varepsilon_1 > 0$, independent of initial values, such that

$$\limsup_{t \rightarrow \infty} \|\Phi(t)\mathbf{u}_0 - (\tilde{S}_0, 0)\|_{L^\infty(\Omega)} > \varepsilon_1.$$

For any $\hat{\varepsilon}_1 > 0$, by contradiction, there exists a $\hat{\mathbf{u}}_0 = (\check{S}_0(x), \check{I}_0(x))$ such that

$$(2.20) \quad \limsup_{t \rightarrow \infty} \|\Phi(t)\hat{\mathbf{u}}_0 - (\tilde{S}_0, 0)\|_{L^\infty(\Omega)} \leq \hat{\varepsilon}_1,$$

where $\Phi(t)\hat{\mathbf{u}}_0 = (\check{S}(t, \cdot), \check{I}(t, \cdot))$.

Take a sufficiently small constant $\varepsilon_2 > 0$. Let $\zeta^*(\varepsilon_2)$ be the principal eigenvalue of the eigenvalue problem

$$\begin{aligned} \nabla \cdot (g(r^{-1}(x))\nabla \tilde{\phi}^*) + \left(\beta(x)\frac{\tilde{S}_0 - \varepsilon_2}{\tilde{S}_0} - r(x)\right)\tilde{\phi}^* + \zeta^*(\varepsilon_2)\tilde{\phi}^* &= 0, & x &\in \Omega, \\ \nabla \tilde{\phi}^* \cdot \mathbf{n} &= 0, & x &\in \partial\Omega, \end{aligned}$$

wherein $\tilde{\phi}^*$ is the corresponding positive eigenfunction. Since $\mathcal{R}_0 > 1$, it follows from Lemma 2.3 that $\zeta^* < 0$; here ζ^* is the eigenvalue of (2.6). Note that $\zeta^*(\varepsilon_2) \rightarrow \zeta^* < 0$ as $\varepsilon_2 \rightarrow 0$. Thus, one can choose a sufficiently small ε_2 such that $\zeta^*(\varepsilon_2) < 0$. By the arbitrariness of $\hat{\varepsilon}_1$, we set $\hat{\varepsilon}_1 = \varepsilon_2$. From (2.20), there exists a $t_0^* > 0$ such that $\check{S}(t, \cdot) \geq \tilde{S}_0 - \varepsilon_2$ and $\check{I}(t, \cdot) \leq \varepsilon_2$ for any $t \geq t_0^*$ and $x \in \Omega$. Consequently, we have

$$\frac{\check{S}\check{I}}{\check{S} + \check{I}} \geq \frac{(\tilde{S}_0 - \varepsilon_2)\check{I}}{\tilde{S}_0 - \varepsilon_2 + \check{I}} \geq \frac{(\tilde{S}_0 - \varepsilon_2)\check{I}}{\tilde{S}_0 - \varepsilon_2 + \varepsilon_2} = \frac{(\tilde{S}_0 - \varepsilon_2)\check{I}}{\tilde{S}_0}$$

for all $t \geq t_0^*$ and $x \in \Omega$.

Moreover, by Proposition 2.1 and the strong maximum principle, there is a $\delta_0 > 0$ small enough such that $\hat{I}(t_0^*, x) \geq \delta_0\tilde{\phi}^*$. One can verify that $\hat{I}_h(t, \cdot)$ is a supersolution of the following system:

$$(2.21) \quad \begin{aligned} \hat{I}_t &= \nabla \cdot (g(r^{-1}(x))\nabla \hat{I}) + \left(\beta(x)\frac{\tilde{S}_0 - \varepsilon_2}{\tilde{S}_0} - r(x)\right)\hat{I} = 0, & t > t_0^*, & x \in \Omega, \\ \nabla \hat{I} \cdot \mathbf{n} &= 0, & t > t_0^*, & x \in \partial\Omega, \\ \hat{I}(t_0^*, x) &= \delta_0\tilde{\phi}^*, & x &\in \Omega. \end{aligned}$$

Note that $\delta_0 e^{-\zeta^*(\varepsilon_2)(t-t_0^*)} \tilde{\phi}^*(\cdot)$ is a solution of (2.21) and $\zeta^*(\varepsilon_2) < 0$. Then, one has

$$\hat{I}(t, x) \geq \delta_0 e^{-\zeta^*(\varepsilon_2)(t-t_0^*)} \tilde{\phi}^*(x) \rightarrow \infty \text{ as } t \rightarrow \infty \text{ for any } x \in \Omega,$$

which contradicts (2.20). Therefore, Claim 3 is true which implies that $\{E_0\}$ is an isolated invariant set for $\Phi(t)$, and $W^S(\{E_0\}) \cap Z_0 = \emptyset$, where $W^S(\{E_0\})$ is the stable set of $\{E_0\}$ w.r.t. $\Phi(t)$.

Combining with Claims 1–3 and [34, Theorem 1.3.1], $\Phi(t)$ is uniformly persistent. Accordingly, (2.19) is true. This completes the proof. \square

Remark 2.2. Lemma 2.7 indicates that the disease will persist.

In a summary, we have the following main conclusion.

THEOREM 2.8. *Suppose (H1)–(H3) hold and $\mathcal{R}_0 > 1$. Then system (1.6) has at least one EE $(S^*(x), I^*(x))$ satisfying $S^*(x), I^*(x) > 0$ on Ω .*

Proof. By Lemma 2.7, system (1.6) is uniformly persistent if $\mathcal{R}_0 > 1$. Thus, applying [18, Theorem 4.7], there is at least one endemic steady state $(S^*(x), I^*(x))$ meeting $S^*(x), I^*(x) > 0$ for $x \in \Omega$ for (1.6). \square

3. The epidemic model with random diffusion. The goal of this section is to investigate the existence and stability of DFE and EE for model (1.5) with random diffusion. Similarly to Proposition 2.1, we have the well-posedness of (1.5) as follows.

PROPOSITION 3.1. *For any $(S_0(x), I_0(x)) \in \mathbb{X}$, system (1.5) has a unique positive solution $(S(t, x), I(t, x))$ fulfilling*

$$(S(t, x), I(t, x)) \in C^{1,2}((0, \infty) \times \bar{\Omega}) \times C^{1,2}((0, \infty) \times \bar{\Omega}).$$

Furthermore, there is a constant $\hat{C} > 0$ independent of the initial values, and $\hat{T}_0 > 0$ such that the solution $(S(t, x), I(t, x))$ satisfies

$$\|S(t, x)\|_{L^\infty(\Omega)} + \|I(t, x)\|_{L^\infty(\Omega)} \leq \hat{C} \text{ for all } t > \hat{T}_0.$$

3.1. Disease-free equilibrium. To discuss the DFE, adding the two equations in (1.5), and then integrating the above equality over Ω yields

$$\partial_t \int_{\Omega} (S(t, x) + I(t, x)) dx = \nabla(f(\beta(x))S + g(r^{-1}(x))I) \cdot \mathbf{n}|_{\partial\Omega} = 0,$$

because $\nabla(f(\beta(x))S) \cdot \mathbf{n} = \nabla(g(r^{-1}(x))I) \cdot \mathbf{n} = 0$ on $\partial\Omega$. Hence,

$$\int_{\Omega} (S(t, x) + I(t, x)) dx = \bar{N}_0, \quad t > 0, \text{ for some constant } \bar{N}_0 > 0.$$

In particular, $\bar{N}_0 = \int_{\Omega} (S_0(x) + I_0(x)) dx$.

LEMMA 3.2. *System (1.5) has a unique DFE*

$$\hat{E}_0 = (\hat{S}(x), 0) = \left(\frac{\bar{N}_0}{f(\beta(x)) \int_{\Omega} \frac{1}{f(\beta(x))} dx}, 0 \right),$$

where $\bar{N}_0 = \int_{\Omega} (S_0(x) + I_0(x)) dx > 0$.

Proof. Suppose that $(\hat{S}(x), 0)$ is a DFE of system (1.5). Then, from the first equation of (1.5), we obtain $\Delta(f(\beta(x))\hat{S}(x)) = 0$. From $\nabla(f(\beta(x))S(x)) \cdot \mathbf{n} = 0$,

it follows that $f(\beta(x))\hat{S}(x) = C$ for some constant C . Then, $\hat{S}(x) = C/f(\beta(x))$. Furthermore, noting that $\int_{\Omega} \hat{S}(x)dx = \bar{N}_0$, we have $C = \bar{N}_0 / \int_{\Omega} (1/f(\beta(x)))dx$. Thus, one gets

$$\hat{S}(x) = \frac{\bar{N}_0}{f(\beta(x)) \int_{\Omega} \frac{1}{f(\beta(x))} dx} > 0 \text{ for all } x \in \Omega,$$

which completes the proof. \square

LEMMA 3.3. Define $g(r^{-1}(\cdot)) = \eta g_1(r^{-1}(\cdot))$, where η and g_1 are a positive constant and $C^3(\bar{\Omega})$ function, respectively, and

$$\lambda^* = \inf_{\Psi \in W^{1,2}(\Omega), \Psi \neq 0} \left\{ \frac{\eta \int_{\Omega} |\nabla \Psi|^2 dx + \int_{\Omega} \frac{r(x) - \beta(x)}{g_1(r^{-1}(x))} \Psi^2 dx}{\int_{\Omega} \frac{1}{g_1(r^{-1}(x))} \Psi^2 dx} \right\}.$$

If $\beta(\cdot) - r(\cdot)$ changes sign on Ω , then

- (i) λ^* is a strictly monotonically increasing function w.r.t. η ;
- (ii) $\lambda^* \rightarrow \min_{x \in \bar{\Omega}} \{ (r(x) - \beta(x))g_1^{-1}(r^{-1}(x)) \}$ as $\eta \rightarrow 0$ for fixed g_1 ;
- (iii) $\lambda^* \rightarrow \int_{\Omega} (r(x) - \beta(x))g_1^{-1}(r^{-1}(x))dx / \int_{\Omega} g_1^{-1}(r^{-1}(x))dx$ as $\eta \rightarrow +\infty$ for fixed g_1 ;
- (iv) if $\int_{\Omega} \beta(x)g_1^{-1}(r^{-1}(x))dx \geq \int_{\Omega} r(x)g_1^{-1}(r^{-1}(x))dx$, then $\lambda^* < 0$ for all $\eta > 0$;
- (v) if $\int_{\Omega} \beta(x)g_1^{-1}(r^{-1}(x))dx < \int_{\Omega} r(x)g_1^{-1}(r^{-1}(x))dx$, then the equation $\lambda^*(\eta) = 0$ has a unique positive root denoted by $\bar{\eta}^*$. Furthermore, if $\eta < \bar{\eta}^*$, then $\lambda^* < 0$, and if $\eta > \bar{\eta}^*$, then $\lambda^* > 0$.

Proof. Linearizing system (1.5) around $(\hat{S}(x), 0)$ yields

$$\begin{aligned} \xi_t &= \Delta(g(r^{-1}(x))\xi) + (\beta(x) - r(x))\xi, & t > 0, x \in \Omega, \\ \nabla(g(r^{-1}(x))\xi) \cdot \mathbf{n} &= 0, & t > 0, x \in \partial\Omega. \end{aligned}$$

Suppose that $\xi(t, x) = e^{-\lambda t} \vartheta(x)$. Then, one obtains

$$(3.1) \quad \begin{aligned} \Delta(g(r^{-1}(x))\vartheta) + (\beta(x) - r(x))\vartheta + \lambda\vartheta &= 0, & x \in \Omega, \\ \nabla(g(r^{-1}(x))\vartheta) \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned}$$

Set $\Psi(x) = g(r^{-1}(x))\vartheta(x)$. Thus, from $g(r^{-1}(\cdot)) := \eta g_1(r^{-1}(\cdot))$, system (3.1) can be reduced to

$$(3.2) \quad \begin{aligned} \eta\Delta\Psi + \frac{(\beta(x) - r(x))}{g_1(r^{-1}(x))}\Psi + \lambda\frac{\Psi}{g_1(r^{-1}(x))} &= 0, & x \in \Omega, \\ \nabla\Psi \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned}$$

Similarly to the analysis of Lemma 2.2, there exists a unique eigenvalue λ^* whose corresponding eigenfunction Ψ^* is positive on Ω . Observe that (λ^*, Ψ^*) satisfies

$$(3.3) \quad \begin{aligned} \eta\Delta\Psi^* + \frac{(\beta(x) - r(x))}{g_1(r^{-1}(x))}\Psi^* + \lambda^*\frac{\Psi^*}{g_1(r^{-1}(x))} &= 0, & x \in \Omega, \\ \nabla\Psi^* \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned}$$

Therefore, λ^* is determined by the variational characterization

$$\lambda^* = \inf_{\Psi \in W^{1,2}(\Omega), \Psi \neq 0} \left\{ \frac{\eta \int_{\Omega} |\nabla \Psi|^2 dx + \int_{\Omega} \frac{r(x) - \beta(x)}{g_1(r^{-1}(x))} \Psi^2 dx}{\int_{\Omega} \frac{1}{g_1(r^{-1}(x))} \Psi^2 dx} \right\},$$

after which the proof is similar to Lemma 2.2. □

To analyze the stability of $(\hat{S}(x), 0)$, we characterize the basic reproduction number \mathcal{R}_0 for system (1.5). Consider the following eigenvalue problem:

$$(3.4) \quad \begin{aligned} \lambda \frac{\beta(x)}{g_1(r^{-1}(x))} \Psi &= -\eta \Delta \Psi + \frac{r(x)}{g_1(r^{-1}(x))} \Psi, & x \in \Omega, \\ \nabla \Psi \cdot \mathbf{n} &= 0, & x \in \partial \Omega. \end{aligned}$$

LEMMA 3.4. *Let λ_0 be the positive eigenvalue of (3.4) with a positive eigenfunction. Then λ_0 is unique and defined by*

$$\lambda_0 = \inf_{\Psi \in W^{1,2}(\Omega), \Psi \neq 0} \left\{ \frac{\eta \int_{\Omega} |\nabla \Psi|^2 dx + \int_{\Omega} \frac{r(x)}{g_1(r^{-1}(x))} \Psi^2 dx}{\int_{\Omega} \frac{\beta(x)}{g_1(r^{-1}(x))} \Psi^2 dx} \right\}.$$

Furthermore, the basic reproduction number \mathcal{R}_0 of (1.5) is defined by

$$\mathcal{R}_0 = \sup_{\Psi \in W^{1,2}(\Omega), \Psi \neq 0} \left\{ \frac{\int_{\Omega} \frac{\beta(x)}{g_1(r^{-1}(x))} \Psi^2 dx}{\eta \int_{\Omega} |\nabla \Psi|^2 dx + \int_{\Omega} \frac{r(x)}{g_1(r^{-1}(x))} \Psi^2 dx} \right\}.$$

Moreover, if $\beta(\cdot) - r(\cdot)$ changes sign on Ω , then \mathcal{R}_0 satisfies the following properties:

- (i) $\mathcal{R}_0 = 1/\lambda_0$ is monotone decreasing w.r.t. η ;
- (ii) $\mathcal{R}_0 \rightarrow \max_{x \in \Omega} \{\beta(x)/r(x)\}$ as $\eta \rightarrow 0$ for fixed g_1 ;
- (iii) $\mathcal{R}_0 \rightarrow \int_{\Omega} \beta(x)g_1^{-1}(r^{-1}(x))dx / \int_{\Omega} r(x)g_1^{-1}(r^{-1}(x))dx$ as $\eta \rightarrow +\infty$ for fixed g_1 ;
- (iv) $\text{sign}(1 - \mathcal{R}_0) = \text{sign } \lambda^*$;
- (v) $\min_{x \in \bar{\Omega}} \{\beta(x)/r(x)\} \leq \mathcal{R}_0 \leq \max_{x \in \bar{\Omega}} \{\beta(x)/r(x)\}$.

Proof. The proof follows the same logic as Lemma 2.3. □

The proof of the following results resemble that of Lemmas 2.4–2.5, we omit the details here.

LEMMA 3.5. *If $\mathcal{R}_0 < 1$, then the DEF \hat{E}_0 is stable; If $\mathcal{R}_0 > 1$, then the DEF \hat{E}_0 is unstable.*

LEMMA 3.6. *If $\mathcal{R}_0 < 1$, then the solution $(S(t, \cdot), I(t, \cdot)) \rightarrow (\hat{S}(\cdot), 0)$ in $C(\Omega)$ as $t \rightarrow \infty$.*

Combining with Lemmas 3.2–3.6, we have the following main results.

THEOREM 3.7. *Assume (H1)–(H3) hold and $\beta(\cdot) - r(\cdot)$ changes sign on Ω . Then there exists a unique DFE $\hat{E}_0 = (\hat{S}(x), 0)$ with fixed \bar{N}_0 . Furthermore, if $\mathcal{R}_0 < 1$, then the DFE is globally asymptotically stable, but if $\mathcal{R}_0 > 1$, then it is unstable.*

3.2. Endemic equilibrium. In this subsection, we explore the existence and uniqueness of EE for (1.5) when $\mathcal{R}_0 > 1$. The steady states of (1.5) satisfy

$$(3.5) \quad \begin{aligned} \Delta(f(\beta(x))S) - \left(\frac{\beta(x)S}{S+I} - r(x) \right) I &= 0, & x \in \Omega, \\ \Delta(g(r^{-1}(x))I) + \left(\frac{\beta(x)S}{S+I} - r(x) \right) I &= 0, & x \in \Omega, \\ \nabla(f(\beta(x))S) \cdot \mathbf{n} = \nabla(g(r^{-1}(x))I) \cdot \mathbf{n} &= 0, & x \in \partial \Omega. \end{aligned}$$

Then it follows that

$$(3.6) \quad \begin{aligned} f(\beta(x))S + g(r^{-1}(x))I &= \mu, & x \in \Omega, \\ \Delta(g(r^{-1}(x))I) + \left(\beta(x) - r(x) - \frac{\beta(x)I}{S+I}\right)I &= 0, & x \in \Omega, \\ \nabla(f(\beta(x))S) \cdot \mathbf{n} = \nabla(g(r^{-1}(x))I) \cdot \mathbf{n} &= 0, & x \in \partial\Omega, \\ \int_{\Omega} (S + I)dx &= \bar{N}_0 \end{aligned}$$

for some constant $\mu > 0$. Setting

$$u = \frac{f(\beta(x))S}{\mu} \quad \text{and} \quad v = \frac{g(r^{-1}(x))I}{\mu},$$

and substituting them into (3.6) gives

$$(3.7) \quad \begin{aligned} u + v &= 1, & x \in \Omega, \\ \Delta v + \mathcal{K}(x, v)v &= 0, & x \in \Omega, \\ \nabla v \cdot \mathbf{n} &= 0, & x \in \partial\Omega, \\ \mu &= \bar{N}_0 \cdot \left[\int_{\Omega} \left(\frac{u}{f(\beta(x))} + \frac{v}{g(r^{-1}(x))} \right) dx \right]^{-1}, \end{aligned}$$

where

$$\mathcal{K}(x, v) = \frac{1}{g(r^{-1}(x))} \left(\beta(x) - r(x) - \frac{\beta(x)f(\beta(x))v}{g(r^{-1}(x))(1-v) + f(\beta(x))v} \right).$$

Clearly, if (u, v) is a positive solution of (3.7), then $(\frac{\mu u}{f(\beta(x))}, \frac{\mu v}{g(r^{-1}(x))})$ is the EE of model (1.5).

LEMMA 3.8. *Assume $\mathcal{R}_0 > 1$. Then system (3.7) admits a nonnegative solution (u, v) satisfying $u(\cdot), v(\cdot) \in C^2(\bar{\Omega})$ and $v(\cdot) \not\equiv 0$ on Ω . Furthermore, the solution is unique, $u(x) > 0$, and $0 < v(x) < 1$ for all $x \in \Omega$.*

Proof. Define $\mathcal{H}(v) := \Delta v + \mathcal{K}(x, v)v$. Then, by (3.7) we obtain

$$(3.8) \quad \begin{aligned} \mathcal{H}(v) &= 0, & x \in \Omega, \\ \nabla v \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned}$$

In the following, we show that there is a sufficiently small constant $\sigma > 0$ such that $\underline{v}(x) = \sigma\Psi^*(x)$ and $\bar{v}(x) \equiv 1$ are the sub- and super-solutions of system (3.8), respectively, where Ψ^* is the eigenfunction of the corresponding eigenvalue λ^* of system (3.3). By Lemma 3.4, $\lambda^* < 0$ recalling that $\mathcal{R}_0 > 1$. Denote

$$\mathcal{K}_1(x, v) = \frac{f(\beta(x))v}{g(r^{-1}(x))(1-v) + f(\beta(x))v}.$$

It is not difficult to verify $\partial_v \mathcal{K}_1(\cdot, v) > 0$ for any v on Ω . That is, $\mathcal{K}_1(x, v)$ is an increasing function of v and $\mathcal{K}_1(\cdot, v) \in [0, 1]$ as $v \in [0, 1]$. According to (3.3) and (3.8), we obtain

$$\mathcal{H}(\underline{v}) = \frac{\sigma\Psi^*}{g(r^{-1}(x))} [-\lambda^* - \beta(x)\mathcal{K}_1(x, \sigma\Psi^*)].$$

Since \mathcal{K}_1 is increasing and $\mathcal{K}_1(x, 0) = 0$, it follows from the continuity of \mathcal{K}_1 that there exists a $\sigma > 0$ sufficiently small so that $\beta(x)\mathcal{K}_1(x, \sigma\Psi^*) < -\lambda^*$ owing to the positivity of $\beta(x)$. Hence, $\mathcal{H}(\underline{v}) > 0$ for $\sigma > 0$ small enough. By $\nabla \underline{v} \cdot \mathbf{n} = 0$ on $\partial\Omega$, we can see that $\underline{v}(x) = \sigma\Psi^*(x)$ is a subsolution of (3.8). In addition, by direct calculations, we have $\mathcal{H}(\bar{v}) = -r(x)/g(r^{-1}(x)) < 0$, $x \in \Omega$ and $\nabla \bar{v} \cdot \mathbf{n} = 0$, $x \in \partial\Omega$. This indicates that $\bar{v}(x) \equiv 1$ is a supersolution of (3.8). For a sufficiently small $\sigma > 0$, $\underline{v} < \bar{v}$ holds. By the definition of sub- and super-solution [27], one has $\underline{v}(x) \leq v(x) \leq \bar{v}(x)$, $x \in \Omega$. Thus, the solutions of (3.8) satisfy $0 < v(x) \leq 1$ on Ω . To prove $v(x) < 1$ on Ω , by contradiction, we assume $v(x_0) = 1$ for some $x_0 \in \Omega$. Then $\Delta v(x_0) \leq 0$ due to the fact $v(x_0) = \max_{x \in \Omega} v(x)$. Hence, we have

$$0 = \mathcal{H}(v(x_0)) = \Delta v(x_0) - \frac{r(x)}{g(r^{-1}(x))} < 0,$$

which is a contradiction. Therefore, $0 < v(x) < 1$ for all $x \in \Omega$.

To cope with the uniqueness of v , suppose system (3.8) has two pairs of solutions, denoted by (u^1, v^1) and (u^2, v^2) , satisfying $v^1 \not\equiv v^2$. From the first equation of (3.7), one has $0 < v^1, v^2 \leq 1$, $x \in \Omega$. By the strong maximum principle, $0 < v^1, v^2 \leq 1$, $x \in \bar{\Omega}$. From the above analysis, we have $\underline{v} \leq v^1(x), v^2(x) \leq \bar{v}$, $x \in \bar{\Omega}$. Let $v^a(x)$ and $v^b(x)$ be the minimal and maximal solutions of (3.8) satisfying $v^a \not\equiv v^b$. Clearly, $\sigma\Psi^* \leq v^a, v^b \leq 1$, $x \in \bar{\Omega}$. Due to $v^1 \not\equiv v^2$, $v^a \leq v^b$ with $v^a \not\equiv v^b$. Applying the strong maximum principle yields that $v^a(x) < v^b(x)$, $x \in \bar{\Omega}$. Substituting v^a and v^b into (3.8), we obtain

$$(3.9) \quad \begin{aligned} \Delta v^a + \mathcal{K}(x, v^a)v^a &= 0, & x \in \Omega, \\ \nabla v^a \cdot \mathbf{n} &= 0, & x \in \partial\Omega, \end{aligned}$$

and

$$(3.10) \quad \begin{aligned} \Delta v^b + \mathcal{K}(x, v^b)v^b &= 0, & x \in \Omega, \\ \nabla v^b \cdot \mathbf{n} &= 0, & x \in \partial\Omega. \end{aligned}$$

Multiplying the equations of (3.9) and (3.10) by v^b and v^a , respectively, and integrating by parts over Ω and then subtracting the resulting equations, one obtains

$$\int_{\Omega} [\mathcal{K}(x, v^a) - \mathcal{K}(x, v^b)]v^a v^b dx = 0.$$

On the other hand, it can be testified $\mathcal{K}(\cdot, v)$ is a decreasing function of v for any $x \in \Omega$. Thus, $\mathcal{K}(x, v^a) > \mathcal{K}(x, v^b)$ for $x \in \Omega$. Therefore,

$$\int_{\Omega} [\mathcal{K}(x, v^a) - \mathcal{K}(x, v^b)]v^a v^b dx > 0$$

due to $v^a, v^b > 0$ which is a contradiction. Consequently, system (3.8) possesses a unique solution (u, v) fulfilling $u > 0$ and $0 < v < 1$ on Ω . \square

By Lemma 3.8, we have the following conclusion.

THEOREM 3.9. *Suppose $\mathcal{R}_0 > 1$. Then system (3.5) admits a nonnegative solution $(S^*(x), I^*(x))$ satisfying $S^*, I^* \in C^2(\bar{\Omega})$ and $I^* \not\equiv 0$ on Ω . Furthermore, the solution is unique, $S^*(x) > 0$, and $I^*(x) > 0$ for $x \in \Omega$, and given by the following formula,*

$$(S^*(x), I^*(x)) = \left(\frac{\mu u(x)}{f(\beta(x))}, \frac{\mu v(x)}{g(r^{-1}(x))} \right),$$

where $u(\cdot)$ and $v(\cdot)$ are defined in Lemma 3.8.

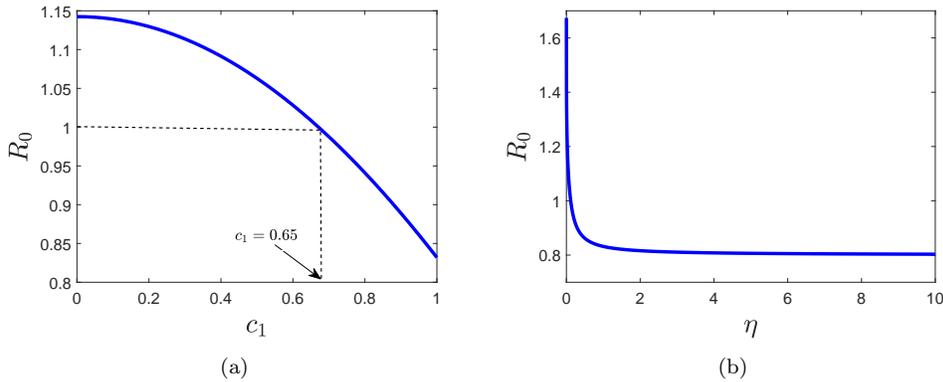


FIG. 2. (a) \mathcal{R}_0 versus c_1 when $\beta(x) = 1.7 - c_1^2x$, $r(x) = 1 + x$, $f(\beta(x)) = \beta(x)$, and $g(r^{-1}(x)) = r^{-1}(x)$, $x \in [0, 1]$; (b) \mathcal{R}_0 versus η when $\beta(x) = 1.7 - x$, $r(x) = 1 + x$, $f(\beta(x)) = \beta(x)$, and $g(r^{-1}(x)) = \eta r^{-1}(x)$, $x \in [0, 1]$.

Remark 3.1. If the diffusion rates $f(\beta)$ and $g(r^{-1})$ are constants in systems (1.5) and (1.6), the relevant results belong to the special case that was analyzed in [1].

4. Numerical computations for threshold dynamics. We first verify the threshold dynamics on the basic reproduction number \mathcal{R}_0 by numerical simulations. Our theoretical results will be verified numerically by choosing functions $\beta(\cdot)$, $r(\cdot)$, $f(\beta(\cdot))$, and $g(r^{-1}(\cdot))$. Specifically, (i) we study the threshold dynamics of systems (1.5) and (1.6) by testing two cases with $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$; (ii) we investigate the effects of \mathcal{R}_0 on the transmission rate $\beta(\cdot)$ and the diffusion rate $g(\cdot)$ to discuss how the spatial heterogeneity and the motility of infected populations affect disease dynamics. For convenience, we consider a one-dimensional space.

4.1. Threshold dynamics of system (1.6). In this subsection, we test the variation of basic reproduction number \mathcal{R}_0 for (1.6) on β and g , and explore the dynamics of (1.6). For numerical examples, we take

$$(4.1) \quad \beta(x) = 1.7 - c_1^2x, \quad r(x) = 1 + x, \quad f(\beta(x)) = \beta(x), \quad \text{and} \quad g(r^{-1}(x)) = \eta r^{-1}(x),$$

and compute the basic reproduction number \mathcal{R}_0 for $0 < c_1 < 1$ and $0 < \eta < 10$. From Figure 2(a), it follows that \mathcal{R}_0 is a decreasing function of c_1 and $\mathcal{R}_0 < 1$ when $c_1 > 0.65$, which implies that we should take necessary isolation measures to make $c_1 > 0.65$ to control the spread of disease. From Figure 2(b), it can be seen that \mathcal{R}_0 is a decreasing function of η . More precisely, $\mathcal{R}_0 \rightarrow 1.6738 \approx \max_{0 < x < 1} \{\beta(x)/r(x)\}$ as $\eta \rightarrow 0$ and $\mathcal{R}_0 \rightarrow 0.8035 \approx \int_0^1 \beta(x)dx / \int_0^1 r(x)dx$ as $\eta \rightarrow 10$, which demonstrate the results in Lemma 2.3.

For the case with $c_1 = 1$ and $\eta = 1$, it is easy to compute $\mathcal{R}_0 = 0.8322 < 1$. By considering one unit of spatial length as one kilometer, we choose the initial densities $S_0(x) = 999$ and $I_0(x) = 1$, $x \in (0, 1)$. The solution is given in Figure 3 for the case $\mathcal{R}_0 < 1$. The infected population tends to zero globally and the susceptible population tends to $\tilde{S}_0 = 1000$, which is consistent with Theorem 2.6.

Next, we choose $\beta(x) = 3 - x$ with other parameters in (4.1) unchanged. Then, we obtain $\mathcal{R}_0 = 1.7008 > 1$ and the solution is given in Figure 4 which describes that

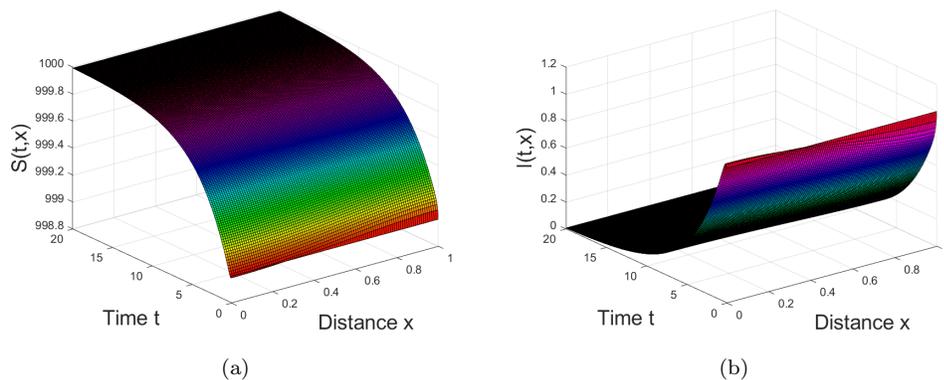


FIG. 3. The dynamics of susceptible and infected populations of (1.6) when $\mathcal{R}_0 = 0.8322 < 1$. (a) $S(t, x)$; (b) $I(t, x)$.

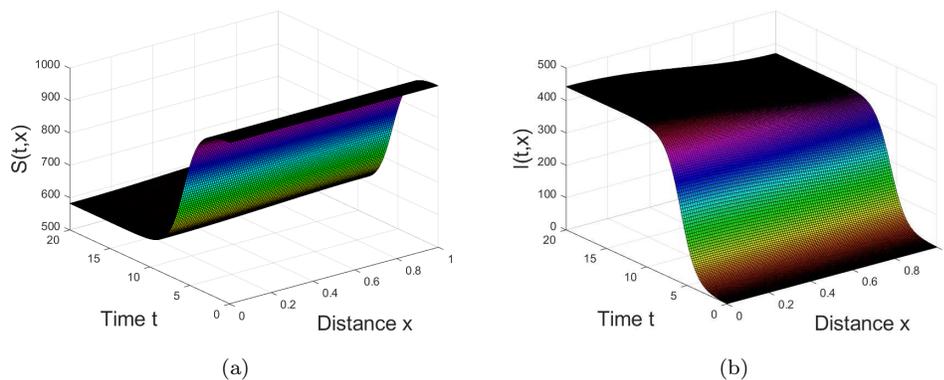


FIG. 4. The dynamics of susceptible and infected populations of (1.6) when $\mathcal{R}_0 = 1.7008 > 1$. (a) $S(t, x)$; (b) $I(t, x)$.

the system (1.6) has an EE when $\mathcal{R}_0 = 1.7008$. This indicates that the disease will persist.

4.2. Threshold dynamics of system (1.5). In this subsection, we test the variation of basic reproduction number \mathcal{R}_0 for (1.5) on β and g , and explore the dynamics of (1.5). For numerical examples, we take the same parameter values as in (4.1).

We sketch the graphs of \mathcal{R}_0 for the system (1.5). In this model, we still have that \mathcal{R}_0 is a decreasing function of c_1 and η . The main difference in using a different diffusion law is the size of the reproduction number. Figure 5(a) shows that $\mathcal{R}_0 < 1$ when $c_1 > 0.54$, which is smaller than the critical value $c_1 = 0.65$ of the Fickian case. Figure 5(b) shows that \mathcal{R}_0 is a decreasing function of η and $\mathcal{R}_0 \rightarrow 1.6738 \approx \max_{0 < x < 1} \{\beta(x)/r(x)\}$ as $\eta \rightarrow 0$ and $\mathcal{R}_0 \rightarrow 0.7398 \approx \int_0^1 \beta(x)g_1^{-1}(r^{-1}(x))dx / \int_0^1 r(x)g_1^{-1}(r^{-1}(x))dx$ as $\eta \rightarrow 10$ (see Lemma 3.4). Note that the limit as $\eta \rightarrow 0$ is the same as the Fickian case and the limit as $\eta \rightarrow 10$ is smaller than in the Fickian case.

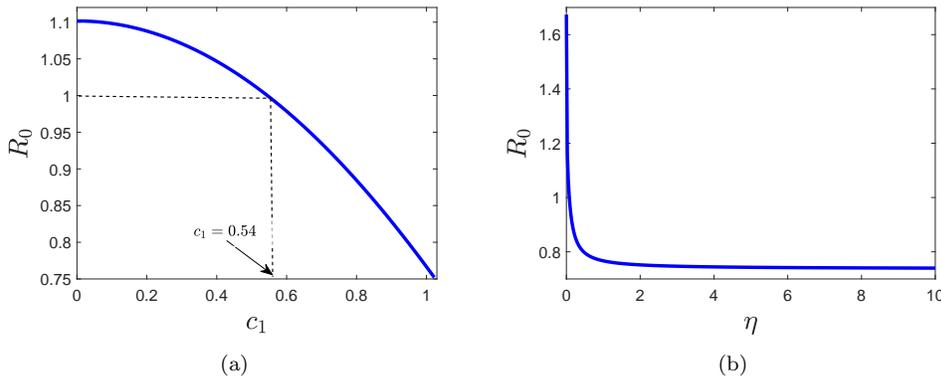


FIG. 5. (a) \mathcal{R}_0 versus c_1 when $\beta(x) = 1.7 - c_1^2 x$, $r(x) = 1 + x$, $f(\beta(x)) = \beta(x)$, and $g(r^{-1}(x)) = r^{-1}(x)$, $x \in [0, 1]$; (b) \mathcal{R}_0 versus η when $\beta(x) = 1.7 - x$, $r(x) = 1 + x$, $f(\beta(x)) = \beta(x)$, and $g(r^{-1}(x)) = \eta r^{-1}(x)$, $x \in [0, 1]$.

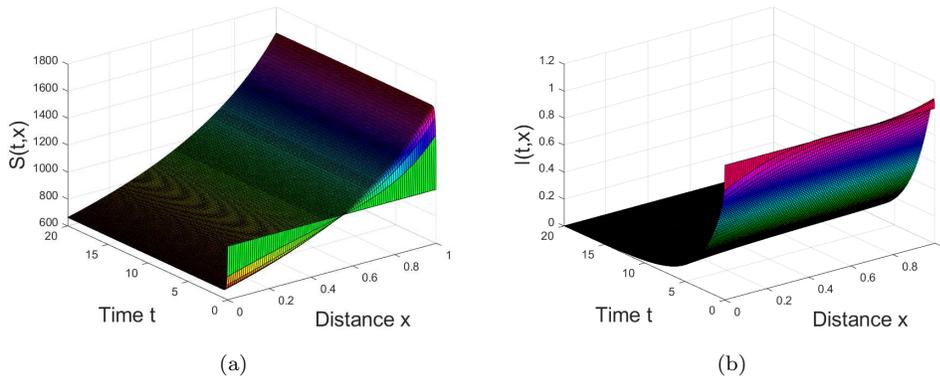


FIG. 6. The dynamics of susceptible and infected populations of (1.5) when $\mathcal{R}_0 = 0.7667 < 1$. (a) $S(t, x)$; (b) $I(t, x)$.

In what follows, we investigate the threshold dynamics of system (1.5). Without loss of generality, fix $c_1 = 1$ and $\eta = 1$ in (4.1). Direct calculations give $\mathcal{R}_0 = 0.7667 < 1$. Moreover, assume the initial densities are $S_0(x) = 999$ and $I_0(x) = 1$, $x \in (0, 1)$. Therefore, Figure 6 presents the corresponding long term behavior of system (1.5) in the case of $\mathcal{R}_0 = 0.7667 < 1$. From Figure 6, one can see that the number of infectives tends to zero and the number of susceptibles tends to the steady state level $\hat{S}(x) = \frac{1000}{(1.7-x)(\ln 17 - \ln 7)}$, which coincides with Theorem 3.7.

In addition, we choose $\beta(x) = 3 - x$ and the other parameter values are the same as Figure 6. Then one has $\mathcal{R}_0 = 1.6041 > 1$. Hence, Figure 7 shows the corresponding long term behavior of (1.5) when $\mathcal{R}_0 = 1.6041$. From Figure 7, system (1.5) admits an EE when $\mathcal{R}_0 > 1$. This implies a disease outbreak.

5. Numerical computations for segregation impacted by cognitive dispersal strategies. Disease spread in an ODE model means an increase in the number of infections under the well-mixed assumption, but not spatial spread. The PDE model is used in the paper to quantify the spatial spread of disease, where diffusion

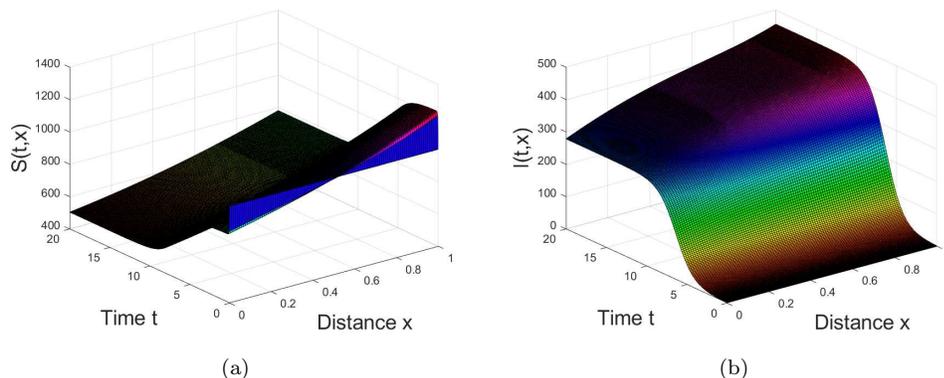


FIG. 7. The dynamics of susceptible and infected populations of (1.5) when $\mathcal{R}_0 = 1.6041 > 1$. (a) $S(t, x)$; (b) $I(t, x)$.

models the average spatial movement of individuals. In this section, we consider three examples of transmission and recovery rates, $\beta(x)$ and $r(x)$, to numerically test the impact of diffusion on the spatial spread of an infectious disease. In the first example, we will see that the heterogeneous random diffusion in the reaction-diffusion system (1.5) may segregate infected and susceptible populations and reduce the infected population. In the second example, random diffusion may reduce the infected population by using disease-free regions even if it reduces the segregation indices. In the third example, we will see that random diffusion may increase the infected population in certain situations. On the other hand, symmetric diffusion in (1.6) behaves similarly to the homogeneous diffusion with constant diffusivity.

5.1. Example 1 (segregation by dispersal). It is generally believed that the diffusion mechanism homogenizes a mixture of substances and reduces the segregation of substances. The reason for such a belief is from the nature of symmetric diffusion models. However, heterogeneous random diffusion models may segregate different substances. In the first example, it will be observed that an epidemic model with random diffusion may segregate the susceptible and the infected populations, and reduce the proportion of the infected population. The infection and the recovery rates of the first example are taken as

$$\beta(x) = 6 \cos x + 6.6, \quad r(x) = \cos x + 1.5, \quad x \in \Omega = (0, 2\pi).$$

The graphs of these two rates are given in Figure 8(a). This is an example that the patterns for $\beta(x)$ and $r(x)$ are similar, where the two have critical points at the same location. The diffusion rates are given by relations

$$f(x) = \beta(x) \quad \text{and} \quad g(x) = r^{-1}(x),$$

where their graphs are given in Figure 8(b). As we observe from the figure, the diffusion rate of the susceptible population is the highest whereas the diffusion rate of the infected population is the lowest. This situation may give extra segregation phenomena caused by the diffusion, which will be observed in the simulations. We assume the diffusivity of the infected population is considerably smaller than the one of susceptible population.

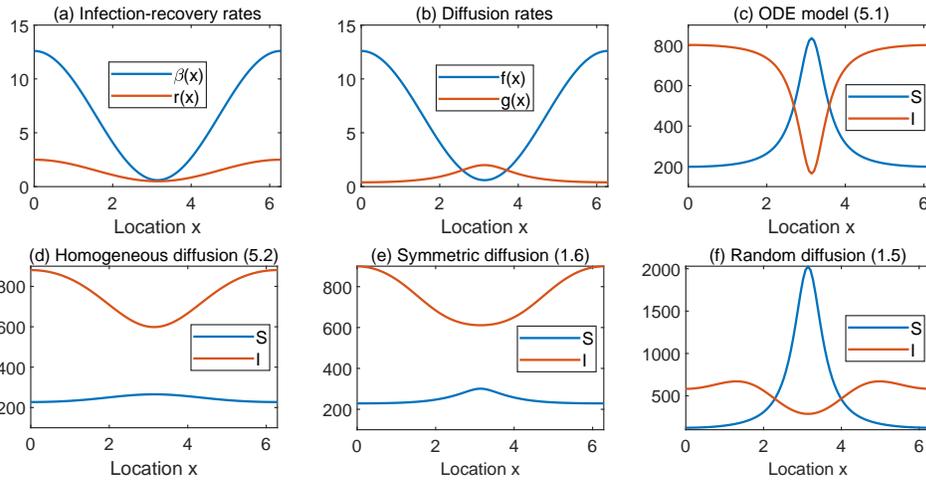


FIG. 8. *Example 1 at steady states. The infection and recovery rates have similar patterns in this example. The random diffusion segregates the two population groups and reduces the infected population.*

To compare the segregation effects of different models, we first consider a model without diffusion:

$$(5.1) \quad \begin{aligned} S_t &= -\left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, \quad x \in \Omega, \\ I_t &= \left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, \quad x \in \Omega, \\ S(x, 0) &= S_0(x) \geq 0, \quad I(x, 0) = I_0(x) \geq 0, & x \in \Omega. \end{aligned}$$

We will compare steady state solutions by solving the initial value problem for $t > 0$ large enough starting from the initial densities:

$$S_0(x) = I_0(x) = 500, \quad x \in \Omega = (0, 2\pi).$$

Note that (5.1) is an ODE model and the steady state is decided algebraically by

$$S(x) = \frac{r(x)}{\beta(x) - r(x)} I(x), \quad S(x) + I(x) = I_0(x) + S_0(x) = 1000,$$

or $I(x) = 0$ and $S(x) = 1000$. The distribution of the steady state is given in Figure 8(c). In this figure, the susceptible and infected populations are segregated due to the heterogeneity of the ratio $\frac{r(x)}{\beta(x) - r(x)}$. There are more susceptibles in a region if the ratio is large in the region and there are fewer otherwise.

Next, we compare the effects of diffusion models. The infected population will increase if the susceptible and infected populations are mixed and will decrease if the two are segregated. For comparison, we consider the homogeneous diffusion model

$$(5.2) \quad \begin{aligned} S_t &= d_S \Delta S - \left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, \quad x \in \Omega, \\ I_t &= d_I \Delta I + \left(\frac{\beta(x)S}{S+I} - r(x)\right)I, & t > 0, \quad x \in \Omega, \\ \nabla S \cdot \mathbf{n} &= 0 = \nabla I \cdot \mathbf{n}, & t > 0, \quad x \in \partial\Omega, \\ S(x, 0) &= S_0(x) \geq 0, \quad I(x, 0) = I_0(x) \geq 0, & x \in \Omega, \end{aligned}$$

TABLE 5.1
The infection fraction of Example 1 with SIs.

Epidemic models	$\frac{\int_0^{2\pi} I dx}{\int_0^{2\pi} (S+I) dx}$ asymptotically	SI- κ	SI- χ ($\times 10^5$)
Infinite-dimensional ODE model (5.1)	0.6890	0.4860	-4.0474
Homogeneous diffusion model (5.2)	0.7563	0.5125	2.1772
Symmetric diffusion model (1.6)	0.7523	0.5046	2.0808
Random diffusion model (1.5)	0.5439	0.5272	-8.8516

where the diffusivity coefficients d_S and d_I are taken as the average diffusivity

$$d_S = \frac{1}{|\Omega|} \int_{\Omega} f(x) dx \quad \text{and} \quad d_I = \frac{1}{|\Omega|} \int_{\Omega} g(x) dx.$$

The distribution of the steady state of the homogeneous diffusion model is given in Figure 8(d). The susceptible and infected populations are more mixed. As a result, the infection fraction increases. It is not surprising that homogeneous diffusion increases infection.

Figures 8(e) and 8(f) plot the distribution of the steady state of the two heterogeneous diffusion models (1.6) and (1.5), respectively. The epidemic model (1.6) with symmetric diffusion shows similar behavior to the model (5.2) with homogeneous diffusion. The main difference is in the pointing shape of the distribution of susceptible population, but the overall size is quite similar. On the other hand, the model (1.5) with random diffusion shows a different behavior. It can be observed that the segregation index (SI) between the two populations increases and the size of the infected population decreases.

In Table 5.1, the infection fraction at the steady state of each model is compared with the SIs κ and χ . The two models with homogeneous or symmetric diffusion have positive SIs $\chi(S, I)$, which implies that the two populations are poorly segregated and κ does not mean much for the two cases. The model with random diffusion and the ODE model have negative SIs $\chi(S, I)$. The index κ is larger for the random diffusion model. This suggests that the random diffusion segregates the two population groups. We can also see that the infection fraction is the smallest when random diffusion is used.

5.2. Example 2 (dispersal toward disease-free region). Dispersal of infected individuals toward susceptible ones usually increases the infected population. However, if the dispersal is toward a region with a small basic reproduction number \mathcal{R}_0 , dispersal may help to mitigate the disease. The second example is for such a case, where the infection and recovery rates are given by

$$(5.3) \quad \beta(x) = -4x + 5, \quad r(x) = 4x + 1, \quad x \in \Omega = (0, 1).$$

The graphs of these two rates are given in Figure 9(a). This is an example where the two rates $\beta(x)$ and $r(x)$ have the opposite monotonicity and intersect at $x = 0.5$. The diffusion rates are given by relations $f(x) = \beta(x)$ and $g(x) = r^{-1}(x)$, where the two diffusion rates have the same monotonicity as we can see from their graphs given in Figure 9(b). Hence, the diffusion pushes both populations toward the same direction. The diffusivity of the infected population is considerably smaller than the diffusivity

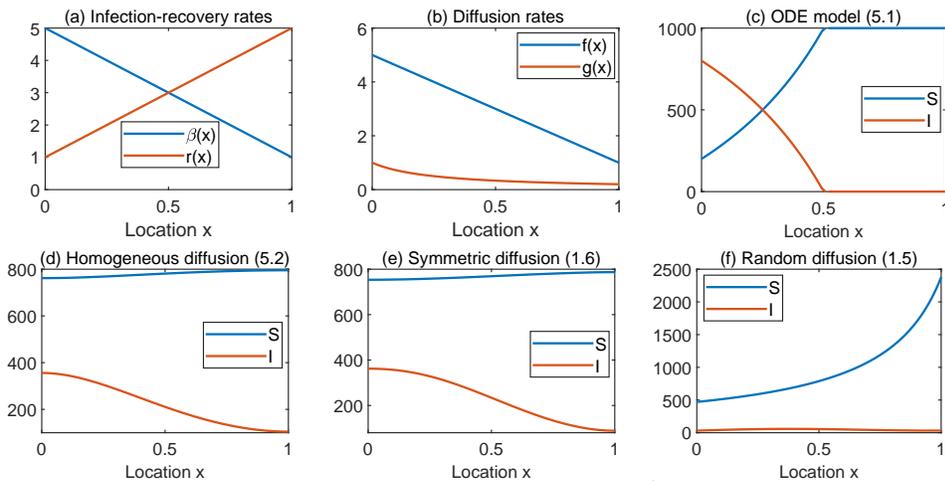


FIG. 9. Example 2 with (5.3) at steady states. The domain $x > 0.5$ is the disease-free region for the ODE model. The random diffusion pushes both populations toward the disease-free region and reduces the infected population.

TABLE 5.2
The infection fraction of Example 2 with SIs.

Epidemic models	$\frac{\int_0^1 I dx}{\int_0^1 (S+I) dx}$ asymptotically	SI- κ	SI- χ ($\times 10^6$)
Infinite-dimensional ODE model (5.1)	0.2355	0.6946	-0.6002
Homogeneous diffusion model (5.2)	0.2201	0.5598	0.2805
Symmetric diffusion model (1.6)	0.2304	0.5392	0.2735
Random diffusion model (1.5)	0.0459	0.9082	1.0311

of the susceptible population as we assumed earlier. The random diffusion will not give an extra segregation effect since f and g have the same monotonicity.

To compare the segregation effects of the previous four models, we compute the steady states for the four models numerically. Figures 9(c)–9(f) picture the distribution of the steady states of the four models. We can see that only the populations of the ODE model are segregated. However, the size of the infected population is the largest in this case. Moreover, the epidemic model (1.6) with symmetric diffusion shows the same behavior again as the model (5.2) with homogeneous diffusion. Nevertheless, the solution of model (1.5) with random diffusion shows a different behavior again. In particular, the size of the infected population is far smaller than the ones of the other three models.

To quantitatively compare the differences of the four cases, the infection fractions are given in Table 5.2 with the two SIs. In this example, the three models with diffusion have positive SIs $\chi(S, I)$, which means that the three cases are not segregated. Only the ODE model has negative SI $\chi(S, I)$. However, the first three models have similar sizes of the infected population and only the solution of (1.5) with random diffusion has a small size of infections. The reason is that (1.5) is the only model that takes advantage of the region $x > 0.5$ (high recovery rate) which has the DFE for the ODE model. In conclusion, we claim that segregation is not the only way to reduce

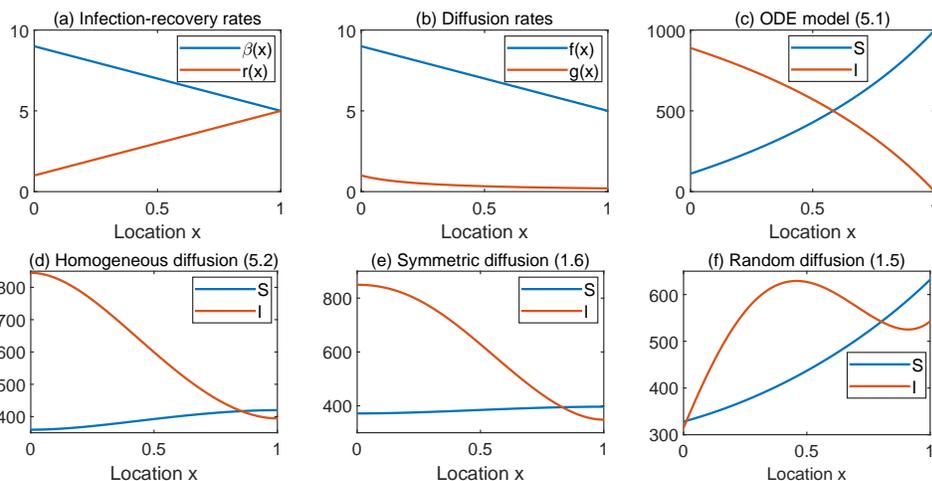


FIG. 10. Modified Example 2 with (5.4) at steady states. If there is no disease-free region as in this example, the advantage of random diffusion disappears.

TABLE 5.3
The infection fraction of modified Example 2 with SIs.

Epidemic models	$\frac{\int_0^1 I dx}{\int_0^1 (S+I) dx}$ asymptotically	SI- κ	SI- χ ($\times 10^5$)
Infinite-dimensional ODE model (5.1)	0.5297	0.4423	-7.7078
Homogeneous diffusion model (5.2)	0.6089	0.2230	-0.1236
Symmetric diffusion model (1.6)	0.6155	0.2419	-0.2303
Random diffusion model (1.5)	0.5483	0.1191	-0.1927

the infection but an appropriate dispersal can reduce the infection depending on the situation.

To check whether the small infected population size under the random diffusion is caused by the disease-free region of the ODE model, we modify Example 2 and remove the disease-free region by taking

$$(5.4) \quad \beta(x) = -4x + 9, \quad r(x) = 4x + 1, \quad x \in \Omega = (0, 1).$$

Then, $\beta(x) > r(x)$ for all $x \in \Omega$ and the steady state of (5.1) is positive on Ω . Figure 10 plots the graphs of the steady states of the four models. The main difference is the size of the infected population. The ODE model has the smallest infected population among all models. The homogeneous diffusion model again shows a similar solution behavior as the symmetric diffusion model.

The infection fraction and the two SIs of the modified Example 2 are given in Table 5.3. In this example, the three models with diffusion have negative SIs $\chi(S, I)$, which is quite small in magnitude. We claim that the segregation of the two population groups is weak in this case. Only the ODE model has a meaningful negative SI $\chi(S, I)$. The sizes of the infected population of the two models with symmetric and homogeneous diffusions are larger than other two cases.

5.3. Example 3 (dispersal toward endemic region). In the whole paper, we assume the susceptible individuals intend to leave an area with a high transmission

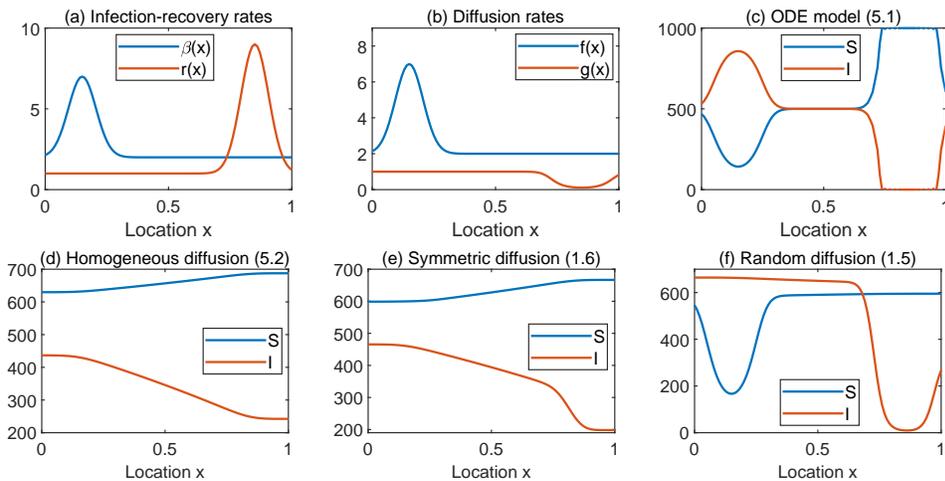


FIG. 11. Example 3 at steady states. The maxima of infection and recovery rates are placed apart in this example. The segregation of the two populations does not help to reduce the infected population in this case.

rate and the infected individuals intend to stay in an area with a high recovery rate. Under this assumption, the diffusivity is given by relations $f(x) = \beta(x)$ and $g(x) = r^{-1}(x)$. This natural assumption may give a better chance to reduce the infected population. In fact, in the previous examples, the heterogeneous random diffusion model gives the smallest infection fraction among all diffusion models. Nevertheless, such a performance is not guaranteed and, in Example 3, we will see the heterogeneous random diffusion increases the infected population. We take the infection and recovery rates as

$$\beta(x) = 5e^{-160(x-0.15)^2} + 2, \quad r(x) = 8e^{-160(x-0.85)^2} + 1, \quad x \in \Omega = (0, 1).$$

The graphs are given in Figure 11(a). In this example, $\beta(x)$ and $r(x)$ have maximum points apart. The diffusion rates are given by relations $f(x) = \beta(x)$ and $g(x) = r^{-1}(x)$ and their graphs are given in Figure 11(b). In this example, the diffusion rate of the susceptible population is highest at $x = 0.15$ and the diffusion rate of the infected population is lowest at $x = 0.85$.

The steady states of the four models are computed numerically and given in Figures 11(c)–11(f). We can see that model (1.6) with symmetric diffusion shows similar behavior to model (5.2) with homogeneous diffusion. However, the solution behavior of the random diffusion model (1.5) is different but partially similar to the one of the ODE model.

The infection fraction and the two SIs of the four models are given in Table 5.4. In this example, the two models with symmetric and homogeneous diffusions have positive SIs $\chi(S, I)$, which means that these two cases are segregated poorly. Nevertheless, the sizes of the infected populations of these two cases are smaller than other two cases. On the other hand, the other two cases with random diffusion and ODE models have negative SIs χ , and the sizes of the infected populations of the model with random diffusion are largest. This implies that the SIs mean little in this example.

TABLE 5.4
The infection fraction of of Example 3 with SIs.

Epidemic models	$\frac{\int_0^1 I dx}{\int_0^1 (S+I) dx}$ asymptotically	SI- κ	SI- χ ($\times 10^5$)
Infinite-dimensional ODE model (5.1)	0.4340	0.4048	-7.3444
Homogeneous diffusion model (5.2)	0.3421	0.3158	0.8594
Symmetric diffusion model (1.6)	0.3697	0.2606	0.6243
Random diffusion model (1.5)	0.4901	0.2704	-2.9092

6. Concluding remarks. In this work, we considered two SIS reaction-diffusion epidemic models, (1.6) with a symmetric diffusion of Fickian diffusion type and (1.5) with a random diffusion of Fokker–Planck diffusion type [24, 19]. We assumed that the diffusion rate $f(x)$ of the susceptible population is proportional to the transmission rate $\beta(x)$ and the diffusion rate $g(x)$ of the infected population is inversely proportional to the recovery rate $r(x)$. The paper consists of a mathematical analysis part and a numerical computation part.

Analytically, we have shown the well-posedness of (1.5) and (1.6) (Propositions 2.1 and 3.1), obtained the basic reproduction number \mathcal{R}_0 using a variational method and its boundedness, and shown the monotonicity and the asymptotic behavior of \mathcal{R}_0 in terms of g (Lemmas 2.3 and 3.4). Next, we have shown the existence, uniqueness, and stability of the DFE of (1.6) (Theorem 2.6). Since the diffusion of model (1.6) is not homogeneous, we could not apply the methods in [1] to study the existence and uniqueness of the EE. Fortunately, we can utilize the persistence theory in [34] to show that system (1.6) is persistent when $\mathcal{R}_0 > 1$, thus ensuring that there exists at least one EE (Theorem 2.8). In addition, we have shown the existence, uniqueness, and stability of DFE and EE for the model (1.5) (Theorems 3.7–3.9). Note that we generalized the relevant results in [1, Lemmas 2.2–2.3] (see Lemmas 2.2, 2.3, 3.3, and 3.4).

In section 4, we numerically tested the analytical results in sections 2 and 3. In order to see the role of the spatial heterogeneity and the diffusion models on the disease transmission, we tested the effects of the infection rate $\beta(x)$ and the diffusion rate $g(r^{-1}(x))$ on \mathcal{R}_0 for models (1.5) and (1.6) (Figures 5 and 2). Moreover, we drew the sample solutions for systems (1.5) and (1.6) when $\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively (Figures 7, 6, 4, and 3). Note that we have obtained the existence of EE for models (1.5) and (1.6), but the stability of EE is open for future study.

In section 5, three examples are numerically tested to see the effect of heterogeneous diffusion in the disease spread. It is widely believed that diffusion homogenizes substances. However, in the first example, we observed that a heterogeneous random diffusion segregates infected and susceptible populations further than an ODE model and thus reduces the size of the infected population (see Figure 8 and Table 5.1). Nevertheless, symmetric diffusion never does that. In the second example, the random diffusion decreases SIs but still reduces the size of the infected population by moving infected individuals to a disease-free region (see Figure 9 and Table 5.2; Figure 10 and Table 5.3). Usually, the heterogeneous random diffusion model takes a smaller infected population size since $f(x) = \beta(x)$ and $g(x) = r^{-1}(x)$. However, depending on the situation, such a strategy may increase the infected population size. The third example is such a case (see Figure 11 and Table 5.4). We should remember the various effects of heterogeneous random diffusion on the disease transmission. The numerical tests in section 5 show some interesting effects, which can help us further understand

the transmission mechanism of diseases and provide effective strategies for disease control.

It should be pointed out that in Theorem 2.8, we only obtain the existence of the EE, not the uniqueness. The uniqueness is therefore left for future investigation. On the other hand, as we all know, many infectious diseases have incubation periods, and populations can move randomly during the period. This means that the infection thereby depends not only on the interaction at the current location and time, but also on the interaction of all possible locations at previous times [29]. The susceptibility of a susceptible highly depends on the distance from each adjacent infectious individual. Such an infection mechanism is often modeled by a nonlocal incidence with a kernel function whose support determines the effective infection area [16]. Therefore, it seems plausible and necessary to incorporate nonlocal effects and/or delay into epidemic modeling. Future endeavor should explore the influences of nonlocality or delay on the segregation phenomenon.

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