Asymptotic profiles of the positive steady state for an SIS epidemic reaction–diffusion model. Part I

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**Abstract**

To capture the impact of spatial heterogeneity of environment and movement of individuals on the persistence and extinction of a disease, Allen et al. in [L.J.S. Allen, B.M. Bolker, Y. Lou, A.L. Nevai, Asymptotic profiles of the steady states for an SIS epidemic reaction–diffusion model, Discrete Contin. Dyn. Syst. Ser. A 21 (1) (2008) 1–20] proposed a spatial SIS (susceptible-infected-susceptible) reaction–diffusion model, and studied the existence, uniqueness and particularly the asymptotic behavior of the endemic equilibrium as the diffusion rate of the susceptible individuals goes to zero in the case where a so-called low-risk subhabitat is created. In this work, we shall provide further understanding of the impacts of large and small diffusion rates of the susceptible and infected population on the persistence and extinction of the disease, which leads us to determine the asymptotic behaviors of the endemic equilibrium when the diffusion rate of either the susceptible or infected population approaches to infinity or zero in the remaining cases. Consequently, our results reveal that, in order to eliminate the infected population at least in low-risk area, it is necessary that one will have to create a low-risk subhabitat and reduce at least one of the diffusion rates to zero. In this case, our results also show that different strategies of controlling the diffusion rates of individuals may lead to very different spatial distributions of the population; moreover, once the spatial environment is modified to include a low-risk subhabitat, the optimal strategy of eradicating the epidemic disease is to restrict the dif-

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1. Introduction

In recent years, it has been commonly recognized that spatial diffusion and environmental heterogeneity are important factors that should be considered in the spread of many diseases, e.g., influenza. More and more works have been devoted to the investigation of the roles of diffusion on the transmission and control of diseases. However, along this direction, very few mathematical models have been successfully developed to capture the effects of environmental heterogeneity on the dynamics.

In order to understand the impact of spatial heterogeneity of environment and movement of individuals on the persistence and extinction of a disease in an analytical aspect, very recently, Allen et al. in [3] proposed a frequency-dependent SIS (susceptible-infected-susceptible) reaction–diffusion model for a population inhabiting a continuous spatial habitat. One of the main features of this SIS model is that the total number of population is assumed to be constant. The habitat is characterized as low-risk (or high-risk) if the spatial average of the transmission rate of the disease is less than (or greater than) the spatial average of its recovery rate. Individual site is also characterized as low-risk (or high-risk) if the local transmission rate of the disease is less than (or greater than) its local recovery rate, which is equivalent to the local reproduction number being less than (or greater than) one.

The main focus of the authors in [3] was on the existence, uniqueness and particularly the asymptotic behavior of the endemic equilibrium as the diffusion (or migration) rate of the susceptible individuals approaches to zero in the case where $\beta(x) - \gamma(x)$ changes sign in the bounded and smooth habitat $\Omega$. Here and in what follows, $\beta(x)$ and $\gamma(x)$ respectively represent the rate of disease transmission and rate of disease recovery. The sign change of $\beta(x) - \gamma(x)$ means that the habitat has both the low-risk and high-risk sites. Their theoretical conclusions exhibit the delicate and of course important influence of spatial heterogeneity and rates of movement of susceptible and infected individuals on the persistence and extinction of the disease. Please refer to the forthcoming sections for the mathematical results of [3] and further discussion.

However, as the authors mentioned, they were unable to derive any stability result (even the local linear stability) for the endemic equilibrium if it exists, and they conjectured that such unique endemic equilibrium should be globally stable. In a recent work [17], Peng and Liu discussed the global stability of the endemic equilibrium in some special cases.

At present, our main purpose of this work is to attempt to provide a further understanding of the impacts of large or small diffusion rates of the susceptible and infected individuals on the spatial distribution of the whole population in the remaining cases, which thereby provide various criteria of the spatial persistence and extinction of the epidemic disease. To this end, we shall have to determine the asymptotic profiles of the endemic equilibrium when the diffusion rate of either the susceptible individuals or the infected ones goes to infinity or zero.

Our study conducted in this work brings some new insights into epidemic models. In particular, as far as the possible application in the theory of disease control is concerned, our findings for system (1.1) show that if the spatial environment can be modified to include low-risk sites (e.g., low transmission rate or high recovery rate of the disease) and if the migration rate of the susceptible or infected individuals is restricted to be small enough, the disease can be nearly eradicated at least in the low-risk area. According to our results it is also surprising to conclude that different strategies of restricting the migration rates may cause very different spatial distribution behaviors of the population. Combined with all of the mathematical results in all limiting cases, one will clearly see that, once such low-risk area is created, the optimal strategy of controlling the disease is to restrict the migration rate of the susceptible individuals rather than that of the infected ones. For more detailed comments on the results derived by this work and [3], please see the final concluding section.

Finally, we would like to mention that in order to derive the desired results, many details of our mathematical analysis in this work are different from that used in [3].
The rest of this work is arranged as follows. In Section 1.1, we shall introduce the background of the concerned SIS epidemic model and list the existing results. In Section 1.2, we are ready to state the main results of the present work. Then, in Section 2, we first recall some useful preliminaries in Section 2.1, and then in Section 2.2 we shall give the proofs of the results of Section 1.2. Finally, in Section 3, we shall carry out necessary discussions of the physical implications of the analytical conclusions and their prospective applications in disease control.

1.1. The model and existing results

Assume that the habitat \( \Omega \subset \mathbb{R}^m \) \((m \geq 1)\) is a bounded domain with smooth boundary \( \partial \Omega \) (when \( m > 1 \)), and \( \nu \) is the outward unit normal vector on \( \partial \Omega \) and \( \frac{\partial}{\partial \nu} \) means the normal derivative along \( \nu \) on \( \partial \Omega \). The SIS epidemic reaction–diffusion model considered by Allen et al. in [3] satisfies the following coupled parabolic system:

\[
\begin{align*}
\frac{\partial S}{\partial t} - d_S \Delta S &= -\frac{\beta(x)SI}{S+I} + \gamma(I) \frac{I}{S} \quad \text{in } \Omega \times (0, \infty), \\
\frac{\partial I}{\partial t} - d_I \Delta I &= \frac{\beta(x)SI}{S+I} - \gamma(I) \frac{I}{S} \quad \text{in } \Omega \times (0, \infty), \\
\frac{\partial S}{\partial \nu} &= 0, \quad \frac{\partial I}{\partial \nu} = 0 \quad \text{on } \partial \Omega \times (0, \infty),
\end{align*}
\]

where \( S(x,t) \) and \( I(x,t) \) respectively represent the density of susceptible and infected individuals at location \( x \) and time \( t \); the positive constants \( d_S \) and \( d_I \) denote the corresponding diffusion rates for the susceptible and infected populations; and \( \beta(x) \) and \( \gamma(x) \) are positive Hölder continuous functions on \( \Omega \) which account for the rates of disease transmission and disease recovery at \( x \), respectively. The homogeneous Neumann boundary conditions mean there is no population flux across the boundary \( \partial \Omega \) and both the susceptible and infected individuals live in the self-contained environment. As mentioned in paper [3], since the term \( SI/(S+I) \) is a Lipschitz continuous function of \( S \) and \( I \) in the open first quadrant, we can extend its definition to the entire first quadrant by defining it to be zero when either \( S = 0 \) or \( I = 0 \). The authors in [3] also assumed that there is a positive number of infected individuals, that is,

\[(A1) \int_{\Omega} I(x,0) \, dx > 0 \text{ with } S(x,0) \geq 0 \text{ and } I(x,0) \geq 0, \quad \text{for } x \in \Omega.\]

By the maximum principle [18], both \( S(x,t) \) and \( I(x,t) \) are positive for \( x \in \overline{\Omega} \) and \( t \in (0, T_{\text{max}}) \), where \( T_{\text{max}} \) is the maximal existence time for solution of (1.1). Then, by the maximum principle again, one also easily sees that both \( S(x,t) \) and \( I(x,t) \) are bounded on \( \overline{\Omega} \times (0, T_{\text{max}}) \). Hence, it follows from the standard theory for semilinear parabolic systems (see, e.g., [7]) that \( T_{\text{max}} = \infty \) and so system (1.1) admits a unique classical solution \((S(x,t), I(x,t))\) for all time. As in [3], let us define

\[
N := \int_{\Omega} [S(x,0) + I(x,0)] \, dx > 0
\]

(1.2)

to be the total number of individuals in \( \Omega \) at \( t = 0 \). We can add two equations in (1.1) and then integrate over \( \Omega \) by parts to obtain

\[
\frac{\partial}{\partial t} \int_{\Omega} (S + I) \, dx = \int_{\Omega} (d_S \Delta S + d_I \Delta I) \, dx = 0, \quad \text{for } t > 0.
\]
This implies that the total population size is a constant, i.e.,

$$\int_{\Omega} [\tilde{S}(x, t) + \tilde{I}(x, t)] \, dx = N, \quad \text{for } t \geq 0. \quad (1.3)$$

Eq. (1.3) shows that both $\|\tilde{S}(\cdot, t)\|_{L^1(\Omega)}$ and $\|\tilde{I}(\cdot, t)\|_{L^1(\Omega)}$ are bounded in $[0, \infty)$. As pointed out in [17], the result in [7] (see Exercise 5 of Section 3.5 there) enables us to assert that $\|\tilde{S}(\cdot, t)\|_{L^\infty(\Omega)}$ and $\|\tilde{I}(\cdot, t)\|_{L^\infty(\Omega)}$ are also bounded in $[0, \infty)$.

By adopting the same terminology as in [3], we say that $x$ is a low-risk site if the local disease transmission rate $\beta(x)$ is lower than the local disease recovery rate $\gamma(x)$. A high-risk site is defined in a similar manner. Let

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

denote the sets of these low-risk and high-risk sites, respectively. We also call that $\Omega$ is a low-risk domain if $\int_\Omega \beta(x) \, dx < \int_\Omega \gamma(x) \, dx$ and a high-risk domain if $\int_\Omega \beta(x) \, dx \geq \int_\Omega \gamma(x) \, dx$.

The authors in [3] were interested mainly in equilibrium solutions of (1.1), that is, the solutions of the following semilinear elliptic system:

$$\begin{align*}
-d_s \Delta \tilde{S} &= -\frac{\beta(x) \tilde{S} \tilde{I}}{S + I} + \gamma(x) \tilde{I} \quad \text{in } \Omega, \\
d_I \Delta \tilde{I} &= \frac{\beta(x) \tilde{S} \tilde{I}}{S + I} - \gamma(x) \tilde{I} \quad \text{in } \Omega, \\
\partial \tilde{S} \over \partial v &= \partial \tilde{I} \over \partial v = 0 \quad \text{on } \partial \Omega.
\end{align*} \quad (1.4)$$

Here, $\tilde{S}(x)$ and $\tilde{I}(x)$, which is called the equilibrium solution of (1.1), denote the density of susceptible and infected individuals, respectively, at $x \in \Omega$. In view of (1.3), it is reasonable to impose the additional hypothesis:

$$\int_{\Omega} [\tilde{S}(x) + \tilde{I}(x)] \, dx = N. \quad (1.5)$$

Throughout this paper, it is always assumed that $N$ is a fixed positive constant.

It is evident that solutions $(\tilde{S}, \tilde{I})$ satisfying $\tilde{S} \geq 0$ and $\tilde{I} \geq 0$ on $\Omega$ are of real interest for us. As in [3] again, we say that $(\tilde{S}, \tilde{I})$ is a disease-free equilibrium (DFE) if $(\tilde{S}, \tilde{I})$ is a nonnegative solution to (1.4) in which $\tilde{I}(x) = 0$ for every $x \in \Omega$; and $(\tilde{S}, \tilde{I})$ an endemic equilibrium if $\tilde{I}(x) > 0$ for some $x \in \Omega$. To distinguish these two types of equilibria, it is convenient to denote a DFE by $(\tilde{S}, 0)$ and an EE by $(\tilde{S}, \tilde{I})$. In fact, we easily observe from (1.4) and (1.5) that $(\tilde{S}, 0) = (N/|\Omega|, 0)$. Here and in what follows, $|\Omega|$ always represents the volume of the domain $\Omega$.

The work in [3] only concentrated on the case that $\beta(x) - \gamma(x)$ changes sign on the underlying domain $\Omega$, i.e.,

(A2) $H^-$ and $H^+$ are nonempty.

By continuity, if (A2) holds, the set $H^0 := \{ x \in \Omega : \beta(x) = \gamma(x) \}$ is nonempty. On some occasions, the paper [3] also assumed that

(A3) $H^0$ consists of finitely many disjoint $C^1$-surfaces or finitely many points if $m = 1$, each of which is a simple root of $\beta(x) - \gamma(x)$.
In establishing the theoretical results, the authors in [3] introduced the basic reproduction number \( R_0 \), which can be characterized by a variational problem:

\[
R_0 := \sup_{\varphi \in H^1(\Omega), \varphi \neq 0} \left\{ \frac{\int_\Omega \beta \varphi^2}{\int_\Omega d_1 |\nabla \varphi|^2 + \gamma \varphi^2} \right\}.
\]

It is a well-known fact that there exists a unique positive function \( \Phi(x) \in C^2(\overline{\Omega}) \) satisfying \( \|\Phi\|_{L^\infty(\Omega)} = 1 \) such that

\[-d_I \Delta \Phi + \gamma(x) \Phi = \frac{\beta(x)}{R_0} \Phi \text{ in } \Omega, \quad \frac{\partial \Phi}{\partial \nu} = 0 \text{ on } \partial \Omega. \quad (1.6)\]

Furthermore, under the hypothesis (A2), the following properties of \( R_0 \) were proved in [3]:

(a) \( R_0 \) is a monotone decreasing function of \( d_I \) with \( R_0 \to \max\{\beta(x)/\gamma(x): x \in \overline{\Omega}\} \) as \( d_I \to 0 \) and \( R_0 \to \int_\Omega \beta / \int_\Omega \gamma \) as \( d_I \to \infty \).

(b) In a low-risk domain (i.e., \( \int_\Omega \beta(x) dx < \int_\Omega \gamma(x) dx \)), there exists a threshold value \( d_i^* \in (0, \infty) \) such that \( R_0 > 1 \) for \( d_I < d_i^* \) and \( R_0 < 1 \) for \( d_I > d_i^* \).

(c) In a high-risk domain (i.e., \( \int_\Omega \beta(x) dx \geq \int_\Omega \gamma(x) dx \)), \( R_0 > 1 \) for all \( d_I \).

For our later purpose, let us denote \( \lambda_1(d, f) \) to be the first eigenvalue of the eigenvalue problem:

\[-d \Delta \varphi + f(x) \varphi = \lambda \varphi \text{ in } \Omega, \quad \frac{\partial \varphi}{\partial \nu} = 0 \text{ on } \partial \Omega, \quad (1.7)\]

where \( d \) is a given positive constant and \( f(x) \in C(\overline{\Omega}) \). Then, it follows that \( R_0 = 1 \) is equivalent to \( \lambda_1(d, \gamma - \beta) = 0 \). In addition, it is also well known that \( \lambda_1(d, f) \) is an increasing function with respect to \( f \) in the sense that \( \lambda_1(d, f_1) < \lambda_1(d, f_2) \) if \( f_1(x) \leq \neq f_2(x) \) on \( \overline{\Omega} \), for any \( f_1 \) and \( f_2 \in C(\overline{\Omega}) \) (see, e.g., [8]).

One of the main conclusions for systems (1.1) and (1.4) obtained by [3] can be stated as follows.

**Theorem A.** Assume that hypothesis (A2) holds. If \( R_0 < 1 \), the DFE \((N/|\Omega|, 0)\) is globally asymptotically stable for (1.1) with initial data (1.2) satisfying (1.3); but if \( R_0 > 1 \), an EE \((\tilde{S}, \tilde{I})\) exists which is also unique, and both \( \tilde{S} \) and \( \tilde{I} \) are positive on \( \overline{\Omega} \).

We have to remark that, as stated in [17], without hypothesis (A2), Theorem A remains true and system (1.4) has a (unique) EE if and only if \( R_0 > 1 \).

Motivated by the direct observation from (1.4) that in the limiting case \( d_S = 0 \) there also exists a family of infinitely many spatially inhomogeneous DFEs \((\tilde{S}, 0)\), each of which satisfies

\[
\tilde{S} \geq 0 \text{ on } \Omega, \quad \frac{\partial \tilde{S}}{\partial \nu} = 0 \text{ on } \partial \Omega, \quad \text{and } \int_\Omega \tilde{S} dx = N. \quad (1.8)
\]

The authors in [3] attempted to inquire about the connection between these spatially inhomogeneous DFEs and the unique EE (if it exists) as \( d_S \) approaches zero. They managed to show that if \( R_0 > 1 \) then the EE approaches such a spatially inhomogeneous DFE as the migration rate of susceptible individuals tends to zero. They wrote this limiting DFE as \((S^*, 0)\) and also considered the distribution of sites for which \( S^* \) is either positive or zero.

To achieve the above goal, instead of making use of (1.4), the work [3] turned to an equivalent system. Let

\[
d_S \tilde{S} + d_I \tilde{I} = \kappa,
\]
where $\kappa$ is some positive constant, and define

$$S(x) := \tilde{S}(x) \kappa \quad \text{and} \quad I(x) := \frac{dI(x)}{\kappa}.$$ (1.9)

then Lemma 3.2 in [3] proved that the pair $(\tilde{S}(x), \tilde{I}(x))$ is a positive solution of (1.4) if and only if $(S, I)$ satisfying $0 < I < 1$ in $\Omega$ is a solution of the following equivalent system:

$$\begin{cases}
dS + I = 1 & \text{in } \Omega, \\
-dI + If(x, I) = 0 & \text{in } \Omega, \\
\frac{\partial I}{\partial \nu} = 0 & \text{on } \partial \Omega, \\
k = \frac{dI N}{\int_{\Omega} (dS + I) \, dx}.
\end{cases}$$ (1.10)

where $f(x, u)$ is given by

$$f(x, u) = \beta(x) \left[ 1 - \frac{dS u}{dI (1 - u) + dS u} \right] - \gamma(x).$$ (1.11)

They claimed that

**Theorem B.** Assume that (A2) is satisfied. Then,

(a) $(\tilde{S}, \tilde{I}) \to (S^*, 0)$ in $C^1(\overline{\Omega})$ as $dS \to 0$ for some $S^*(x) \in C^1(\overline{\Omega})$ satisfying (1.8);

(b) $\Omega^+ := \{ x \in \overline{\Omega} : S^*(x) > 0 \}$ contains $H^-$;

(c) $\Omega^0 := \{ x \in \overline{\Omega} : S^*(x) = 0 \}$ has positive measure and is contained in $\overline{H^+}$;

(d) if (A3) also holds, then $\Omega^+$ contains $\overline{H^-}$, and the set $\Omega^+ \setminus H^-$ has positive measure. Moreover, if $m = 1$, then the measure of $\Omega^+ \setminus H^-$ tends to zero as $dI \to 0$.

Indeed the authors showed that $S^*$ belongs to $C^1(\overline{\Omega}) \cup C^2(\Omega^+)$, and satisfies the free boundary problem:

$$-dI \Delta S^* = (S^* - C)(\beta(x) - \gamma(x)) \quad \text{in } \Omega^+, \quad S^* = \nabla S^* = 0 \quad \text{on } \partial \Omega^+, \quad \int_{\Omega^+} S^* \, dx = N,$$

where $C$ is a positive constant.

We would like to remark here, if we assume that $H^-$ is connected with respect to $\Omega$, then one can apply the argument similar to that in the proof of our Theorem 1.2 below to the equivalent system (1.10) to assert that $\Omega^+$ is also connected.

In the discussion section of [3], Allen et al. proposed the open problem concerning the local and global stability of EE if it exists; and they conjectured that such unique EE should be globally asymptotically stable for (1.1) under the initial data $\tilde{S}(x, 0)$ and $\tilde{I}(x, 0)$ satisfying (A1).

In a recent work, the author and Liu in [17] obtained partial results for the global stability of DFE $(N/|\Omega|, 0)$ and EE in some special cases. In the following, when mentioning the global stability of this DFE or EE, we always mean that the initial data $\tilde{S}(x, 0)$ and $\tilde{I}(x, 0)$ satisfy the condition (A1). In particular, we considered the case of $\beta(x) = r \gamma(x)$ for some positive constant $r \in (0, \infty)$. In this situation, it should be observed that when $r > 1$, the unique EE can be explicitly expressed as

$$(\tilde{S}, \tilde{I}) = \left( \frac{1}{r} \frac{N}{|\Omega|}, \frac{r - 1}{r} \frac{N}{|\Omega|} \right).$$
Therefore, by constructing a nontrivial Lyapunov functional, we proved the global stability of \((\bar{S}, \bar{I})\) for \(r > 1\). More precisely, we have

**Theorem C.** The following conclusions hold.

(a) Assume that \(d_S = d_I\). Then if \(\int_{\Omega} \beta(x) \, dx > \int_{\Omega} \gamma(x) \, dx\), the EE is globally asymptotically stable for system (1.1); and if \(\int_{\Omega} \beta(x) \, dx \leq \int_{\Omega} \gamma(x) \, dx\), then the DFE \((N/|\Omega|, 0)\) is globally asymptotically stable.

(b) Assume that \(\beta(x) = r \gamma(x)\) on \(\Omega\) for some positive constant \(r \in (0, \infty)\). Then, if \(r > 1\), the EE is globally attractive, while the DFE \((N/|\Omega|, 0)\) is globally asymptotically stable if \(r \leq 1\). In addition, assume that both \(\beta\) and \(\gamma\) are positive constants, the EE is also globally asymptotically stable if \(r > 1\).

As a concluding remark of this subsection, we mention that in another related reference [2], Allen et al. investigated a continuous time SIS epidemic path model and derived many analogous results as in [3]; and the interested readers may refer to that work for the details.

### 1.2. The statement of our main results

In this subsection, we shall state the results to be obtained in the present paper. From now on, unless otherwise specified, it is always assumed that the endemic equilibrium \((\bar{S}, \bar{I})\) exists. As stated in [17], without the assumption (A2), system (1.4) possesses the unique endemic equilibrium if and only if \(R_0 > 1\). Therefore, we note that, from Section 1.1, the existence of \((\bar{S}, \bar{I})\) is guaranteed either if \(H^+\) is nonempty when \(d_I\) is sufficiently small or if

\[
\int_{\Omega} [\beta(x) - \gamma(x)] \, dx > 0. \tag{1.12}
\]

Turning now to present the main results, we start with the case when both of the diffusion rates of the susceptible and infected individuals vary simultaneously and at least one of the diffusion rates tends to zero or infinity, and determine the asymptotic profile of the endemic equilibrium \((\bar{S}, \bar{I})\) in each subcase. From now on, we define

\[
(s)_+ = \max\{s, 0\},
\]

and for \(d_0 \in [0, \infty)\), we also denote

\[
A(d_0; x) = \begin{cases} 
0 & \text{if } x \in H^-; \\
\frac{d_0(\beta - \gamma)}{d_0(\beta - \gamma) + \gamma} & \text{if } x \in H^+. 
\end{cases}
\]

We extend the definition of \(A(x)\) to \(d_0 = \infty\) in the natural way:

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

Clearly, \(0 \leq A(d_0; x) \leq 1\) and \(A(d_0; x)\) is a continuous function on \(\overline{\Omega}\) if \(d_0 \in [0, \infty)\).

In the present case, we are able to establish the following result.

**Theorem 1.1.** The following conclusions hold:

1. Assume that (1.12) holds and let \(d_S, d_I \to \infty\), then

\[
(\bar{S}, \bar{I}) \to \left(\frac{N \int_{\Omega} \gamma}{|\Omega| \int_{\Omega} \beta}, \frac{N}{|\Omega|} \left(1 - \frac{\int_{\Omega} \gamma}{\int_{\Omega} \beta}\right)\right) \text{ in } C^2(\overline{\Omega}).
\]
Assume that (A2) is satisfied and denote \( \frac{d_I}{dx} = d \). Let \( d_I \to 0 \) and \( d \to d_0 \in [0, \infty] \).

(i) If \( d_0 = 0 \), then
\[
\tilde{S} \to \frac{N}{\int_O (1 + \beta - \gamma)^{-1}} =: S^*
\]
and
\[
\tilde{I} \to \frac{N(\beta - \gamma)^{-1}}{\int_O (1 + \beta - \gamma)^{-1}} =: I^*
\]
uniformly on \( \overline{O} \).

(ii) If \( d_0 \in (0, \infty) \), then
\[
\tilde{S} \to \frac{N d_0 (1 - A(d_0; x))}{\int_O (A(d_0; x) + d_0 (1 - A(d_0; x)))} =: S^*
\]
and
\[
\tilde{I} \to \frac{N A(d_0; x)}{\int_O (A(d_0; x) + d_0 (1 - A(d_0; x)))} =: I^*
\]
uniformly on \( \overline{O} \).

(iii) If \( d_0 = \infty \), then \( \tilde{I} \to 0 =: I^* \) uniformly on \( \overline{O} \), and
\[
\tilde{S} \to \frac{N (1 - A(\infty; x))}{\int_O (1 - A(\infty; x))} =: S^*
\]
uniformly on any compact subset of \( H^- \) and \( H^+ \), respectively.

When it comes to case (2) of Theorem 1.1, the results seem very interesting. We give the complete characterization of the precise spatial location of the existence and extinction of the susceptible and infected population. Let us set
\[
\Omega_S^+ := \{ x \in \overline{O} : S^*(x) > 0 \}, \quad \Omega_S^0 := \{ x \in \overline{O} : S^*(x) = 0 \},
\]
and
\[
\Omega_I^+ := \{ x \in \overline{O} : I^*(x) > 0 \}, \quad \Omega_I^0 := \{ x \in \overline{O} : I^*(x) = 0 \}.
\]
Then, in view of the definition \( A(d_0; x) \), conclusions (i)–(iii) in (2) of Theorem 1.1 imply that

**Corollary 1.1.** Assume that (A2) is satisfied, then the following holds.

(i) If \( d_I \to 0 \) and \( d \to d_0 \in [0, \infty) \), then
\[
\Omega_S^+ = \overline{O} \quad \text{and} \quad \Omega_S^0 = \emptyset,
\]
and
\[
\Omega_I^+ = H^+ \quad \text{and} \quad \Omega_I^0 = \overline{H^-}.
\]
(ii) If \( d_I \to 0 \) and \( d \to d_0 = \infty \), then

\[
\Omega^+_S = H^- \quad \text{and} \quad \Omega^+_0 = H^+,
\]

and

\[
\Omega^+_I = \emptyset \quad \text{and} \quad \Omega^+_0 = \overline{\Omega}.
\]

As far as Theorem 1.1 is concerned, we want to make some further comments. For more explanation in epidemiology, one may refer to the final discussion section.

**Remark 1.1.**

(a) Conclusion (1) of Theorem 1.1 and its proof below imply that the limiting equilibrium is independent of the order of letting \( d_I, d_S \to \infty \). In other words, the limiting equilibrium is the same in all of the three cases: letting \( d_I \to \infty \) and then \( d_S \to \infty \); letting \( d_S \to \infty \) and then \( d_I \to \infty \); and letting \( d_I \to \infty \) and \( d_S \to \infty \) simultaneously.

(b) However, the very surprising phenomenon will occur when \( d_I, d_S \to 0 \). Conclusion (2) of Theorem 1.1 shows that in this situation, the ratio \( d_I/d_S \) of the two diffusion coefficients will play the critical role in determining the limiting equilibrium. In particular, according to (2)(i) of Theorem 1.1, if the susceptible individuals move comparatively faster than the infected ones, the eventual spatial distribution of the two types of individuals is independent of the diffusion of the infected once \( d_I \to 0 \).

(c) Simple observation shows that the conclusion (2)(i) of Theorem 1.1 continues to hold if we let \( d_I \to 0 \) first and then let \( d_S \to 0 \). On the other hand, we recall, in Theorem B the case of letting \( d_S \to 0 \) and then letting \( d_I \to 0 \) was discussed. However, in these two cases, the limiting equilibrium is totally different. Of course, it is worthwhile to mention that our assertion (2) is an interesting complement of Theorem B. Taking into account the assertion (ii) of Corollary 1.1, we believe that, in Theorem B, the measure of \( \Omega^+ \setminus H^- \) tends to zero as \( d_I \to 0 \) in all dimensions, where \( \Omega^+ \) was defined there.

(d) It is also interesting to notice that the value

\[
\frac{S^*}{I^*} = \frac{d_0(1 - A(d_0; x))}{A(d_0; x)} = \frac{\gamma(x)}{\beta(x) - \gamma(x)}
\]

is invariable with respect to \( d_0 \in [0, \infty) \) for \( x \in H^+ \). By means of the expressions of \( A(d_0; x) \) and \( I^* \) obtained in (2) of Theorem 1.1, meticulous calculation gives that, as \( d_0 \) increases, the total number \( \int_{\Omega} I^* \) of the infected population declines and so the total number \( \int_{\Omega} S^* \) of the susceptible population increases.

When it comes to the case where both of the diffusion rates of the susceptible and infected individuals vary simultaneously and at least one of the diffusion rates tends to zero or infinity, the subcases that Theorem 1.1 and Theorem B have not covered include:

(I) \( d_I \to \infty \) and \( d_S \to d_{S,0} \in (0, \infty) \);
(II) \( d_S \to \infty \) and \( d_I \to d_{I,0} \in (0, \infty) \);
(III) \( d_I \to \infty \) and \( d_S \to 0 \).

Obviously, we can take \( d_S > 0 \) and \( d_I > 0 \) to be fixed in the first two cases, respectively, since they have no essential effect on the asymptotic profiles of the endemic equilibrium.

For these three cases, our main results can be summarized as follows.
Theorem 1.2. Assume that (1.12) is satisfied. Then the following conclusions hold:

1. Let \( d_S \to \infty \), then

\[
(\tilde{S}, \tilde{I}) \to \left( \frac{d_I N}{\int_{\Omega} (d_I + w^*)}, \frac{N w^*}{\int_{\Omega} (d_I + w^*)} \right) \quad \text{in} \quad C^2(\overline{\Omega}),
\]

where \( w^* \) is the unique positive solution of the semilinear elliptic equation:

\[
-d_I \Delta w = w \left[ \beta(x) \left( 1 - \frac{w}{d_I + w} \right) - \gamma(x) \right] \quad \text{in} \ \Omega, \quad \frac{\partial w}{\partial \nu} = 0 \quad \text{on} \ \partial \Omega. \tag{1.13}
\]

2. Let \( d_I \to \infty \), then

\[
(\tilde{S}, \tilde{I}) \to (S^*, I^*) \quad \text{in} \quad \left[ C^2(\overline{\Omega}) \right]^2,
\]

where \( S^* \) is a positive function over \( \overline{\Omega} \) and \( I^* \) is a positive constant. Moreover, \( (S^*, I^*) \) satisfies

\[
\begin{cases}
-d_S \Delta S^* = I^* \left[ \gamma(x) - \frac{\beta(x) S^*}{S^* + I^*} \right] & \text{in} \ \Omega, \\
\frac{\partial S^*}{\partial \nu} = 0 & \text{on} \ \partial \Omega, \\
\int_{\Omega} (S^* + I^*) \, dx = N.
\end{cases} \tag{1.14}
\]

Furthermore, if assumption (A2) holds and letting \( d_S \to 0 \) in (1.14), then

(a) \( I^* \to 0 \) and \( S^* \to S_* \) in \( C^1(\overline{\Omega}) \) for some \( S_*(x) \in C^1(\overline{\Omega}) \) satisfying (1.8);

(b) \( \Omega^+ := \{ x \in \Omega: S_*(x) > 0 \} \) contains \( H^- \); and in \( \Omega^+ \), \( S_* \) satisfies

\[
-\Delta S_* = \tau \left[ \gamma(x) - \beta(x) \right] \quad \text{in} \ \Omega^+, \quad S_* = \nabla S_* = 0 \quad \text{on} \ \partial \Omega^+, \quad \text{and} \quad \int_{\Omega^+} S_* \, dx = N \tag{1.15}
\]

for some positive constant \( \tau \);

(c) \( \Omega^0 := \{ x \in \overline{\Omega}: S_*(x) = 0 \} \) has positive measure and is contained in \( \overline{H^+} \);

(d) if (A3) also holds, then \( \Omega^+ \) contains \( \overline{H^-} \) and the set \( \Omega^+ \setminus H^- \) has positive measure.

Moreover, if further assume that \( H^- \) is connected, then \( \Omega^+ \) is also connected.

One should notice that, in each limiting case discussed in Theorems 1.1 and 1.2, we have completely determined the spatial location of the existence or extinction of the infected individuals.

By checking the details of the proof of (2) in Theorem 1.2, we easily see that conclusions (a)–(d) also hold true in the case of \( d_I \to \infty \) and \( d_S \to 0 \) simultaneously. Hence, we believe that the limiting equilibrium does not depend on the order of taking the limit in the case \( d_I \to \infty \) and \( d_S \to 0 \).

We also point out here, problem (1.13) admits a positive solution if (1.12) holds; this result can be proved in the same way as in [5,11]. A similar result will be stated in Lemma 2.4 below.

As a concluding remark of this subsection, we would also like to mention that, if no low-risk subhabitat is created, the susceptible and infected population will coexist in the entire habitat. That is,
Remark 1.2. When $\beta(x) > \gamma(x)$ on $\tilde{\Omega}$ holds, one can use Lemma 2.1 below to derive that the endemic equilibrium $(\tilde{S}, \tilde{I})$ satisfies

$$\min_{\tilde{\Omega}} \left\{ \frac{\beta - \gamma}{\gamma} \right\} \leq \frac{\tilde{I}(x)}{\tilde{S}(x)} \leq \max_{\tilde{\Omega}} \left\{ \frac{\beta - \gamma}{\gamma} \right\}, \forall x \in \tilde{\Omega}.$$  

Then, combined with (1.5), we find that the disease always persists in the whole habitat, regardless of what the diffusion rates of the susceptible and the infected individuals are.

2. Some preliminaries and proofs of main results

In this section, we first recall some well-known facts, and then present detailed proofs of our main results stated in Section 1.2.

2.1. Some preliminary results

This subsection is devoted to the statement of some basic results, which will be frequently used in the forthcoming subsection.

To begin with, we recall a maximum principle for elliptic equations, which is due to Lou and Ni [12].

Lemma 2.1 (Maximum principle). Suppose that $g \in C(\tilde{\Omega} \times \mathbb{R})$.

(i) Assume that $w \in C^2(\Omega) \cap C^1(\tilde{\Omega})$ and satisfies

$$-\Delta w(x) \leq g(x, w(x)) \text{ in } \Omega, \quad \frac{\partial w}{\partial \nu} \leq 0 \text{ on } \partial \Omega.$$  

If $w(x_0) = \max_{\tilde{\Omega}} w$, then $g(x_0, w(x_0)) \geq 0$.

(ii) Assume that $w \in C^2(\Omega) \cap C^1(\tilde{\Omega})$ and satisfies

$$-\Delta w(x) \geq g(x, w(x)) \text{ in } \Omega, \quad \frac{\partial w}{\partial \nu} \geq 0 \text{ on } \partial \Omega.$$  

If $w(x_0) = \min_{\tilde{\Omega}} w$, then $g(x_0, w(x_0)) \leq 0$.

Next is a Harnack inequality for weak solutions (see, e.g., [9] or [14]), whose strong form was obtained in [10].

Lemma 2.2 (Harnack inequality). Let $c(x) \in L^q(\Omega)$ for some $q > m/2$. If $w \in W^{1,2}(\Omega)$ is a nonnegative weak solution of the boundary value problem

$$-\Delta w + c(x)w = 0 \text{ in } \Omega, \quad \frac{\partial w}{\partial \nu} = 0 \text{ on } \partial \Omega,$$

then there is a constant $C$, determined only by $\|c\|_q, q$ and $\Omega$ such that

$$\sup_{\tilde{\Omega}} w \leq C \inf_{\tilde{\Omega}} w.$$
In the proof of the forthcoming section, we shall also use the following elliptic estimates for the linear Neumann problem:

\[-\Delta w + w = g \text{ in } \Omega, \quad \frac{\partial w}{\partial \nu} = 0 \text{ on } \partial \Omega.\]  

**Lemma 2.3.** For problem (2.1), the following holds.

(a) Let \( g \in L^1(\Omega) \) and let \( w \in W^{1,1}(\Omega) \) be a weak solution of (2.1). Then, \( w \in W^{1,q}(\Omega) \) for all \( q \in \left[ 1, \frac{m}{m/(m-1)} \right) \) and

\[
\|w\|_{W^{1,q}(\Omega)} \leq C \|g\|_{L^1(\Omega)}
\]

with \( C \) independent of \( w \).

(b) Let \( g \in L^r(\Omega) \) with \( 1 < r < \infty \) and let \( w \in W^{1,1}(\Omega) \) be a generalized solution of (2.1). Then, \( w \in W^{2,r}(\Omega) \) and satisfies

\[
\|w\|_{W^{2,r}(\Omega)} \leq C \|g\|_{L^r(\Omega)}
\]

with \( C \) independent of \( w \).

**Lemma 2.4.** Assume that \( d_1, d \) are positive constants, and let \( \beta(x) \) and \( \gamma(x) \) be continuous functions on \( \overline{\Omega} \) with \( \beta(x) > 0 \) on \( \Omega \). Then, the elliptic problem

\[-d_1 \Delta w = w \left[ \beta(x) \left( 1 - \frac{w}{d(1-w)+w} \right) - \gamma(x) \right] \text{ in } \Omega, \quad \frac{\partial w}{\partial \nu} = 0 \text{ on } \partial \Omega
\]

has a positive solution if and only if \( R_0 > 1 \), which is also unique, denoted by \( \tilde{w} \). Moreover, \( 0 < \tilde{w}(x) < 1 \) on \( \overline{\Omega} \), and \( \tilde{w}(x) \) monotonely increases in \( d \). Furthermore, we have:

(i) If \( d_1 \to 0 \) and \( d \to d_0 \in [0, \infty) \), then

\[
\tilde{w} \to A(d_0; x) \text{ uniformly on } \overline{\Omega}.
\]

(ii) If \( d_1 \to 0 \) and \( d \to \infty \), then

\[
\tilde{w} \to A(\infty; x)
\]

uniformly on any compact subset of \( H^- \) and \( H^+ \), respectively.

(iii) If \( d_1 \to \infty \) and \( d \to d_0 \in [0, \infty) \), then

\[
\tilde{w} \to \frac{d_0[1 - (\int_{\Omega} \beta)^{-1} \int_{\Omega} \gamma]}{1 + (d_0 - 1)[1 - (\int_{\Omega} \beta)^{-1} \int_{\Omega} \gamma]} \text{ in } C^2(\overline{\Omega}).
\]
(iv) If \(d_I \to \infty\) and \(d \to \infty\), then

\[ \tilde{w} \to 1 \text{ in } C^2(\overline{\Omega}). \]

The above result is an extension of the one in [5] and [11], where \(d \equiv 1\) was studied. As in Theorem 1.1 of [11], we can establish an analogous weaker result regarding the asymptotic behavior of the solution under weaker conditions of \(\beta\) and \(\gamma\). But the present form is enough for our later use. The sufficient condition for the existence, and the uniqueness of solution and the properties that \(0 < \tilde{w}(x) < 1\) on \(\Omega\), and \(\tilde{w}(x)\) monotonely increases in \(d\) can be proved in a similar way as in Lemmas 3.3 and 4.1 of [3]; and the necessary condition for the existence of solution was given in [17]. For the asymptotic behaviors when \(d_0 \in [0, \infty)\) stated in our lemma, the argument is similar to that in the case of \(d = 1\), we omit the details here. For \(d_0 = \infty\), using \(0 < \tilde{w} < 1\) and the monotone property of \(\tilde{w}\) in \(d\), we can also easily obtain the desired conclusions.

We observe that, when \(d_I, d \to 0\), the conclusion (i) of Lemma 2.4 claims that \(\tilde{w}\) uniformly on \(\Omega\); and when \(d_I \to \infty\) and \(d \to 0\), the conclusion (ii) of Lemma 2.4 claims that \(\tilde{w} \to 0\) in \(C^2(\overline{\Omega})\).

2.2. Proofs of main theorems

From now on, we begin to give the detailed proof of the results in Section 1.2.

**Proof of (1) in Theorem 1.1.** Instead of using the equivalent system (1.10) and (1.11), we consider the original system (1.4). Because we are concerned with the asymptotic behavior of \((\tilde{S}, \tilde{I})\) when \(d_I, d_S \to \infty\), in the analysis below, we always restrict \(d_I, d_S \geq 1\).

In virtue of Lemma 2.2, from the equation of \(\tilde{I}\) in (1.4), we can find a positive constant \(C_1\), depending only on \(\beta, \gamma\) and \(\Omega\), such that for any positive solution \((\tilde{S}, \tilde{I})\) of (1.4) the following holds:

\[ \max_{\overline{\Omega}} \tilde{I} (x) \leq C_1 \min_{\overline{\Omega}} \tilde{I} (x). \]  

Therefore, there is a positive constant \(C_2\) which depends only on \(\beta, \gamma\) and \(\Omega\), such that

\[ \| \tilde{I} \|_{L^\infty(\Omega)} \leq C_2. \]  

Otherwise, we can find a sequence \(\{d_{I,n}\}_{n=1}^\infty\) with \(d_{I,n} \to \infty\) as \(n \to \infty\) and the corresponding positive solution sequence \(\{(\tilde{S}_n, \tilde{I}_n)\}\) to (1.4), satisfying \(\|\tilde{I}_n\|_{L^\infty(\Omega)} \to \infty\), and thus \(\tilde{I}_n(x) \to \infty\) uniformly on \(\overline{\Omega}\) by (2.2), which thereby contradicts (1.5).

Now, noting

\[ \left\| \left\frac{\beta(x) \tilde{S} \tilde{I}}{\tilde{S} + \tilde{I}} - \gamma(x) \tilde{I} \right\right\|_{L^\infty(\Omega)} \leq C_3 \]

for some positive constant \(C_3\) depending only on \(\beta, \gamma\) and \(\Omega\), together with the equation which \(\tilde{I}\) satisfies, we can apply the standard theory for elliptic equations and the embedding theorems to see that there exists a sequence \(\{(\tilde{S}_n, \tilde{I}_n)\}\) of positive solutions to (1.4) with \(d_I = d_{I,n}\) and \(d_{I,n} \to \infty\) as \(n \to \infty\), such that \(\tilde{I}_n(x) \to I^*\) in \(C^1(\overline{\Omega})\) as \(n \to \infty\), and \(I^*\) satisfies

\[ -\Delta I^* = 0 \text{ in } \Omega, \quad \frac{\partial I^*}{\partial \nu} = 0 \text{ on } \partial \Omega. \]

Obviously, \(I^*\) must be a nonnegative constant.
Next, we want to assert that \( I^* \) is a positive constant. To this end, we have to turn to the equation of \( \tilde{S}_n \). Let us denote \( \tilde{S}_n(y_0) = \min_{\Omega} \tilde{S}_n(x) \). By the equation of \( \tilde{S} \), the maximum principle of Lemma 2.1 tells us that
\[
\frac{\beta(y_0)\tilde{I}_n(y_0)}{\tilde{S}_n(y_0) + \tilde{I}_n(y_0)} + \gamma(y_0)\tilde{I}_n(y_0) \leq 0,
\]
from which it follows that
\[
\min_{\Omega} \gamma(x)\tilde{I}_n(x) \leq \gamma(y_0)\tilde{I}_n(y_0) \leq \max_{\Omega} \beta(x)\tilde{S}_n(x),
\]
for all \( x \in \Omega \). Therefore, by (2.2), it is easily seen that there exists a positive constant \( C_4 \), depending only on \( \beta, \gamma \) and \( \Omega \), such that
\[
\tilde{I}_n(x) \leq C_4 \tilde{S}_n(x), \quad \text{for all} \quad x \in \Omega. \tag{2.5}
\]

We rewrite the equation of \( \tilde{S}_n \) as
\[
-d_{S,n}\Delta \tilde{S}_n = h_n(x)\tilde{S}_n \quad \text{in} \quad \Omega, \quad \frac{\partial \tilde{S}_n}{\partial v} = 0 \quad \text{on} \quad \partial \Omega, \tag{2.6}
\]
where \( h_n(x) \) is given by
\[
h_n(x) = \frac{\tilde{I}_n}{\tilde{S}_n} \left( \gamma(x) - \frac{\beta(x)\tilde{S}_n}{\tilde{S}_n + \tilde{I}_n} \right).
\]
From (2.5), we can find a positive constant \( C_5 \) which does not depend on \( \tilde{S}_n, \tilde{I}_n \) and \( d_{I,n}, d_{S,n} \), such that
\[
\|h_n\|_{L^\infty(\Omega)} \leq C_5. \tag{2.7}
\]
Hence, (2.6) and the Harnack inequality ensure that there is a positive constant \( C_6 \) independent of \( \tilde{S}_n, \tilde{I}_n \) and \( d_{I,n}, d_{S,n} \geq 1 \), satisfying
\[
\max_{\Omega} \tilde{S}_n(x) \leq C_6 \min_{\Omega} \tilde{S}_n(x). \tag{2.8}
\]
If \( \|\tilde{S}_n\|_{L^\infty(\Omega)} \to \infty \), (2.8) shows \( \tilde{S}_n(x) \to \infty \) uniformly over \( \Omega \), contradicting (1.5). When \( \|\tilde{S}_n\|_{L^\infty(\Omega)} \to 0 \), then thanks to (2.5), we have \( \tilde{S}_n(x), \tilde{I}_n(x) \to 0 \) uniformly on \( \Omega \), and again this is a contradiction against (1.5). Hence, \( \|\tilde{S}_n\|_{L^\infty(\Omega)} \) has positive upper and lower bounds independent of \( d_{I,n}, d_{S,n} \geq 1 \).

The above analysis allows us to claim that, up to a further subsequence of \( \{(\tilde{S}_n, \tilde{I}_n)\} \), relabeled by itself, with \( (d_{S,n}, d_{I,n}) = (d_{S,n}, d_{I,n}) \) and \( d_{S,n}, d_{I,n} \to \infty \) as \( n \to \infty \), such that \( \tilde{S}_n(x) \to S^* \) in \( C^1(\Omega) \) as \( n \to \infty \), and \( S^* \) satisfies the same equation (2.4) as \( I^* \). Hence, \( S^* \) is a positive constant. Combined with the well-known regularity theory for elliptic equations, this also implies that \( (\tilde{S}_n, \tilde{I}_n) \to (S^*, I^*) \) in \( [C^2(\Omega)]^2 \) as \( n \to \infty \).

If \( I^* = 0 \), we take
\[
\tilde{I}_n = \frac{\tilde{I}_n}{\|\tilde{I}_n\|_{L^\infty(\Omega)}}.
\]
Then, $\hat{I}_n$ solves

$$-d_{l, n} \Delta \hat{I}_n = \left[ \frac{\beta(x) \tilde{S}_n}{\tilde{S}_n + \hat{I}_n} - \gamma(x) \right] \hat{I}_n \quad \text{in } \Omega, \quad \frac{\partial \hat{I}_n}{\partial \nu} = 0 \quad \text{on } \partial \Omega. \quad (2.9)$$

Arguing as before, passing up to a further subsequence if necessary, we may assume that $\hat{I}_n \to \hat{I}$ in $C^1(\overline{\Omega})$, and $\hat{I}$ is a nonnegative constant and also satisfies (2.4). Notice $\|\hat{I}_n\|_{L^\infty(\Omega)} = 1$ for each $n \geq 1$. Thus, it is necessary that $\hat{I} = 1$.

Recall that we have verified that $\tilde{S}_n \to S^*$ in $C^2(\overline{\Omega})$ as $n \to \infty$ for some positive constant $S^*$. Then, if $I^* = 0$, by integrating (2.9) over $\Omega$ by parts and then taking the limiting, we have

$$\int_{\Omega} \left[ \beta(x) - \gamma(x) \right] d\Omega = 0,$$

which is a contradiction with (1.12). As a result, $I^* > 0$.

Now, we have shown that $S^*$ and $I^*$ are both positive constants. Then, integrating the equation of $\tilde{S}_n$ over $\Omega$ and then letting $n \to \infty$, together with (1.5), it is clear that $(S^*, I^*)$ satisfies

$$\int_{\Omega} (S^* + I^*) d\Omega = N,$$

and

$$\int_{\Omega} \left[ \gamma(x) - \frac{\beta(x) S^*}{S^* + I^*} \right] d\Omega = 0,$$

from which we finally obtain

$$(S^*, I^*) = \left( \frac{N}{|\Omega|} \int_{\Omega} \gamma \frac{N}{|\Omega|} \left( 1 - \frac{\int_{\Omega} \gamma}{\int_{\Omega} \beta} \right) \right).$$

The proof of conclusion (1) in Theorem 1.1 is complete. \qed

**Proof of (2) in Theorem 1.1.** To prove the desired results, we shall use the transformed problems (1.10) and (1.11). According to (1.10) and (1.11), we have

$$\begin{aligned}
-\frac{d_I}{dS} \Delta I &= I \left[ \beta(x) - \gamma(x) - \frac{\beta(x) I}{d(1-I) + I} \right] \quad \text{in } \Omega, \\
\frac{\partial I}{\partial \nu} &= 0 \quad \text{on } \partial \Omega, \\
0 < I < 1 & \quad \text{in } \Omega.
\end{aligned} \quad (2.10)$$

In what follows, we need to consider three different cases.

**Case 1: $d_I \to 0$ and $d = \frac{d_I}{dS} \to 0$.** In this case, we make a scaling:

$$w := \frac{dS I}{dI} = \frac{I}{d}, \quad (2.11)$$
and then from (2.10), \( w \) solves
\[
-d_I \Delta w = w \left[ \beta(x) - \gamma(x) - \frac{\beta(x)w}{1 + (1 - d)w} \right] \text{ in } \Omega, \quad \frac{\partial w}{\partial \nu} = 0 \text{ on } \partial \Omega. \tag{2.12}
\]

Arguing as in the proof for Lemma 2.4, one can easily assert that
\[
w \to \frac{(\beta - \gamma)}{\gamma} \text{ uniformly on } \overline{\Omega}, \text{ as } d_I \to 0 \text{ and } d \to 0. \tag{2.13}
\]

On the other hand, using (1.9), (1.10) and (2.11), we yield
\[
\tilde{S} = \kappa S = \frac{d_I NS}{\int_\Omega (d_I S + I)} = \frac{N(1 - d)w}{\int_\Omega (1 + (1 - d)w)} \tag{2.14}
\]
and
\[
\tilde{I} = \frac{\kappa I}{d_I} = \frac{NI}{\int_\Omega (d_I S + I)} = \frac{Nw}{\int_\Omega (1 + (1 - d)w)} \tag{2.15}
\]

Consequently, by means of (2.13), (2.14) and (2.15), elementary computations give
\[
\tilde{S} \to \frac{N}{\int_\Omega (1 + (\beta - \gamma) + \gamma^{-1})}
\]
and
\[
\tilde{I} \to \frac{N(\beta - \gamma) + \gamma^{-1}}{\int_\Omega (1 + (\beta - \gamma) + \gamma^{-1})}
\]
uniformly on \( \overline{\Omega} \) as \( d_I \to 0 \) and \( d \to 0 \).

Case 2: \( d_I \to 0 \) and \( d = \frac{d_I}{d_S} \to d_0 \in (0, \infty) \). In this case, we can use (2.10) directly. From (1.9), (1.10), it follows that
\[
\tilde{S} = \kappa S = \frac{d_I NS}{\int_\Omega (d_I S + I)} = \frac{N(1 - I)}{\int_\Omega (I + d(1 - I))} \tag{2.16}
\]
and
\[
\tilde{I} = \frac{\kappa I}{d_I} = \frac{NI}{\int_\Omega (d_I S + I)} = \frac{NI}{\int_\Omega (1 + d(1 - I))}. \tag{2.17}
\]

As \( I \to A(d_0; x) \) uniformly on \( \overline{\Omega} \) as \( d_I \to 0 \) and \( d \to d_0 \in (0, \infty) \), (2.16) and (2.17) ensure that
\[
\tilde{S} \to \frac{N(1 - A(d_0; x))}{\int_\Omega (A(d_0; x) + d_0(1 - A(d_0; x)))}
\]
and
\[
\tilde{I} \to \frac{NA(d_0; x)}{\int_\Omega (A(d_0; x) + d_0(1 - A(d_0; x)))}
\]
uniformly on \( \overline{\Omega} \) as \( d_I \to 0 \) and \( d \to d_0 \).
Case 3: $d_I \to 0$ and $d = \frac{d_I}{d_S} \to \infty$. In this case, we first observe that $0 < I < 1$ in $\Omega$, thus

$$w = \frac{I}{d} \to 0 \text{ uniformly on } \overline{\Omega}, \text{ as } d_I \to 0 \text{ and } d \to \infty.$$ 

Furthermore, in this case, Lemma 2.4 gives $I \to 0$ uniformly on any compact subset of $H^-$. Hence,

$$\tilde{I} = \frac{NI}{\int_\Omega (d_I S + I)} = \frac{NW}{\int_\Omega (1 - I + w)} \to 0$$

uniformly on $\overline{\Omega}$ as $d_I \to 0$ and $d \to \infty$.

For the limit of $\tilde{S}$, we yield

$$\tilde{S} = \frac{N(1 - I)}{\int_\Omega (1 - I + d^{-1} I)} \to \frac{N(1 - A(\infty; x))}{\int_\Omega (1 - A(\infty; x))}$$

uniformly on any compact subset of $H^-$, and $H^+$, respectively.

Until now, we complete the proof of Theorem 1.1. □

In the following, we shall prove all of the conclusions stated in Theorem 1.2. Since we need to deal with two different cases, for the sake of clarity, we divide our analysis into two smaller parts.

**Proof of (1) in Theorem 1.2.** To verify our result, we have to turn to (2.10) again. For our purpose, in this case, we need to let $w = d_S I$, then $w$ solves

$$-d_I \Delta w = w \left[ \beta(x) \left( 1 - \frac{w}{d_I (1 - d_S^{-1} w) + w} \right) - \gamma(x) \right] \text{ in } \Omega, \quad \frac{\partial w}{\partial \nu} = 0 \text{ on } \partial \Omega. \quad (2.18)$$

We first claim that $\|w\|_{L^\infty(\Omega)}$ has a uniform bound independent of $d_S$. Actually, let $w(x_0) = \max_{\Omega} w(x)$. Thus, Lemma 2.1 enables us to obtain

$$w(x_0) \left[ \beta(x_0) - \gamma(x_0) - \frac{\beta(x_0) w(x_0)}{d_I (1 - d_S^{-1} w(x_0) + w(x_0))} \right] \geq 0.$$ 

This implies

$$\gamma(x_0) w(x_0) \leq d_I [\beta(x_0) - \gamma(x_0)] (1 - d_S^{-1} w(x_0)) < d_I [\beta(x_0) - \gamma(x_0)],$$

and thus we get

$$w(x) \leq d_I \max_{\overline{\Omega}} \{ (\beta - \gamma) \gamma^{-1} \}.$$ 

Now, we prove that $\|w\|_{L^\infty(\Omega)} \to 0$ as $d_S \to \infty$ is also impossible. Otherwise, there exists a solution sequence $w_n$ such that $w_n \to 0$ uniformly on $\overline{\Omega}$ as $d_{S,n} \to \infty$.

To produce a contradiction, we set

$$\hat{w}_n = \frac{w_n}{\|w_n\|_{L^\infty(\Omega)}}.$$
Then, simple argument indicates that there is a subsequence of \( \hat{w}_n \), still denoted by itself, satisfying \( \hat{w}_n \to \hat{w} \) in \( C^1(\Omega) \) as \( n \to \infty \). Moreover, \( \hat{w} \) is a positive solution of

\[
-d_1 \Delta \hat{w} = \left[ \beta(x) - \gamma(x) \right] \hat{w} \quad \text{in} \quad \Omega, \quad \frac{\partial \hat{w}}{\partial \nu} = 0 \quad \text{on} \quad \partial \Omega.
\]

Therefore, we have \( \lambda_1(d_1, \gamma - \beta) = 0 \) and so \( \mathcal{R}_0 = 1 \) due to (1.6). As \( \mathcal{R}_0 \) is independent of \( d_S \) and (1.4) admits a (unique) EE if and only if \( \mathcal{R}_0 > 1 \), then a contradiction occurs.

From the above discussion and the standard theory, it follows that there is a positive solution sequence \( w_n \) of (2.18) corresponding to \( d_S = d_{S,n} \) with \( d_{S,n} \to \infty \) as \( n \to \infty \), such that \( w_n \to w^* \) in \( C^2(\Omega) \) as \( n \to \infty \), where \( w^* \) is the unique positive solution of (1.13). Clearly, the uniqueness of \( w^* \) shows \( d_{S,n}I_n = w_n \to w^* \) in \( C^2(\hat{\Omega}) \) holds for \( d_S \to \infty \). Hence, \( I_n \to 0 \) in \( C^2(\hat{\Omega}) \). Using (1.10), it is also easy to note that \( d_{S,n}S_n + I_n = 1 \) implies \( d_{S,n}S_n \to 1 \) in \( C^2(\hat{\Omega}) \) as \( d_{S,n} \to \infty \). Thus, applying the scaling (1.9), together with the expression of \( \kappa \) in (1.10), we derive that

\[
\tilde{S}_n = \kappa S_n = \frac{d_1 NS_n}{\int_{\Omega} (d_1 S_n + I_n)} = \frac{d_1 Nd_{S,n} S_n}{\int_{\Omega} (d_1 d_{S,n} S_n + d_{S,n} I_n)} \to \frac{d_1 N}{\int_{\Omega} (d_1 + w^*)}
\]

and

\[
\tilde{I}_n = \frac{\kappa I_n}{d_1} = \frac{N I_n}{\int_{\Omega} (d_1 S_n + I_n)} = \frac{Nd_{S,n} I_n}{\int_{\Omega} (d_1 d_{S,n} S_n + d_{S,n} I_n)} \to \frac{N w^*}{\int_{\Omega} (d_1 + w^*)}
\]

hold in \( C^2(\Omega) \) as \( d_{S,n} \to \infty \). This verifies the assertion in (1) of Theorem 1.2. \( \square \)

**Proof of (2) in Theorem 1.2.** The proof for the limiting equilibrium as \( d_1 \to \infty \) is very similar to that for conclusion (1) of Theorem 1.1. We still use the same notations as there. By checking the argument for proving conclusion (1) of Theorem 1.1, it is easy to see that (2.2), (2.3), (2.5), (2.8) remain true in our present case. Hence, up to a sequence of \( d_1 \), we may assume that \( (\tilde{S}, \tilde{I}) \to (S^*, I^*) \) in \( [C^2(\Omega)]^2 \) as \( d_1 \to \infty \), where \( S^* \) is a positive function and \( I^* \) is a positive constant on \( \Omega \). Furthermore, using the equations of \( \tilde{S} \) and \( \tilde{I} \), we also know that \( (S^*, I^*) \) solves (1.14).

In what follows, we are ready to discuss the asymptotic behavior of \( (S^*, I^*) \) as \( d_S \to 0 \). Since \( I^* \) is a positive constant, we may assume that, up to choosing a sequence,

\[
I^* \to I_* \in [0, N/|\Omega|], \quad \text{as} \quad d_S \to 0,
\]

where \( I_* \) is a constant.

For the sake of clarity, we break our proof into five steps.

**Step 1.** The proof of (iii)(a) of Theorem 1.2. To this end, we first have to assert that \( \frac{I_n}{d_{S,n}} \) has positive upper and lower bounds as \( d_S \to 0 \).

We begin with the claim that \( \frac{I_n}{d_{S,n}} \) has a positive lower bound as \( d_S \to 0 \). Suppose that this claim is false, then there is a sequence \( d_{S,n} \) satisfying \( d_{S,n} \to 0 \) as \( n \to \infty \), such that

\[
\frac{I_n}{d_{S,n}} \to 0, \quad \text{as} \quad n \to \infty.
\]

Then, it is obvious that \( I_n^* \to I_* = 0 \) as \( n \to \infty \). Since \( S_n^* \) satisfies

\[
\int_{\Omega} S_n^* \, dx \leq N, \quad \text{for each} \quad n \geq 1,
\]
and \( S^*_n \) solves
\[
-\Delta S^*_n + S^*_n = \frac{I^*_n}{d_{S,n}} \int_\Omega \left( \gamma(x) - \frac{\beta(x)S^*_n}{S^*_n + I^*_n} \right) \, dx \quad \text{in} \quad \Omega, \quad \frac{\partial S^*_n}{\partial \nu} = 0 \quad \text{on} \quad \partial \Omega. \tag{2.19}
\]
using Lemma 2.3 and the standard argument, we can conclude that there is a further subsequence of \( S^*_n \), still labeled by itself, such that \( S^*_n \to S^*_* \) in \( C^2(\Omega) \) as \( n \to \infty \), where \( S^*_* \) is a nonnegative constant. We notice that \((S^*_*, I^*_*)\) satisfies
\[
\int_\Omega (S^*_* + I^*_*) \, dx = N, \tag{2.20}
\]
and
\[
\int_\Omega \left[ \gamma(x) - \frac{\beta(x)S^*_*}{S^*_* + I^*_*} \right] \, dx = 0. \tag{2.21}
\]
It is apparent that \( S^*_* > 0 \); otherwise, \( S^*_* = I^*_* = 0 \) would contradict (2.20). If \( S^*_* > 0 \), from (2.21), it follows that
\[
\int_\Omega \left[ \gamma(x) - \beta(x) \right] \, dx = 0, \tag{2.22}
\]
and again this is a contradiction. Hence, the previous claim holds.

We then prove that \( \frac{I^*_n}{d_{S,n}} \) has a positive upper bound as \( d_S \to 0 \). By an indirect argument again, suppose that there is a sequence \( d_{S,n} \) with \( d_{S,n} \to 0 \) as \( n \to \infty \), such that
\[
\frac{I^*_n}{d_{S,n}} \to \infty, \quad \text{as} \quad n \to \infty.
\]
For any fixed point \( x_* \in H^- \), we define \( B_\rho(x_*) \) to be the ball with center \( x_* \) and radius \( \rho \). Then, we can take \( \rho \) to be small enough so that \( \bar{B}_\rho(x_*) \subset H^- \) and \( \gamma(x) - \beta(x) \geq r_0 \) for all \( x \in \bar{B}_\rho(x_*) \), where \( r_0 \) is a positive constant which is independent of \( d_{S,n}, S^*_n \) and \( I^*_n \). Thus,
\[
\gamma(x) - \frac{\beta(x)S^*_n}{S^*_n + I^*_n} = \frac{\gamma(x)I^*_n + (\gamma(x) - \beta(x))S^*_n}{S^*_n + I^*_n} \geq r_0, \quad \text{for all} \quad x \in \bar{B}_\rho(x_*).
\]
Hence, for such chosen \( \rho \) and each \( n \geq 1 \), by (2.19), \( S^*_n \) satisfies
\[
-\Delta S^*_n \geq \frac{I^*_n}{d_{S,n}} r_0 \quad \text{in} \quad B_\rho(x_*), \quad S^*_n > 0 \quad \text{on} \quad \partial B_\rho(x_*).
\]
Now, we denote \( \psi \) to be the unique solution of the following elliptic equation:
\[
-\Delta \psi = 1 \quad \text{in} \quad B_\rho(x_*), \quad \psi = 0 \quad \text{on} \quad \partial B_\rho(x_*). \tag{2.23}
\]
Then \( \psi > 0 \) in \( B_\rho(x_*) \) and \( \Psi_n = \frac{I^*_n}{d_{S,n}} r_0 \psi \) satisfies
\[
-\Delta \Psi_n = \frac{I^*_n}{d_{S,n}} r_0 \quad \text{in} \quad B_\rho(x_*), \quad \Psi_n = 0 \quad \text{on} \quad \partial B_\rho(x_*). \tag{2.24}
\]
Note that $S_n^*$ and 0 is a pair of upper and lower solution of (2.24). So together with the uniqueness of solution to (2.24), from the well-known iteration argument one can assert that $\Psi_n \leq S_n^*$ in $B_\rho(x_*)$. Therefore, $S_n^* \to \infty$ in any compact subset of $B_\rho(x_*)$ as $n \to \infty$ due to $\frac{I_n^*}{dS_n} \to \infty$. This fact directly leads to
\[
\int_\Omega S_n^*(x) \, dx \to \infty, \quad \text{as } n \to \infty,
\]
which is an obvious contradiction with (2.20). Our analysis shows $\frac{I_n^*}{dS_n}$ must also be bounded from above. As a consequence, up to a sequence, $I_n^* \to 0$ and $S_n^* \to S^*$ in $C^1(\Omega)$, as $dS \to 0$.

Step 2. Next, we verify $S_n^*(x) > 0$ in $H^-$. In virtue of the result in Step 1, we can find a sequence of $I_n^*$, denoted by $\{I_n^*\}_{n=1}^\infty$, such that
\[
\frac{I_n^*}{dS_n} \to \tau \in (0, \infty), \quad \text{as } n \to \infty.
\]
Clearly, $I_n^* \to 0$ as $n \to \infty$. By (2.19), we may also assume that $S_n^* \to S_*$ in $C^1(\Omega)$ as $n \to \infty$, where $S_*$ is a nonnegative function on $\Omega$, and
\[
\frac{\partial S_*}{\partial \nu} = 0 \quad \text{on } \partial \Omega.
\]
(2.25)

As in the proof of Step 1, for any fixed point $x_* \in H^-$, we choose $B_\rho(x_*)$ to be as before and let $\rho$ be so small that $\bar{B}_\rho(x_*) \subset H^-$ and $\gamma(x) - \beta(x) \geq r_0$ for all $x \in B_\rho(x_*)$ with $r_0$ being a positive constant independent of $dS_n, S_n^*$ and $I_n^*$. Then, the analysis similar to Step 1 yields, for all large $n$, that $S_n^*$ satisfies
\[
-\Delta S_n^* - \frac{1}{2} \tau r_0 \psi > 0 \quad \text{in } B_\rho(x_*), \quad S_n^* > 0 \quad \text{on } \partial B_\rho(x_*).
\]
Moreover, by simple upper-lower solution argument as in the last paragraph of Step 1, one easily sees that $S_n^* \geq \frac{1}{2} \tau r_0 \psi > 0$ in $B_\rho(x_*)$ for all large $n$. Here, $\psi$ is uniquely defined by (2.23). Consequently, $S_* \geq \frac{1}{2} \tau r_0 \psi > 0$ in $B_\rho(x_*)$, and thus the arbitrariness of $x_* \in H^-$ implies that $S_*(x) > 0$ in $H^-$. Furthermore, in $\Omega^+ = \{x \in \overline{\Omega}: S_*(x) > 0\}$, $S_*$ satisfies (1.15).

Step 3. The measure of $\Omega^0 = \{x \in \overline{\Omega}: S_*(x) = 0\}$ is positive. Suppose that the measure of $\Omega^0 = \{x \in \overline{\Omega}: S_*(x) = 0\}$ is zero. Then, $S_*>0$ almost everywhere in $\Omega$, and so by (2.21) it is easily seen that in this case (2.22) holds, contradicting our assumption.

Step 4. Under the additional condition (A3), we prove that $S_*>0$ on $\overline{H^-}$. Now, using Eq. (2.19), we know that $S_*$ satisfies
\[
-\Delta S_* = \tau \left[ \gamma(x) - \beta(x) \right] > 0 \quad \text{in } H^-.
\]
Then, the interior regularity theory for elliptic equations guarantees \( S_* \in C^2(H^-) \) and so \( S_* \in C^2(H^-) \cap C^1(\overline{\Omega}) \). Suppose that there is a point \( x_* \in \partial H^- \) such that \( S_*(x) = 0 \). If \( x_* \in \partial H^- \setminus \partial \Omega \), then since the boundary \( \partial H^- \) is of \( C^1 \)-smoothness, the celebrated Hopf boundary lemma infers

\[
\frac{\partial S_*}{\partial \ell}(x_*) < 0,
\]

where \( \ell \) is the outward unit normal vector on \( \partial H^- \) with respect to \( H^- \). Thus, along the outward direction of \( \ell(x_*) \), elementary analysis deduces \( S_*(x) < 0 \) on a small relative neighborhood of \( x_* \) with respect to \( \ell(x_*) \cap \Omega \). This is impossible since \( S_*(x) \geq 0 \) on \( \overline{\Omega} \). If \( x_* \in \partial \Omega \), due to the Hopf boundary lemma again, we have

\[
\frac{\partial S_*}{\partial \nu}(x_*) < 0,
\]

which contradicts (2.25). Hence, the fact of \( S_* > 0 \) on \( \overline{H^-} \) has been verified.

Step 5. When \( H^- \) is connected, we show the connectedness of \( \Omega^+ = \{ x \in \overline{\Omega} : S_*(x) > 0 \} \). Arguing by contradiction, we assume that \( \Omega^+ \) is disconnected with respect to \( \Omega \). Denote by \( \Omega_1 \) the connected component of \( \Omega^+ \) containing \( H^- \). Then, by the result of Step 2, we can find a connected subset \( \Omega_2 \) of \( \Omega^+ \) such that \( \Omega_1 \cap \Omega_2 \) is an empty set. Obviously, \( \Omega_2 \subset H^+ \). Furthermore, in \( \Omega_2 \), we notice that

\[
-\Delta S_* = \tau \left[ \gamma(x) - \beta(x) \right] < 0 \quad \text{in} \quad \Omega_2. \tag{2.26}
\]

As before, by the interior regularity theory for elliptic equations, \( S_* \in C^2(\Omega_2) \cap C^1(\overline{\Omega}) \). Then, owing to (2.26), the well-known strong maximum principle concludes that the maximal value of \( S_* \) on \( \overline{\Omega_2} \) must be attained at some point of the boundary \( \partial \Omega_2 \), say \( y_* \). According to the definition of \( \Omega_2 \), we must have \( y_* \in \partial \Omega \); otherwise \( y_* \in \partial \Omega_2 \setminus \partial \Omega \) would imply \( S_*(y_*) = 0 \), a contradiction. Now, noting that \( \partial \Omega \) is smooth and applying Eq. (2.26) again, the Hopf boundary lemma implies

\[
\frac{\partial S_*}{\partial \nu}(y_*) > 0,
\]

and once again this causes a contradiction against (2.25). As a result, \( \Omega^+ \) is connected provided that \( H^- \) is connected.

It is clear that Theorem 1.2 follows from all of the above conclusions. \( \Box \)

3. Conclusion

In this work, we are concerned with a frequency-dependent SIS (susceptible-infected-susceptible) reaction–diffusion model for a population inhabiting a continuous spatial habitat. This system was firstly proposed and studied by Allen et al. in [3]. There the authors paid much of their attention to the analysis of the asymptotic behavior of the unique endemic equilibrium as the diffusion rate \( d_1 \) of the susceptible individuals goes to zero. Their result shows that the epidemic disease can be effectively eradicated if a so-called low-risk spatial environment is included and the movement of the susceptible individuals is controlled to zero.

First of all, in terms of epidemiology, let us present a description of the main results in [3] (namely, Theorems A and B in our Section 1.1). Firstly, the authors introduced a basic reproduction number \( R_0 \), which can be characterized by a variational problem. They discussed the relationship between this basic reproduction number and the heterogeneity of the spatial domain. In particular, it was shown that for low-risk domains, \( R_0 < 1 \) if and only if the mobility \( d_1 \) of infected individuals lies above a threshold value. Secondly, they proved that a unique spatially homogeneous disease-free equilibrium always exists and is globally asymptotically stable if \( R_0 < 1 \). Moreover, the authors in [3] derived that if \( R_0 > 1 \), a unique endemic equilibrium exists. And then, they demonstrated that when the
endemic equilibrium exists, it approaches a spatially inhomogeneous disease-free equilibrium as the diffusion rate $d_S$ of the susceptible individuals goes to zero. This limiting disease-free equilibrium has a positive number of susceptible individuals at all low-risk sites and also at some (but not all) high-risk sites. Furthermore, in one-dimensional case, they proved the number of susceptible individuals at all high-risk sites approaches zero when the diffusion rate $d_I$ of infected individuals further tends to zero.

In a recent work [17], Peng and Liu discussed the global stability of the endemic equilibrium in some special cases by using the upper–lower solution argument and the Lyapunov functional method, respectively. The precise mathematical result was collected in Theorem C of Section 1.1. In addition, [17] also pointed out that, no matter $\beta(x) - \gamma(x)$ changes sign or not, $R_0 > 1$ is the necessary and sufficient condition for the existence of the unique endemic equilibrium.

As the objective of the present work, we provide further understanding regarding the roles of large or small migration rates of the susceptible and infected population on the spatial persistence and extinction of the epidemic disease in the other cases. This leads us to establish the results for the asymptotic profiles of the endemic equilibrium as either $d_S$ or $d_I$ tends to infinity or zero. In what follows, we shall explain our main conclusions in terms of epidemiology and also make more comments. From now on, it is always assumed that the endemic equilibrium exists.

(B1) If $d_I \to 0$ and $d_I/d_S \to 0$, the statement in (2) of Theorem 1.1 shows that the endemic equilibrium converges to the limiting state where the susceptible individuals spatially homogeneously exist at each location of the habitat; meanwhile the disease only dies out at the low-risk sites and the infected population distributes spatially heterogeneously in high-risk area. More surprisingly, this limiting equilibrium is independent of the diffusion rate $d_S$. Hence, from a disease control point of view, once the migration rate of the susceptible is far away from zero, it is not enough to just restrict the movement of the infected individuals to completely eradicate such disease in the whole habitat.

If $d_I \to 0$ and $d_I/d_S \to d_0 \in (0, \infty)$, as shown in (ii) of Corollary 1.1, the susceptible population distributes in the entire habitat and the infected survives only in the high-risk region. However, in this case, both of the susceptible and infected individuals are heterogeneous in their respective domain of survival. We have confirmed that (see (2)(ii) of Theorem 1.1) the limiting value $d_0$ will play the decisive role in determining the profiles of spatial distribution of the two types of individuals. Our results show that the total number of the infected declines and the total number of the susceptible increases as $d_0$ increases in $[0, \infty)$. In this process, it is worthwhile to mention that, though the profile of spatial distribution of each of the individuals undergoes delicate change, for the limiting equilibrium, the ratio of the densities of the infected and susceptible in each high-risk site is constant, independent of $d_0 \in [0, \infty)$ (see (d) of Remark 1.1).

When $d_I \to 0$ and $d_I/d_S \to \infty$, a radical and fantastic phenomenon happens. In such extreme case, the disease will become entirely extinct on the habitat and the whole population will heterogeneously reside only in each low-risk site. This may be explained in such a way: because the infected population has been kept nearly immovable, certainly the disease will die out eventually. As for each individual (including the susceptible and the one recovering from the disease), due to the weak mobility of itself and the occurrence extinction of the disease, it is natural that it prefers to live in the more favorable low-risk zone.

From the viewpoint of disease control, the above results seem to coincide with the realistic intuition. This information strongly suggests that, as the infected moves very slowly, the more slowly the susceptible moves, the more the number of population recovering from this epidemic disease is, and the more the susceptible population tends to move the safe and favorable region (namely, the low-risk region). Eventually, the disease is completely extinct and the smart susceptible individuals live only in the low-risk region.

We would also like to compare the results above in the case of $d_I, d_S \to 0$ simultaneously with those in the two other cases. If taking $d_I \to 0$ first and then $d_S \to 0$, (b) in Remark 1.1 mentions that the limit of the endemic equilibrium is the same as in the case of $d_I \to 0$ and $d_I/d_S \to 0$, and so the infected remains in the high-risk sites. In sharp contrast, Theorem B points out that, in the case of taking $d_S \to 0$ first and then $d_I \to 0$, the disease will be extinguished in the whole habitat. What is more, it is striking to notice that, though the susceptible and infected population heterogeneously distributes in these three cases, the limiting profiles of their heterogeneity may dramatically differ.
from each other. As a result, we conclude that, in the case of \( d_I, d_S \to 0 \), the qualitative properties of the spatial distribution of the population heavily relies upon the orders of controlling the movements of the two types of individuals.

(B2) If \( d_S \to \infty \), (1) of Theorem 1.2 says that the endemic equilibrium tends to a coexistence limiting equilibrium, of which the component of susceptible individuals is a positive constant while the component of infected individuals is a spatially inhomogeneous limiting state. This suggests that the disease stays uneven in the whole habitat provided that the diffusion of the susceptible individuals is very fast. If both \( d_S \) and \( d_I \) diffuse to infinity, from (1) of Theorem 1.1 it follows that the limiting equilibrium approaches a spatially uniform coexistence state, and therefore the disease homogeneously persists everywhere. Note that, such a spatially uniform coexistence state is independent of the order of passing the limits of \( d_S \to \infty \) and \( d_I \to \infty \), as pointed out by (a) of Remark 1.1. These results also seem reasonable because, for example, the susceptible individuals will tend to live as homogeneously as they can if they are driven to move so fast.

(B3) If \( d_I \to \infty \), conclusion in (2) of Theorem 1.2 states that the endemic equilibrium approaches a coexistence limiting equilibrium in which the susceptible exists spatially heterogeneously but the infected exists homogeneously, respectively, in the whole habitat. As a more interesting result, Theorem 1.2 tells us that if further taking \( d_S \to 0 \), the limiting endemic equilibrium approaches to a spatially inhomogeneous disease-free equilibrium, which has a positive number of susceptible individuals at all low-risk sites and also at some (but not all) high-risk sites. Furthermore, in this case we have rigorously proved that the distribution area of the susceptible is a connected domain with respect to the habitat \( \Omega \) under an additional but necessary hypothesis that the set of all low-risk sites is connected, and this limiting disease-free equilibrium satisfies a free boundary elliptic equation.

From the discussions (B2) and (B3), we can conclude that large diffusion of either the susceptible or infected will help to cause the homogeneousness of spatial distribution of the corresponding individuals. On the contrary, slow diffusion of either the susceptible or infected will tend to contribute to the possibility of its heterogeneity of spatial distribution. Moreover, according to the discussion (B1) above, we have found that reducing the movement of the susceptible to zero will lead to the extinction of the disease in the entire habitat; while reducing the movement of the infected can cause the disease to disappear only in the low-risk area. However, it seems impossible to get rid of the disease by increasing the migration rate of either (or both) of them. Hence, reducing at least one of the diffusion rates to zero is necessary in order to eliminate the disease at least in the low-risk region.

On the other hand, if each site of the habitat \( \Omega \) is of high-risk, then it follows from Remark 1.2 that, no matter what measures are taken to limit or increase the mobility of either the susceptible or the infected, the disease remains persistent at any location of the whole habitat. In other words, the creation of low-risk area is also necessary to extinguish the epidemic disease.

In light of the threats of newly emerging diseases such as SARS, west Nile virus, etc., there is an increased need to precisely understand and model spatial spread of epidemics as well as optimal vaccination or (and) control strategies.

For system (1.1), to sum up, our findings here show that, if the spatial environment can be modified to include low-risk sites (e.g., low transmission rate or high recovery rate) and if the migration rate of the susceptible or infected individuals can be restricted, then it will become very likely to eradicate the disease at least in the low-risk area. More importantly, our results strongly suggest that, once a low-risk area is created, the optimal strategy of the disease eradication is to restrict the migration rate of the susceptible individuals rather than that of the infected ones.

In the two forthcoming works [15,16], we will continue to analyze the influence of the diffusion of the susceptible and infected population on the asymptotic behaviors of the endemic equilibrium in some other different heterogeneous environments.

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